



THE UNIVERSITY OF
SYDNEY

THE SYDNEY TEXTBOOK OF PSYCHIATRY

Edited By

Philip Boyce

Anthony Harris

Gin S. Malhi

sydney.edu.au

First edition published in Australia in 2024
by The University of Sydney
NSW 2006 Australia
sydney.edu.au

© 2024 The University of Sydney

All rights reserved. No part of this publication may be reproduced, distributed, or transmitted in any form or by any means, including photocopying, recording, or other electronic or mechanical methods, without the prior written permission of the publisher, except in the case of brief quotations embodied in critical reviews and certain other non-commercial uses permitted by copyright law.

Designations used by companies to distinguish their products are often claimed as trademarks. All brand names and product names used in this book are trade names, service marks, trademarks or registered trademarks of their respective owners. The publisher is not associated with any product or vendor mentioned in this book unless otherwise specified.

The authors and publisher have taken care in the preparation of this book, but make no expressed or implied warranty of any kind and assume no responsibility for errors or omissions. No liability is assumed for incidental or consequential damages in connection with or arising out of the use of the information contained herein.

The Sydney Textbook of psychiatry / edited by Philip Boyce, Anthony Harris, and Gin S. Malhi.

Includes bibliographical references and index.

ISBN: 9798329583977

The University of Sydney. Sydney Medical School. Specialty of Psychiatry

Editor Biographies

Philip Boyce AM MBBS (Lond) MD (UNSW) FRANZCP FRCPsych

Philip Boyce is Emeritus Professor of Psychiatry, University of Sydney and Emeritus Consultant Psychiatrist at Westmead hospital and adjunct Professor at Charles Darwin University. He was formerly the head of the Specialty of Psychiatry at the University of Sydney. He sits on the Mental Health Review tribunal and is an examiner for the AMC.

He has a clinical and research interest in mood disorders, particularly in the management of bipolar disorder over the perinatal period and research into the triggers for bipolar relapse. He is still involved in research and is a co-investigator in an MRFF perinatal psychiatry research project and an NHMRC bipolar disorders project. He ran perinatal psychiatry services in Western Sydney (at the Nepean and Westmead hospitals) for over 30 years.

He was a member of the working groups that developed and updated the RANZCP's Mood Disorders Clinical Practice Guideline and was a member of the group that developed the Clinical Practice Guideline for treatment of the anxiety disorders and was an adviser for the COPE perinatal mental health guidelines. He was President the RANZCP 2003-2005 and President of the International Marce society (a society focused on perinatal mental health) from 2000-2002. He was an associate editor of the Australian and New Zealand Journal of Psychiatry for 10 years. He was made a Member of the Order of Australia in the King's birthday list in 2023.

Anthony Harris AM MBBS PhD FRANZCP

Anthony Harris is a Professor of Psychiatry and the present head of the specialty of Psychiatry at the University of Sydney. He is based at Westmead Hospital and the Westmead Institute for Medical Research. He is the consultant psychiatrist for the Prevention Early Intervention and Recovery Service, a mental health service for young people within Western Sydney Local Health District Mental Health Services. He has a long-term interest in medical education, and over 30 years' experience in clinical research into the aetiology and treatment of severe mental illness, particularly schizophrenia. He is a director of Mind Australia, a leading NGO in the mental health field. He became a Member of the Order of Australia for his work in 2024.

Gin S. Malhi MBChB (Manc) BSc (Hons) MSt (Oxf) MD (UNSW) FRCPsych (UK) FRANZCP (Aus)

Upon appointment to The University of Sydney in 2006, Professor Malhi took up the role of Head of Discipline (Specialty) and directed the Sydney Medical School psychiatry curriculum for four years (2007-2011). Based at Royal North Shore Hospital (RNSH), he has led The Department of Academic Psychiatry at Northern Clinical School for 18 years, during which time he has provided the only state-wide, public hospital outpatient service for mood disorders (www.cadeclinic.com), which uses telehealth (Mood-T) to provision regional NSW. During his tenure he also directed for over a decade the Advanced Research Clinical High-field Imaging (ARCHI) Facility for neuroscience research – funded by the NHMRC.

Alongside these clinical and research endeavours he maintains a keen interest in education. In the past decade he has twice led the publication of the RANZCP Mood Disorders Guidelines (2015 and 2020), and regularly publishes seminars and reviews in top tier journals. He has written several professional examination aides and educational textbooks, and his commitment to teaching has been recognised both nationally and internationally by way of several awards. To advance academic psychiatry he has also served as Editor-in-Chief of *Acta Neuropsychiatrica*, *The Australian and New Zealand Journal of Psychiatry*, and *Bipolar Disorders* – the official journal of the International Society for Bipolar Disorders (ISBD.org), for which he was elected President (2020-2023).

In addition to his clinical interest in mood disorders, which spans three decades, Professor Malhi has maintained an active research program and published over 800 articles as well as scientific book chapters and books. Citations to his work and an H-index of over 100 have led to his recognition as a Highly-Cited researcher (ISI, Clarivate). Building on his passion for research and education he is presently Deputy Director of the GALENOS project (funded by Wellcome, UK) and a Visiting Professor at The University of Oxford. He is also The Royal College of Psychiatrists (UK) College Editor, and The Editor-in-Chief of *The British Journal of Psychiatry*.

Acknowledgement

In addition to thanking the many contributors to this text we would like to thank Dr Daniel Talbot for assisting with the production of the book, a task started by Cassandra Ma. Dr Joe Garside reviewed and proofread the final draft of all chapters. Erica Bell not only contributed to several chapters but developed and adapted the figures throughout the text. Jayashree Adhikari helped with transcription and adaptation of text, Dr Sarah Jadidi for her contributions to an initial draft of the Psychopathology and Phenomenology Chapter, and Alison Griffiths helped with referencing and indexing. We would also like to express our deep gratitude to Anne Naylor, for not only contributing to one of the mood disorder chapters, but also generously allowing us to use her artwork throughout our book. Her work “Vivid” is the cover of our book.

Artist's Statement

Anne Naylor (BEd, MSpecEd, NAATI) is an author, educator and ambassador for the CADE Clinic. She enthusiastically supports and promotes the work of CADE, and is committed to raising awareness of mental health.

Anne is also a contemporary visual artist who works in a variety of media. Her subjects include abstract landscapes and still life, and much of her work is inspired by neuroscience.

'Vivid' embodies connections to people and places and pays homage to two iconic Sydney and Australian symbols, the Sydney Harbour Bridge and the Sydney Opera House. At first glance, the painting is simple and the subjects obvious, but the meaning has greater depth. Embedded in the painting are the initials of CADE and of some of the inspirational individuals who work there.

The birds on the wire represent a meeting of minds and the unique friendships that are built on mutual admiration and respect. The birds on the bridge climb represent the struggles we all face in our personal lives, in our careers and in the unique challenges of dealing with mood disorders.

Many of Anne's artworks, including 'Vivid', are on permanent display at CADE in Royal North Shore Hospital at St Leonards in Sydney.

Anne's memoir, 'Art From Adversity: A Life with Bipolar', is available through Interactive Publications at ipoz.biz, or via Anne's website: annenaylorart.com or email: anne@annenaylorart.com

Anne was born, and currently resides, in Sydney, Australia.

List of Contributors

Erica Bell

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

ORCID: [0000-0002-8483-8497](https://orcid.org/0000-0002-8483-8497)

Phillip Boyce

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia, Westmead Institute of Medical Research, Australia, and Faculty of Health, Charles Darwin University, Australia.

ORCID: [0000-0002-9816-7356](https://orcid.org/0000-0002-9816-7356)

Vlasios Brakoulis

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia.

ORCID: [0000-0002-4188-4370](https://orcid.org/0000-0002-4188-4370)

Richard Bryant

School of Psychology, The University of New South Wales, Australia.

ORCID: [0000-0002-9607-819X](https://orcid.org/0000-0002-9607-819X)

Nicholas Burns

Greater Western New South Wales Local Health District, Australia.

ORCID: [0009-0006-5284-8312](https://orcid.org/0009-0006-5284-8312)

Huw Davie

Concord Drug Health Services, Sydney Local Health District, Australia, and Specialty of Psychiatry, The University of Sydney, Australia.

Angela Dixon

The Children's Hospital at Westmead, Australia

Juliette Drobny

Specialty of Psychiatry, The University of Sydney, Australia.

Joe Garside

Specialty of Psychiatry, The University of Sydney, Australia, and Australian Capital Territory, Australia.

Sara Ghaly

Specialty of Psychiatry, The University of Sydney, Australia, and New South Wales
Director of Advanced Training and Chair of Binational Subcommittee of Advanced
Training Consultation-Liaison Psychiatry RANZCP.

Anthony Harris

Specialty of Psychiatry, Sydney Medical School, The University of Sydney,
Australia, Westmead Institute for Medical Research, Australia, and Western
Sydney Local Health District Mental Health Services, Australia.
ORCID: [0000-0002-8617-4962](https://orcid.org/0000-0002-8617-4962)

Philip Hazell

Specialty of Psychiatry, The University of Sydney, Australia, and School of
Medicine, Charles Darwin University, Australia.
ORCID: [0000-0002-8085-2996](https://orcid.org/0000-0002-8085-2996)

Ralf Ilchef

Specialty of Psychiatry, Sydney Medical School, The University of Sydney,
Australia, and Royal North Shore Hospital, Northern Sydney Local Health
District, Australia.

Cornelia Kaufmann

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School,
Faculty of Medicine and Health, The University of Sydney, Australia, and CADE
Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health
District, Australia.
ORCID: [0009-0007-3014-5269](https://orcid.org/0009-0007-3014-5269)

Peter Kelly

Specialty of Psychiatry, Sydney Medical School, The University of Sydney,
Australia

Amy Kitajima

Concord Drug Health Services, Sydney Local Health District, Australia, and
Specialty of Addiction Medicine, The University of Sydney, Australia.

Rebecca Koncz

Specialty of Psychiatry, The University of Sydney, Australia, and Sydney Local
Health District Mental Health Services, Australia.
ORCID: [0000-0002-1412-1423](https://orcid.org/0000-0002-1412-1423)

Anthony Korner

Westmead Psychotherapy Program, Western Sydney Local Health District, and
Specialty of Psychiatry, The University of Sydney, Australia.

ORCID: [0000-0001-6162-9812](https://orcid.org/0000-0001-6162-9812)

Haley M. LaMonida

Brain and Mind Centre, The University of Sydney, Australia

ORCID: [0000-0002-6563-5467](https://orcid.org/0000-0002-6563-5467)

Lisa Lampe

School of Medicine and Public Health, The University of Newcastle, Australia.

ORCID: [0000-0001-5540-8810](https://orcid.org/0000-0001-5540-8810)

Kiran Lele

Specialty of Psychiatry, The University of Sydney, Australia.

ORCID: [0000-0002-7353-8042](https://orcid.org/0000-0002-7353-8042)

Gin S. Malhi

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School,
Faculty of Medicine and Health, The University of Sydney, Australia, CADE Clinic
and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District,
Australia, Visiting Professor, Department of Psychiatry, The University of Oxford,
UK, and Oxford Uehiro Centre for Practical Ethics, Faculty of Philosophy, The
University of Oxford, Oxford, UK.

ORCID: [0000-0002-4524-9091](https://orcid.org/0000-0002-4524-9091)

Prashanth Mayur

Specialty of Psychiatry, The University of Sydney, Australia, and Cumberland
Hospital, Western Sydney Local Health District, Australia

ORCID: [0000-0002-0872-5714](https://orcid.org/0000-0002-0872-5714)

Loyola McLean

Westmead Psychotherapy Program, Western Sydney Local Health District, and
Specialty of Psychiatry, The University of Sydney, Australia.

ORCID: [0000-0002-6004-0093](https://orcid.org/0000-0002-6004-0093)

Lauren Monds

Drug and Alcohol Services, South Eastern Sydney Local Health District, Australia,
and Specialty of Addiction Medicine, The University of Sydney, Australia.

ORCID: [0000-0002-2592-7238](https://orcid.org/0000-0002-2592-7238)

Loren Mowszowski

Brain and Mind Centre, The University of Sydney, Australia

ORCID: [0000-0002-7534-5017](https://orcid.org/0000-0002-7534-5017)

Sharon L. Naismith

Charles Perkins Centre, The University of Sydney, Australia, and Brain and Mind Centre, The University of Sydney, Australia.

ORCID: [0000-0001-9076-2778](https://orcid.org/0000-0001-9076-2778)

Shalini Narchal

Western Sydney Local Health District Mental Health Services, Australia.

Nick O'Connor

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia.

Brian O'Toole

Brain and Mind Centre, The University of Sydney, Australia, and Specialty of Psychiatry, The University of Sydney, Australia.

ORCID: [0000-0002-3825-0772](https://orcid.org/0000-0002-3825-0772)

Carmelle Peisah

Specialty of Psychiatry, The University of Sydney, Australia, and School of Clinical Medicine, The University of New South Wales, Australia.

ORCID: [0000-0002-7934-7001](https://orcid.org/0000-0002-7934-7001)

Iain E. Perkes

School of Clinical Medicine, The University of New South Wales, Australia.

ORCID: [0000-0001-8028-9032](https://orcid.org/0000-0001-8028-9032)

Zoe Quick

Formerly with The Children's Hospital at Westmead, Australia

Christopher James Ryan

Specialty of Psychiatry, The University of Sydney, Australia, The University of Sydney, Australia, School of Clinical Medicine, The University of New South Wales, Australia, and School of Medicine, The University of Notre Dame Sydney, Australia.

ORCID: [0000-0003-2158-9427](https://orcid.org/0000-0003-2158-9427)

Robyn Shields

Justice Health and Forensic Mental Health Network, New South Wales Health, Australia.

Nerissa So

Specialty of Psychiatry, The University of Sydney, Australia.

John Snowden

Specialty of Psychiatry, The University of Sydney, Australia, and School of Clinical Medicine, The University of New South Wales, Australia.

Ankur Srivastava

Westmead Mother and Baby Unit, Westmead Hospital, Western Sydney Local Health District, Australia, and Statewide Outreach Perinatal Services, New South Wales, Australia.

Janine Stevenson

Specialty of Psychiatry, The University of Sydney, Australia.

Vladan Starcevic

Specialty of Psychiatry, The University of Sydney, Australia,
ORCID: [0000-0002-6772-6995](https://orcid.org/0000-0002-6772-6995)

Jean Starling

Specialty of Psychiatry, The University of Sydney, Australia.
ORCID: [0000-0002-3761-4810](https://orcid.org/0000-0002-3761-4810)

Daniel Talbot

Westmead Hospital, Western Sydney Local Health District, Australia, and
Specialty of Psychiatry, The University of Sydney, Australia.
ORCID: [0000-0002-5505-4474](https://orcid.org/0000-0002-5505-4474)

Stephen Touyz

Inside Out Institute, The University of Sydney, Australia, and Sydney Local Health District, Australia.
ORCID: [0000-0002-1290-6243](https://orcid.org/0000-0002-1290-6243)

John Towney

School of Rural Health, The University of Sydney, Australia.

Anne Wand

Specialty of Psychiatry, The University of Sydney, Australia, and School of Clinical Medicine, The University of New South Wales, Australia.
ORCID: [0000-0002-7811-5225](https://orcid.org/0000-0002-7811-5225)

Emma Walke

School of Public Health, The University of Sydney, Australia, and School of Medical Sciences, The University of Sydney, Australia.

ORCID: [0000-0001-6962-4231](https://orcid.org/0000-0001-6962-4231)

Danielle Winch

Specialty of Psychiatry, The University of Sydney, Australia

Contents

Editor Biographies	ii
Acknowledgement	iv
Artist's Statement	v
List of Contributors	vi
Chapter 1: Introduction	4
Chapter 2: Medical Student Self-Care and Culture	11
Chapter 3: Classification in Psychiatry	22
CLINICAL SKILLS	31
Chapter 4: The Psychiatric History	32
Chapter 5: The Mental State Examination	49
Chapter 6: Bedside Cognitive Testing	58
Chapter 7: Formulation	71
Chapter 8: Introduction to Involuntary Care	81
Chapter 9: Psychopathology and Phenomenology	85
RESEARCH AND THEORETICAL MODELS	125
Chapter 10: Phenomenology - A Philosophical Perspective	126
Chapter 11: Psychiatric Epidemiology	141
Chapter 12: Research Methods in Psychiatry	153

Chapter 13: Attachment	167
Chapter 14: Neurobiological Models in Psychiatry	178
CLINICAL DISORDERS	183
Chapter 15: Schizophrenia	184
Chapter 16: Depressive Disorders	200
Chapter 17: Bipolar Disorder	221
Chapter 18: Anxiety Disorders	239
Chapter 19: Obsessive-Compulsive and Related Disorders	256
Chapter 20: Posttraumatic Stress Disorder	264
Chapter 21: Feeding and Eating Disorders	272
Chapter 22: Personality Disorders	286
SPECIFIC CLINICAL GROUPS	307
Chapter 23: Australian Aboriginal and Torres Strait Islander Psychiatry	308
Chapter 24: Perinatal Psychiatry	333
Chapter 25: Child and Adolescent Psychiatry	352
Chapter 26: Old Age Psychiatry	383
Chapter 27: Emergency Psychiatry	407
Chapter 28: Consultation-Liaison Psychiatry	422
Chapter 29: Comorbidity Between Mental and Substance Use Disorders	439

Chapter 30: Comorbidity of Psychiatric Illness and Physical Illness	450
TREATMENTS	459
Chapter 31: Sleep Wake Cycle Management	460
Chapter 32: Structured Problem Solving and Behavioural Activation	467
Chapter 33: CBT and the Cognitive Behavioural Model	475
Chapter 34: Psychotherapy	508
Chapter 35: Basic Principles of Psychopharmacology	531
Chapter 36: ECT and Other Brain Stimulation Techniques	545
REFERENCES	555
INDEX	612

Chapter 1: Introduction

Philip Boyce

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia, Westmead Institute of Medical Research, Australia, and Faculty of Health, Charles Darwin University, Australia.

and

Gin S. Malhi

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia, Visiting Professor, Department of Psychiatry, The University of Oxford, UK, and Oxford Uehiro Centre for Practical Ethics, Faculty of Philosophy, The University of Oxford, Oxford, UK.

From 1922 to 1926, Sir John Macpherson served as the Chair and Foundation Professor of Psychiatry at The University of Sydney. During this period, of just over four years he established the Diploma of Psychological Medicine. Nowadays, in Sydney Medical School (SMS), psychiatry is taught as a *Discipline*. Over the years, all three editors of this book have served as Head of Discipline and as such, have been involved in the teaching and development of the curriculum and the examination of medical students. In addition to being academics and conducting research, all three editors have also maintained active clinician roles within their respective areas of expertise. We mention these roles because fulfilling diverse responsibilities requires discipline, as does writing and editing a book.

In a similar vein acquiring the knowledge and skills to become a doctor also necessitates discipline and this is particularly true of psychiatry – a subject matter that is intellectually stimulating and endlessly interesting, but one that is difficult to circumscribe because of its inherently fuzzy boundaries. Nevertheless, defining its scope is precisely what we have endeavoured to achieve. However, our aim has not been to cover “everything you need to know about psychiatry”, instead, we have focussed on that which you must know and are most likely to need, both in your psychiatry training as an undergraduate, and eventually as a doctor, irrespective of which specialty you ultimately pursue.

The idea of creating a psychiatry textbook for Sydney Medical Students is not new, and the notion had been considered previously on several occasions. However, this time the idea emerged during a conversation between Philip Boyce and Gin Malhi, who at the time were writing the mood disorders guidelines for

psychiatrists. They both lamented the lack of practical guidance for good clinical psychiatry and arrived at the conclusion that the foundations of good training lay within the teaching of medical students. To address this, the vision for a book materialised and soon the project began to take shape. Academics and experienced clinicians were steadily recruited, and as writing gained momentum a third ‘Amigo’ (Professor Anthony Harris) was invited to assist with the endeavour. However, while the editors planned, contributed, and edited the book, the project would not have come to fruition, were it not for the stellar efforts made by a multitude of authors.

Psychiatry concerns mental illness. It is the clinical specialty within medicine that is responsible for diagnosing abnormalities of the mind and identifying the best means of clinical management. This requires understanding the mind – to enable the reliable identification of when things are no longer as they should be. The mind is complicated and has been described as the most sophisticated entity known to us. Fortunately, each of us has an equally highly developed mind that equips us to understand each other. This is the essence of psychiatry - the fundamental goal, namely, to be able to fully appreciate the subjective experience of another person. To do this, we necessarily draw on our own experiences but at the same time, it is necessary to have instruction - a framework of ‘how to go about it’. This is one of the purposes of this book; to provide you with the knowledge to navigate the pathway, that leads to having a meaningful understanding of another person's thoughts, beliefs, emotions, and desires.

Another key aim of this book is to provide instruction as to how to organise the information gathered through history taking and examination of the mind so that the process of piecing these elements together is straightforward and logical. The aim is to compose a picture that tells a complete story; one that reflects the person's narrative and experiences so that it may reveal the problems that have caused them difficulty. This process of formulation entails critical thinking. But clinically, formulation is just the beginning. The next steps are equally challenging such as determining the potential causes of a mental illness that has been diagnosed and selecting the optimal treatments to prescribe.

If ‘doing psychiatry’ is beginning to sound difficult then you are right and are paying attention, as it is no easy feat to make the correct diagnosis and successfully manage a person with a severe mental illness, while still maintaining a therapeutic relationship. However, it is important to emphasise that despite these challenges psychiatry is immensely rewarding, both intellectually and from a humanistic perspective, and this perhaps explains why so many young doctors are drawn to this specialty.

We thank our many colleagues without whom this book would not have been possible. We thank them for their contributions but also for their patience and for allowing us to tinker with their thoughts, views, and ideas. Collectively, they have provided a glimpse of why psychiatry is fascinating. The subject attempts to integrate the biological underpinnings of psychiatric disorders with the unique psychology of the patient as well as their social environment. The scientific basis of psychiatry, and how scientific developments are being translated into clinical practice is critical; we have had major advances in treatments over the years, although we still have a long way to go. Keeping up with developments can be a challenge and we hope that in addition to providing you with the basic knowledge you need, the additional reading, and references will encourage you to explore further to get a deeper understanding of the various disorders and their treatment.

The book is structured broadly into sections to assist you in your reading. We have put the full list of references at the end of the book, with crucial reading listed at the end of each chapter. The reference list at the end of the book is extensive but it is there for you to seek out further information if you so desire.

Sometimes, what the patient has experienced, can impact us and evoke strong feelings, which can help us empathize and understand, but at the same time be distressing. This is not a trivial issue but is something that we need to discuss with someone to fully appreciate what may have happened. To this end, in the introductory section of this book, Peisah *et al.* provide important information regarding medical student self-care, and we recommend that you read this chapter and heed its advice.

At the outset, the key issue of psychiatric taxonomy using official classificatory systems (ICD and DSM) is discussed by Malhi and colleagues who in this opening section of the book provide an overview of the major psychiatric disorders and set the scene for the remainder of the text.

Clinical Skills

The *first section* on clinical skills is essential reading when embarking on the rotation. It can be daunting the first time you sit down to interview a patient and there is no doubt that the psychiatric history is more complex than taking a history from a patient complaining of chest pain, but, if you are prepared to listen and remain curious, it is an enriching experience. Kelly & Ryan provide a comprehensive step by step guide as to how to take a psychiatric history. Their chapter aims to assist you in anamnesis and make the process easier. The skills they cover in taking a psychiatric history can be readily applied whenever you take a detailed history from any patient in medical practice. Putting your observations together and presenting

these in a systematic way is the essence of the mental state examination (MSE); and it is precisely this that Brakoulias addresses in his chapter and provides the necessary framework for performing a systematic MSE and presenting it clearly.

In the next chapter, LaMonida and colleagues, provide a comprehensive guide on how to test cognition at the bedside; a skill that will serve you well throughout your medical career. This is because assessing cognition is not unique to psychiatry. Indeed, it is an essential skill both in primary care and in general medicine overall. We anticipate that if you read and take in these crucial chapters, then you will not only gain competency in interviewing patients, you will also find that the experience is more fulfilling.

Putting your thoughts together, and bringing together the biological, psychological and social factors contributing to the patient's illness, is an essential skill in psychiatry and essential in treatment planning. This is termed the psychiatric formulation. The process of formulating a patient is covered in a chapter by Boyce and colleagues.

A critical issue in psychiatry is that at times we have to treat patients against their will. From time to time, patients with mental illness, are at risk of harming themselves or others and therefore require treatment. In such circumstances, we can treat them against their will - as involuntary patients, using the Mental Health Act. Garside, in his chapter, highlights the key points about involuntary treatment – a matter you need to know about when working in any area within acute medicine.

Research and Theoretical Models

In the chapter on the mental state exam, you will have been introduced to the major signs of mental illness. Further details about specific signs and symptoms, such as the different types of delusion or hallucination, are comprehensively covered by Malhi and colleagues in the chapter on psychopathology. This is coupled to Winch's phenomenology chapter, which delves into the more philosophical aspects of understanding a patient's experience; it goes beyond the descriptive step of identifying mental state signs and symptoms.

We often hear that 1 in 5 people will experience a mental illness each year. O'Toole gives us a detailed critical review of psychiatric epidemiology that will give you a good understanding of the field and the approaches that have been used to determine the prevalence and incidence of psychiatric disorders. Koncz and So, provide an overview of the common research approaches used in psychiatry. These chapters are also essential reading as they will assist you in taking an evidence-based approach to the treatment of psychiatric disorders.

The book then shifts and addresses some of the theoretical models that underpin psychiatric disorders. One area of recent interest is ‘trauma’ focussed care, which has evolved from attachment theory. McLean discusses the key aspects of this theory and how it may help guide us in our understanding of patients. In addition to this model there are psychological models grounded in cognitive, emotional, and behavioural theories. The most dominant of these that underpins cognitive behavioural therapy (CBT) is covered in the chapter on this treatment. Similarly, the historical psychodynamic model, is covered in the chapter on psychotherapy. To complete the picture, Harris in his chapter on psychobiological models reviews the major biological models that contribute to psychiatric disorders.

Clinical Disorders

Having discussed some of the key elements and foundations of psychiatry, we then move on to the *third section* of the book: the specific disorders. These chapters review the clinical features, presentation, diagnostic criteria, causal factors and treatments of the major disorders that you will encounter during the psychiatric rotation. These are essential reading, as you do need to know the fundamental aspects of schizophrenia, depressive disorders, bipolar disorder, anxiety disorders, obsessive compulsive disorder, eating disorders, post-traumatic stress disorder and personality disorders. This is necessary for your undergraduate psychiatry training but also for your career in medicine as you will come across patients with these disorders regularly in whatever specialty you choose. Knowing about the presentation and management of these disorders is essential to you becoming good doctors.

Specific Populations

The *fourth section* of the book focusses on the psychiatric considerations in different populations and comorbidity. Our Aboriginal and Torres Strait Islander Australians, with their ancient history, connection to the land and understanding of wellbeing are an important population to have some understanding of. In their chapter on Indigenous Psychiatry, Towney and colleagues explain the essential aspects of the importance of social and emotional wellbeing that is necessary for us to be able to make sense of how indigenous individuals experience mental ill-health. They trace, for us, the history of what has happened to our first nations peoples, especially critical issues of dispossession and enforced childhood separations. They discuss the alarming data about the prevalence of mental disorders in the indigenous population and discuss how culturally sensitive approaches are necessary in planning treatments.

Different approaches are required in the management of psychiatric disorders over the lifespan. The presentation of psychological illness in children and adolescents is different than what we see in adults as are the approaches to management, especially the crucial role of involving the whole family. Assessment of children and adolescents must take a different approach. These issues, along with the epidemiology of child and adolescent disorders are covered in the chapter by Hazell and colleagues. The disorders at the other end of the spectrum are discussed in detail by Stevenson and colleagues in their chapter on old age psychiatry, in which they discuss the crucial issues of being able to assess and investigate and manage patients with cognitive decline. They also discuss the issue of assessment of capacity – an essential skill in all aspects of medicine. Women over the perinatal period are particularly at risk of developing mental illness, such illness can have an effect on the mother-infant relationship with downstream effects of the infant; these conditions are discussed in the Chapter by Srivastava & Boyce. Consultation and Liaison Psychiatry is a subspecialty of psychiatry that not only deals with comorbidity, but how patients with a medical illness cope with or responds to their illness; these issues are discussed by Ghaly & Ryan. Patients with mental illness frequently present to the emergency department, and Ilchef, provides a helpful framework for the assessment and management of such psychiatric emergencies.

Patients with psychiatric disorders often have comorbid physical disorders and by the same token, patients presenting with medical conditions can have an underlying psychiatric disorder. These important comorbidities are reviewed in the chapter by Talbot & Harris. All too often in psychiatric practice, especially in the public health system we see patients with psychiatric disorders using illicit drugs and alcohol, complicating their illness. Monds and colleagues discuss critical issues relating to these comorbidities.

Treatments

The *fifth and final section* of the book deals with treatment approaches. The fundamental issues underpinning psychodynamic psychotherapy are discussed in detail in the chapter by Korner. Drobny, explains the cognitive model as an explanatory model for depression and anxiety – detailing cognitive behaviour therapy, the most widely used evidence-based model of therapy. Lampe describes the problem-solving approach to management – a straightforward and effective management approach for the common mental health problems and one that can be readily used in primary care. She also discusses the basics on sleep wake cycle management and the essence of good sleep hygiene - an essential aspect of non-pharmacological treatment. Turning then to pharmacological treatment, Boyce & Harris outline the basic principles concerning the use of medication in psychiatry. Finally, Mayur, addresses Electroconvulsive Therapy (ECT) – arguably the most

well-known psychiatric treatment. ECT was once a much-maligned form of treatment, in part due to its (incorrectly) dramatic portrayal in films. This chapter is therefore important as it points out that ECT is an effective and safe therapy when used correctly and for specific psychiatric disorders. It is important to learn about ECT as it is still widely used in clinical practice and therefore it is a therapy that you need to be familiar with and ideally you should aim to observe a session of ECT during your training.

Thus, as promised the book takes you, the reader, on a journey through the essentials of psychiatry - at least for the purposes of undergraduate medical training. However, the book is just a guide and an indication of the knowledge you need to acquire. The real learning takes place when interacting with patients and for this there is no substitute. However, equipped with the knowledge in this book you can be confident that you will know how to approach the subject matter, and indeed patients themselves, and with practice you should be able to make sense of the lived experiences of others and gain a deeper understanding of the suffering they endure.

Finally, this book would not have been possible without its many contributors and we thank them sincerely, however we also take this opportunity to thank in particular Daniel Talbot for assisting with the typesetting and production of the book, Joe Garside for proof reading its final drafts, Erica Bell for assistance with development and adaptation of figures, Jayashree Adhikari for transcription and adaptation of text and Anne Naylor, for generously allowing us to use her artwork throughout.

Professor Philip Boyce and Professor Gin S. Malhi

Chapter 2: Medical Student Self-Care and Culture

Carmelle Peisah

Specialty of Psychiatry, The University of Sydney, Australia, and School of Clinical Medicine, The University of New South Wales, Australia.

Kiran Lele

Specialty of Psychiatry, The University of Sydney, Australia.

and

Loyola McLean

Westmead Psychotherapy Program, Western Sydney Local Health District, and Specialty of Psychiatry, The University of Sydney, Australia.

Introduction

The health and well-being of medical students matter. The development of their professional identity matters, as does their learning and work culture. Why? Because as educators we owe a duty of care to our students as well as a responsibility to improve and shape future medical culture, which lies in their hands (Selvaraj and John, 2020). In this chapter, we explore the nature and determinants of medical student distress and impairment, and the evolution of professional identity, its interaction with student culture and the wider medical culture. Finally, we provide a brief guide to the Australian Health Practitioner Regulation Agency (AHPRA) regulatory processes and the National Law in relation to students and provide a guide to available resources to support students. We aim to raise awareness of these issues amongst students and educators alike while debunking the myth of omnipotence and shame associated with mental disorder that seems to start early in a medical career.

As will be seen below, much of our understanding of the Australian medical student comes from the largest Australian survey of its type, the National Mental Health Survey of Doctors and Medical Students reported in 2013 and updated in 2019. The sample comprised 6,658 medical students: the final response rate being 27% yielding 1,811 respondents (National Mental Health Survey of Doctors and

Students, 2019). Similar insights can also be found in the United Kingdom (UK) General Medical Council (GMC) review of the experience of medical students working and learning within the UK's healthcare systems (West and Coia, 2019).

Distress and Burnout

The stressors facing medical students are well-known. Beyond the obvious examination stresses, there is also perceived mistreatment by consultant staff, financial and family issues (Gaughran et al., 1997; Tennant, 2002). Focus groups with UK medical students identified generic factors shared by all students including the transition from home and school to university life; the lack of a support network (family, friends); fear of disclosing mental health issues; competition with peers reinforced by social media; and self-care issues (e.g., with nutrition or sleep). These are compounded by stressors specific to the medical student's experience such as witnessing ill or dying patients; being part of a serious untoward incident or mistreatment by supervising doctors (West and Coia, 2019).

These issues are even more keenly experienced by the international medical student, who often faces isolation from family while at the same time trying to meet family expectations and aspirations, as well as difficulties with social acculturation in the hegemonic culture both at university and socially. International medical students report more stress symptoms, achieve poorer results in written, oral, and practical exams, have extended study duration and higher dropout rates (Huhn and Nikendei, 2018).

Although the concept of burnout is often associated with practising doctors, burnout can begin in the early years of medical training. A systematic review of literature on burnout in the United States showed that at least half of all medical students experienced burnout (IsHak et al., 2013). These results have been echoed in the Beyond Blue National Mental Health Survey of Doctors and Medical Students which found that students reported high rates of burnout and emotional exhaustion (National Mental Health Survey of Doctors and Students, 2019). Such burnout has practice implications, even in students, in its association with both unprofessional conduct and less altruistic attitudes regarding physicians' responsibility to society (Dyrbye et al., 2010).

Impairment

This distress frequently reaches levels of frank disorder. The National Mental Health Survey showed that levels of very high psychological distress were substantially higher in medical students than in the general population (9.2% and 3.1% respectively). Rates of current (8.1%) and lifetime depression (18.1%) were

substantially higher in medical students compared with the general population (12-month prevalence 6.2%, lifetime prevalence 15.0%). Similar patterns were found for anxiety, although it was noted that these prevalence rates for anxiety and depression were shared by all university students. In contrast, while male students showed higher levels of moderate and high-risk drinking behaviour than female students, medical students have lower levels of harmful drinking (4%) in comparison to the general student population. Moreover, a decline in moderate drinking with age suggests that these behaviours may improve over time (Said et al., 2013). Most sobering is that approximately 20% of medical students have had thoughts of suicide in the previous 12 months, a rate substantially higher than that observed in the general population (National Mental Health Survey of Doctors and Students, 2019).

Gender Differences in Impairment

The National Mental Health Survey (National Mental Health Survey of Doctors and Students, 2019) has revealed a number of concerning gender differences in mental health amongst Australian medical students. For example, with regards to burnout, females reported a slightly higher rate of burnout in all three domains (emotional exhaustion: 57.1%, cynicism: 26.4%, low professional efficacy: 31.2%) compared to male students (emotional exhaustion: 44.2%, cynicism: 24.4%, low professional efficacy: 25.6%). Female students have higher levels of psychological distress and minor psychiatric disorders and report more specific mental health diagnoses than male students. Female students show higher levels of distress than male students (26% and 18.2% reported high or very high distress respectively). Most troubling of all is that female students were more likely than male students to have had suicidal thoughts in the previous 12 months (20.5% vs. 17.1%) and prior to the previous 12 months (34.3% vs. 27.3%) (National Mental Health Survey of Doctors and Students, 2019).

These gender differences have been replicated elsewhere (Dahlin et al., 2005; Said et al., 2013) and the causes can be thought of as falling into three broad groups: gender differences in the population at large, differences in relation to the experience of medical training, and measurement error or bias. With regard to the population at large, the Australian Bureau of Statistics National Health and Wellbeing Survey (2007) showed that for lifetime prevalence of anxiety disorders (32% female vs 20% male) and affective disorders (18% female vs 12% male), there was a marked female predominance. Without a non-medical control group for the Beyond Blue survey, it is difficult to know how much of the gender differences could be accounted for by these differences.

Various hypotheses have been put forward to account for differences specific to medical students including: a greater expectation on women to manage

domestic duties, perceptions of guilt and institutional disincentives around pregnancy and maternity leave, some ongoing disparities in the presence of female role models in positions of power, and exposure to harassment. Some of these indicators are rapidly changing with improving gender balance in medical schools both amongst students and teaching staff. Females comprised 51.3% of all medical students in Australia and New Zealand in 2019-20 (Medical Deans Australia and New Zealand Inc, 2020). Nevertheless, in certain specialties the gender difference remains profound. For example, of all medical practitioners working in the fields of General Surgery, Orthopaedic Surgery and Neurosurgery the proportion of females were 11.6%, 3% and 10.9% respectively (Walton, 2015). In a study of women surgeons 87% experienced gender-based discrimination in medical school. Notably, 40% of these discriminatory events came from other women (Bruce et al., 2015) suggestive of a “transgenerational” transmission of adverse medical culture, which isn’t simply addressed by greater female representation.

A longstanding issue of bias remains unresolved in the collection of many of these statistics however, and that is that females may be reporting their distress in greater numbers. It may also be that the expressions of distress that are being measured (e.g. depression, anxiety) are more in line with the ways that females are socialised to express their distress in comparison to other predominantly male ways (antisocial behaviour, substance use). Looking further back, socialisation in early family life may encourage more affiliative orientations in females and more self-made independent orientations in males. This could create some discrepancies in perceptions of role performance and self-efficacy in females while also enabling them to seek help and voice their emotional struggles more openly (Richman and Flaherty, 1990).

LGBTIQA+ Issues

The vast majority of LGBTIQA+ (Lesbian, Gay, Bisexual, Transgender, Intersex, Queer/questioning, Asexual and other minority gender identities and sexualities not explicitly included within the term) people in Australia live healthy and happy lives. Nevertheless, they are disproportionately impacted by mental ill health when compared to their cisgender peers. LGBTIQA+ young people (16 to 27) are five times more likely to make a suicide attempt than young people in the general population (Robinson et al., 2014). Unfortunately, LGBTIQA+ populations were not assessed separately in The National Mental Health Survey (National Mental Health Survey of Doctors and Students, 2019) and statistics around the mental health outcomes of this group amongst medical students are largely unknown.

Given the long history of homophobia in our society, it is difficult to know what proportion of Australian students or doctors identify as LGBTIQA+. There

has been a revolution in the institutions of medicine and in society at large, which started with the removal of homosexuality as a DSM diagnosis in 1973. LGBTIQ+ doctors and students come from every ethnic and religious group and many still face discrimination from their own communities over and above what they experience in medicine (Myers and Gabbard, 2008). The ongoing barriers faced include the lack of access to healthcare due to perceived and real stigma; harassment in the workplace by colleagues, superiors and patients; and a lack of data which leads to difficulties making evidenced-based arguments for change.

Australian Aboriginal and Torres Strait Islander Issues

Australian Aboriginal and Torres Strait Islander participation in professional life and medicine is on the rise, albeit with challenges. However, data that speaks to the experience is still lacking. In the National Mental Health Survey of Doctors and Students (2019) approximately 0.2% of doctors and 1.2% of students responding identified as being of Australian Aboriginal and Torres Strait Islander background. The sample sizes were small and comparisons with the non-Aboriginal and Torres Strait Islander group were necessarily cautious. Yet among the Aboriginal and Torres Strait Islander Australian student group 73% were classified as having a high likelihood of a minor psychiatric disorder, a much higher proportion than the non-Aboriginal and Torres Strait Islander Australian group (43%). Very high psychological distress was reported in 27% versus 9% in the non-Aboriginal and Torres Strait Islander Australian group. Although the small sample made it harder to be clear about depression and anxiety levels in doctors and students, suicidal ideation in this group of students was high with 45% reporting suicidal ideation in the previous year. Substance use was however low in the Aboriginal and Torres Strait Islander population.

With respect to burnout Aboriginal and Torres Strait Islander Australian doctors reported similar levels to the general doctors' group, but the student group reported high levels of emotional exhaustion (59.1%). In terms of sources of stress, 21.7% were very stressed by bullying, a higher level than the general doctors' population. In line with other work on Aboriginal and Torres Strait Islander Australian health, more Aboriginal and Torres Strait Islander Australian students reported experiencing a broad range of stressful events (all of those listed in the survey) compared with non-Aboriginal and Torres Strait Islander Australian students. Cumulative trauma and loss are important in this group (see Chapter 23 Australian Aboriginal and Torres Strait Islander Psychiatry), with the most commonly reported event being the death of a family member or friend. There is much that could be said about the historical, cultural, traumatic and bereavement issues at play in this group, where the concept of saturation with trauma and loss is relevant (Atkinson, 2002) but good data-gathering and collaborative approaches to

research and service remain essential to moving forward together to enable equity and well-being in our First Nation peoples.

Regulatory Responses to Student Impairment

We discuss regulation not to incite fear but to educate and raise awareness. A triad of weighty issues are at stake here, recognised by the Tribunals in their responses to medical student impairment. Firstly, the safety of the public is at stake and remains a relevant and primary consideration even for medical students. Secondly, there is recognition of both the onerous path to medical school and the enormous emotional and financial investment in a medical education, not to be thrown away easily. Thirdly, there is the potential consequences of future practice on an impaired practitioner (Peisah and Haysom, 2021).

Health practitioners supervising students in clinical training must be mindful of obligations under the Health Practitioner Regulation National Law (National Law) for mandatory reporting which explicitly include students. However, the threshold for reporting is very high for the specific purpose of ensuring that students get help and are not frightened of accessing such due to fear of notification. The circumstances for mandatory reporting under the National Laws 141 are that if a registered health practitioner who, in the course of practising their profession, forms a reasonable belief that a student has an impairment that, in the course of the student undertaking clinical training, may place the public at substantial risk of harm, they must, as soon as practicable after forming the reasonable belief, notify the Australian Health Practitioner Regulation Agency (AHPRA) of the student's impairment, unless they know or reasonably believe, that AHPRA has already been notified (e.g. by the university, who have equal obligations under the Act).

This 'reasonable belief' must be based on direct knowledge or observation, or report from a reliable source (who might consider making a mandatory notification themselves) about the incident or behaviour that led to the concern. The "reasonable belief" should not be based on suspicion, speculation, gossip or rumours (Australian Health Practitioner Regulation Agency (AHPRA), 2020). There is protection from civil, criminal and administrative liability for persons who, in good faith, make a notification under this Law. Conversely, while failure to notify does not constitute an offence it may constitute behaviour for which action may be taken (Australian Health Practitioner Regulation Agency (AHPRA), 2020).

An important distinction to make here is that in this section we are referring to big "I" Impairment, i.e., legal Impairment. Impairment for students is also defined

separately under section 5 of the Health Practitioner Regulation National Law, namely:

A physical or mental impairment, disability, condition or disorder (including substance abuse or dependence) that detrimentally affects or is likely to detrimentally affect a student, the student's capacity to undertake clinical training—

- (i) *as part of the approved program of study in which the student is enrolled; or*
- (ii) *arranged by an education provider.*

The threshold to make a notification to AHPRA is further determined by risk to the public. This is why early identification and treatment of the struggling student is so important (Australian Health Practitioner Regulation Agency (AHPRA), 2020). As with practising doctors, just having an illness (or small “i” impairment) that does not detrimentally impact on the student’s capacity to undertake clinical training, does not constitute Impairment. Even with an Impairment, not all Impairments need to be reported. A student may carry out clinical training with a mental health condition, physical health condition or physical illness, but that is not enough to trigger a mandatory notification. If the student’s impairment affects their capacity to carry out clinical training but does not place the public at substantial risk of harm, there is no need to make a mandatory notification. As with practising doctors, effective safeguards to manage the impairment may include treatment, break from study, such as sick leave, modified scope of practice, strategies used to manage impacts of impairment, compliance with monitoring and supervision, or a reasonable belief that AHPRA has already been notified.

Clearly, there are responsibilities of students, supervisors and universities alike under the National Law. However, the intent of the National Law is not to punish students or practitioners for impairment, but to protect the public by ensuring that impairment is managed responsibly. We neither do ourselves nor our students a favour by pretending this Law doesn’t exist. Compassionate responses to this issue require bravery and honesty. Implicit in our responsibilities as educators is the early identification of struggling students, both for their own sake to ensure access to treatment, but also with respect to transparency with regards to career trajectory. Honest feedback to students who are struggling emotionally and providing relevant support or even diverting those, who despite maximal treatment are unable to manage training into a more strength-based career direction, is of far more value than propping up, or worse still, turning a blind eye to a persistently impaired student out of misguided compassion (Peisah and Haysom, 2021).

This approach seems to be echoed by the United Kingdom (UK) General Medical Council (GMC):

“We heard that in some circumstances minimum requirements for attendance were too rigid and didn’t allow for life events during training. We have also received feedback that, for some students, re-taking a year was in their best interests and meant they were not struggling to keep up going forward. Although students may not be keen to take time out or re-take a year, medical schools should be assisting students to consider what is best and consistent with achieving GMC outcomes and meeting the demands of the course” (West and Coia, 2019).

Student Identity and Professionalism

Montroux and Rees (2017) describe the concept of medical professionalism as comprising personal virtues, professional attributes of competence and knowledge, professional interpersonal practice (including continuous professional development, respect for diversity, ethical values, and safe practice) and wider societal responsibilities. This professional identity starts budding in medical school. In their exposure to the clinical environment, students progressively internalise professional ways, practices and behaviours. This includes how they view the clinical world, including patients, doctors and other health care professionals.

As professionalism (or lack of) in the learning environment is observed and absorbed by students, their responses to such can be consonant (natural or comfortable) or dissonant (unfamiliar, problematic or confronting (Montroux and Rees, 2017). From their extensive research based on medical student narratives, Montroux and Rees (2017) explore the notion of the dissonant experience. This often arises out of identity-related, patient-safety related, and patient-dignity related professional dilemmas, and by witnessing workplace abuse, all of which can be sources of medical student distress. Examples of identity-related dilemmas include students being mistaken for doctors, sometimes used to coerce patient consent for student involvement, and at other times leading to patients seeking advice from students. Patient-safety related, and patient-dignity related professional dilemmas that often result in moral distress for the student include communication failings and asymmetrical power relationships between doctors and patients. Understanding professionalism and its interaction with medical student well-being is now considered a priority in medical education.

Student Culture and the Wider Medical Culture

Medical student culture is individualistic, not collectivist in nature. Henderson et al. (2012) have observed:

“The competitive medical environment reinforces the need for toughness and self-reliance which has become more of a wider cultural phenomenon.”

It is also perfectionistic, and marks are equated with self-worth. There is a fear of failure and of being failed (Peisah, 2017). This paves the way for medical cultures characterised by rigid hierarchies, teaching by humiliation, denial of vulnerability and omnipotence (Peisah, 2020). With a lack of generativity and commitment to teaching in some medical cultures, it is not surprising that medical students frequently perceive their place “at the bottom of the ladder” (West and Coia, 2019) or “in the way.” The UK GMC describes the ideal teaching and learning environment as a compassionate culture, one that provides compassionate and inclusive supervision that offers reasonable flexibility for students with mitigating circumstances, and a culture that enables students to speak up about concerns enabling and supportive supervisory support focused on removing the obstacles in the workplace, rather than creating directive, controlling cultures that focus more on blame rather than on learning and accountability (West and Coia, 2019).

Responses to Distress and Impairment: What Are We Doing About These Issues?

General Principles

From around 1997, policies for medical schools developed as part of the NSW Doctors' Mental Health Program have included:

- Promoting importance of mental health by embedding it in the curriculum.
- Encouraging a caring culture within the medical school.
- Identifying suitable staff to provide assistance to students with psychological and social problems; and
- Assisting students to access good medical and psychological care (Tennant, 2002)

It is one thing to offer such services, it is another for students to actually access these. The myth of omnipotence that doctors are “invincible” and the consequent self-stigmatisation associated with illness is well-documented in doctors (Henderson et al., 2012) and equally evident in students. The National Mental Health Survey (National Mental Health Survey of Doctors and Students, 2019) described a range of barriers to students seeking help including embarrassment, impact on registration and right to practice (34.6%) concern about career development/progression (37%); privacy and confidentiality (49.9%) and reliance on self/ don't want help (47.7%). Not surprisingly, only 56% of students who felt seriously depressed, or had received a diagnosis of depression, and 40% of those who felt seriously anxious or who had been diagnosed with an anxiety disorder sought treatment. Counselling was the most frequently used treatment in younger students,

while older students were more likely to be treated with both counselling and medication. The most common sources of support for students with depression and anxiety were general practitioners, family and friends, and the internet. Notably, few students with depression or anxiety seek support from faculty services, attributed to perceptions of stigmatising attitudes or concerns regarding privacy and confidentiality.

Preventative small group work with medical students may address some of these barriers. For example, there are promising findings from an open-label, single-session, cognitive-behavioural intervention for medical students, focused on improving mindfulness, emotional awareness, and cognitive flexibility (Bermudez et al., 2019). Small group prevention work in the form of Reflective Practice groups is currently delivered at the Sydney Medical School through the Psychiatry and Addiction Medicine (PAAM) term in the curriculum (Lele et al., 2023).

If we are going to make a difference, there needs to be change. Henderson and colleagues (Henderson et al., 2012), have advocated for cultural change from medical school onwards to allow emerging doctors to recognise their vulnerabilities to allow them more easily to recognise and manage illness of any kind, to which we are all vulnerable. Understandably and appropriately so, healthcare professionals often feel an extra responsibility to students with health concerns (Fertleman and Carroll, 2013). As educators we have a responsibility to act as “counter-weights” to some of the unhealthy myths that abound in medical culture and model self-care and recognition of this vulnerability. Finally, there is evidence that dealing with mental health problems at an early stage of training can help promote resilience and reduce burnout later (West and Coia, 2019).

Resources

There are a variety of services available to students in need, ranging from university-based to externally provided support services.

Acute Distress

If it is urgent, namely there are concerns of self-harm/suicide or psychosis, students should attend the local Emergency Department (ED), (and), or the local hospital psychiatry services on call. In light of the aforementioned stigma, shame and reluctance to seek help, if a clinician is involved in the referral they should ring ahead to discuss and ensure that the student makes contact in urgent cases. Emergency departments are understanding of, and compassionate towards, medical students and colleagues who are in distress. The person will be seen in a private area to ensure their confidentiality as best as is possible and consultation liaison psychiatry will often

provide advice and support, usually at a senior level. Many students prefer to be seen outside their teaching hospital environment and if this is possible it should be arranged. If it is not urgent, and the person can guarantee their own safety, consider other options such as the individual's private GP.

Further Reading

- Lele, K., Leung, K. C., & Peisah, C. (2023). A reflective practice group tutorial series for medical students. *Australasian Psychiatry*, 31(4).
- Leung, K. C., & Peisah, C. (2023, June). A mixed-methods systematic review of group reflective practice in medical students. In *Healthcare* (Vol. 11, No. 12, p. 1798). MDPI.
- Peisah C. & Haysom G. Chapter 18. Professional Capacity. In: O'Neill N., Peisah C. (2021) Capacity and the law. 4th Edition Australasian Legal Information Institute Communities
<http://austlii.community/wiki/Books/CapacityAndTheLaw/>.

Chapter 3: Classification in Psychiatry

Gin S. Malhi

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia, Visiting Professor, Department of Psychiatry, The University of Oxford, UK, and Oxford Uehiro Centre for Practical Ethics, Faculty of Philosophy, The University of Oxford, Oxford, UK.

and

Erica Bell

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

Introduction

The need to describe and define mental disorders and have a classificatory system that is logically organised is undeniable. The first problem one faces however, when embarking upon such a task, is what precisely should be included, and how should the description of things that are to be included be organised so that the architecture of the taxonomy makes sense. These uncertainties remain a matter of considerable debate, and this is not surprising given that in addition to discriminating between different kinds of mental illness, it is necessary first to differentiate abnormal experiences and behaviours from what is generally regarded as normal – whatever that may be. This is necessarily difficult and requires careful consideration, noting that there is no absolute normal and that most boundaries in psychiatry are subject to judgement.

The two main taxonomies that are used worldwide to define mental illnesses are the American Psychiatric Association Diagnostic and Statistical Manual (DSM) of mental disorders (American Psychiatric Association, 2013b; American Psychiatric Association, 2022), which is now in its 5th edition (DSM-5) and has been further revised (DSM-5-TR) (American Psychiatric Association, 2022), and the mental, behavioural or neurodevelopmental disorders section of the International Classification of Diseases (ICD) (World Health Organization, 2019). The latter has also been updated recently, and its 11th version, (ICD 11) was released in 2022.

When DSM-5 was published a decade ago in 2013, the US National Institute of Mental Health (NIMH) proposed a new approach to ‘capture’ psychiatric phenomena. The novel taxonomy was abbreviated to RDoC, which stands for Research Domain Criteria (The National Institute of Mental Health (NIMH), 2013). It was intended largely for research, pursuing transdiagnostic phenomena such as irritability (Bell et al., 2021b; Bell et al., 2021a), however, it was also meant to inform clinical decision-making and practice.

Alongside these main taxonomies, there are many local manuals that describe different cultural interpretations and traditions, however in this textbook, reference is made mainly to DSM and ICD. In this chapter, we examine a number of examples of diagnoses and disorders within these classificatory systems and the controversies surrounding them, to illustrate some of the challenges that arise when trying to define and classify psychiatric disorders.

Dimensional or Categorical?

As a doctor, one of the primary considerations when diagnosing any illness is to determine whether a set of symptoms meet a specified threshold to define an illness or disorder. This is easier if there is pathology that is known to be associated with the illness. The clinical diagnosis can then be confirmed based on the findings of a relevant test (e.g. plasma levels of a hormone) or investigation (e.g. an x-ray examination of the chest). Alternatively, there may be signs that can be reliably elicited and are highly characteristic of the illness, perhaps even pathognomonic.

In psychiatry, this is seldom the case, and most diagnoses rely predominantly on symptoms and occasionally some signs, but to date there are no tests that can definitively diagnose a psychiatric disorder such as depression. Nevertheless, for classificatory purposes and even more importantly, to communicate with patients, it is necessary to invoke the concept of a ‘category’. This is because patients, quite reasonably, want to know if what they are experiencing is abnormal, and if so, whether it is an illness. In clinical practice this means sorting individuals into those that have an illness or disorder and those that do not. The problem with this approach when considering psychiatric conditions is that it is often difficult to identify a clear demarcation between what is normal and abnormal. A good example of where this distinction is challenging concerns the symptom of anxiety, which is most often a normal physiological response that is necessary for day-to-day functioning. However, it can be deemed abnormal if severe or sustained and it begins to limit functioning. Conversely, extremely low mood may be ‘normal’ in the context of a severe loss such as bereavement. But even when there is a *prima facie* understandable reason for feeling low such as a stressful life event, it still may be thought of as depression, i.e. a psychiatric disorder. In other

words, a clear and confident delineation between normality and abnormality on the basis of symptoms and signs alone may simply not be possible.

Therefore, in addition to having set criteria in terms of specific symptoms and their duration, there is also a requirement for some degree of functional impairment. Nevertheless, in practice these judgements remain complicated, as the process of definition is also dependent on individual characteristics, and thresholds of distress differ from one individual to the next. For example, some stoic individuals may have a higher pain threshold than others and similarly resilience in dealing with stress varies considerably across the population - especially as it is also a composite construct (Malhi et al., 2019). However, in practice, it is necessary to be able to differentiate between those that do have an illness so that they can be helped and offered treatment, and those that do not require any special medical or psychological assistance.

In addition to differentiating disorders from normality, and distinguishing between disorders, further subcategorisation occurs *within* certain disorders, to define specific *kinds* of illness. Again, it is important to note that the 'depth' of any such distinction is largely descriptive in most cases, and only occasionally is it causal. Staging and subtyping are common terms that are used to describe entities within psychiatric disorders. For example, depression may be 'mild' or 'severe', it may be described as 'atypical' or 'melancholic', or there may be different types of depression such as that occurring in bipolar disorder (bipolar depression) and that featuring psychotic symptoms (psychotic depression). However, it should be noted once again that these so-called categories do not have crisp boundaries and that many seemingly separate categories necessarily overlap, and some delineations remain highly disputed.

An alternative approach is to describe illnesses dimensionally. This certainly accords with clinical experience, where sadness for example varies by an imperceptible degree as does anxiety. So too the number of symptoms and their duration seems to lie on a continuum. However, there are exceptions, for example the emergence of hallucinations or clear-cut delusions is often 'all or nothing'. But the dimensional perspective, whilst perhaps more accurate in reflecting reality, makes the distinction of normal and abnormal difficult and hinders the stratification of disorders in line with treatments and therefore boundaries need to be defined, and hence this is why current approaches artificially partition the dimensional experiences of psychiatric phenomena into categories. Having accepted that this is a somewhat theoretical and pragmatic way forward, the resulting classificatory system does for all intents and purposes provide a useful way of conceptualizing psychiatric diagnoses. Within this framework, diagnoses are symptoms that have been aggregated into syndromes and labelled as disorders, provided they meet a certain

threshold, whilst noting that many of them overlap with other disorders and probably lie on a fundamental substrate that is essentially continuous. In other words, our clinical schema for psychiatric diagnoses likely does not reflect the psychological and biological mechanisms underlying mental distress.

Biological or Contextual?

Yet another way of defining and understanding psychiatric disorders is through understanding their causes. This aetiological perspective assumes that there are specific causes that lead to psychiatric manifestations. It draws upon the biomedical model and in particular the microbial model of illness and disease, in which a single bacterium for example can be the primary cause of an illness. The complexity of how the disease manifests is evident by considering the effects of the microbium within physiological processes, for example infections often lead to changes in temperature, and gut sensitivities resulting in nausea and vomiting. Infections also often cause lymphadenopathy, pain, and inflammation. Classification solely on the basis of these downstream sets of symptoms is likely to be potentially inaccurate and misleading, and hence why knowing the pathogen is of tremendous importance. The classification of psychiatric disorders, or at least the identification of some psychiatric disorders that are thought to have a discrete pathophysiology, is modelled on similar thinking, that is, specific causal pathways can perhaps be mapped and a deeper understanding of the processes that lead to the illness can be determined.

Enthusiasm for this line of inquiry has been fuelled by the enormous advances in technology over the past three decades. To this end, research using the latest technological advances and insights in medicine, such as those afforded by genetics and neuroimaging, have provided many clues as to the perturbations that are associated with psychiatric disorders. They have also led to greater understanding of many biological processes and the impact of treatments. However, alas, as yet, *a complete and satisfactory pathophysiology of any psychiatric disorder is yet to be achieved* and thus *classification according to causes is not possible*. Despite this, in formulating psychiatric disorders, many of the diagnoses are discussed in terms of their potential causes, particularly when planning management. In depression for example, it is often the case that a life event or stressor is closely associated with the onset of an episode of illness or that the illness is being maintained by such contextual factors. At the same time, many illnesses that seem to cluster in families are described as familial and thought to be hereditary – the implication being that there is a biological component such as a genetic predilection.

The separation of various causal factors is inherently difficult and even if this were possible, whether individual causal elements reliably inform treatment

remains, unclear. Naturally, if an obvious stressor is compounding an illness or is the direct cause of a set of symptoms, as is often the case in phobias, then this can be addressed directly. However, in most psychiatric diagnoses, such as mood disorders and psychoses, such causal links are less direct, especially as some putative causal factors can also be self-driven adaptations and attempts to ameliorate symptoms. For example, substance misuse clearly impacts mental health, but often excessive use begins as a strategy to overcome symptoms of an underlying illness in the first place. Hence, the strong reciprocal associations between mood disorders and alcohol use, for example.

Nevertheless, despite all these limitations, the biological versus contextual approach does provide a schema for exploring the associated factors of any psychiatric disorder and assists in formulating the clinical management plan. To this end a biopsychosocial approach is often useful as it provides a broad framework for different sets of interventions and provides a richer contextual understanding of the illness whilst respecting the fact that many factors are unknown and that some of these are likely to be intrinsic.

Mixed Mood States

In some instances, the assumptions and approximations made to define categories, or the adoption of a purely pragmatic approach, can mean that clinical diagnoses become somewhat estranged from reality. Put another way, real disorders are not captured by the diagnostic label. For example, in the chapters on depressive disorders (Chapter 16), you will read about ‘mixed mood states’ along with the polar extremes of melancholia (depression) and mania, even though mixed mood states are not formally captured in DSM-5 TR. Instead, they are referred to broadly and simply as ‘features’ or ‘specifiers’. This is a problem, as mixed mood states are common in clinical practice and feature in up to a third of bipolar presentations (Judd and Akiskal, 2003). The fundamental problem here is that the classificatory framework for mood disorders is contingent on having two poles - depression and mania - and the symptoms of mood are necessarily constrained as they have to be diametrically opposed and be assigned to either pole. In other words, using this framework, all symptoms are categorical and mutually exclusive. Instead, if symptoms are conceptualized dimensionally, it becomes evident that almost all mood symptoms exist on a spectrum that extends from depression to mania and that symptoms can also occur independent of their polar groupings. In other words, not all the symptoms of depression necessarily co-occur and that at times symptoms of both mania and depression may coexist thus creating a ‘mixed mood state’. To accurately capture this, a dimensional perspective is needed and at the same time it helps if the symptoms can be put into domains as has been done by the ACE (Activity,

Cognition and Emotion) model for bipolar disorders and more recently by ICD-11 for depressive episodes (Malhi et al., 2018b; Malhi and Bell, 2023).

The ACE model (Malhi et al., 2018b), which is discussed further in the chapter on bipolar disorder (Chapter 17), shows that by grouping symptoms into domains of activity, cognition, and emotion and regarding individual symptoms as functionally independent, the occurrence of mixed states can be explained. In this way, the ACE model provides a useful means of capturing the clinical presentations of mood disorders as well as targeting treatments. The model also helps explain how various mood symptoms become uncoupled and how this may be brought about by interventions such as antidepressants. This is important because the prescription of antidepressants in bipolar disorder can precipitate what is commonly referred to as a ‘treatment-induced mixed state’. This arises because some of the domains of symptoms are more susceptible to the effects of the antidepressant and therefore symptoms that usually occur in unison become separated. For example, the person is full of energy and has no need for sleep and yet simultaneously feels low in mood and is unable to enjoy themselves. Therefore, in a categorical model such as DSM-5-TR, mixed mood states warrant being a separate category, but because the symptoms themselves are not conceptualised dimensionally, the mere existence of such a category cannot be acknowledged. This is illustrative of the difficulty of needing to have diagnostic entities that have practical boundaries while trying to capture a clinical picture that comprises continua and is intrinsically dimensional in nature.

Schizoaffective Disorder

In contrast to mixed states, wherein current diagnostic labels fail to capture a distinct entity, some DSM-5-TR psychiatric disorders serve as examples of diagnostic labelling attempting to categorise discrete, separate disorders which do not necessarily have any evidence base to support their separation. For example, schizoaffective disorder (SAD), within both DSM-5-TR and ICD-11, essentially describes an overlap of psychotic phenomena and mood episodes, wherein an individual meets the criteria for both schizophrenia and a mood episode (these can be either depressive or manic) (Malhi et al., 2008). However, despite being introduced to DSM-III over 40 years ago, the diagnosis lacks both reliability and validity and provides no specific insight as regards appropriate treatments, illness course or prognosis (Malhi and Bell, 2019). Indeed, no clinical practice guidelines for the management of schizoaffective disorder have been produced in this time period, despite the diagnosis being widely applied in specialised mental health services (Morgan et al., 2012b). Thus, clinically, the management of schizoaffective disorder is generally an amalgamation of strategies for managing schizophrenia and mood disorders, with no strategies specific to schizoaffective disorder itself.

This lack of separation between schizoaffective disorder and schizophrenia in particular is evident within the classificatory systems themselves. In ICD-11 for example, the ‘first-episode’ specifier of schizoaffective disorder cannot be applied if the patient has experienced a prior episode of schizoaffective disorder or schizophrenia, as the disorders are apparently considered interchangeable in this regard.

Schizoaffective disorder is emblematic of the use of psychiatric classificatory systems beyond the scope for which they were originally developed. Rather than accurately capturing diagnoses that provide insights regarding aetiology, course, prognosis and treatments, some diagnostic labels such as schizoaffective disorder have been misused to facilitate the administration of treatments such as antipsychotics, while circumventing a more accurate diagnosis such as schizophrenia, because it carries significant stigma or conveys a poor prognosis. In and of itself this is not necessarily problematic, and it is understandable that early in the course of an illness a definitive diagnosis may not be positive, but once a label is applied it is seldom revised and rarely removed, and this is a problem.

Conclusion

This chapter has briefly introduced and outlined the primary purpose of classificatory systems in psychiatry, which is, to describe and define mental disorders to inform diagnosis and treatment and to provide an understanding of prognosis and illness course. However, it has also highlighted the limitations and flaws within our current taxonomies and illustrated these using some contemporary examples. The principal point to bear in mind is that these classificatory systems are not written in stone, nor are they necessarily reflective of clinical reality, and therefore in clinical practice they must always be viewed critically and utilised judiciously.

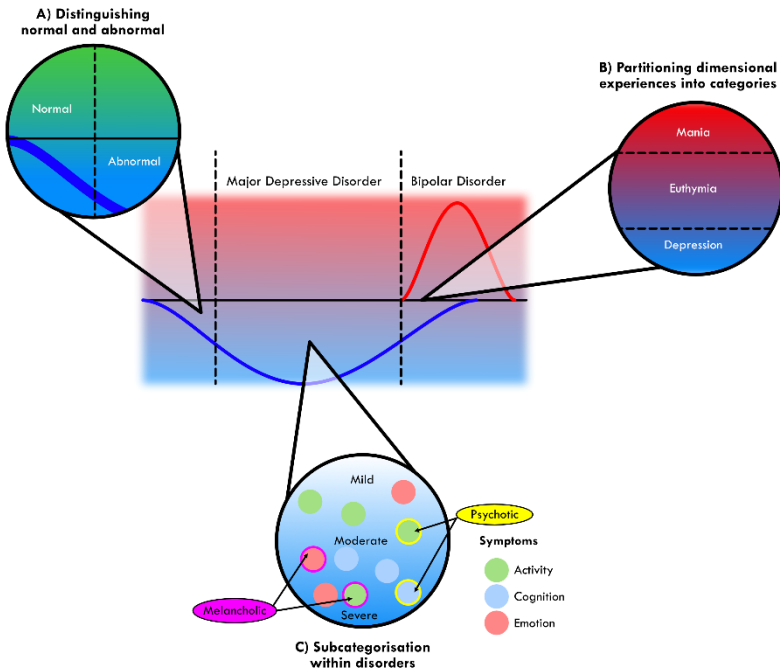


Figure 3.1. Schematic illustrating dimensional and categorical conceptualisation of psychiatric disorders. A) Primary purpose of diagnosis: the distinction of normal experiences from those that are deemed abnormal. B) Artificial partitioning of a spectrum of experiences into distinct diagnostic categories such as a depressive or a manic episode. C) Subcategorisation within disorders. For example, depression can be further specified (subcategorised) in terms of severity (e.g. ‘mild’, ‘moderate’ or ‘severe’) or according to descriptors derived from the pattern of how specific symptoms manifest such as ‘melancholic’ or ‘psychotic’. Finally, models such as the ACE model capture clinical presentations of depression by grouping symptoms according to the domains of activity, cognition and emotion; a similar approach has been adopted by ICD-11 with the development of clusters.

Further Reading

Guloksuz, S., & van Os, J. (2018). The slow death of the concept of schizophrenia and the painful birth of the psychosis spectrum. *Psychological Medicine*, 48(2), 229-244.

CLINICAL SKILLS



Artist: Anne Naylor

Title: *Landscape of the Mind*

Medium: Acrylic Paint on Canvas

Dimensions: 700 x 1000 mm

Chapter 4: The Psychiatric History

Peter Kelly

Specialty of Psychiatry, Sydney Medical School, The University of Sydney, Australia.

and

Christopher James Ryan

Specialty of Psychiatry, The University of Sydney, Australia, The University of Sydney, Australia, School of Clinical Medicine, The University of New South Wales, Australia, and School of Medicine, The University of Notre Dame Sydney, Australia.

“The good physician treats the disease; the great physician treats the patient who has the disease.”

William Osler

Introduction

At the beginning their psychiatry term, many medical students are demoralised when confronted by what they see as a completely different approach to taking a history. In fact, though, the approach to taking a good psychiatric history is, in essence, exactly the same as the approach to taking a good medical history. It is just that in psychiatry many of the elements that were not obvious in taking a history from most medical and surgical patients are brought into sharp relief.

A psychiatric interview has three main aims: to gather information sufficient to allow for formulation and diagnosis of the person’s predicament and to speculate on its origins; to develop and maintain the therapeutic relationship; and to communicate information so that a management plan can be negotiated.

These are, of course, exactly the same aims as any medical interview. Just like any patient with a medical or surgical problem, the way people with mental illness experience their maladies are heavily influenced by their personality styles, coping mechanisms and the extent of their family and social supports. Just like any

medical or surgical patient, the predicaments that psychiatric patients find themselves in are diverse and multifaceted, going far beyond the simple impact of a series of symptoms. Like many medical and surgical patients, many psychiatric patients will not readily fall into the patient role and accept that everything suggested by their doctor is for the best or that all directions should be slavishly adhered to. And, like many medical and surgical patients, many psychiatric patients will not understand management suggestions without considerable effort on the part of the doctor and will not accept suggested plans without negotiation.

Leaving to one side the fact that all doctors will frequently see patients with mental illness, you may think of your psychiatry term as a way of gaining advanced interviewing skills that will be vital when engaging with all of your future patients. By the time they do their psychiatry term, most medical students are relatively proficient at eliciting a history of a disease. During psychiatry you'll be practicing eliciting the history of the person who has the disease. In Osler's terms, you'll be learning the skills to become a great physician.

In this chapter we will provide a step-by-step guide to taking a psychiatric history. We shan't be focussing too much on exactly what symptoms to elicit – details on those will be given in the chapters to come. Instead, we'll be examining more general issues – how to begin, what areas to cover and how to continue when things seem hard.

We'll start with examining what needs to be attended to even before the interview begins. We'll then go on to review the beginning of the interview and the way to elicit the presenting complaint. Most of the chapter will focus on the history of the presenting illness and the ways in which the approach to that might differ from what you've become used to. The rest of the chapter will run quickly through the other headings of the history, focusing on two issues given special prominence in psychiatry: personality; and insight and capacity.

Frequently the secret to a great interview is hidden in the subtleties of how questions are phrased, so from time to time we'll suggest a form of words that might be useful in a particular situation. When we do, we'll place that text in a little box like the one on the following page. These are suggestions, and not meant to be slavishly learnt. Ideally, the student will develop their own preferred words and phrases.

“Hello Ms Pappenheim, I’m Bill Osler. I’m a fourth-year student at Sydney University working on the ward here with Dr Jung. I was wondering if I could ask you a few questions about how things are going?”

The Set Up

In some ways taking a history is just another medical procedure and, like all medical procedures, setting things up properly before you begin is vital.

People in the grip of psychiatric illness may become very agitated, may be prone to misinterpret cues in their environment, or may lose control of their actions. As a result of those realities, very rarely staff and students on psychiatric wards are assaulted, sometimes seriously. This is not something that you should lie awake at night worrying about – serious incidents are rare, but it is something to keep in mind.

To minimise the chances of such incidents all psychiatry wards will have some rules or guidelines on how interviews should be conducted. These are not always obvious and may not be written down anywhere. They might include the need to carry a duress alarm, or they may place restrictions on who students can, and can’t, see. Most likely your supervisor will let you know about these rules when you first arrive on the ward, but if you’re not sure, always ask. In the same way, you should check with a staff member before you begin interviewing any patient. Some patients may be particularly agitated or upset at that moment, or may have particularly acute privacy issues, or may simply have seen hundreds of students and have requested a break.

Psychiatric patients don’t spend the day sitting conveniently by their beds waiting for doctors and students to approach them. Generally, you’ll find patients in common rooms or courtyards, and having secured their agreement to be interviewed, you’ll usually need to find somewhere that the patient will be comfortable talking to you. On occasion, a quiet area of the courtyard will be fine, but often it will mean retiring to the patient’s room or an interview room on the ward. Many patients will be on routine observations that require they be sighted by a staff member every 10 to 30 minutes, so if you take a patient away somewhere, make sure you tell the nursing staff where you’re going. This will also give the nursing staff the opportunity to tell you that it is not a good idea to take this particular patient to the isolated interview room at the end of the corridor.

Before you begin the interview proper make sure the room is arranged so that everyone will be comfortable. Almost always this will mean allowing the patient to choose where they would like to sit and then arranging a chair for yourself. Multiple studies have shown that patients perceive doctors who sit during interviews as more compassionate and caring and as spending more time with their patients, even when the actual time spent was the same (Gupta et al., 2015; Swayden et al., 2012). Consequently, you should always sit during interviews even if, on the medical wards, that means scooting off first to find a chair. Don't sit on the patient's bed without asking permission.

Once you've established the environment you may begin the interview proper. Introduce yourself again, re-affirm your role, thank the patient again for their time and set out the purpose of the interview, which in your case will usually be primarily so that you can become a better doctor - "this is part of my training". Let patients know the broad areas you intend to cover and apologise in advance for asking them some of the same questions they would have been asked many times before. If you've not looked at the patient's medical record, you might want to be explicit about that so that the patient understands why you're asking about things that have already been recorded by others.

Refer to the patient by their honorific and their second name (e.g., Mr Lanzer) and only use their first name if invited to do so. If you are not sure if its "Miss" or "Mrs", "Ms" is usually safest.

Beginning the Interview Proper – Demographics and Presenting Complaint

Begin the interview by gathering some basic demographic data. Not only will this be of great of assistance in coming to understand the patient's situation, but since demographic questions tend to be routine, it will give you more time to deepen your rapport before getting into more sensitive areas.

Ask for the patient's age and occupation or, if unemployed how they support themselves financially. If you're not familiar with what the patient's job entails, ask. Once again this will give you a greater understanding of the patient's circumstances and will demonstrate that you're interested in the patient's life, not just his or her disease. Ask patients where they live, and who lives with them. Avoid asking if the patient has a "husband" or "wife" as these questions usually assume things about the patient's sexual orientation that are likely to distance you from same-sex attracted patients.

As a general rule you should take notes as you go. This tends to be much faster and more accurate than making notes later and allows you to copy down some important patient responses verbatim. It also gives you a little more time to consider the responses you get and formulate your next question. Taking notes as you go though is a skill in itself and (like everything) you'll get better with practice. While with some patients it might be a barrier to building rapport, the reverse is also true, and many patients appreciate the attention you appear to be giving to their utterances. Obviously be circumspect about eagerly scribbling down patient descriptions of deeply personal experiences and be aware that some patients with persecutory ideation may be suspicious of avid note taking.

By the time you've obtained their demographics, patients should be reasonably comfortable in your presence and hopefully you in theirs. Now is the time to explore the presenting complaint.

Of course, in psychiatry, the presenting complaint may well have been a complaint of someone else - not the patient. On occasion patients are perplexed or even dumbfounded as to why the ambulance or police were called or why their partner insisted they should come to hospital. In those instances, simply note down what the patient recalls of that time.

"May I ask ... how did you come to be in hospital here? What happened for you to be admitted?"

Staking Out the History of the Presenting Illness

At the front end of the history of the presenting illness, your focus should remain on open-ended questions, with the occasional exception of some closed questions aimed to anchor the patient's experience in time. Be cautious about asking questions that begin "Why did you ..." as they may be taken as implying criticism and provoke a defensive response.

Taking the history of the presenting illness can often seem a daunting task for medical students. Commonly students feel a tension between allowing patients to express themselves and the desire to gain as much relevant information as possible. The sheer length of time required to gain the history requires a certain level of adaptation, as it is expected that a psychiatric history will require much more time than the sort of focused history usually encouraged in other specialities. Experienced psychiatrists will usually set aside an hour or so to take a history from a new patient,

and even they realise that this will be insufficient to cover everything that will need to be addressed.

“Ok. I see. And then what happened?”
“Can you tell me more about that?”
“Can you say more about that?”
“Could you fill me in on that?”
“Right, what did you make of that?”
“When was that exactly?”
“When was the last time you felt completely yourself?”
“Avoid questions beginning with “Why?””

For at least the first five minutes or so, try to let the patient lead the way. Even if you hear something extremely unusual (perhaps suggesting a delusion, say), try not to interrupt but instead note it down as an area to return to clarify. If you focus in on one aspect of the history too early, you risk patients never revealing other vital elements of their experience.

Only after patients have staked out the general territory of their problems, should you zoom in to try to clarify what each part of that territory really looks like. Sometimes efforts at closer surveillance will open up whole new vistas of concerns, and in that case, it might be wise to zoom out again, until this new piece of real estate is also marked out. The discipline to modulate between structure and flexibility, between big picture and fine detail, is another skill that only comes with exposure and with practice.

Though it hardly seems worth saying, take care to listen to what the patient is actually saying. Remember that a patient’s aims and motivations in giving a history are not necessarily the same as your aims in taking it. Patients don’t necessarily know what’s important and sometimes it is a feature of their illnesses that they do not recognise the importance of what would be to us crucial pieces of information. We regularly see students completely miss clear diagnostic markers or bright red flags important in a patient’s future management, simply because patients gave no weight to them in their utterances and students were distracted by formulating their next question.

In many ways your aim in eliciting the first part of the history of the present illness is to come to as good as understanding as possible of what it is like to be the patient. There are two main ways of pulling this off.

First, after the patient has set the ground of his or her complaints, return to specific important events in that ground and take the patient through them in detail. It is important to get the time sequence of events and when symptoms appeared. Summarise this back to the patient and check with them that you have it correct. Probably the most common example of an important event prior to an admission is a suicide attempt that precipitated the admission. Focus in on the attempt, taking the patient back to a time before they decided to act, and then ask them to describe events as they unfolded. Details are crucial here. The patient who, after an argument, took a handful of tablets in the bathroom after announcing their intention and slamming shut the bathroom door, may have been in a very different state of mind to the patient who, after an argument, slipped into the bathroom unseen, closing the door quietly behind him. By the end of this history of the event, you should be able to construct a movie in your mind's eye of the patient's experience – a movie complete with subtitles explaining what the patient was thinking and feeling as the events unfolded (Ryan et al., 2015c).

The second approach to understanding what it is like to be the patient is to ask for clarification. Remember that patients do not simply relay their symptoms as detached factual reportage; they also try to interpret and make sense of them. The chapters that follow will deal with the variety of clinical features found within psychiatry's myriad syndromes and specialities, but for the purposes of this chapter it is important to emphasise that as each new symptom emerges, you should do your level best to clarify exactly what the patient means when they say that they had that experience or feeling.

“Different people mean different things when they say depression. Depression affects people in different ways. Can you tell me more about how your depression affects you? You said you were having trouble sleeping. Can I ask, what time do you usually go to bed? ... And do you usually go straight to sleep? ... What time do you wake up? ... Is that early for you? ... Do you ever get back to sleep again?”

Although students will always feel pressure to move on from the history of the presenting illness, and eventually of course other areas must be covered, you should feel very reluctant to leave any particular piece of symptomatology without nailing exactly (or as exactly as possible) what the patient meant when he or she reported it.

Before moving on to the second phase of the history of the presenting illness it is worth dealing explicitly with one anxiety-provoking situation that occasionally arises. Sometimes you can be nine or ten minutes into taking the history and despite

your best efforts, you still have no real notion what this patient's problem is, or even how the patient came to be in hospital. There are two main ways of dealing with this.

First, consider the possibility that the reason that you cannot discover the reason for the patient's admission may relate to the patient himself being unable to recall it. Many psychiatric disorders seriously impair patients' cognitive abilities, and this may not be readily apparent until their cognition is formally tested. Though in the normal course of events one would not perform formal cognitive testing until toward the end of the interview, this situation might prompt you to embark on cognitive testing much earlier, midway through the history of the presenting illness. If it turns out that the patient is significantly cognitively impaired, then that will place any historical details you elicit under a cloud, and the focus of interview may turn to performing the best mental state examination possible.

The second approach to the patient whose presentation still remains a mystery nearly 15 minutes in, especially one where cognitive impairment is not the problem, involves what might be called a "jump-out question". A jump-out question is like a meta-question, a question to the patient about the interview process.

"I have got to tell you Ms Bauer, I'm confused. You see we've been chatting for about 10 minutes and you've been very kind in answering all my questions, but despite your best efforts, well ... this is a psychiatric ward and I'm still not at all clear what happened for you to be admitted here."

Sometimes by clearly putting your dilemma on the table patients can come to see this as an issue for the first time, and sometimes having seen what exactly you're after they are able to address that issue, with a sort of "why didn't you tell me that's what you wanted to know" attitude. Many times, it must be said, even this approach fails to clarify the situation, but even in that event, if you are unfortunate enough to encounter such a patient during an observed exam, at least the question signals to the examiner that you know you're having trouble and you're doing all you can to get out of it.

More of the History of the Presenting Illness

Once the patient has provided their version of the history of the presenting illness and you have clarified that as much as you can, it is time to move onto other questions designed to enrich your understanding and to challenge or confirm your

emerging assumptions about diagnosis and the factors that led to the patient's current predicament. This will involve using a range of fairly standardised enquiries.

By this time in the history of the presenting illness, you're likely to have hypothesised that much of the patient's experience represents features of one or more psychiatric syndromes. This is now the time to find out if the patient has any of the other features associated with the various diagnoses you're considering.

No medical student interview should be completed without having broached the possibility of the patient being suicidal. Concerns about suicidality will often have been the reason for a patient being admitted, so suicidality will be a common (but sometimes unvoiced) issue among the patients you see on the wards. In the exam situation, few examiners will feel comfortable passing a student if he or she thought that the patient may have experienced suicidal ideation, but the student did not explore this.

"You told me that sometimes you got very depressed/sad/frightened. Sometimes when people feel like that, they begin to think that it's not worth going on. Have you ever felt like? Have you ever considered ending it all?"

If there is some hint that the patient might have considered suicide, you cannot leave this topic, until, as much as possible, you understand the whole of patient's thinking on it. These questions are important and now is not the time for euphemisms such as *"you wouldn't do anything silly would you?"*. Be sensitive about these enquiries but be direct.

Two things ought to be said about questions into a person's suicidality. First, though it is a common concern among students, there is no evidence, and no reason to think, that simply asking patients about suicide makes them any more likely to try to suicide. The hope is, of course, that such enquiries may *decrease* the likelihood of patients suiciding as they may feel relief in being able to talk about their fears and distress. Try to look comfortable asking about suicide (even if that's not exactly how you feel). Practice helps here.

“Have you thought of killing yourself?”

“Have you thought how you would do this?”

“Have you (ever) made any plans or taken any action?”

“Do you still feel like that now?”

“What might stop you from killing yourself if those thoughts came back?”

Second, many textbooks of psychiatry state that it is important to ask questions about suicide so that the interviewer may make an estimate of the patient’s suicide risk. That makes no sense. It *is* important to ask about suicide, but its importance has nothing to do with gauging the likelihood of the patient eventually dying by suicide.

While all psychiatric inpatients are, and will continue to be, at many times the population at risk of completed suicide, countless studies have now demonstrated that there are no clinical features, either alone or in combination, that can be used to usefully divide this group of patients into those at even higher risk of suicide and those at relatively lower risk (Large and Ryan, 2014). The futility of efforts to do so is a direct result of the extremely low base rate of suicide even in this very high relative risk population. Despite its popularity in textbooks and even some government policies, useful suicide risk assessment of acute psychiatric patients is simply impossible (Ryan et al., 2010).

It is important to ask about patients’ suicidality, and about all the clinical features relevant to that, not because we can then predict who is more likely to die by suicide, but because that enables us to better understand the patient, and understanding the patient is our primary tool in caring for and minimising the chances of suicide in all our patients.

In a similar vein, and for essentially the same reasons, it is impossible to usefully classify psychiatric patients into those more or less likely to harm someone else (Paton et al., 2014). Nonetheless, if there is any suggestion that a person might come to harm another, similar questions need to be pursued.

“Have you thought of hurting other people?”

“Have you thought how you would do this?”

“Have you ever hurt others physically?”

Having clarified *your* views on the patient's clinical state, now is the time to clarify the *patient's* views on the patient's clinical state.

"What do you think is wrong?"

"What have the doctors said the problem is? Have they suggested a diagnosis? What do you think of that?"

"What do you think could be helpful?"

"The doctors have put you on this medication. Do you think that that is helping? Many people simply forget to take all the medications they are prescribed, do you find that you often forget to take your tablets?"

Try too to gain the patient's perspective on the impact of their illness. How has it affected his or her life? How is it still causing disability and to what extent has he or she been affected by the stigma still so closely attached to psychiatric illness?

The final task in gathering the HPI is some sort of "systems review" looking for features of the major psychiatric illnesses that did not come up as the patient presented the history and that might never come up unless explicitly explored. The exact nature of the systems review will vary from patient to patient but might include screening questions for features of:

1. Psychosis – delusions or hallucinations (see Chapter 15)
2. Mania – elevated or irritable mood, increased energy or racing thoughts (see Chapter 17)
3. Depression – low mood, anhedonia, guilt, sleep or appetite changes (see Chapter 16)
4. Anxiety – worry, obsessions, panic (see Chapter 18)

The Bulk of the Psychiatric History

The history of the presenting illness successfully negotiated, students can run through much of the rest of the psychiatric history covering pretty familiar ground. The rest of the history is after all just the standard medical history with a few added headings and some slight changes in emphasis.

Under the heading of "past psychiatric history" review any previous episodes of illness, any previous suicide attempts or any previous treatments or hospitalisations. It is useful to know if previous episodes have involved treatment under the mental health act, or the use of electroconvulsive therapy, lithium or

clozapine, as these reveal the views of previous psychiatrists about the nature of the patient's disorder.

Review the patient's "substance use history" carefully gaining details on any type of substance ever used including, of course, tobacco. In many instances, substance use can overlap with the History of the Presenting Illness; for example, in those whose episodes of illness were precipitated by substances, or where increased substance use is a symptom of a disorder (see Chapter 29). It is important to remember that co-morbid substance use may be the rule, rather than the exception, in severe psychiatric disorders. Remember too that alcohol misuse is the most common substance use disorder and that its manifestations can mimic many other psychiatric disorders. This is also the place to ask whether the patient gambles. Given that you've just asked about illicit drugs, whose use, of necessity, involves breaking the law, this is also the time to ask about any "forensic history".

"Have you ever had any trouble with the law?"

Next move onto "medical history" focusing particularly on those illnesses that have particular psychiatric relevance – epilepsy, closed head injury, intellectual disability, diabetes, cancer, thyroid disease and chronic pain disorders. Then onto "family psychiatric and medical history".

"Anyone in the family have problems with their nerves?"

"Anyone in the family have anything to do with psychiatrists, psychologists or social workers?"

"Are there any heavy drinkers in the family? [Bear in mind that alcohol or other substance use disorders are frequently not seen as psychiatric problems]"

"Did anyone in the family die by suicide as far as you know?"

Though a "personal history" and "social history" are often formal parts of a medical interview, these are usually given much greater focus in psychiatry. Enquire into where the patient was born and their position in the family. It could be relevant to ask about birth trauma and achieving milestones.

“Are you the oldest or youngest in the family?”

“What was the atmosphere like in your family when you were growing up?”

“What was the worst thing that happened to you when you were young?” “Did really bad things happen to you?”

“Tell me about your Mum/Dad. What sort of a person was she/he? What was your relationship like with them growing up? What’s that relationship like now?”

Ask about the patient’s school performance, not only academically, but also socially and on the sporting field. Find out when the patient left school and if it was younger than expected, why? Take an occupational history, focusing on what jobs were done and how long they were held. Try to discover any pattern if the patient moved from job to job. Ask about the patient’s relationships and sexual history looking again at stability and any patterns that might be discernible or that the patients themselves have identified. Ask if religion or spirituality is important to the person, and if so try to find out in what ways. For those from cultural backgrounds markedly different from yours, it can be helpful to ask the patient about demands and expectations related to this, bearing in mind that people are not always faultless interpreters of their own culture.

Premorbid Personality

This is not a standard section of a general medical interview, though arguably it should be. As will become obvious in the chapters to come, a person’s personality style will colour the way they give their history and must be accounted for when designing and communicating a management plan. Failure to take account of the person’s customary style of interacting with others, the world and their own internal world is likely to cause the best laid plans to go awry.

Many of your judgements about a person’s personality style are made from responses given over the course of history. Right back at the time of gathering demographic data it should have been obvious that the person who reported that he worked as an actuary was unlikely to be a devil-may-care free spirit, and similarly that a professional exotic dancer was unlikely to be shy and retiring (though it’s best not to make too many assumptions on this sort of thing without further evidence). Some mental state features too will be strongly suggestive of certain personality types.

“This is often quite a hard question, but if you were asked to give a thumbnail sketch of yourself (as you are usually, not perhaps right now), how would you describe yourself?”
“How do you think others would describe you?”
“What do you like about yourself? What are you proud of?”
When you get very angry / sad, how do you cope with that? What do you do?”

In this “premorbid personality” section of the history the issue of personality style is dealt with head on. It need hardly be said of course that patients’ responses to these questions do not necessarily translate into accurate descriptions of their personality traits. The patient who responds to an inquiry as to how others see him with “modest, despite all my many achievements; modesty is my middle name” is probably revealing a great deal about his personality, but his revelations don’t necessarily match his words.

Sometimes you can ask a series of questions to test out any hypotheses about the person’s personality you formed while taking the rest of the history.

“Would you say you’re a worrier?”
“Some people like everything to be just right: “A place for everything and everything in its place”. Are you a bit like that?”
“Some people like to spend a lot of time on their own. Often times they find that they don’t really get other people.”
“Some people would like to spend time with others, but they find that that never seems to work.”
“Some people like to be the life of the party.”

Insight and Decision-Making Capacity

While both these issues can be central to the assessment of medical and surgical patients, it is the nature of some psychiatric illnesses that both of these can be seriously impaired. Insight refers to the level of understanding that patients have into various elements of their own predicament. Never be tempted to comment that a patient’s insight is “absent” or “poor”. Insight is much more complicated than that.

Consider patients’ insight into the nature of their symptoms. Do they, for example, think that their delusions or hallucinations reflect reality? Consider their insight into the nature of their illness. Would they agree that they might be unwell in some way and if they would, what do they feel is wrong? Consider their insight

into any need for treatment. Oddly all these three dimensions of insight are, to some extent, independent (Diesfeld, 2003). It is not that unusual to meet a person with schizophrenia, who knows that his voices are hallucinations, but denies vehemently they are due to schizophrenia, but is nonetheless happy to take antipsychotics to assist with the troubling voices.

Decision-making capacity is related to insight, but the two do not neatly overlap. The assessment of decision-making capacity is relevant only to a particular decision at a particular time. No one loses the capacity to make *all* decisions, unless they become unconscious or profoundly catatonic.

In adults an assessment of decision-making capacity begins with a presumption that capacity is intact. However, this is a presumption that can be rebutted if either the person cannot comprehend and retain the information relevant to the decision, or if he or she is unable to use and weigh that information to come to a decision (Bird, 2011).

Assessing a patient's ability to comprehend and retain information is usually as simple as providing the patient with that information and then asking them to paraphrase it back to you. Obviously, the information has to be given in a form that maximises the patient's chances of understanding it. This might involve getting an interpreter or, even in native English speakers, ensuring the facts and opinions are delivered in plain and simple terms.

Assessing the ability to use and weigh is a little more tricky. Basically, someone can use and weigh the relevant information if they can consider it and hold the pros and cons in the balance in a normal fashion. Importantly the assessment of capacity, is the assessment of the *process* of coming to a decision, and not an assessment of the decision itself. A person with decision-making capacity is allowed to make a decision which others might view as foolish or irrational and it does not matter that the refusal of some treatment might be thought to be risky or harmful (Ryan et al., 2015b).

Assessments of decision-making capacity are particularly important in psychiatry because it is not at all unusual for a patient to refuse psychiatric treatment. In some Australian jurisdictions psychiatric patients cannot be forced to have treatment if they competently refuse it and are deemed to have decision making capacity (Callaghan and Ryan, 2016). In others, such as New South Wales, doctors can override competent refusals but only in very unusual circumstances (Ryan and Callaghan, 2016).

Concluding the Interview

As mentioned above the psychiatric interview will often conclude with questions related to cognitive testing and the rest of the mental state exam, and these will be covered in Chapters 5 and 6. Before finishing though it is often wise to check that nothing important has been missed or that patients themselves don't have any lingering questions.

“Is there anything I haven't asked that you think it's important that I should know?”
“Is there anything you'd like to ask me?”

Conclusion

You can't be a great doctor without being great at taking a history. The only way to get better at interviews is to practice. The more you practice, the better you'll get and the less anxious you'll be when things don't go exactly as planned. It is important to note that rapport with the patient during the interview increases the information given. Rapport can be increased by paying attention to the patient's distress and their view of their situation. There is little to be lost, and much to be gained, by showing warmth and empathy. For those who have learnt to use it, humour can also have an important part to play. It is important not to say things you believe to be false, even if circumstances require that you do not fully reveal all that you believe to be true. One should avoid premature reassurance.

Rapport can be increased by using language suitable to the patient: that is, plain English, not jargon. A patient shouldn't be asked if his “affect has lifted now that he is ambulatory”, but if he feels “better now that he is walking”. Patients shouldn't be asked if they have “siblings” or are “experiencing hallucinations”, not using those words anyway.

History taking is not just about obtaining information. A patient interview, conducted with respect and sensitivity is frequently therapeutic, that is, it makes the patient feel better. They may also be psychoeducational, generating insight and understanding for the patient. It will also be easier to negotiate an agreed management plan from someone who has felt understood.

Further Reading

Othmer, E., and Othmer, S.C. (2001) *The Clinical Interview Using DSM-IV-TR. Volume 1: Fundamentals*. American Psychiatric Press, Washington DC

Othmer, E., and Othmer, S.C. (2001) *The Clinical Interview Using DSM-IV-TR. Volume 2: The Difficult Patient*. American Psychiatric Press, Washington DC

Chapter 5: The Mental State Examination

Vlasios Brakoulis

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia.

Introduction

The mental state examination is an essential component of a psychiatric assessment and involves a systematic way of recording observations of the patient as well as specific questions about psychiatric phenomena. It is structured and refined. It requires practice, experience and the learning of a new vocabulary of descriptive terms (psychopathology). The mental state examination is a cross-sectional assessment of an individual's mental state. It can provide an easily understood and objective evaluation of how unwell a patient is, and this provides a good reference point for comparing the mental state from one time point to another.

The format of presenting a mental state examination

- Appearance
- Behaviour
- Speech
- Mood
- Affect
- Thought form
- Thought content
 - Includes assessment of suicidal ideation
- Perception
- Cognition
- Insight
- Judgement

Appearance

The mental state examination begins with a detailed description of the individual's appearance. It can help to imagine how you might describe this person's appearance if you were introducing a character in a novel. The description usually begins with physical features from general features to more specific features. It then comments on added features such as hair, nails, bruises, scars, tattoos, piercings, make up, clothing and other apparel. All of which may point to psychopathology.

For example, *“On mental state examination, Mr Smith is a middle-aged male with a stooped posture and a pale sickly appearance. Despite his thin limbs he has considerable central adiposity. He is dishevelled with matted blonde hair, long dirty nails and he is somewhat malodourous. He wears a torn and discoloured suit coat with shorts and no shoes. His T-shirt has an image of a skull with the letters “R.I.P.”*”

Behaviour

The description of behaviour should begin with an assessment of the individual's attitude to the examiner and their current situation, followed by any psychomotor changes, abnormal movements and any overt behaviours. The description of behaviour should commence from the time of referral (e.g., repeated phone calls, waiting room behaviour). For example, *“Mr Smith was guarded and hostile. He was suspicious towards the accompanying nursing staff, displaying hypervigilance and psychomotor agitation. He had mild oral tardive dyskinesia, but no other obvious abnormal movements or behaviours.”*

Terms used to describe behaviour.

- **Mannerisms:** A repeated, habitual movement of the face, hands or body.
- **Stereotypies:** Rhythmic, repetitive and predictable movements, e.g., rocking associated with autism.
- **Catatonic posturing:** The maintenance of an inappropriate posture for a prolonged period of time, like a statue.
- **Echopraxia:** The imitation of movements performed by an examiner or another person.

Speech

Speech can be described in terms of its volume, quantity, rate and quality. Prosody or the stress, rhythm or intonation of speech can also be commented on.

Terms used to describe speech

- **Pressured speech:** Rapid speech with increased quantity of speech that is difficult to interrupt.
- **Poverty of speech:** Little substance is conveyed in the speech despite a normal quantity of speech. This may be due to vagueness or repetitive empty phrases.
- **Stuttering:** Repetition or prolongation of sounds and/or syllables to the extent that the content of speech takes longer to be conveyed.
- **Alogia:** Very few words are uttered. This usually occurs in the context of negative symptoms of schizophrenia or dementia.

Mood

Mood is defined as an emotional state that influences the patient's behaviour and outlook. Mood can be assessed by asking the patient how they feel. It can also be assessed by aspects of their behaviour and speech. For example, a patient who is psychomotor retarded and their thought content is characterised by negative ideas is likely to have depressed mood.

Terms used to describe mood

- **Anxious:** An emotional state characterised by fear, dread and concern.
- **Depressed:** An emotional state characterised by sadness and a negative outlook.
- **Euthymic:** A normal mood state which is neither depressed nor manic.
- **Elated:** An emotional state characterised by joy, energy and optimism.
- **Euphoric:** Intensely elated mood with excitement, but without associated psychomotor agitation or pressured speech.
- **Expansive:** Unrestrained emotional expression with excessive enthusiasm, friendliness and superiority.
- **Alexithymic:** An inability to describe emotions or mood.
- **La belle indifference:** Incongruent feelings of indifference towards one's disability or problem, as in conversion disorder.

Affect

Affect refers to the range of emotional expression observed by the examiner in the individual.

Terms used to describe affect

- **Restricted:** Reduced intensity and range of emotions.
- **Blunted:** Severe reduction in intensity and range of emotions.
- **Flat:** Absent or near absent emotional expression.
- **Labile:** Rapidly alternating range of emotional expression from depressed and tearful to manic and laughing.
- **Incongruent:** Emotional expression is not consistent with what is expressed verbally, e.g. laughing when describing the tragic death of a loved one.

Thought Form

Thought form refers to the way that ideas or thought content are associated. The main object of assessment of thought form is to evaluate whether thought content flows in a logical and coherent manner. Disorder of thought form usually indicates the presence of a psychotic disorder (see Chapter 15).

Terms used to describe abnormal thought form

- **Tangential:** Questions are never really answered. Patient may start to answer the question, but goes off on a tangent.
- **Derailing:** Questions begin to be answered, but train of thought abruptly changes to another topic.
- **Circumstantial:** Content is over-inclusive and the individual takes a long time to eventually answer the question.
- **Loosening of associations:** Flow of ideas are not well connected or are very loosely associated.
- **Clang associations:** Flow of ideas is not connected by meaning, but by the sound of the words, e.g. “the cold mould rolled”.
- **Flight of ideas:** Ideas tend to be connected, but rapidly change from one topic to another.
- **Thought blocking:** The abrupt termination of the flow of thought. When asked, the individual says that the thought disappeared.
- **Word salad:** An incomprehensible collection of words.
- **Echolalia:** The repetition of another person’s words or phrases.
- **Neologism:** A word created by the patient that is unlikely to be used by another person.
- **Perseveration:** The repetition of the same words or phrases, despite different questions.

Thought Content

Thought content attempts to assess “what is on the person’s mind”. Thought content might describe one’s preoccupations, obsessions, over-valued ideas or delusions. It should also include an assessment for thoughts that might be dangerous, e.g., suicidal ideation, self-harm ideation or homicidal ideation. Ideas are more transitory (e.g., “Wouldn’t it be nice to go to the beach today.”), whereas beliefs are more enduring (e.g., “It’s bad luck to go to the beach at night.”). A belief is defined as a feeling that something is true when there is neither concrete evidence

to support or refute the belief, for example, the belief that life exists on another planet in the universe. A belief becomes a delusion when it is held with absolute conviction and is not amenable to any change even when an individual is presented with evidence that might suggest that the belief is not true. Although most definitions regard delusions as false beliefs that are not in keeping with the beliefs of others from a similar socio-cultural or religious background, delusional beliefs can sometime be proven true. Thus, delusions are better defined by the way beliefs are held and by the behaviours associated with the belief of the individual. For example, people are frequently admitted to psychiatric units with persecutory delusions where they may strongly believe that their neighbour is attempting to seriously hurt them. During the course of the admission, it might be discovered that the neighbour did indeed threaten to kill the patient. The conviction with which the belief is held, the patient's inability to consider viable alternatives, their associated distress and their associated behaviours, for example, covering the windows with foil, are factors that would support them having had a delusional belief despite the belief being true.

Overvalued ideas are held with less conviction than delusional beliefs and tend to be more acceptable or understandable. Overvalued ideas tend to dominate over other ideas over long periods of time and to be associated with changes in behaviour and functioning. Some regard overvalued ideas occurring on a spectrum between delusions and obsessions.

Obsessions (see Chapter 19 Obsessive-Compulsive and Related Disorders) are defined as recurrent intrusive thoughts, images or impulses which tend to be ego-dystonic (in contrast to the wishes and desires of the individual). An example would be the recurrent thought that a mother might give her children food poisoning if she doesn't count to one hundred when washing her hands. With an obsession, the mother views this belief as completely irrational, whereas with an overvalued idea, the mother will not view this belief as irrational. If the mother holds this belief with absolute conviction, she will be regarded as having a delusional belief.

Content of thought needs to be contextualised to the patient's condition and may be mood congruent or incongruent. A depressed individual may have negative cognitions that include negative views of themselves (themes of worthlessness and guilt), negative views of the future (themes of hopelessness) and negative views of the world around them (pessimism). When negative thoughts are repeatedly experienced, they are commonly termed depressive ruminations.

Terms associated with abnormal thought content

- **Delusional mood:** The perplexing feeling that something (which is not clearly definable) has changed in one's environment.
- **Ideas of reference:** The idea that the television, radio or newspaper is making special reference, e.g., "when the news presenter smiled at the end of the programme it was as if they knew that my life wasn't funny".
- **Thought insertion*:** The belief that thoughts are being put into one's mind from an external source.
- **Thought withdrawal*:** The belief that one's thoughts are being withdrawn by an external source.
- **Thought broadcast*:** The belief that one's thoughts are being broadcast (as if through speakers) so that they are audible to others.
- **Thought echo (echo de la pensées)*:** The experience of one's thoughts being repeated within one's mind, like an echo.
- **Passivity phenomena*:** The belief that one's movements are being controlled not by them, but by an external force.
- **Persecutory delusion:** A belief that one is under threat or surveillance from others or as part of a conspiracy.
- **Folie à deux:** When a delusion is shared among two people.
- **Grandiose delusion:** A belief that one has more power, prestige, intelligence, beauty or wealth than is actually true.
- **Religious delusion:** A belief that one is God, a Saint or a religious figure, or that one has a special connection with a religious figure.
- **Hypochondriacal delusions:** A belief that one has a terrible disorder, e.g., cancer or a disorder of the bowels.

***Schneiderian first rank symptoms:** Together with delusional perception (where external events have special meaning), hallucinations of running commentary and hallucinations where the voices talk to each other, these symptoms form what are termed Schneiderian first rank symptoms. The presence of these symptoms support (but are not necessarily pathognomonic of) a diagnosis of schizophrenia.

Perception

Perceptual disturbances refer to either hallucinations or illusions. Hallucinations are false sensory perceptions that can occur in all of the five senses (auditory, visual, tactile, olfactory and gustatory). Auditory hallucinations occur more frequently than perceptual disturbances in the other senses in schizophrenia. If hallucinations in other senses are prominent, organic causes should be considered. Illusions are considered a misperception of a real stimulus, for example, mistaking the folds in one's bedsheets for a snake.

Terms associated with perceptual disturbances

- **Hypnopompic hallucination:**
Hallucination occurs on waking from sleep.*
- **Hypnagogic hallucination:**
Hallucination occurs on falling asleep.*
- **Pseudohallucination:** There is awareness that the hallucination is not real and that it arises from within oneself.
- **Derealisation:** The external world feels unreal, distant or unfamiliar.
- **Depersonalisation:** One's own self and/or body feels unreal, unfamiliar or unusual.

* Not necessarily related to psychopathology.

Cognition

The assessment of cognition includes a variety of factors, such as level of consciousness, alertness, orientation, concentration, memory, abstract thinking, construction, disinhibition and intelligence. Testing is tailored to the subject's history and mental state examination thus far. Cognitive testing may be very brief in a fit and healthy medical student where no cognitive impairment is suspected, but it can be very comprehensive in a forgetful 85-year-old hospitalised nursing home resident. Comprehensive testing (see Chapter 6) is often aided by assessment tools such as the Folstein Mini Mental State Examination (MMSE) (Folstein et al., 1975b) or the Montreal Cognitive Assessment (MoCA) (Nasreddine et al., 2005).

At a basic level, delirium should always be excluded by observing for fluctuating levels of consciousness (i.e., may be coherent at one time and disorientated and perplexed at others) and by assessing orientation (e.g., "Can you tell me where you are?"). A test of concentration, for example serial sevens (where one is asked to subtract 7 from 100 and to continue to subtract 7 from the answer) or asking one to spell the word "WORLD" backwards is also important as depression and states of distress can significantly impair concentration. If the subject is found to be forgetful when the history is taken, memory impairment and dementia can be assessed with a brief test of memory, for example asking the subject to recall three objects after five minutes. A good indication of an individual's level of intelligence can be obtained by considering the extent of their vocabulary when they are narrating their history to you.

Insight

Insight refers to the individual's level of awareness that they are unwell and/or that they require treatment. It is not uncommon for patients with schizophrenia to be completely insightless. They may have no insight into their illness, for example, "there is nothing wrong with me.", their belief, e.g. "I am not delusional" and to their need for treatment, for example "Let me out of here doctor, I am not taking those psych drugs because I am not crazy...". Sometimes, patients have partial insight, in that for instance, they may be aware of the need to stay in hospital but disagree with the doctor that they need to be given medication."

Judgement

Judgement refers to the ability of an individual to appropriately consider and weigh up pros and cons and make good decisions. Judgement is evaluated during history taking by considering whether various actions described in the course of taking the history displayed good judgement or not. If a patient with limited finances tells you that they were looking to buy a new expensive luxury sports car because the price of public transport had increased, then the patient is likely to be displaying poor judgement. Imaginary scenarios can also be given to individuals to assess judgement. Common examples include: "What would you do if you received an email that was addressed to a very good friend of yours?", "What would you do if you were in a cinema and you could smell smoke?".

Further Reading

Sims, A. (1988). *Symptoms in the mind: An introduction to descriptive psychopathology*.

Bailliere Tindall Publishers.

Trzepacz, P. T., & Baker, R. W. (1993). *The psychiatric mental status examination*.

Oxford University Press.

Chapter 6: Bedside Cognitive Testing

Haley M. LaMonica

Brain and Mind Centre, The University of Sydney, Australia.

Loren Mowszowski

Brain and Mind Centre, The University of Sydney, Australia.

Stephen Touyz

Inside Out Institute, The University of Sydney, Australia, and Sydney Local Health District, Australia.

and

Sharon L. Naismith

Charles Perkins Centre, The University of Sydney, Australia, and Brain and Mind Centre, The University of Sydney, Australia.

Introduction

Cognitive assessment is an integral part of a mental status examination (see Chapter 5) as it can assist with differential diagnosis, treatment planning, and monitoring of response to intervention. Cognitive assessment typically incorporates measures across the various cognitive domains, including attention and working memory, processing speed, executive functions, memory (across all stages, i.e., encoding, storage, and retrieval), language, visuospatial skills, and praxis (i.e., purposeful movements). The following chapter provides an overview of the available screening tools and tests that can easily or practically be used at bedside to identify the presence, severity, and pattern of cognitive dysfunction. While many of these assessment tools are applicable across a range of diagnoses, this chapter is aimed primarily at screening for (but not diagnosis of) neurodegenerative disorders.

Screening Tests

Several screening tools are available to grossly assess cognition at bedside. Whilst these tools tend to have limited utility with respect to identifying more subtle cognitive changes or to evaluating specific cognitive domains, they can certainly be useful in identifying the overall presence of cognitive impairment, and they are widely used within the broader medical profession and therefore worthwhile to recognise and understand.

One important issue to note is that when it comes to assessing memory, most screening tests tend only to evaluate the patient's ability to learn or encode new material over a brief period (e.g., within five minutes), and therefore do not provide an opportunity to reliably evaluate information storage or retrieval, which requires a more substantial delay to allow time for the information to be consolidated and stored within the brain. More comprehensive neuropsychological tests of memory typically include a delayed recall component of at least 20-30 minutes after initial learning. This is an important limitation to bear in mind when assessing patients for evidence of neurodegenerative disorders such as Alzheimer's disease, which in its earlier stages, more prominently affects memory storage and retrieval, and therefore might be 'missed' on global screening.

Two of the most widely recognised screening tests are the Mini-Mental State Examination (MMSE) (Folstein et al., 1975a) and the Montreal Cognitive Assessment (MoCA) (Nasreddine et al., 2005), although others are also described below.

Mini-Mental State Examination

The MMSE is a brief, objective, 11-item measure of cognitive functioning, which takes approximately 5-10 minutes to complete, and which yields a maximum score of 30. The MMSE is typically used to estimate the degree of cognitive decline experienced by a patient, track progression over time through serial assessments, and monitor the general effects of interventions on cognition. The cognitive functions assessed include orientation, registration (i.e. encoding or new learning), attention, working memory, recall, language functions (i.e., naming, phrase repetition, reading, and writing), and constructional praxis (i.e. copying a simple line-drawing). The MMSE is available in several different languages (e.g. Spanish, German, Dutch and Hindi) and has alternate forms for serial assessment.

Despite the broad use of the MMSE in clinical and research settings, this global screening tool has several limitations. For example, the MMSE does not

effectively assess frontal-executive functions, such as mental flexibility and verbal abstraction; therefore, it is insensitive to the frontal deficits characteristic of acquired brain injury or diseases with prominent executive dysfunction (e.g., fronto-temporal dementia, Lewy body dementia, and schizophrenia). Similarly, it does not incorporate a measure of processing speed which is important in the assessment of conditions such as depression and multiple sclerosis. Additionally, the two objects used to assess naming skills are high frequency words, hence even participants with a naming deficit may perform normally on this task. As mentioned above, the test does not evaluate delayed recall (only new learning) and therefore may not elicit evidence of memory impairment until the later stages of some dementias, such as Alzheimer's disease. Finally, the test is copyrighted and, therefore, needs to be purchased.

With regard to use with specific patient populations, the MMSE is one of the most commonly used tests for the screening of dementia; however, it is important to interpret the scores with caution as some individuals experiencing a neurodegenerative process may still score normally on this test. The cut-off score used to define normal cognition is typically set at 24, which has been found to have a sensitivity of 85% and a specificity of 90% for dementia (Creavin et al., 2016). In other words, it is expected that 15% of patients will be incorrectly classified as not having dementia on the basis of a reasonable MMSE score. Furthermore, in relation to predicting conversion from Mild Cognitive Impairment (MCI) to all-cause dementia, a Cochrane review of 11 studies, including a total of 1569 participants, reported variable sensitivity of MMSE scores ranging from 23% to 76%, and specificity ranging from 40% to 94% (Arevalo-Rodriguez et al., 2015). It has also been found that a higher cut-off score of 27 may be more appropriate for highly educated individuals (O'Bryant et al., 2008).

In light of these concerns, it is always essential that MMSE scores are interpreted in the context of a patient's sociodemographic history and day-to-day functioning. If discrepancies arise between the MMSE score and other aspects of the patient's presentation, clinicians are encouraged to refer patients for more comprehensive neuropsychological assessment to assist with differential diagnosis and ensure appropriate management.

Montreal Cognitive Assessment

The MoCA is another brief screening tool that was specifically developed to accurately identify individuals with Mild Cognitive Impairment, who may otherwise perform within the normal range on the MMSE (Nasreddine et al., 2005). The MoCA takes approximately 10 minutes to administer and assesses a range of cognitive domains, including executive functions, visuospatial skills, attention,

working memory, language, (i.e., naming, repetition and phonemic fluency), recall and orientation. The total possible score is 30 points and a cut-off score of 26 is typically used to define normal cognition. The MoCA is available in the public domain (www.mocatest.org) and has multiple versions for serial testing. Additionally, the MoCA has been translated into and validated in a variety of languages (e.g., Spanish, Dutch, and Mandarin). With that being said, it is still important to recognise that the MoCA is only a brief screening tool and does not evaluate all cognitive domains. Additionally, similar to the previous criticism of the MMSE, some areas, such as naming and processing speed, are inadequately assessed, thus potentially missing individuals experiencing cognitive decline.

Several direct comparisons have been conducted between the MMSE and the MoCA. In one such study, it was found that the MoCA score accurately detected 90% of participants with Mild Cognitive Impairment when using a cut-off score of 26, whereas the MMSE score had a sensitivity of only 18%. Furthermore, the MMSE had a sensitivity of 78% and the MoCA had a sensitivity of 100% to detect mild Alzheimer's disease (Nasreddine et al., 2005). It has also been shown that for any threshold set on the MMSE, there is a cut-off score on the MoCA that yielded higher sensitivity and specificity (Damian et al., 2011). While the MoCA is therefore a superior screening tool to the MMSE for the detection of Mild Cognitive Impairment and mild dementia, its ability to accurately identify domain-specific cognitive impairment is inadequate for diagnostic purposes (Coen et al., 2016; Moafmashhadi and Koski, 2013). Therefore, a referral for a neuropsychological evaluation is recommended when a detailed cognitive profile is needed for diagnostic and/or patient planning or rehabilitation purposes.

Addenbrooke's Cognitive Assessment, Third Edition

The Addenbrooke's Cognitive Examination, Third Edition (ACE-III) is a 100-point screening test that was designed for the detection and classification of dementia (Hsieh et al., 2013). The ACE-III takes approximately 15 minutes to complete and assesses five cognitive domains, including attention, memory, verbal fluency, language (comprehension, writing, repetition, naming and reading) and visuospatial skills. A cut-off score of 88 has been shown to have high sensitivity (1.0) and specificity (0.96) for dementia syndromes (Hsieh et al., 2013). Furthermore, the ACE-III has been shown to reliably discriminate between healthy controls and patients with Mild Cognitive Impairment (Matias-Guiu et al., 2017). The ACE-III is available for free in several languages (e.g., Arabic, Japanese, and Hindi) as well as in alternate forms.

Importantly, certain cognitive domains on the ACE-III have been shown to be highly correlated with standardised neuropsychological tests (Matias-Guiu et al.,

2017). For example, the total score from the language-based tasks on the ACE-III correlate significantly with the Sydney Language Battery total score ($r = 0.86$, $p < 0.001$) (Hsieh et al., 2013) and the Boston Naming Test ($r = 0.74$, $p < 0.001$) (Matias-Guiu et al., 2017). Please refer to the original papers for more detailed results.

Rowland Universal Dementia Assessment Scale

The Rowland Universal Dementia Assessment Scale (RUDAS) was specifically developed as a 30-point screening tool for individuals from culturally and linguistically diverse backgrounds (Storey et al., 2004). Gender, years of education and preferred language are not independent predictors of scores on the RUDAS, reflecting an advantage over the MMSE (Rowland et al., 2006). The 6-item test takes 10-15 minutes to complete and assesses multiple cognitive domains, including memory, visuospatial orientation, praxis, visuoconstruction, judgment and language (semantic fluency). Importantly, the RUDAS can be translated directly into several other languages without impacting on the structure or format of the test. Additionally, patients are encouraged to respond in the language with which they are most proficient and comfortable. A cut-off score of 23 has been shown to have a sensitivity of 89% and a specificity of 98% for the detection of dementia (Storey et al., 2004). Of note, the results of a recent study found that the ACE-III had the best diagnostic accuracy in relation to distinguishing between healthy controls and patients with Alzheimer's disease relative to the MMSE, MoCA, and RUDAS (Matias-Guiu et al., 2017).

Diagnosis-Specific Screening Tests

It is worthwhile to note that more specialised screening measures have been designed for administration within specific acute settings, including the National Institute of Health Stroke Scale, Brief International Cognitive Assessment for Multiple Sclerosis, International HIV Dementia Scale, and Confusion Assessment Method for the ICU; however, a detailed description of these tools is beyond the scope of this chapter.

Domain-Specific Cognitive Tests

It is often important to supplement brief screening tools with additional cognitive tests to examine specific domains in greater detail (Table 6.2). It is important to recognise that some of the tests described below have been incorporated into the MMSE, MoCA, ACE-III and RUDAS.

Attention and Working Memory

There are several ways in which to assess attention and working memory at bedside. Specifically, asking patients to say the months of the year in order or repeat a string of numbers in forward sequence (digit span) are simple measures of basic attention. A forward digit span of six or more numbers is generally considered a normal performance, with some variability associated with age and level of education. Working memory skills, or the ability to mentally manipulate information, can be assessed by asking a patient to repeat digit strings in reverse sequence, with a span of four or more digits reflecting intact performance. Importantly, impairments in attention and working memory are relatively non-specific, frequently occurring in patients presenting with mood and behavioural disorders, sleep abnormalities, neurodegenerative disorders or more focal brain lesions, such as those sustained in a traumatic brain injury.

Hemi-neglect refers to an attentional abnormality in which the patient does not attend to visual or sensory information presented in one-half of their field of vision or affecting one-half of their body. The line bisection test is commonly used to evaluate spatial neglect and requires patients to place a mark at the midpoint of a series of lines presented on a page. Neglect is typically associated with dysfunction in the right (non-dominant) parietal lobe, resulting in neglect of the left side of space.

Orientation

Questions about orientation, including the patient's name, current location and the full date (i.e. day of the week, date, month and year), provide information about recent and longer-term memory.

Memory

Memory can be assessed at bedside by assessing orientation but also awareness of current affairs and personal and/or family information. Ask the patient how long they have been in hospital, why are they in hospital, what treatment(s) have they received and what has been happening in the news lately. It is often helpful to also ask staff how well they are laying down new memories and ask family members if they are recalling information, they have discussed from one visit to the next. Some test batteries such as the ACE-III ask the patient to remember a name and address and then recall it again at the end of the test. This can be a useful bedside assessment. It is important to note that neuropsychological testing may be required to assess in more detail the specific conditions upon which a person is forgetting information, or the particular pattern of memory impairment, as this may be relevant for differential diagnosis as well as management. Neuropsychologists use a suite of different memory tests to assess both the ability to learn as well as to

remember new information such as prose passages, lists of related or unrelated words, and the extent to which a person benefits from repetition, and multiple choice options. This can help to pinpoint where a memory difficulty lies such as in the encoding, retention or retrieval of new information.

Executive Functions

‘Executive functions’ is an umbrella term that refers to a range of higher-order thinking skills localised to the frontal lobes (see Table 6.1), such as mental flexibility, cognitive inhibition (i.e. withholding an automatic or inappropriate response), abstract reasoning, problem-solving, planning and organisation. There are several simple measures that can be administered at bedside to grossly assess aspects of executive functions. For example, a ‘go-no-go’ paradigm can be used to evaluate inhibition. A patient is instructed to hold up two fingers when the clinician holds up one finger and vice versa, with elevated error rates reflecting dysfunction. An abbreviated version of the Trail Making Test is included in the MoCA as a measure of mental flexibility. Patients are instructed to alternate between numbers and letters to put them in order (1 – A – 2 – B...). Verbal fluency, an aspect of executive functioning, is also commonly impaired in patients with frontal lobe dysfunction. In some screening batteries, such as the ACE-III, verbal fluency is included. Patients are asked to generate as many words as possible in one minute that begin with a specified letter or belong to a designated category (such as animals or items found in a kitchen). Average scores vary in relation to age and education.

Table 6.1. Relationship between executive dysfunction and neuroanatomy.

Impaired Executive Function	Brain Region(s)
Inhibition	Orbitofrontal cortex Anterior cingulate
Mental flexibility	Dorsolateral prefrontal cortex
Verbal fluency	Dorsolateral prefrontal cortex (dominant hemisphere) <i>Note: semantic fluency is also heavily dependent on the medial temporal lobes</i>
Perseveration	Dorsolateral prefrontal cortex
Abstract reasoning and logic	Dorsolateral prefrontal cortex
Motor programming	Prefrontal cortex Dorsolateral prefrontal cortex
Planning and organization	Dorsolateral prefrontal cortex Orbitofrontal cortex

Individuals with frontal lobe dysfunction are often perseverative, meaning that they struggle to switch between actions. Specifically, if asked to copy an alternating sequence of squares and triangles that are open at the bottom (Luria's 'rampart' design, shown in Figure 6.1), these patients may only draw one of the shapes rather than switching between the two. Additionally, they may be unable to switch from one motor movement to the next when performing the Luria Manual Sequencing Task, a 3-step sequencing test that requires the patient to alternate repeatedly from placing a fist on the table, followed by their palm and finally the side of their hand. Patients with severe frontal dysfunction may be unable to learn the 3-step sequence at all, let alone continue to repeat the actions independently after initially copying the clinician's actions.



Figure 6.1. Luria's 'rampart' design task.

Tests of logic and abstract reasoning are also common tools used to evaluate frontal lobe dysfunction. For instance, verbal abstraction is frequently assessed by asking patients to identify or describe the way in which two items or concepts are similar, such as "How are a horse and a tiger alike?". Patients with frontal lobe disorders tend to respond with concrete responses (i.e., they have four legs), rather than being able to identify the abstract link between the two objects (i.e., animals). Alternatively, patients may be unable to identify any association between the items at all, often describing how they are different (i.e., one is brown and one has stripes). Asking a patient to interpret proverbs such as "One in the hand is worth two in the bush" or "The squeaky wheel gets the grease" is another means by which to examine reasoning skills. Importantly, educational and cultural background must always be considered in relation to these tests. Another aspect of reasoning can be evaluated at bedside with a simple set of hypothetical problems. By presenting the patient with realistic scenarios (e.g. "What would you do if you accidentally took two tablets of your medication instead of one", or "What would you do if you were heating up some soup and a small fire broke out on the stovetop"), the clinician can also assess the patient's functional or everyday problem-solving skills, which has particular relevance for decisions regarding safety upon discharge and rehabilitation.

The Frontal Assessment Battery (FAB) (Dubois et al., 2000) is a bedside screening battery specifically designed to improve the detection of executive dysfunction characteristic of some neurodegenerative disorders including fronto-temporal dementia, vascular dementia, and progressive supranuclear palsy as well as acquired brain injuries. This brief screening battery takes approximately 10 minutes to complete and includes measures of verbal abstraction, phonemic fluency, motor programming, susceptibility to interference, inhibition, and prehension behaviour.

Language Functions

Both receptive and expressive aspects of language can be easily assessed at bedside, often times through simple conversation with the patient. For example, observe the rate, fluency and prosody (patterns of intonation) of spontaneous speech. Does the patient consistently stammer or stumble over words, become easily frustrated while communicating, or rely on non-verbal gestures to get their point across? Is the content of speech marked by grammatical errors, word finding problems, paraphasic errors (semantic paraphasia, e.g. ‘dog’ for cat’, or phonemic paraphasia, e.g. ‘cap’ for ‘cat) or neologisms (i.e., made up words)? In order to assess comprehension, it is important to observe whether or not a patient understands simple questions. Additionally, ask the patient to follow single (‘Pick up the pen.’) and multi-step commands (‘Take this paper in your right hand, fold it half and put it on the floor.’). To assess naming, ask the patient to identify easy as well as more difficult objects in the room, such as a pen, watch and stethoscope. Repetition can be evaluated by asking the patient to repeat single words (‘hippopotamus’) and sentences (‘All that glitters is not gold.’). To evaluate reading skills, ask the patient to read single words and a brief passage, such a paragraph from the newspaper, aloud. Subsequently, ask questions about what the patient has read as an additional test of comprehension. Finally, as a measure of writing skills, ask the patient to write their name and a simple sentence. Language impairments are often associated with dysfunction in the dominant (typically left hemisphere) frontal lobe, including Broca’s area, and the parietal and temporal lobes, including Wernicke’s area; however, involvement of subcortical structures as well as the non-dominant hemisphere cannot be ruled-out.

Language problems are common in various neurodegenerative diseases and are often the presenting complaint from patients (‘I can’t remember familiar words in conversation.’). Dysnomia and impairments in semantic fluency are hallmark signs of Alzheimer’s disease, often presenting early in the disease process alongside significant forgetfulness. Primary progressive aphasia (PPA) (including the non-fluent agrammatic variant, logopenic variant, and semantic variant) are characterised by prominent language difficulties. For example, effortful and agrammatic speech is the core diagnostic feature of non-fluent PPA whereas patients with semantic PPA

present with impairments in confrontation naming and single-word comprehension. The logopenic variant is characterised by primary word finding problems and impaired repetition of sentences and phrases. While language testing at bedside may suggest a progressive language disorder, it is not sufficient for diagnostic purposes. In such cases, a referral for a comprehensive neuropsychological assessment in conjunction with neurological and neuroradiological investigations is essential to ensure accurate diagnosis and classification, with a view to appropriate prognosis and management.

Visuospatial and Constructional Skills

Most screening tools, including the MMSE and the ACE-III, include basic measures of spatial analysis and visual processing. Patients are asked to copy simple geometric figures (e.g., intersecting pentagons or a cube) to assess their ability to accurately perceive and construct the designs. The Clock Drawing Test is another measure of visuoconstruction frequently used to screen for cognitive decline and dementia. The patient is asked to draw the face of a clock including all of the numbers and make the hands of the clock show 10 past 11. Accuracy of the drawing is assessed based on the contour of the clock face, the spatial orientation and inclusion of all the numbers, and the placement of the hands. Of note, the Clock Drawing Test is also a useful measure of neglect, as these patients frequently arrange all of the numbers in one half of the clock face; as well as concept formation, as patients with executive dysfunction can often accurately draw the clock face and numbers but have considerable trouble depicting the specified time. Difficulties on these visuospatial tasks are typically associated with dysfunction in the frontal and parietal systems. Impairments in visuospatial skills are characteristic of a range of neurodegenerative disorders, including Alzheimer's disease, Parkinson's disease dementia and Lewy body dementia. Table 6.2 summarises cognitive tests by domain.

Table 6.2. Summary of Cognitive Tests by Domain

Domain	Cognitive Test
Attention and Working Memory	Digit span Line bisection (neglect)
Learning and Memory	Orientation, current events, and details of hospitalisation and treatment Name and address
Executive Functioning	Go-No-Go paradigm Trail Making Test Verbal fluency Luria's 'rampart' design Luria Manual Sequencing Task Abstract reasoning Proverbs Functional problem-solving skills
Language	Expressive language (volume, rate, prosody) Content of speech (grammar, logic, word finding problems, paraphasic errors, neologisms) Comprehension Command following Repetition of words and phrases Reading Writing Naming Verbal fluency
Visuospatial	Copy simple geometric designs Clock Drawing Test
Praxis	Demonstrate complex movements

Apraxia

Apraxia refers to the inability to carry out purposeful motor commands in the absence of primary motor deficits. Ask the patient to demonstrate complex movements, such as “Pretend to cut a loaf of bread” or “Pretend to stir a cup of coffee.” Patients with apraxia will perform awkward movements that do not align with the command. Often times, patients substitute their own body part for an object specified in the instructions, such as using their hand as a knife when pretending to slice bread; this is referred to as ideomotor apraxia. The clinician may also ask nursing staff or the patient’s family/friends about difficulties with everyday activities such as dressing (e.g., pulling one’s arms through sleeves or doing up buttons) or using cutlery whilst eating.

General Tips for Bedside Cognitive Testing

In addition to selecting a suite of measures to evaluate cognition at bedside, the clinician should also be aware of the following factors which may impact on the patient's performance and may therefore over- or under-estimate their cognitive performance:

- 1. Is this an appropriate time to complete cognitive testing?** It is important to consider the patient's state of alertness as well as environmental factors. For example, if they are in or recovering from a delirium, under sedating medication, or in an acute psychotic episode, cognitive testing is not appropriate. It is also preferable to complete testing in a quiet, calm environment with minimal distractions from visitors, other patients, or other medical staff.
- 2. Has the patient completed these measures before?** Often, patients may have completed the same test (particularly screening measures such as the MMSE) on several occasions even within the same admission. This may result in familiarity with the test items and reasonable performance due to 'practice effects'.
- 3. Does the patient have any sensory impairments which may impact on test performance?** Patients with hearing impairment may perform poorly on memory tests simply because they have not heard the information well enough to encode it. Check whether your patient usually wears a hearing aid, glasses or other sensory aids and if so, insist that they use them for the duration of the cognitive assessment to avoid any potential disadvantage.
- 4. Is the patient proficient in English?** It is important to conduct cognitive assessments in a patient's native language which may require the use of an interpreter. Ideally, it is important to provide the interpreter with an overview of the standardised instructions and normal or expected responses to cognitive tests; however, this is not always practical given time and cost restrictions. The examiner must, therefore, be alert to unexpected irregularities in a patient's test performance that may be better accounted for by errors in translation as opposed to a cognitive deficit. The use of casual interpreters, such as friends and family members, is strongly discouraged as they may not possess the language skills necessary to translate at a level required for accurate assessment and may seek to protect the patient from performing poorly on the tests, thus limiting the value of the results as a means to inform patient care. Results from assessments completed with the aid of an interpreter should be evaluated with caution.

Conclusion

Cognitive testing at bedside can help to document the presence and severity of objective cognitive impairment. Evaluating the pattern of dysfunction across cognitive domains often assists with differential diagnosis as well as treatment and discharge planning. It is important that cognitive test results are not interpreted in isolation, but rather that they are examined in the context of a comprehensive history of the patient's presenting illness and current symptoms, medical history, family history, and sociodemographic factors, including educational background and occupational status. Factors such as delirium should always be considered in the hospital patient and it is crucial that either interview-based or questionnaire-based screening tests for depressive symptoms are also administered, since depression commonly impacts upon performance. Whilst they offer practical utility, there are several limitations associated with screening tools used at bedside, including floor and ceiling effects, variability in diagnostic accuracy, and insensitivity to cultural and linguistic factors. As noted, those with high levels of education often perform well above the traditionally used cut-off scores. Therefore, referral for a comprehensive neuropsychological evaluation is often valuable for the purposes of differential diagnosis and treatment planning.

Further Reading

- Norris, L., & Cobbs, E. L. (2018). Bedside cognitive assessment. *The Medical Journal of Australia*, 208(5), 200-201.
- Sagiadinou, M., Plerou, A. (2020). Brief Cognitive Tests in the Case of Dementia and Alzheimer's Disease Early Diagnosis. In: Vlamos, P. (eds) *GeNeDis* 2018. *Advances in Experimental Medicine and Biology*, vol 1195. Springer, Cham.

Chapter 7: Formulation

Gin S. Malhi

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia, Visiting Professor, Department of Psychiatry, The University of Oxford, UK, and Oxford Uehiro Centre for Practical Ethics, Faculty of Philosophy, The University of Oxford, Oxford, UK.

Philip Boyce

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia, Westmead Institute of Medical Research, Australia, and Faculty of Health, Charles Darwin University, Australia.

and

Erica Bell

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

Making a correct diagnosis is a necessary first step when planning clinical management, however, it is insufficient for the development of a comprehensive management plan. This is because a diagnosis does not inform us about *why* a person develops a disorder, *what* the underlying vulnerabilities are, or indeed *which* factors are maintaining the symptoms. In management planning we need to have a more detailed understanding of why the individual before us is presenting with a particular set of symptoms or problems, and formulate what it is they are suffering from. This is where a formulation is helpful as it facilitates a comprehensive understanding of the patient, and importantly, where to target specific interventions to help the patient most effectively.

The origins of psychiatric disorders are complex and multifaceted. Their development is rarely attributable to a single cause and often involves interactions between an individual's biology (genetic predisposition), psychological makeup (including personality, coping mechanisms and thinking styles) and their social environment as well as their support networks. Therefore, to understand a patient comprehensively requires making use of the *biopsychosocial model*. This model was introduced by George Engel (a psychiatrist and a physician), as a result of his

dissatisfaction with the basic biomedical model of disease, which he regarded as insufficient to explain, or understand, psychosomatic disorders (Engel, 1977).

Formulation Strategies

The essence of being able to formulate a patient lies in careful history taking (formally known as anamnesis). The psychiatric history needs to be more than just eliciting the current symptoms the patient is manifesting - a *cross-sectional* approach that is necessary to make a DSM diagnosis but one that tells us little else. Instead, we need to adopt a *longitudinal* approach to explore what predisposes the person to develop a psychiatric disorder and what triggers there are for the onset of the disorder. To do this a detailed family and developmental history is essential as it provides the necessary context to understand the individual's present predicament and future life trajectory. Here we need to cast the net wide and search for the full range of biological, psychological, social and cultural factors that could play a part in the patient's illness.

The Five P's

The formulation makes use of the 5 'Ps' approach, trying to tease out **p**redisposing, **p**recipitating, **p**erpetuating and **p**rotective factors that contribute to the presenting **p**roblem (see Figure 7.1). There is another P that describes the pattern of illness, and this is particularly important for those with recurrent illnesses (see chapters on Depressive Disorders and Bipolar Disorder). Once all the necessary information has been gathered it is then possible to construct a formulation grid (see Table 7.1).

Table 7.1. The formulation grid.
A typical example of factors that may impact the manifestation of a psychiatric disorder are shown.

	Predisposing	Precipitating	Perpetuating	Protective
Biological	Mother has Psychiatric disorder			
Psychological		Recent loss of father		Psychologically minded and resilient
Social			Work related stress	Supportive partner

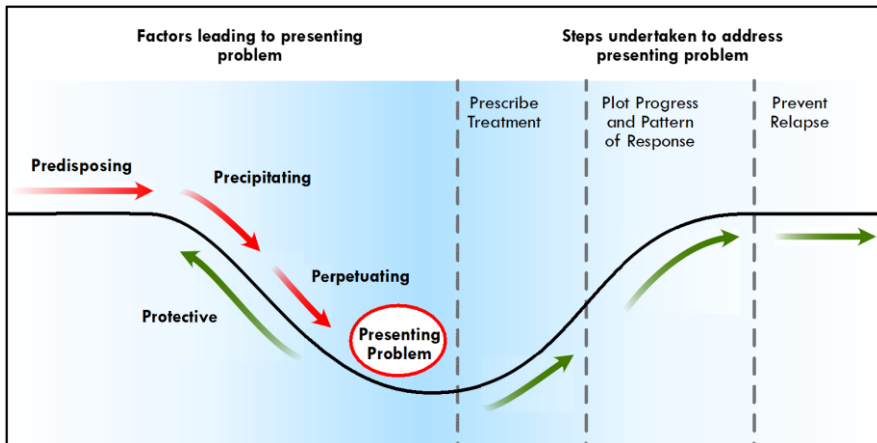


Figure 7.1. 5P Model. In this figure an expanded version of the conventional 5P Model approach to formulation is shown, which includes the three phases of management, adapted from Malhi et al. (2015).

Predisposing Factors

Individuals are predisposed to developing a psychiatric disorder if they have been exposed to, or inherited, a risk factor distal to the onset of the disorder. Identifying predisposing factors requires taking a careful developmental history that assumes a longitudinal perspective (see Figure 7.2 below). The aim is to find elements in the patient's life story that may have contributed to their vulnerability to develop a psychiatric disorder. This requires using open ended questions enquiring about the patient's cultural background, socioeconomic status, family environment, schooling, social networks, intimate relationships, personality functioning and coping mechanisms as well as pursuing biological factors such as genetic endowment, physical illness and exposure to potential neurotoxins such as alcohol, tobacco and illicit substances.

While listening to their history, it is important to start considering hypotheses about possible predisposing factors that can then be tested out by formulating more targeted questions to the patient. These risk factors will have an evidence base coming from longitudinal cohort studies, family studies, twin studies and epidemiological studies that identify risk factors for specific illnesses.

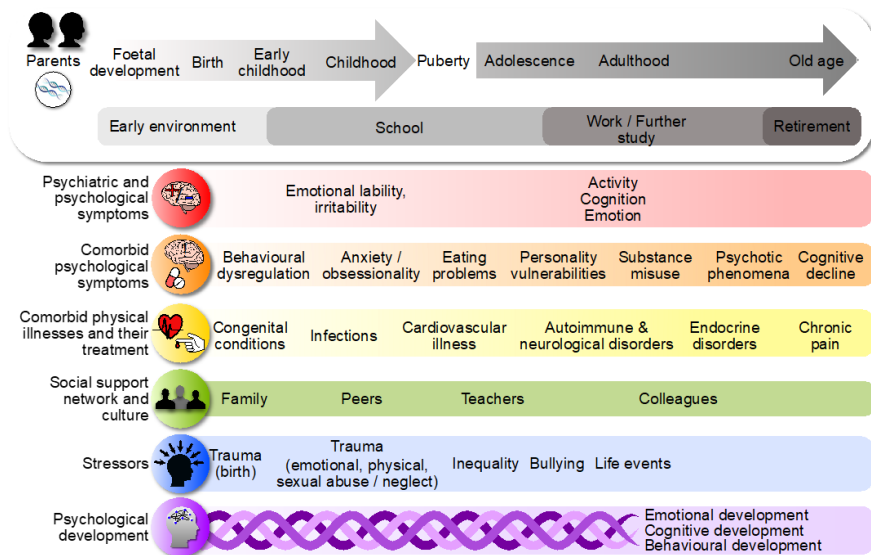


Figure 7.2. An example of predisposing factors for a patient with a mood disorder, from Malhi et al. (2020b). A framework for assessing the clinical factors that contribute to depression. The growth and development of an individual is depicted at the top of the figure and this ‘life-line’ provides a timeframe for changes across a number of domains. The domains are shown on the left and within these key aspects that should be considered and assessed are depicted according to the approximate stage of life. This then provides a framework for eliciting the clinical factors that can putatively contribute to the development and maintenance of depression.

When adopting a biopsychosocial (and cultural) approach it is important to keep in mind that there is an interplay between biological factors, the social environment, and internal psychological factors as these are not independent factors and they cannot be fully compartmentalised. For example, socioeconomic disadvantage (a social factor) can be a risk factor on its own, but we know that it also contributes to chronic illness, which may then further contribute to the risk of developing a mental illness. Even a seemingly distinct psychological risk factor such as the personality trait of shyness is likely to be associated with the individual having a limited social network, which is, in and of itself, a risk factor for mental illness.

There are several predisposing risk factors for the development of a psychiatric disorder that should be considered when taking a history. Table 7.2 lists the major domains of predisposing factors, with some select examples in each.

Table 7.2. Examples of Predisposing Factors.

Biological	Genetic endowment (<i>e.g. family history of schizophrenia</i>)
	Neurodevelopment
	Intrauterine (<i>e.g. maternal influenza</i>)
	Brain trauma/injury (<i>e.g. hypoxia</i>)
	Physical health (<i>e.g. chronic disease</i>)
	Medication (<i>e.g. steroids</i>)
	Illicit drugs and alcohol (<i>e.g. methamphetamine</i>)
Psychological	Temperament (<i>e.g. impulsivity</i>)
	Attachment style (<i>e.g. anxious attachment</i>)
	Loss (<i>e.g. bereavement</i>)
	Childhood adversity (<i>e.g. childhood abuse</i>)
	Intelligence (<i>e.g. intellectual disability</i>)
	Health literacy (<i>e.g. treatment non-adherence</i>)
	Personality style (<i>e.g. neuroticism</i>)
	Self-identity (<i>e.g. risk taker</i>)
Social	Self-esteem
	Habits (<i>e.g. gambling</i>)
	Socio-economic (<i>e.g. poverty</i>)
	Housing (<i>e.g. homelessness</i>)
	Socio-cultural (<i>e.g. marginalisation</i>)
	Migration
	Religious affiliation (<i>e.g. persecution</i>)
	Family environment (<i>e.g. dysfunction and conflict</i>)
	Interpersonal relationships (<i>e.g. domestic violence</i>)
Lifestyle	Social network (<i>e.g. limited social support</i>)
	Activity level (<i>e.g. sedentary lifestyle</i>)
	Diet (<i>e.g. unhealthy diet</i>)
	Sleeping pattern (<i>e.g. poor sleep hygiene, insomnia</i>)
	Social media (<i>e.g. excessive use</i>)
	Substance use
	Smoking
	Alcohol use

Precipitating Factors

In addition to identifying predisposing factors, it is important to work out what has led to the illness now. In other words, what has precipitated the illness? Can you identify the potential precipitating factors that have led to the onset of the illness. This requires taking a detailed account of any life events that may have

happened prior to the onset of symptoms. It is also important to find out if there have been any changes in the person’s life (such as physical illness) or a change in their medication, as is often the case in severe mental illness. Select examples of precipitating factors are shown in Table 7.3.

Table 7.3. Examples of Precipitating Factors.

Biological	• Medical illness ○ Infections ○ Inflammation ○ Childbirth • Medications • Non-adherence to medication
Psychological	• Interpersonal conflict • Severe trauma • Family conflict • Experience of loss or significant change in ○ Relationship ○ Occupation ○ Retirement
Social	• Relationship problems • Community discord/unrest
Socioeconomic	• Unemployment • Homelessness
Lifestyle	• Substance misuse • Alcohol misuse • Sleep dysfunction

Perpetuating Factors

Once a disorder emerges and becomes established, there are a range of factors that may maintain the illness. Such perpetuating factors need to be addressed to achieve full remission, recovery and ultimately return to full functioning. Select examples of perpetuating factors are shown in Table 7.4.

Table 7.4. Examples of Perpetuating Factors.

Biological	• Physical illness/comorbidity • Medication ○ Non-adherence ○ Insufficient dose ○ Pharmacokinetic problems ○ Incorrect medication ○ Non-psychotropic drugs maintaining illness
Psychological	• Illness behaviour ○ Malingering • Denial • Poor mental health literacy • Lack of insight
Social	• Poor social support • Family disharmony • Relationship problems • Domestic Violence • Lack of access to care e.g. because of rurality • Inability to afford care • Unemployment • Poverty • Homelessness • Marginalisation
Lifestyle	• Substance misuse especially alcohol • Smoking • Poor sleep hygiene • Poor diet • Lack of exercise

Protective Factors

As well as determining risk factors it is important to identify the elements in a person’s life that are protective and shield the patient against the development or recurrence of a mental illness. Once again, the protective factors that need to be considered, can be grouped using the biopsychological and lifestyle framework (see Table 7.5). It is essential to emphasise these in management planning, as the protective factors can be developed and/or enhanced to engender greater resilience. These protective factors contribute to the broader construct of

resilience, which has in-built (intrinsic) components in addition to those that are acquired through experiences. For further information on resilience, see Malhi et al. (2019).

Table 7.5. Examples of Protective Factors.

Biological	• Healthy • Intact cognition • Accepting of medication
Psychological	• Mental health literacy • Effective coping strategies • Able to establish working therapeutic relationship
Social	• Strong and diverse social support network • Supportive intimate relationship • Supportive family • Supportive work environment • Stable housing • Financial stability • Access to mental health services
Lifestyle	• Exercising • Healthy diet • Good sleep hygiene and quality sleep • Not drinking alcohol to excess • Not smoking or taking illicit substances

Formulation

After teasing out the predisposing, precipitating, perpetuating factors that are contributing to the patient’s current illness, along with the protective factors that will assist the patient’s recovery, the various pieces of relevant information need to be integrated to provide a cohesive narrative that explains the person’s current circumstances and how their current situation has come about. If done properly, this should provide a useful framework upon which a clear management plan for the patient can be constructed. We have provided a relatively straightforward example (See below).

Formulation Example

John is a 24-year-old single unemployed male who has presented with a relapse of schizophrenia (hallucinations, delusions and thought disorder). These symptoms emerged one month after he was made redundant from his job as a labourer (*psychological/social precipitant*). After he lost his job, he stopped taking his antipsychotic medication as he thought it wasn't helping him (*biological precipitant*) and since then he has started to use methamphetamine (*biological precipitant*).

He was first diagnosed with schizophrenia two years earlier and made a good response to antipsychotic medication and had been functioning well in his job. He formed a positive and supportive relationship with his psychiatric case manager.

He has a *genetic vulnerability* for schizophrenia as his father has schizophrenia for which he has received treatment. His early life was characterised by family dysfunction, with little parental care (*social predisposing factor*). School was difficult because of being marginalised and his family having financial hardship. He found it difficult to make close friends and was bullied (*social predisposing factors*). Adolescence was characterised by poor school performance (*psychological predisposing factor*) and experimentation with illicit drugs (*biological predisposing factor*). After school, he was only able to hold down unskilled work and had fleeting intimate relationships.

Protective factors are that when John was engaged with treatment, he adhered to medication (*biological protective factor*) and with the support of his case manager (*social/psychological protective factor*) he is able to cease substance misuse (*biological protective factor*).

Therefore, in sum, he has schizophrenia, which is responsive, and provided substance misuse can be avoided he is likely to benefit from medication in conjunction with psychosocial interventions.

For further, comprehensive examples of case formulations:
(<https://onlinelibrary.wiley.com/doi/10.1111/bdi.13350>).

Conclusion

Eliciting the signs and symptoms of mental illness (anamnesis) and conducting a mental state examination can be regarded as threads, each providing a theme that contributes to an overall story – the narrative that the person experiences. Weaving all these threads together to create a tapestry that accurately

reflects the person's life is the sophisticated process of formulation. Diagnosis sits within formulation and although it informs treatment to some extent, it is formulation that provides the basis for a comprehensive management plan – one which draws on the past and present to meaningfully prognosticate.

Further Reading

- Engel, G. L. (1977). The need for a new medical model: a challenge for biomedicine. *Science*, 196(4286), 129-136.
- Parker, S., Nhieu, K., Warren, N., Dark, F., Cutbush, J., & Suetani, S. (2023). Formulation in psychiatry—a guide to developing competence. *Australasian Psychiatry*, 31(1), 99-104.

Chapter 8: Introduction to Involuntary Care

Joe Garside

Specialty of Psychiatry, The University of Sydney, Australia, and Australian Capital Territory, Australia.

Providing treatment to individuals without obtaining consent is not limited to psychiatry, individuals can be treated in medical emergencies when they are unable to give consent. However, involuntary treatment is a very visible aspect of the practice of psychiatry. Involuntary treatment means taking away a person's freedom and autonomy and provides the treating team with the legal right to impose treatment – something that in other circumstances could be considered to be assault.

Australia has one of the highest rates of involuntary inpatient admissions in the world averaging around 227 involuntary admissions per 100,000 people per year in 2016 (Sheridan Rains et al., 2019). An international comparison found that only Austria exceeded this with 282 involuntary admissions per 100,000 of the population. This rate was double the midpoint value of involuntary admissions across 22 other European countries and New Zealand (Sheridan Rains et al., 2019).

In New South Wales, 54% of admissions to public mental health care units were involuntary in 2022, and the primary factors related to being made involuntary were suffering from psychosis, an organic mental disorder, or a mood disorder. A comorbid substance use disorder also contributed to the risk of involuntary care (Corderoy et al., 2024). Being referred from legal services, being from a non-English speaking background and a socially disadvantaged area all increased the risk of involuntary care as did being from a First Nations people (Australian Institute of Health and Welfare, 2024a; Corderoy et al., 2024).

Each state and territory in Australia have their own Mental Health Act that outline the laws and regulations pertaining to involuntary treatment. Generally, involuntary treatment can be imposed on individuals if they are suffering from a mental illness, at risk of harm to themselves or others, and the involuntary treatment is the least restrictive alternative. This chapter will not focus on any one state's Mental Health Act but provide general ideas about

involuntary treatment. A simple comparison of the Involuntary Commitment and Treatment (ICT) Criteria in Australian and New Zealand Mental Health Acts as of June 2017 can be found at www.ranzcp.org/legislation (Royal Australian and New Zealand College of Psychiatrists, 2017).

Involuntary Transport to Hospital

The journey to involuntary treatment often starts with being transported to a hospital by police for an assessment (Ryan et al., 2015a). Depending on the jurisdiction, typically police and ambulance officers, mental health clinicians and medical practitioners are able to determine that someone needs to be detained at a hospital for an assessment and are able to arrange transport for that to happen.

If the person is unwilling to go to hospital for assessment, almost the only way of transporting them is via a police vehicle. Sometimes, it is possible to use an ambulance escorted by the police, however no specialised ambulance unit is available to transport people under the Mental Health Act who do not wish to comply. This process can be very traumatic for the patient, and, in some instances, for the police or paramedics charged to bring the patient to hospital.

If the patient has evidence of a mental disorder and it is associated with risk that can only be managed in hospital, but they refuse to stay then it will be appropriate for them to be detained as an involuntary patient.

Involuntary Inpatient Admission

In Australia the evolving standard as to whether someone should remain an involuntary patient is whether they have decision making capacity. To not have decision making capacity: 'he or she must be unable to do one or more of the following: 1. Understand the information relevant to the decision; 2. 'use or weigh' that information as part of the process of making the decision; 3. communicate the decision.' (Ryan et al., 2015a)

It is important to understand that lack of insight is not the same as impaired decision-making capacity. A patient with impaired decision-making capacity may argue it is pointless trying to talk them into staying in hospital because they are not unwell, and nothing works and there will be no adverse consequences if they are allowed to go home. A person with impaired insight might argue there is nothing at all wrong with them, but the tablets helped somehow last time so they're willing to give an admission a go. These are very simplistic examples, but it is important to not conflate decision making capacity with insight.

From the outset it should be assumed that a person has decision making capacity and it is the role of an examination to test this. Also, patients need to be given all possible assistance to demonstrate they have decision making capacity by using simple language, an interpreter if necessary, and the support of family or friends if willing and available.

Other key requirements state that involuntary care should be the least restrictive option in which it is safe to treat the patient, that is the person can't be treated safely or effectively in the community, and that the treatment should be effective and available at the treating facility.

The Mental Health Review Tribunal

When someone has been detained as an involuntary inpatient in a mental health unit, they must in due course be presented to a tribunal to review if they indeed have a mental illness and require involuntary treatment. This is usually done by a Mental Health Review Tribunal (MHRT) which has the responsibility to periodically review a person detained under a Mental Health Act. Mental Health Review Tribunals generally comprise a panel of a lawyer, a psychiatrist and another member. At a tribunal hearing the patient will be offered the service of a solicitor and, if necessary, an interpreter.

The MHRT has a number of responsibilities depending upon the jurisdiction. One role of the MHRT is to make sure capacity has been tested. Other duties may include ensuring that the person detained has been provided with information about their rights, their carers have been told about the hearing, that medication hasn't interfered with the ability of a person to communicate and that cultural considerations have been taken into account when a diagnosis of a mental illness has been arrived at. The MHRT can discharge the person, make an order for continued inpatient care as an involuntary patient or discharge the person but with a continuing community treatment order. The Tribunal may also make a decision about the person's capacity to manage financial affairs.

Involuntary Community Psychiatric Care

The process of receiving involuntary care in the community is the same as receiving involuntary care as an inpatient but the least restrictive setting for care has changed. The treating team presents evidence to the MHRT about the presence of a mental illness, risk, and impaired decision-making capacity. Importantly, when asking for an involuntary order for a patient in the community there must be a

treatment that works and for which the mechanisms are in place to provide that treatment.

The rates of community treatment order (CTO) use in Australia are very high by international standards (Light, 2019). Concerns about the long-term restriction of people's freedom of choice brought about by CTOs have combined with evidence casting doubt on the efficacy of CTOs in achieving their stated aims and led to considerable controversy about their continued high usage (Brophy et al., 2021). The effectiveness of community treatment orders are debated with evidence suggesting that they are most likely to reduce the risk of readmission if sustained for at least 2 years (Kisely et al., 2021), but used less frequently (Kisely et al., 2023). The Australian Institute of Health and Welfare reported that in 2022 15% of patients managed in public community mental health teams were subject to involuntary orders (Australian Institute of Health and Welfare, 2024a).

Summary

The role of involuntary care in mental health is controversial. Rates of use vary substantially from country to country which suggests that the high rates of involuntary care seen in countries like Australia may be unnecessary, particularly if the “less restrictive option” of community care is properly funded and developed (Kelly, 2019). Most patients receiving psychiatric care are in the community and are accepting it voluntarily. And most involuntary inpatients eventually become voluntary community patients. International agencies support a human rights approach that emphasises personal autonomy and community inclusions (World Health Organisation and the United Nations, 2023). The change of mental health law in Australia to be based around capacity is supportive of that human rights approach.

Further Reading

Micale, M. S., & Porter, R. (Eds.). (1994). *Discovering the history of psychiatry*. Oxford University Press, USA.

Chapter 9: Psychopathology and Phenomenology

Gin S. Malhi

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia; CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia; Visiting Professor, Department of Psychiatry, The University of Oxford, UK, and Oxford Uehiro Centre for Practical Ethics, Faculty of Philosophy, The University of Oxford, Oxford, UK.

Cornelia Kaufmann

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia; and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

and

Erica Bell

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia; and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

Introduction

This chapter aims to provide an account of the psychopathology and phenomenology of the psychiatric disorders that you are most likely to encounter as a medical student. It contains both essential knowledge for your training as a doctor and that which you may need once you have qualified. In other words, it is more comprehensive than what you require for your psychiatry attachment, but it will be useful, beyond this time, in your clinical practice as a physician. The chapter comprises several sections that address different themes. Within these sections, important terms are given primacy and others are usually listed alphabetically for ease of reference.

The essence of psychiatry is the exploration of the subjective experiences of individuals with mental disorders. An appreciation of what people are thinking and how they are feeling enables a more nuanced empathetic understanding of distress and its diverse manifestations. Other chapters in this book discuss psychiatric history-taking (anamnesis), mental state examination (MSE) and formulation with a

view to developing a management plan. To achieve this, the first step is to elicit the signs and symptoms that contribute to a diagnosis of a psychiatric disorder. This requires phenomenological knowledge and the ability to identify psychopathology.

Psychopathology refers to deviations in mental states, processes, and experiences, and it entails the systematic study of abnormal experiences, thoughts and behaviours. In other words, it is the study of the ‘products’ of the disordered mind (Sims, 1995: Page 1.). Psychopathology can be examined from three perspectives: *experimental* (biological aspects), *experiential* (phenomenological aspects), and *explanatory* (psychological factors), however in practice, it is useful to regard it as having two main components:

1. The empathic appraisal of subjective experience and,
2. The observation of behaviour.

The latter requires careful attention and involves more than merely counting symptoms. It also necessitates objectivity.

To assess the subjective experience of an individual the clinician needs to make use of *empathy*. However, it is important not to confuse this with *sympathy*, which is the relational resonance we have with someone else’s experience. In contrast, empathy is a concept within descriptive psychopathology that is used to gain insight and assess another person’s internal mental state. It draws on the observer’s capacity for cognition and emotional experiences, and these are used as a comparative measure. Typically, it involves the psychiatrist questioning the patient until they are able to create a sense of the patient’s subjective internal experience. This is then relayed to the patient and through successive iteration the experience is accurately captured.

Psychopathology is a broad term that encompasses all abnormal experiences, and within this, *descriptive psychopathology* specifically characterises and categorises those experiences that are described by the patient and observed in their behaviour.

Karl Jaspers (1883-1969) is widely regarded as the father of descriptive psychopathology, even though he was inspired in much of his thinking by the philosopher Edmund Husserl. Jaspers was a psychiatrist as well as being a philosopher. Both he and Husserl believed that the direct investigation of mental phenomena and their atheoretical description, without aetiological exploration, provided deep understanding. Therefore, in keeping with this approach, the signs and symptoms of mental disorders should be defined purely on the basis of

description. In other words, in the absence of any assumptions about aetiology and potential consequences.

Phenomenology is the study of the framework of consciousness – specifically, as it is experienced from the first-person perspective. Notably, it involves *precise description* and nothing more; it does not entail explanation.

German Berrios provides four meanings for phenomenology (Berrios, 1993) and, of these, it is the usage employed by Karl Jaspers, that involves the description of mental states in an empathic and theoretically neutral manner, that is used here (Jaspers, 1963).

It is vital to note that it is not possible to learn the empathic method of eliciting symptoms from a book. As well as being equipped with the necessary knowledge of what to look for, it is essential to see patients and interview them and assess their mental state with the aim of understanding their subjective experience. Therefore, eliciting symptoms in a clinical context is critical to gaining a true appreciation of psychiatric phenomena. In addition, written and sometimes video examples can also be useful and for detailed explanations we recommend Sim's *Symptoms in the mind: an introduction to descriptive psychopathology* (see the recommended readings at the end of this chapter for full details). For video materials it is worthwhile searching online for particular phenomena of interest, or specific disorders within which they manifest.

Mental State Examination (MSE)

The MSE is discussed in Chapter 5, and this should be read in conjunction with this chapter on psychopathology and phenomenology. A template for the format of the MSE is provided in Chapter 5, and we have adhered to this framework to a large extent. There are however a few variations, which reflects the fact that there is no absolute standard for reporting the mental state examination. This is because the MSE, like all medical examinations, is not limited to a particular part of the interaction with a patient. For example, when observing a patient at the bedside who is perspiring profusely, and complaining of a feeling that they are “burning up”, it is immediately apparent that the patient is likely to have a raised body temperature. However, this can only be confirmed by taking the patient's temperature using a thermometer. In other words, while what can be elicited upon examination was evident from history-taking and a preliminary interaction, a formal assessment was still needed to confirm the change. Similarly, in psychiatry, important aspects of the mental state of an individual will necessarily become apparent during history-taking, for example whether the person maintains eye contact, is restless and unable to sit still, or is easily distracted, as well as how they communicate and to what extent

they understand the questions being put to them. An impression on these matters will already have been formed to some extent by the time a formal MSE is conducted.

Further, even within the MSE, global assessments such as whether the person has insight, how they behave and interact, and whether they have decision-making capacity are best decided towards the end of the examination, once more specific aspects have been elicited. The structure of the MSE and when and where to position various components can be confusing.

Always	Appearance └ <i>awareness</i>
Be	Behaviour └ <i>movement</i>
SMarT	Speech Mood └ <i>anxiety</i> └ <i>risk</i>
	Thoughts └ <i>suicide</i>
PerCI	Perception Cognition Insight └ <i>judgement</i>

Figure 9.1. Outline for noting and presenting psychopathology and phenomenology as part of the mental state examination. Note, suicide is an idea and therefore belongs within *thoughts*. However, suicidal thinking and ideation are features of depression and therefore, asking about suicidal ideation in the context of mood is both logical and clinically more practical. Hence why *risk* has been inserted within mood - although risk is a broader term, and it includes harm to self and others.

Therefore, it is important to remember that the MSE is a snapshot of how the person is *now*, and if they are being assessed in hospital, their mental state is likely to have changed considerably from the time they were first admitted. For example, a person who presented agitated and with suicidal ideation some days ago may now be calm and no longer thinking about harming themselves. If assessing them now, then it is the latter (an absence of suicidal thinking) that is reported in the MSE. Hence, there is some fluidity as regards when various aspects of the MSE are assessed, and in which order the assessment is conducted. The format, however,

applies to reporting the findings in the medical notes and summarising these as part of a formal presentation.

The psychopathology and phenomenology in this chapter has been grouped according to seven of the components of the MSE: appearance and behaviour, speech, mood – which includes the assessment of anxiety and risk, thoughts – which again includes the assessment of suicidal thinking, perception, cognition and insight. These headings and the order in which they may be noted can be committed to memory and recalled using the following: Always Be SMarT PerCI (see Figure 9.1).

Appearance and Behaviour

It goes without saying that while noting a person's appearance and behaviour, a record should be made regarding their level of consciousness, for example whether their awareness is compromised or if they seem unusually vigilant or alert. **Awareness** and **consciousness** are related but distinct concepts. Awareness refers to the recognition and perception of information, while consciousness encompasses a wider range of experiences (see Cognition).

In terms of **appearance**, this has a practical purpose in that it allows individuals to be identified in hospital settings, but otherwise it also provides a general indication of whether an individual is taking care of themselves, for example, with adequate grooming and taking an interest in how they appear.

As with appearance, **behaviour** should be documented briefly but may require detailed observation and noting if it reflects the individual's mental state. For example, eye contact, facial expression and general attitude towards others are good indicators of how the person is regarding others. A note should be made of how the patient relates to others and the degree of rapport that is established in the interview.

It is here under appearance and behaviour that any notes regarding **movement** should be made. A general comment about the amount of movement, especially if excessive or limited, is helpful in addition to specific abnormalities of movement. Consideration should be given, however, to the fact that patients may be nervous about being interviewed and therefore, some agitation is to be expected, especially at the outset. However, an excess of motor activity wherein the person is restless, fidgeting, and even perhaps getting up and pacing is unusual, as is a noticeable reduction of activity and slowness of movement. Occasionally, the person may be completely immobile and say nothing (mute).

The abnormalities of movement can be considered under two broad headings: *adaptive* and *non-adaptive* movements. Adaptive movements

encompass **expressive movements** involving the upper body – face, hands, arms and trunk. Expressive movements are often altered in disorders such as depression and schizophrenia – for example, in severe depression (melancholia) the person may exhibit psychomotor retardation in which, along with slowing of thought, the person has reduced and slowed gestures and bodily movements. **Reactive movements** are also adaptive and refer to the automatic and immediate adjustments a person makes in response to a stimulus. For example, flinching when startled by a loud sound. This can also reflect heightened anxiety. Conversely reactivity may be diminished in catatonia and Parkinson's disease. Disorders of **goal-directed movements** are also adaptive movements and may or may not indicate mental illness. For example, mannerisms are not necessarily pathological.

Non-adaptive movements include **spontaneous movements** and **abnormal induced movements**. Spontaneous movements include face touching or scratching, stroking the face or touching or pulling the nose. At one time these actions were goal-directed but have gradually lost purpose and have become spontaneous. They are akin to displacement activity seen in animals and usually triggered by frustration and anxiety. They include tremors, tics and stereotypies. **Abnormal induced movements** are thought to be either a reaction to the environment or a consequence of excessive obedience. Many of these occur in catatonia, which can be categorised according to whether the motor symptoms feature automatic phenomena e.g., automatic obedience; repetitive phenomena e.g., perseveration; withdrawal phenomena e.g., mutism, posturing; and agitated phenomena e.g., impulsivity.

In addition to the two major headings (adaptive/non-adaptive), the individual's **posture** should be assessed and noted whether it is abnormal. Further, in psychiatric patients it is also important to examine for **treatment associated movement disorders**. These most often occur with antipsychotic (neuroleptic) medications.

Beyond these relatively straightforward patterns of abnormal behaviour there are many **complex patterns of abnormal behaviour** that can occur in psychiatric disorders. These can also be divided into goal-directed abnormal patterns of behaviour, such as aggressive and disruptive behaviour that sometimes occurs in psychotic and organic brain disorders, and non-goal-directed abnormal patterns of behaviour that include excitement and stupor, which are sometimes seen in catatonia and extremes of mood (mania and depression) as well as delirium.

Disorders of movement are important as they can often provide useful insights as regards a person's mental state. If for instance a person is especially fidgety, appears somewhat agitated, and is finding it hard to sit still, it suggests that

they may be feeling anxious and restless. Disorders of movement can be noted at the outset of the MSE especially if they have pertinence to the examination as a whole e.g., “the person was difficult to assess as they could not sit for long and were having to pace from time-to-time throughout the interview”. They can also be noted under cognition especially if describing the level of consciousness e.g., drowsy or alert.

Key terms describing movement phenomena are listed in Table 9.2 at the conclusion of this chapter. This table includes descriptions of various kinds of tremor, which can be broadly divided into: **action** and **resting tremors**, based on whether they are brought about by voluntary contraction of muscles (action) or not.

Speech

Speech is the most sophisticated and immediate means through which we not only express ourselves but also gain insights into the minds of others. Therefore, our current understanding of disordered thoughts derives primarily from the speech-related impairments they cause. However, the terminology used to describe disordered thoughts often revolves around abnormalities in speech, and this can sometimes lead to imprecision.

Further, while speech provides valuable insights into the inner workings of the mind, a comprehensive understanding of a person’s thoughts requires consideration of additional factors such as the individual’s cognitive processes, emotional experiences, and social context. By adopting an integrated approach, psychiatrists endeavour to construct a more precise and nuanced understanding of disordered thinking.

- When engaging a patient, it is necessary to first determine whether they are able to speak.
 - Occasionally a person does not speak at all, even though they have the ability to do so (**mutism**). More commonly, they may be impaired in their ability to vocalise speech (**dysphonia**) or articulate speech (**dysarthria**) or comprehend or express language (**dysphasia**).
 - If English is not their first language this should be noted even if they have a good command of it.
- Note the **form, amount, rate, volume** and **tone** of speech (see Figure 9.2). Both the amount and rate of speech can be increased or decreased. These are termed **logorrhoea**, and **poverty of speech** with respect to amount, and **pressure of speech** and **speech retardation** with respect to rate.

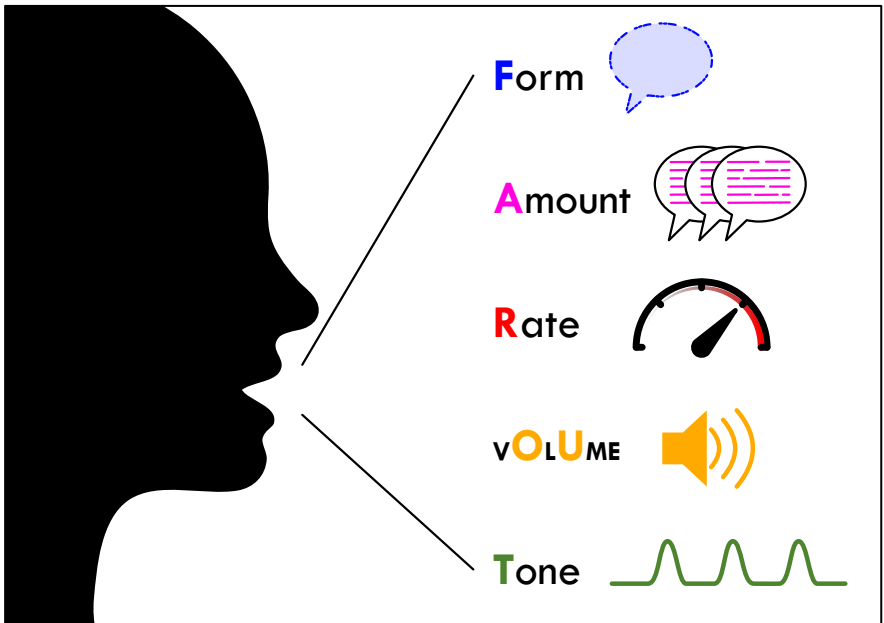


Figure 9.2. The illustration shows a person stating “FAR-OUT” which colloquially refers to something that is exceptional or outstanding. More specifically, it means something is unorthodox or unconventional and this is exactly what needs to be determined when assessing a person’s speech. The aspects of speech that need to be noted can be committed to memory and recalled using ‘FAR-OUT’ as a mnemonic for Form, Amount, Rate, vOLUme and TOne of speech.

Form

Thoughts are necessarily expressed in speech, and some thoughts produce characteristic patterns of speech. It is important to note in these instances that the disorder is that of thought but that it is reflected in speech. This can cause confusion and so below we have provided two common examples of speech patterns (tangential and circumstantial) that are in fact abnormalities of thought form.

Agrammatism: The syntax of speech is lost and as such it becomes meaningless. The person experiences difficulty or an inability to use grammatically correct speech, and often omits conjunctions and neglects tense and word order. Agrammatic speech is often telegraphic and effortful, and while individual words or phrases may still be understandable, the overall meaning of what is being said is lost or is hard to comprehend. In **paragrammatism** speech may still contain well-constructed sentences. Further, while agrammatism usually affects propositions, articles and conjunctions within speech, paragrammatism usually involves

substitution errors in pronouns and verb tense, suggesting that it impacts a later stage in the process of sentence formation.

Clang association: the association of words based on the common sound of syllables rather than meaning. It usually results in nonsensical rhyming speech patterns.

Echolalia: The immediate, automatic (involuntary) and pointless repetition of words or phrases of another person's speech. Note, **echologia** is similar in that the person repeats what has been said but without using the exact same words.

Linguistic stereotype: a tendency to employ a specific word or phrase repeatedly in everyday speech, despite it being unnecessary or simply to excess. It is noteworthy that such usage can be considered normal within the realm of colloquialisms or specific dialects.

Metonym: an existing word with a new meaning or one that is used in an idiosyncratic manner that does not conform to language norms.

Neologism: a totally new word that has been constructed by the individual or the abstraction of words to create a new word.

Perseverative speech: the persistent and involuntary repetition of a particular word or phrase, beyond the point that it is relevant or appropriate. It differs from verbal stereotype in which the same word or phrase is used excessively irrespective of context.

Schizophasia (word salad or confused speech): Individuals produce a jumble of unrelated words, phrases, and nonsensical language that produces incoherent and fragmented speech that is difficult to understand. There is a loss of association between words.

Overlapping phenomena:

Tangentiality and **circumstantiality** are patterns of speech that are indicative of formal thought disorder. These are described in more detail under Thoughts, specifically under Form of Thoughts.

Amount

Alogia: This is also referred to as **poverty of speech**. It can be severely (and noticeably) limited to the extent that the person uses only single words to

respond. It is important to note that it is a functional, non-organic inability to speak, and that less severe cases may be referred to as dyslogia. Note, in some individuals the amount of speech is not necessarily reduced, it may simply require greater prompting.

Logorrhoea: increased quantity of speech, often described as voluble and garrulous. It often lacks meaningful content or coherence and is difficult to understand. Note, there is no increase in the *rate* of speech.

Poverty of content refers to instances where the quantity of speech is adequate, but the content is vague and lacks substance. Poverty of speech may be seen in schizophrenia and severe depression.

Rate

Rapid and slow speech: the rate of speech can be increased or decreased. In rapid speech the rate of speaking is markedly increased, and the quantity of speech may be noticeably greater. This is seen in mania where the person is talking rapidly and is difficult to interrupt. The opposite can also occur where speech is slowed. This is common in depression and seen in **psychomotor retardation**. It can be referred to more specifically as **speech retardation**.

Pressured speech is separate from rapid speech. When speech is pressured the pauses that normally occur in speech are lost and speech essentially becomes continuous. In other words, there is no break or pause and the person is difficult to interrupt. Speech when pressured may also be faster than usual but this is not necessary. Pressured speech is often seen in mania but may sometimes occur in psychosis and extreme agitation or anxiety.

Mutism: the complete loss and absence of speech despite being fully conscious. It can occur in adults in a wide variety of psychiatric disorders such as depression and schizophrenia. In children it can occur if they choose to refuse to speak in certain situations, or to particular people, and this is termed **selective mutism**.

Psychomotor retardation: a slowing down of psychological and physical processes that can be both subjectively and objectively perceived, which results in reduced cognition, speech, and movement, with particular difficulty in initiating action. It is typically a feature of melancholia.

Volume

It is important to note whether the person speaks loudly or softly and whether this is in accordance with the environment. It may be appropriate for example for someone to be speaking loudly if in a noisy emergency department, or softly if imparting particularly sensitive information.

Tone

This usually applies where a person's tone conveys additional meaning. For example, they may be upset and angry and have an 'aggressive tone'. The intonation of speech may also reflect the person's mood and how they are feeling: speaking excitedly for example when elated and having a monotonous tone without inflexion when depressed. These emotional characteristics of speech are valuable and should be used alongside the formal appraisal of mood itself (see below).

Mood, Anxiety, Risk

Our emotions involve a complex interplay of cognitive, physiological, and subjective elements, and colloquially they are referred to as feelings. Emotions comprise elements of mood, affect and temperament that are distinct psychological concepts. Each of these relates to different aspects of an individual's emotional experience and behaviour (see recommended readings).

The emotional predisposition of an individual, refers broadly to their emotional temperament and their mood in general. It can be described as normal (*euthymia*), predominantly depressive (*dysphoria*) or predominantly euphoric (*hyperthymia*). Other changes in mood can be regarded as *emotional reactions* and these include *irritability*, *anxiety* and *apathy* as well as *anhedonia* and indeed, *depression* and *mania*. How emotions are expressed by an individual usually provides additional information and therefore, when noting affect, it should be specified whether it is *blunted*, or whether there is a *dissociation* such that the affect is inappropriate with respect to thought content and/or it is incongruous such that the individual's affect does not accord with the magnitude of events. Note, marked dissociation or incongruity of affect is suggestive of psychosis and warrants further exploration.

Emotions and Feelings

Emotions are colloquially referred to as one's feelings. Emotions are composites of a number of states that have different characteristics and so it is important to separate these when appraising how someone is feeling. The three main emotional components are mood, affect and temperament (MAT) (See Figure 9.3).

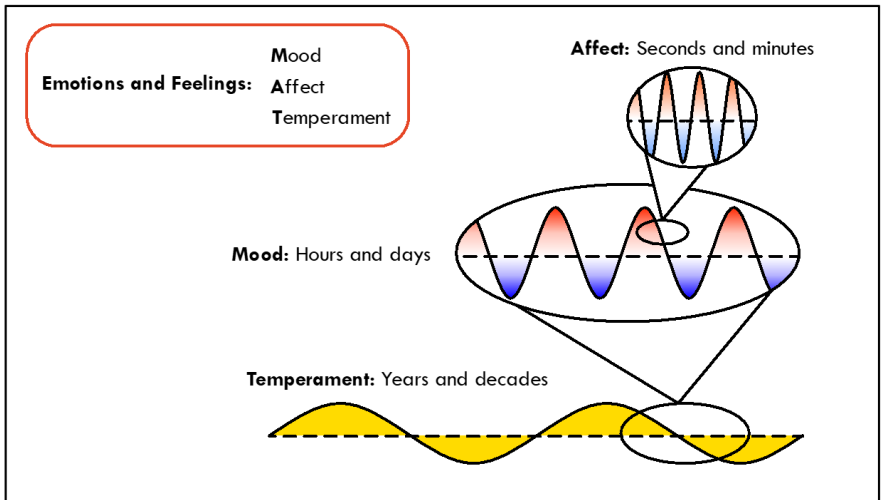


Figure 9.3. The relationship between mood, affect and temperament. Emotions (feelings) comprise mood, affect and temperament. Mood reflects a prolonged emotional state, affect pertains to outward emotional expression, and temperament refers to an individual's inherent and stable behavioural tendencies.

Mood is a sustained emotional state that influences a person's overall outlook and perception of the world. A particular mood state typically lasts for hours and sometime days.

When examining mood, in addition to recording the current mood, its severity should also be rated. If an abnormality of mood is suspected, then after suitable exploration, both one's impression of the patient's mood (objective mood) as well as the report provided by the patient (subjective mood), should be recorded.

Anhedonia: the subjective inability (or reduced ability) to gain pleasure or experience joy from thoughts and activities that the person would usually find enjoyable and rewarding. It is important to note that this symptom refers to the sensation of experiencing emotion (c.f. affect) and it is best elicited by asking specifically about the experiences the person normally finds pleasurable such as watching their favourite sporting team or spending time playing with their children/grandchildren. Questions therefore need to be tailored accordingly.

Apathy: a lack or complete loss of interest, enthusiasm, or motivation towards activities, goals, or life in general. Individuals experiencing apathy have a diminished experience of the sensation of emotion and may exhibit reduced emotional responsiveness and a diminished sense of purpose or engagement. Clinically, the impact of apathy is typically more pervasive than anhedonia.

Dysphoria: an unpleasant mood state characterised by discomfort, dissatisfaction, or unease. It occurs in depression and anxiety and is the opposite of euphoria.

Elation: an elevation of affect or mood.

Euphoria: a happy and pleasant mood when occurring within normal limits. In pathological states is used to signify an extreme change with excessive feelings of happiness and well-being. The latter is also sometimes referred to as **ecstasy**. Excessive and sustained euphoria that does not reflect the reality of one's situation is a feature of mania.

Euthymia: normal contented mood that is neither elevated nor depressed. The term is often used in bipolar disorder to refer to mood status where there are no symptoms of depression or mania.

Lability of mood: the tendency of mood to fluctuate rapidly and unpredictably. Described colloquially as “mood swings”. When the changes in mood are extreme and lead to a complete loss of emotional control, this can be described as **emotional incontinence** and suggests an organic aetiology. More commonly, oscillations and variations in mood indicate the possibility of a bipolar illness (See Chapter 17).

Affect is the observable emotional expression and responsiveness that an individual displays in their demeanour, facial expressions, voice tone, and body language. It is subject to variation over short periods of time (minutes and seconds) and denotes the outward manifestation of a person's emotional state. Clinically, a distinction should also be drawn between mood and affect and the latter if not normal can be further specified as inappropriate, incongruent, flat, blunted or expansive. Dissonance between a person's mood (i.e. their subjective appraisal of their mood) and their affect (i.e. their observable emotional state) is referred to as **incongruity of affect/mood**. [It is important to note that in some psychology texts and publications the term affect has a broader meaning and is used interchangeably with emotion, as this can cause confusion].

It is important to assess emotional responsivity, as it can be diminished, resulting in a reduction in the range and intensity of emotional expression. Individuals may appear emotionally dulled such that their facial expressions, tone of voice, and overall demeanour lack the usual variations of emotion. Terms such as “emotional numbing” are sometimes used, and affect can also be described as being constricted. The main difference is that of degree. **Restricted affect** is the mildest

change. The person is less expressive than usual and experiences a reduction in their emotional responsiveness. It is sometimes referred to as **constricted affect**. **Blunted affect** is significantly compromised in their ability to express emotion, more so than someone with restricted affect but less than that which occurs in flattening of affect. A **flat affect** refers to a complete or nearly complete lack of emotional expression and responsiveness. Note these are all terms applied to affect; decrements in mood – the experience of emotion, are described using terms such as apathy and anhedonia.

Temperament refers to elements of personality, and it reflects an individual's innate and enduring predisposition or style of behaviour, emotional response, and interaction with the environment. It is thought to be biologically anchored and therefore tends to be consistent over time. Aspects of temperament have emotional components and can contribute to feelings of *irritability* and perhaps also the inability to experience emotion altogether (alexithymia).

Aptly, *alexithymia* is sometimes described as **emotional blindness** as it refers to an inability to identify and comprehend one's own emotions and express them verbally. It has been described as 'having no words for one's emotions' and there is both scientific and taxonomic uncertainty as to whether it is symptom of mood for example, or whether it is a facet of personality and whether it should be regarded as pathological.

Similarly, the classification of **irritability** is in question. Clinically, it is a transdiagnostic phenomenon. It is technically conceptualised as a heightened sensitivity to stimuli, coupled with a lowered threshold for responding with anger and aggression to typically less vexing stimuli. It manifests within a wide range of disorders, is present across the lifespan, and is the only phenomenon that spans both poles of mood (see further readings).

Anxiety

Anxiety is a normal but unpleasant affective state. Colloquially, it is often referred to as stress. It is an emotional state of unease, worry, apprehension or intense inner tension and fear that is occasionally accompanied by physiological symptoms (increased heart rate, muscle tension, rapid breathing, dry mouth, and restlessness). It usually occurs because of an anticipated threat, which induces fear without adequate reason. Experientially it is separate and quite distinct from mood but it often cooccurs. Therefore, when assessing mood, it is customary to assess **anxiety**. This is logical, as anxiety is also often a prelude to the emergence of mood symptoms. Further, many of the treatments for mood disorders, both psychological and pharmacological, are also effective in relieving anxiety. If upon inquiry the person reports they are feeling anxious then both the psychological and physical

symptoms of anxiety should be explored further (see Chapter 18 on anxiety disorders, for greater detail).

Risk

A final but important component of assessing mood is to appraise the level of **risk**. This is because thoughts of suicide occur most commonly in mood disorders. These need to be explored sensitively but thoroughly. It is sometimes helpful to link questions about suicide, to symptoms of mood and anxiety. For example, *“You mentioned that your mood is especially low in the morning, is it ever so low that you sometimes wish you had not woken up?”*.

Alternatively, *“do you ever feel so anxious or low that the thought of ending your life crosses your mind?”*.

These questions are difficult and ideally, they should only be put to the patient once sufficient rapport has been established. When noting the psychopathology of emotion, it is useful to have the following subheadings - predispositions, reactions and expressions.

Thoughts

Thoughts have both form and content. The proper terms to describe disorders pertaining to these two facets are: **formal thought disorder** (FTD), which refers specifically to the formation of an individual's thoughts and **content thought disorder**, which refers specifically to what the individual is thinking – their ‘thought content’. Problems regarding the formation of thoughts (FTD) impact the process of thinking, and manifest in the individual's pattern of speech. In contrast, problems regarding thought content are not discernible by examination of speech patterns and therefore a necessary distinction is drawn (See Table 9.1).

In addition to separating thought from speech it is important to understand the different aspects of problems concerning thinking. This requires an appreciation of the nature of thoughts, which as yet, is not fully known. Therefore, thoughts have to be conceptualised, and one example is to liken the process of thinking to that of a **stream** of flowing ideas. The ideas and the stream emanate from our thought processes within the mind, and they flow according to rules that govern our thinking processes. The rate at which thoughts occur is the rate at which the stream is flowing. The substance of the stream and its function is determined by its **content** e.g., the nature and the purpose of our ideas. The structure of the stream depends on the relationship between various ideas and this gives it shape – in other words, it is the **form** of our thoughts.

Table 9.1. Framework for assessing phenomenology of thoughts.

	Thought	Speech
Formal Thought Disorder (FTD)	Process (<i>how the person thinks</i>)	Pattern (<i>how the person speaks</i>)
Content Thought Disorder	Content (<i>what the person thinks</i>)	Subject Matter (<i>what the person says</i>)

When disrupted by disorders, thoughts may not be forthcoming or may be interrupted or slowed; they may be completely lacking or hindered significantly; or alternatively, they may be fast flowing and uncontrollable. These are disorders of the *stream of thought*. In addition, the thoughts themselves (the ideas) may be unusual or abnormal, and cause for concern, and this may include all manner of variations – for example fears, odd ideas and made-up beliefs. However, in all of these cases, it is *thought content* that makes the thoughts abnormal. Further, the thoughts themselves may not be linked in a way that makes sense – having lost their cohesion, their connections and associations, and therefore presenting with odd patterns of thought that are difficult to follow and comprehend. These are aberrations of *thought form* and although the patterns appear in speech, they are primarily disorders of thought. Figure 9.4 illustrates these three properties of thoughts. Each of them should be assessed and noted separately.

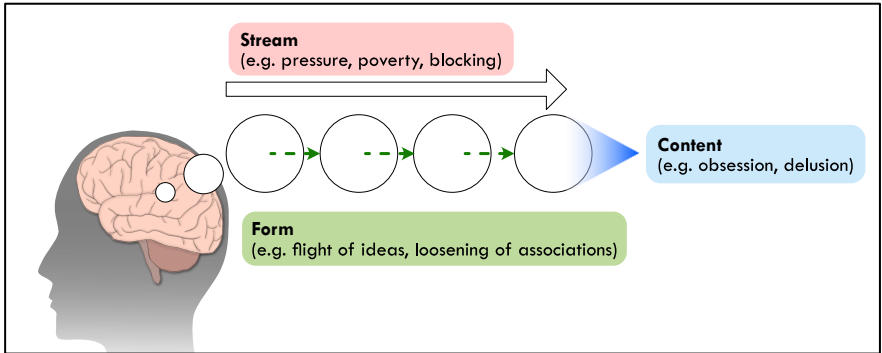


Figure 9.4. Schematic illustrating the components of the psychopathology of thoughts. Thought *stream* (red) reflects the flow of thoughts e.g., phenomena such as pressured speech, poverty of thought and thought blocking. Thought *form* (green) denotes the process of thinking and how thoughts are linked together. Formal thought disorder (FTD), where these processes begin to breakdown, may manifest as loosening of associations. Thought *content* (blue) refers to what the individual is thinking. It is the substance of thought – in terms of its meaning. Therefore, disorders of thought content include over-valued ideas, obsessions and delusions.

Stream of Thoughts

When the stream of thought comes to an abrupt stop – sometimes mid-sentence and the person themselves feels that they have not only lost their train of thought, but their mind has gone completely blank and become devoid of thoughts altogether, this kind of disruption is referred to as **thought block** (or thought blocking, *entgleiten*). The person often experiences and describes a ‘mental block’ during which time they felt unable to continue speaking, despite wanting to do so. The pause may be brief (a few seconds) but can be much longer and the phenomenon is also sometimes referred to as **thought obstruction**. Usually, when they resume thinking /speaking they do so with a totally new train of thought. It is a symptom of schizophrenia but can also occur in severe anxiety.

Disruptions to the flow of thought may also manifest with too few or too many thoughts. In other words, changes to the amount or quantity of thoughts. The term **poverty of thought** is used to describe the phenomenon of having too few thoughts and it manifests as poverty of speech (e.g., *alogia*, see earlier under speech). The opposite of this phenomenon is having the experience of too many thoughts – termed ‘**pressure of thought**’, which then presents as pressured speech (see earlier under Speech).

Form of Thoughts

Problems with the formation of thought are called **formal thought disorder (FTD)**. The term should *not* be abbreviated to ‘thought disorder’ as this lacks specificity and encompasses disorders of thought content. Further, it should be noted that FTD is not synonymous with the descriptor ‘psychotic’. This is important nowadays in clinical practice where patients experiencing a psychotic episode are often wrongly assumed to have FTD when in fact they do not. In other words, it is possible to have severe psychosis and not have FTD. Usually, FTD occurs in schizophrenia and organic brain disorders, and it should only be used to describe disorders of the *form of thinking*.

According to Bleuler, the key problem in schizophrenia is that the associations linking thoughts become disordered and this leads to features such as **displacement**, **condensation** and **misuse of symbols** (Jerónimo et al., 2018). In displacement, one idea displaces another that is loosely associated. In condensation two associated ideas are melded into one idea that is false. In misuse of symbols, there is concrete thinking, such that the person fails to grasp the symbolic meaning. The loosening of associations between thoughts is a central feature. Cameron describes this as **asyndesis** - pointing to a disintegration of connections between thoughts (Jerónimo et al., 2018). In addition, he noted **over-inclusiveness** in

patients with schizophrenia in which the person is unable to maintain conceptual boundaries and also often uses imprecise terms (*metonyms*).

Schneider defined five features of FTD: fusion, omission, derailment, substitution and drivelling (Jerónimo et al., 2018). **Fusion** (*verschmelzung*) is akin to condensation except that the elements of thought that are conjoined are not necessarily linked. **Omission** is straightforward and involves simply leaving out some part of a thought or a whole thought altogether. **Derailement** (*entgleisen*) refers to the shifting of thought ‘off its tracks’ to another subsidiary theme, and **substitution** is a loose variant of this which involves the complete replacement of a major thought with a more subsidiary one. **Drivelling** is the incomprehensible intermingling of the various parts of a complex thought.

Schneider also proposed three key features of healthy thoughts namely, *constancy*, *organisation*, and *continuity* and suggested that in schizophrenia each of these is impaired and that this results in a transitoriness of thinking (*transitory thinking*), an absence of normal thought organisation (*drivelling thinking*) and an interruption to the continuity of thought with sudden ideas forcing their way into the normal thoughts of the individual (*desultory thinking*).

Normally, as part of our existence we act on the world – in other words, we do things to the world and to ourselves and others - we have a sensation of intentionality that is innate to our existence and subjective experience. However, with thought insertion and withdrawal the individual feels that something other than themselves is in control and doing things to them. This displaced locus of control in which the person believes they are being controlled is also the basis of passivity phenomena involving desires, feelings, and actions. Regarding thoughts, passivity phenomena may manifest as thought insertion, withdrawal and broadcasting.

Thought insertion is the subjective experience of thoughts or ideas being “placed” in one’s mind. The thoughts are inserted without the person’s consent and are not under their control. **Thought withdrawal** is the subjective experience of one’s thoughts being removed by an external force. The person experiences the ‘sensation’ of the process of removal of the thought and is left with a sense of loss of control over their thinking. **Thought broadcasting** is also called ‘*thought diffusion*’ and sometimes more informally referred to as ‘*thought leakage*’. The person feels that their thoughts are accessible to people around them, and that somehow, they are passively escaping their mind and therefore effectively being broadcast. They may even feel that this is taking place via means of various technologies such as radio, television or the internet. The feelings this induces are usually negative and typically the phenomenon is accompanied by great concern as the person fears others will come to know their thoughts, which may be of an aggressive or embarrassing nature.

Other Terms for Disorders of Thought Form

Dereistic thinking refers to idiosyncratic thoughts that are not in keeping with reality but are also not being falsified by reality per se. For example, daydreaming.

Tangential thoughts are apparent in a corresponding pattern of speech that constantly pursues irrelevant topics and fails to arrive at the main point. Verbal responses are oblique and only glance at the gist.

Circumstantial thoughts are apparent in a corresponding pattern of speech wherein the conversation meanders, delving into unnecessary details as it progresses towards its ultimate goal. Importantly, the point that the person intends to make is finally reached although in the process they have usually incorporated many irrelevancies and focused on unnecessary details. It can occur in schizophrenia and those with a learning disability and is associated with obsessional personality traits. Ultimately, their account often includes many trivial details that are largely irrelevant. However, in contrast to **tangentiality**, a person being circumstantial eventually arrives at their main point (their intended destination).

In ***loosening of associations***, thoughts shift from one idea to another but lack connection. The person experiences this as their speech being illogical and their thoughts being confused.

Flight of ideas is best thought of as a combination of either tangential thinking or loosening of associations with pressure of speech. It is important to note that the latter is not simply rapid speech (see earlier).

Thought Content

Ruminations: These consume the individual's thought processes and disrupt normal thinking. They are persistent, repetitive thoughts that often centre on past events (as opposed to future events, which usually characterise worry) hence they often occur in anxiety, depression and obsessive-compulsive disorders.

Phobias: an irrational and persistent fear of an object, activity, or situation that goes beyond normal bounds and is difficult to control. It leads to conscious avoidance of the feared entity.

Pseudologia fantastica (mythomania): the person lies fluently and plausibly, hence often referred to as 'pathological lying'.

Obsessions: these are persistent and intrusive thoughts, images, or impulses, that usually causes distress in the form of anxiety and sometimes guilt. The obsessional thoughts themselves are seen as senseless but cannot be controlled by the individual. Indeed, they seem to occur against the person's will and cannot be resisted. Hence, moods and hallucinations are not described as obsessional, whereas thoughts, ideas, impulses and fears as well as mental images can all be described as obsessions. Obsessions differ from normal worries in that they persist beyond the point of relevance or usefulness and seem to occur without purpose. Their content is often repugnant to the individual. For example, religious individuals may be tormented by blasphemous thoughts and those that are shy may experience ruminations of a sexual nature. Other common examples include fears of contamination, the need for order, and aggressive thoughts. **Compulsions** are often regarded as separate from obsessions but in essence, compulsions are simply obsessional motor acts. They can be driven directly by obsessional thinking or occur in response to an obsessional thought or image. Usually, compulsions involve a behavioural or mental activity that is undertaken to alleviate anxiety or distress associated with an obsession. Typically, compulsions provide no pleasure or gratification, but offer temporary relief to the individual from the anxiety and/or distress associated with an obsessional thought. Common examples of compulsions include behaviours such as hand washing or checking, and mental activities such as counting, praying or repeating words or phrases to oneself in one's mind.

Ideas of reference: a belief that neutral events or stimuli in the environment have a special and personal significance directed towards oneself. Individuals experiencing ideas of reference may interpret ordinary occurrences or unrelated actions of others as being specifically related to them. This manifests as a feeling that external incidents and causal events have reference to oneself. If this feeling transmutes to a fixed idea or belief, then it may be referred to as a ***delusion of reference*** (see below).

Overvalued idea: A preoccupation that is held intensely by an individual, and while they believe it to be justified, it remains open to counterargument or evidence. In other words, the idea itself is acceptable and comprehensible but is problematic because it dominates the person's thoughts and behaviour. Further, the idea is not commonly shared by others with similar backgrounds. Notably, an overvalued idea differs from an obsessional thought as it is not regarded as being senseless and it differs from a delusion because the belief is not fixed.

Delusion: a fixed, and indeed, unshakeable, false belief that persists despite evidence to the contrary. Delusions are irrational and not in line with cultural, religious, or societal norms. Note, delusions are not always and necessarily

false, but they arise through a process that is illogical and often bizarre. Delusions can be primary or secondary. A **primary delusion**, also termed an autochthonous delusion, or delusional intuition, is one that is psychologically irreducible. It is not in any way linked to previous ideas or events and it appears fully formed. A **secondary delusion** is understandable in the context of previous events or ideas and arises from such prior events or ideas. Delusions have many themes (see below) and typically develop gradually through various stages (see Figure 9.5).

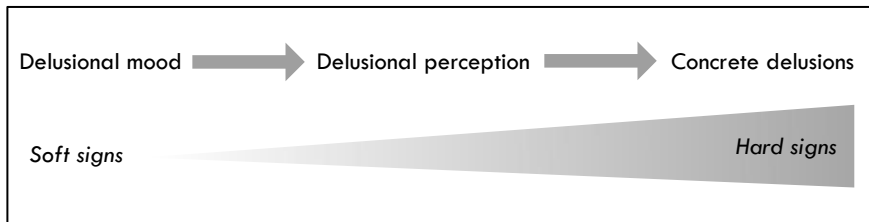


Figure 9.5. Schematic illustrating the typical development and progression of delusions. Delusions commonly occur as part of **psychosis**, a process that is usually marked by ‘hard signs’, which also include hallucinations and formal thought disorder. Typically, the **hard signs** of psychosis do not appear abruptly. Instead, they usually emerge gradually indeed often insidiously, beginning with ‘soft signs’. The first step is the emergence of a **delusional mood** (Wahnstimmung) in which the individual begins to sense (feel) that something, that they can’t put their finger on, is not quite right. Sights and sounds might become more intense, and the environment appears more vivid and clearer to them than ever before. Certain matters take on a special significance and the individual generally feels unsettled and has a sense of foreboding. Over time, delusional mood gives way to **delusional perception** in which the percept is normal, but its interpretation is abnormal. In other words, delusional significance is attributed to normal percepts. Finally, delusional perception and delusional mood are superseded by **concrete delusional ideation**. Now the person ‘knows’ for sure what they are thinking is true – and their beliefs become fixed.

Delusional Themes

Jealousy: A mistaken conviction that one’s spouse or partner is being unfaithful. The person remains vigilant for signs supporting this belief, fabricates proof if none is present, and dismisses contradictory evidence. This condition is also referred to as **morbid jealousy**, or **pathological jealousy** and is sometimes described as *Othello syndrome*.

Being controlled (passivity phenomena): a false belief that external forces, such as machines or other people, are controlling one’s thoughts, feelings, or actions. Delusions of control are of various types:

- Passivity of affect, desire, or action
- Somatic passivity
- Thought broadcast
- Thought insertion
- Thought withdrawal

Grandeur: A false belief in one's exceptional ability, knowledge, worth, identity, prestige, power, importance, or achievements far beyond reality. This belief is also referred to as a **grandiose delusion**.

Persecution: a false and persistent belief that one is being targeted, harmed, or threatened by others. Individuals experiencing this delusion interpret neutral or benign actions as evidence of malicious intent to harm them.

Reference: the belief that neutral or unrelated events, objects, or actions hold special significance that is specifically directed toward oneself. Individuals experiencing this delusion may interpret everyday occurrences as having hidden messages or meanings intended for them (see also *ideas of reference*).

Somatic: a false belief concerning one's body or physical health. Individuals experiencing somatic delusions believe they have a medical condition, deformity, or infestation, despite evidence to the contrary. These delusions often centre around bodily functions or appearance.

Hypochondriacal: a fear or a preoccupation with the belief that one has a serious physical illness or abnormality based on an incorrect interpretation of bodily symptoms. The person cannot be reassured and repeatedly seeks help.

Nihilistic: the irrational belief that oneself, others, or the world as a whole do not exist, and that these things lack meaning or significance. Individuals experiencing this delusion may feel detached from reality and may deny the existence of essential aspects of life. In severe psychotic depression a nihilistic delusion may occur in combination with a somatic delusion in which the individual believes they don't exist or are dead. This is called Cotard's syndrome (*le délire de negation*) after Jules Cotard.

Querulent: a persistent and unwarranted belief that one is being unjustly persecuted, harassed, or conspired against by others. Individuals with querulent delusions may obsessively pursue legal action or make repeated complaints to authorities, even when evidence contradicts their claims.

Perception

Perception is the process by which sensory information is received, organised, and interpreted by the brain to create a meaningful understanding of the external world. It involves the integration of sensory inputs, such as sight, hearing, touch, taste, and smell, along with cognitive processes, emotions, and past experiences, to construct a coherent and subjective representation of one's surroundings.

The phenomenology of perception refers to the subjective experience and interpretation of sensory information by an individual. It explores how sensory stimuli are processed, organised, and given meaning, influencing one's perception of the world and interactions within it.

Changes in perception that occur because of a change in the quality or intensity of a stimulus, or its spatial form are termed **sensory distortions**. These include changes in the threshold for sensations such that, for example, normal sounds may seem louder to the point they are distressing (*hyperacusis*), or alternatively, they may be noticeably dampened as can happen in depression with the dulling of taste and colour. The colours of objects may be altered altogether – although this is usually a consequence of drug-taking or the toxic effects of medications. In such instances, the spatial form of objects may be distorted with things seeming much larger or smaller. Mood and anxiety can also distort our experience of time (personal judgement of time passing). When low in mood for example, most people complain of time passing slowly whereas when happy time seems to pass quickly. Sensory distortions are interesting and noteworthy, but it is sensory deceptions that are the mainstay of perceptual psychiatric phenomena.

Sensory Deceptions

Generally, there are two distinct kinds of sensory deceptions: illusions and hallucinations.

Illusions

Illusions are misperceptions of real external stimuli. They occur when sensory inputs are correctly perceived but are interpreted by the brain inaccurately. Illusions can involve any of the sensory modalities (e.g., vision, hearing, touch) and are specified accordingly. Visual illusions are the most common and although they do not usually indicate psychopathology they can occur in delirium and in psychiatric disorders. Therefore, it is important to differentiate them from hallucinations (see below), in particular functional hallucinations where the hallucination and stimulus

are perceived simultaneously. In the latter instance the two remain differentiated whereas in an illusion the stimulus becomes part of the new perception.

In addition to subtyping illusions according to sensory modality, they can also be defined based on the mechanisms involved. Three types of illusions have been described.

Affect illusions arise in the context of specific mood states, for example, in melancholia, severely depressed individuals are more prone to misperceive negatively things that are said to them.

Completion illusions involve completing an incomplete percept, such as reading a word that is missing a letter as if it were complete. This usually arises because of inattention.

Pareidolia are illusions that arise without effort. They are usually vivid and occur within poorly defined stimuli such as a fire or the clouds in the sky resulting in images and shapes.

Hallucinations

Hallucinations are perceptual distortions within any sensory channel without any corresponding external stimuli or trigger. They can therefore involve seeing, hearing, feeling, tasting, or smelling things that are not actually present. Typically, hallucinations are described and categorised based on the sensory modality involved, and in psychiatry auditory hallucinations are the most common type.

The definition of hallucinations is important. Put succinctly they are false perceptions or a perception without an object. A more detailed definition is that a hallucination is a percept that seems real and is therefore experienced as emerging from the sensory organs and not from *within* the mind. The key is that there is no real external object, and the hallucination is being generated internally. Hallucinations can be confused with vivid mental images, which can appear to be real and exist externally. However, these are recognised as not being real by the person perceiving the mental image. In contrast, a person experiencing a hallucination fails to make this distinction and regards it as real.

Auditory Hallucinations

Auditory hallucinations are described as follows:

Auditory Hallucinations: Hearing distinct words, phrases, or sentences spoken by voices or entities, often directed towards the individual.

These can be categorised into three perspectives:

- *First Person* ('I'): The individual hears voices referring to themselves using "I." Voices make statements, give instructions, or converse as if speaking from their own perspective.
- *Second Person* ('you' or direct address): Hallucinatory voices directly address the individual using "you" or their name. They command, criticise, or comment on the person's thoughts, actions, or behaviour.
- *Third Person* ('he/she' or 'him/her'): Voices refer to the individual in the third person, using pronouns like 'he/she' or 'him/her.' Multiple speakers may engage in conversations, discussing the person's actions and behaviour.

Non-Verbal Auditory Hallucinations: Perceiving sounds, noises, or music without distinct verbal content. These may include buzzing, ringing, footsteps, or other auditory sensations.

Command Hallucinations: Voices instructing the individual to perform specific actions, often with varying degrees of urgency or authority.

Conversational Hallucinations: Hearing voices engage in dialogue with each other or with the person experiencing the hallucination.

Commentary Hallucinations: Voices providing a running commentary on the person's thoughts, actions, or surroundings.

Somatic Auditory Hallucinations: Hearing internal bodily sounds, such as one's heartbeat or digestive noises, as if they were originating externally.

Thought Sonorisation subsumes two slightly different phenomena.

Gedankenlautwerden is a composite German word that means 'thoughts becoming audible'. It refers to the experience of hearing one's own thoughts spoken out loud while having the thoughts. The two occur simultaneously. In contrast, in *echo de la pensée* the person hears their thoughts *after* they have been produced. Therefore, *thought echo* is a specific type of auditory hallucination in which an individual hears their own thoughts being spoken aloud, often as if echoed by another voice.

Thought echo can be further specified as **direct**, where the person hears their thoughts repeated exactly as they occur in their mind, resembling an immediate echo. And **indirect** where the person hears their thoughts being spoken aloud, but with variations or additions that may change the original content.

Visual Hallucinations

Visual hallucinations are perceptual distortions in which an individual sees objects, images, or scenes that are not actually present in their external environment. These hallucinations involve the sense of sight and can vary from simple shapes to complex and detailed visual experiences. Types:

Simple Visual Hallucination: Basic, unformed visual perception, such as flash of light, geometric shapes, or colours.

Complex Visual Hallucination: Detailed and elaborate visual experience involving recognisable objects, scenes, or people.

Lilliputian Hallucination: Visual hallucination where objects or people are perceived as smaller than they should be.

Autoscopic Hallucination: involves perceiving images of one's own body or face projected into the surrounding environment.

Negative Autoscopy: the individual feels as though their own presence or body is missing, leading to a sense of disembodiment. Interestingly, the person reports they cannot see an image of themselves in a mirror.

Tactile Hallucination: false or disordered perception of touch or physical contact that may involve sensations felt on the skin such as animals or insects crawling (formication), or the feeling of being touched by a person who is not present. The hallucination may also involve the sensation of organs moving within the body, or the skin being stretched or pulled.

Gustatory Hallucination: false or distorted perception of taste and flavour that can vary from pleasant to unpleasant sensations.

Olfactory Hallucination: perceptual distortions in which an individual perceives smells or odours in their environment.

Somatic Hallucination: perceptual distortions involving false sensations experienced in the body, such as touch, pressure, movement, or internal bodily functions. May vary from subtle sensations to intense and distressing experiences.

Phantom limb sensation is a somatic hallucination where an individual continues to feel sensations, such as pain or movement, in a limb that is no longer present (e.g. amputated). This phenomenon occurs due to the brain's continued representation of the missing limb, leading to perceptions within the brain even though the limb is absent.

Specific kinds of hallucination

Some hallucinations occur in relation to sleep. **Hypnagogic Hallucinations** occur immediately before falling asleep (gogic – going to bed). Common themes include sensations of falling, floating or seeing geometric shapes

or patterns. **Hypnopompic Hallucinations** occur immediately before waking (pompic – popping awake). Often these include a sense that another person is in the room, or sensations of weightlessness or flying.

A **pseudo-hallucination** is a perceptual experience that has sensory qualities similar to a hallucination but is *recognised by the individual* as not originating from external stimuli. It occurs within the mind's eye or mental imagery, often lacking the sense of reality present in true hallucinations.

A **functional hallucination** is triggered by an environmental stimulus from within the *same* sensory modality. For example, an auditory hallucination that is triggered by the sound of a particular piece music. In contrast, a **reflex hallucination** is triggered by a stimulus in a *different* sensory modality.

Depersonalisation and **derealisation** are considered disturbances in the subjective experience of reality rather than psychotic symptoms, although they sometimes occur in psychotic states. Hence, they are considered here. These symptoms can also occur in the context of emotional states, organic brain disease and substance misuse. Usually, **depersonalisation** involves a subjective sense of detachment from oneself, leading to a feeling of unreality regarding one's thoughts, sensations, body awareness, and perception of time. Despite this change, individuals still experience and recognise low mood and/or anxiety, which often accompanies this phenomenon, and it is this associated distress that prompts them to seek assistance. The phenomenon is often accompanied by **derealisation** in which the individual perceives their external environment, including other people, as unreal, distant, or distorted. It involves a sense of detachment from the surroundings as if the world around them has taken on a dream-like or surreal quality.

Cognition

A comprehensive and formal assessment of cognition (see definition below) requires sophisticated testing that takes considerable time (hours) and requires specialist expertise. Bedside, the appraisal of cognition involves assessing the level of consciousness and awareness and then conducting simple tests to assess *orientation, attention and concentration, memory and general mental abilities*. More details about cognition and cognitive testing are provided in Chapter 6 but in essence orientation to time, place and person should be noted along with the results of a straightforward test of attention/concentration such as serial sevens. This involves subtracting 7 from 100 and then repeating this for as long as possible (e.g., 100, 93, 86, 79, ...). The time taken and any errors are noted. Short-term memory is assessed as well as recent and remote memory before evaluating the grasp the person has on current affairs. Only if the person struggles with these tests is a more comprehensive assessment indicated.

In addition to the formal appraisal of **cognition** – *the mental process of gaining knowledge through experience and thought to achieve understanding* – it is important to be familiar with terms that define the general awareness of an individual. These are less specific to psychiatry and are used in medicine more generally.

Awareness refers broadly to whether a person is attentive, and it involves recognising and perceiving information, whether internal (thoughts, emotions) or external (environment, stimuli). In contrast, **consciousness** refers to the broader and more complex state of being awake and having an ongoing subjective experience. It encompasses awareness and self-awareness and entails the sense of one's own existence through perception, thoughts and emotions. When an individual has diminished awareness of the environment, and becomes confused, and has difficulty in maintaining concentration the sensorium is said to be clouded (*clouding of consciousness*). If this progresses to a profound state of unconsciousness this is termed a *coma*. The latter can be rated using the Glasgow Coma Scale. Altered consciousness occurs in delirium – an acute confusional state that is characterised by a fluctuating level of consciousness, disorientation, and impaired attention. Delirious individuals often exhibit restlessness and bizarre behaviour. Delirium is typically caused by underlying medical conditions, such as infections, adverse reactions to medications or metabolic imbalances. (Note: in contrast, dementia involves a global impairment of executive functioning in a *clear state of consciousness*. Though of course delirium and dementia can cooccur and often do.) An important component of being aware is self-awareness, which is necessary for insight and the execution of judgement (See below).

Insight

An understanding that one is sick and that one requires treatment is basic to health and help seeking but is often absent or only partial in psychiatry. Being self-aware and having an understanding of one's own thoughts, emotions, behaviours, and mental health condition is described as having **insight**. It involves recognising and acknowledging one's strengths, weaknesses, and motivations. It also involves understanding the impact of one's actions on oneself and others and that these actions may constitute an illness. Not only that it may be an illness but that this requires change or treatment. Hence, it plays a crucial role in therapeutic and self-improvement processes. Insight can be graded as being full, partial or absent (lacking).

Insight is also linked to **judgement**, the ability to make well-considered decisions. This is also referred to as *deliberative capacity* and it refers to the individual having the ability to weigh up contingencies and arrive at a reasoned decision. Like insight, having good judgement is important for making decisions regarding clinical

care. In psychiatry the judgement of patients may be overridden by that of health professionals to maintain their mental well-being and ensure their safety (See also Chapter 8).

Table 9.2. Phenomena of movement

Voluntary Purposeful Movements	
Mannerism	The unnecessary repetition of normal (goal diverted) movements or habitual poses, such as blinking or flicking one's hair. It can also involve the adoption of exaggerated postures and the strange use of words or expressions that are not in keeping with the situation with excessive frequency.
Dyspraxia	The impaired ability to perform a familiar task despite having intact coordination, sensation, and motor pathways. It involves difficulties in executing coordinated movements efficiently.
Compulsive act	An unnecessary action that is performed as a means of relieving inner tension. It is typically a specific task or behaviour carried out repetitively. In contrast, a motor tic (see later) is generally characterised by the presence of repetitive and involuntary movements without a specific purpose. Note, obsessions and compulsions can be considered as disorders of behaviour as they involve the disruption of the normally subjective experience that entails the progression of thoughts and actions.
Voluntary Non-Purposeful Movements	
Stereotypy	A non-goal direction action that results in purposeless repetitive movements, such as excessive foot-tapping or rocking, that are frequently observed in an individual to the point that it appears characteristic for them. Can occur in organic states and catatonia. Sometimes a vestige of purposeful movement remains and may be discernible. Thus, can be confused with mannerisms (see above).
Involuntary Movements	
Akathisia	A subjective feeling of restlessness, that entails an irresistible urge to move the legs or body. Individuals with akathisia find it challenging to stay still or sit, and often exhibit repetitive movements or pacing. The distress is relieved temporarily by movement of the affected part of the body – most commonly the legs. It is a side effect of some medications (e.g., antipsychotics, see Chapter 35 on psychopharmacology).

Ambitendency	The individual experiences conflicting urges between carrying out and resisting a specific action. There is the coexistence of contradictory actions or thoughts, resulting in hesitation or difficulty in decision-making. It can be conceptualised as a combination of altering automatic obedience and negotiation.
Athetosis	Slow, writhing, and twisting movements of the limbs, face, and sometimes the trunk. The movements are sinuous fluid and continuous, and muscle contractions and relaxations are evident.
Automatism	Involuntary and unconscious actions, behaviours, or movements that occur without the person's deliberate control or awareness. These actions can range from simple movements such as lip-smacking or repeatedly using the same phrase to more complex behaviours such as sleepwalking and automatic writing.
Automatic obedience (command automatism)	The individual carries out another person's instructions without a subjective appraisal or consideration of the consequences. The actions are executed automatically and reflexively, without questioning motives. Mitmachen is a form of automatic obedience (cooperation) in which the individual, allows their body to be freely positioned even though the individual has been told to resist all movements. Once the displacing force is removed the body part returns to its original resting position. Mitgehen: an exaggerated and extreme form of Mitmachen that is characterised by the individual exhibiting movement in response to minimal force/pressure in any direction. Individuals with this condition perceive even slight pressure as forceful and respond with involuntary movement, again despite instructions to resist.
Blepharospasm	Tonic spasms of the eyelid muscle, leading to involuntary blinking or closure of the eye. This involuntary movement can be persistent and disruptive, causing discomfort and functional impairment.
Bruxism (bruxomania or stridor dentium)	The persistent habit of grinding, clenching, or gnashing of teeth, typically occurring during sleep. It is often associated with feelings of tension, anger, frustration, or fear.

Catalepsy (catatonic rigidity, flexibilitas cerea or waxy flexibility)	This refers to the condition in which the limb of a patient can be moved (positioned) by another person to any posture. The individual then maintains this position or a fixed body posture for an extended period. It is observed in catatonic schizophrenia.
Cataplexy	A sudden loss of muscle tone, which can be either localised or generalised. Localised cataplexy may lead to the loss of grip or nodding of the head, while generalised cataplexy causes the collapse of the entire body. The condition is usually temporarily and typically triggered by intense emotional stimuli, such as overwhelming anxiety, excitement, or anger. It is also a symptom of narcolepsy.
Catatonia	This involves an increase in resting muscle tone. In contrast to the visibility caused by Parkinson's disease and extrapyramidal side effects, the increase in muscle tone is not present on active or passive movement. It is a psychomotor syndrome that can be categorised into two main subtypes. Catatonia of the retarded type is characterised by immobility, mutism, staring, and rigidity. Excited catatonia is less common and involves prolonged periods of psychomotor agitation. While catatonia was previously considered a subtype of schizophrenia, it is now known to occur in various medical and psychiatric conditions, especially affective disorders.
Chorea	Rapid, jerky, and irregular movements that flow unpredictably from one muscle group to another. These movements are often described as dance-like and can affect various parts of the body, including the face, limbs, and trunk. The migration of movement across muscle groups can make the action appear voluntary.
Cog-wheeling	A term used to describe a distinctive Parkinsonian sign. It involves the combination of lead-pipe rigidity, which is a constant resistance to passive movement, with superimposed tremor. This phenomenon results in a jerky, interrupted movement resembling the turning of gears, hence the term "cog-wheeling."
Copropraxia	The involuntary expression of obscene or socially inappropriate gestures, e.g., offensive or obscene hand

	movements or gestures. It may cause significant distress and impairment in social interactions. The movements can be resisted but only temporarily and resistance causes significant anxiety. Seen in Gilles de la Tourette's syndrome (see Named Syndromes below).
Dyskinesia (dyskinesia)	A broad term encompassing various involuntary, abnormal, and often repetitive movements. These movements can include tics, chorea, athetosis, and other motor abnormalities. Dyskinesia can result from neurological disorders, medication side effects, or other underlying conditions.
Dystonia	Impairment of normal muscle tone, leading to recurrent prolonged muscle contractions that cause abnormal postures, twisting, or repetitive movements. While dystonia typically manifests in a focal area, it can also spread to other parts of the body. An example of dystonia is opisthotonus, characterised by an arched posture caused by spasms in the paraspinal muscles.
Echopraxia	Automatic mimicry. The involuntary mechanical imitation or repetition of another person's actions, gestures, or movements. It persists even after being told to stop.
Grasp reflex	An involuntary, automatic response observed in infants and some neurological conditions. When an object is placed in the palm of the hand or touches the fingers, the individual's fingers close around the object and grip it tightly.
Grimacing	The involuntary exaggerated contraction of facial muscles, resulting in distorted or contorted facial expressions.
Lead-pipe rigidity	A type of muscle hypertonicity characterised by constant, uniform resistance to passive movement throughout the range. It is commonly seen in conditions like Parkinson's disease and is caused by basal ganglia dysfunction.
Myoclonus	Sudden, brief involuntary twitching or jerking of a muscle or group of muscles. Occurs spontaneously and cannot be suppressed by the individual.
Nystagmus	Involuntary, rapid oscillating movement of the eyes. Motion may be rotatory, horizontal, vertical, or a mixture.

Oculogyric crises	Involuntary, spasmodic movements of the eyeballs, typically in an upward direction. These episodes, lasting minutes to hours, may be accompanied by increased blinking, pain, and associated symptoms like neck dystonia and tongue protrusion. Neuroleptics and dopamine receptor antagonists are two identified triggers for oculogyric crises.
Oppositional paratonia	Also known as gegenhalten (a German word meaning resisting), is characterised by involuntary resistance to passive movement of the limbs.
Parkinsonism	A motor syndrome characterised by movement abnormalities similar to those seen in Parkinson's disease. Typically manifests as tremors, bradykinesia (slow movements) and rigidity (stiffness). Commonly occurs as the extra-pyramidal side effect of medications (see Chapter 35, Basic Principles of Psychopharmacology).
Posturing	Individuals adopt bizarre or inappropriate body postures for an extended period of time. Observed in psychotic disorders and may hold delusional significance.
Psychological pillow	Individual holds their head a few centimetres above the floor/bed while lying on their back. The person is able to maintain this position for a prolonged period of time even though it is uncomfortable.
Schnauzkrampf	The involuntary contraction of facial muscles, particularly those around the mouth, leading to a grimacing or pained expression resembling pouting. It is also known as a 'snout spasm'.
Tardive dyskinesias	A group of late-onset dyskinesias associated with prolonged antipsychotic therapy. Characterised by disabling and potentially irreversible symptoms, including tremors, choreoathetoid movements, and spasticity in orofacial and limb muscles. Most commonly there is continuous involuntary movement of the lower face and tongue, more severe cases extend to the upper face and limbs.
Tic	Abrupt and involuntary movements that occur despite efforts to suppress them. Tics can be simple (isolated muscle movement) or complex (coordinated movement of multiple muscles) and typically involving jerking actions affecting the face, neck, and trunk. They tend to

	worsen during periods of marked anxiety and emotional distress.
Titubation	A rhythmic, involuntary movement of the head characterised by a back-and-forth or side-to-side nodding motion. Titubation can be observed in various neurological conditions, including disorders affecting the cerebellum or its connections.
Torticollis	Also known as “wry neck,” is characterised by involuntary and sustained contraction of neck muscles, causing the head to tilt to one side and the chin to rotate in the opposite direction. It can be a side effect of some neuroleptic medications.
Trismus (lockjaw)	Sustained, tetanic spasm of masticatory muscles causing reduced mandible motion.
Waxy flexibility (catalepsy; catatonic rigidity; flexibilitas cerea)	Prolonged maintenance of fixed body posture or physical attitude, with a plastic tone allowing the body to be positioned and held in various positions for an extended duration (see Catalepsy).
Tremors	
Postural tremor	Occurs when an individual sustains a position against gravity, such as holding their arms outstretched.
Kinetic tremor	Manifests during the movement of a specific body part, such as moving the wrists up and down. It becomes more pronounced with purposeful motion and tends to diminish or disappear when the limb is at rest.
Intention tremor	Trembling of a body part that occurs towards the end of a purposeful, voluntary movement. For example, it may be observed when an individual attempts to touch their finger to their nose. The tremor becomes more noticeable as the intended movement target is approached. Intention tremor is often associated with disorders affecting the cerebellum or its connections.
Task-specific tremor	Occurs specifically during the performance of highly skilled, goal-oriented tasks. Examples of such tasks include handwriting, playing a musical instrument, or performing fine motor activities. The tremor is typically isolated to the specific task and may not be present during rest or other activities.
Isometric tremor	Arises during a voluntary muscle contraction without any associated movement of the affected body part. The shaking or oscillations occur within the contracted

	muscle itself. It can occur in various muscles and can be observed in tasks that involve sustained muscle contractions, such as gripping an object tightly or maintaining a fixed position against resistance.
Essential tremor	An action tremor, either postural or kinetic in character, mainly affecting the hands. It is usually bilateral, with a frequency of 4 Hz to 12 Hz and largely symmetrical. It is hereditary and the most common type of movement disorder.
Physiological tremor	A normal occurrence in all individuals when muscles are activated. It is typically postural and has a frequency of 8-12 Hz. It is often so subtle that it is barely visible to the naked eye and does not disrupt daily activities. The frequency of physiological tremors tends to decrease when the limb is subjected to larger loads or inertia.
Increased physiological tremor	The heightened visibility of a high-frequency, postural tremor that occurs without any indication of an underlying neurological disorder. This type of tremor can be resolved by addressing the underlying cause. Various factors can exacerbate physiological tremors, such as stress and anxiety, especially in situations involving public performance. Other conditions that can amplify physiological tremors include thyrotoxicosis, pheochromocytoma, hypoglycaemia, withdrawal from opioids or sedatives and taking Valproic acid or Lithium.
Resting tremor	Resting tremor manifests when the affected body part is at rest and not engaged in any voluntary movement. It is typically more prominent when the individual is relaxed or sitting still. Resting tremor is often associated with conditions such as Parkinson's disease, where it is considered a hallmark symptom.
Parkinsonian tremor	The most prevalent type of resting tremor. In Parkinson's disease, the tremor typically manifests when the affected body part is at rest and diminishes during voluntary movement. Initially, the tremor is observed in the distal upper extremity and gradually progresses proximally. Subsequently, it may also affect the opposite upper extremity, following a pattern from distal to proximal.

Named Syndromes

Table 9.3. Named Syndromes. Named syndromes refer to specific sets of signs and symptoms that occur together and are recognised as distinct clinical entities.

Capgras syndrome	A form of delusional misidentification in which the individual falsely believes that familiar people such as their loved ones have been replaced by identical-looking strangers. The person therefore regards them as impostors. It occurs rarely in schizophrenia or psychotic mood disorders. The syndrome was named after the psychiatrist Jean Marie Joseph Capgras, who first described it in 1923.
Charles Bonnet syndrome	Characterised by recurring and detailed visual hallucinations, commonly depicting animals or people in various settings. These hallucinations occur in elderly individuals who maintain clear consciousness and possess intact insight and cognitive abilities. The syndrome may have an organic basis, often associated with visual impairments. It is named after the Swiss naturalist and philosopher Charles Bonnet.
Cotard's syndrome	Individuals usually suffering from psychotic depression have nihilistic and/or hypochondriacal delusions and firmly believe that their bodies, or even reality itself, have disintegrated or no longer exist. The person often claims they are already dead, and they are rotting internally. Usually seen in elderly people. It was first reported by Jules Cotard in 1880.
Couvade syndrome	The occurrence of abdominal pain or other non-specific physical symptoms in male partners of pregnant women such as nausea, vomiting and food cravings. It typically manifests during the third and ninth months of pregnancy, and the man does not actually believe himself to be pregnant. It is a conversion symptom, not a delusion.
Pseudocyesis	A false pregnancy in which a person, most commonly, a woman, exhibits the typical symptoms and sometimes signs of pregnancy, e.g. amenorrhea, abdominal distension, and breast enlargement, although conception has not occurred. It can be psychogenic in some cases, or it can be attributed to medical conditions like tumours or endocrine disorders.
Da Costa's syndrome (effort syndrome or	A somatisation disorder involving the cardiovascular system. Initially observed in soldiers during the American Civil War, it is now characterised as panic disorder that features symptoms of fatigue, heart palpitations, chest pain, and

soldier's heart)	difficulty breathing. It is named after the U.S. surgeon Jacob Méndes Da Costa.
De Clérambault's syndrome (Erotomania)	A delusional belief in which an individual is convinced that someone of higher status and therefore inaccessible to them is deeply in love with them. It is important to note that the syndrome does not necessarily involve a desire for a sexual relationship with that person. Named after Gaëtan Gatian de Clérambault, a French Psychiatrist, artist and photographer.
Dhat syndrome	The belief that passing semen in the urine causes a non-specific malaise, often attributed to excessive sexual activity. It is culturally influenced and may share similarities with other post-orgasmic disorders such as post-coital tristesse, post-orgasmic illness syndrome, and sexual headache.
Post-coital tristesse, or "post-sex blues"	A phenomenon characterised by feelings of sadness, melancholy, or even crying shortly after sexual activity, typically following orgasm. This emotional response can occur despite positive or satisfying sexual experiences.
Diogenes syndrome (senile squalor syndrome)	Extreme self-neglect, domestic squalor, social withdrawal, apathy, hoarding of useless objects or animals, and a lack of shame. May be caused by an organic condition and is seen in schizophrenia, depression, OCD and may occur in response to stress in predisposed personality. The condition was named after Diogenes of Sinope, an ancient Greek philosopher known for his minimalist lifestyle, which contradicts the hoarding behaviour seen in this syndrome.
Ekbom syndrome (delusional parasitosis)	A hypochondriacal monosymptomatic delusional disorder characterised by a strong belief of being infested by parasites crawling under, in, or on the skin. It can manifest as delusions, tactile hallucinations, or over-valued ideas. It is important to note that Ekbom's syndrome can also refer to restless legs syndrome, a separate condition involving discomfort and fidgeting in the legs, distinct from akathisia. It was named after Karl-Axel Ekbom (1907-1977), a Swedish neurologist mostly known for his detailed description of restless legs syndrome.
Fregoli syndrome	A form of delusional misidentification in which the individual mistakenly identifies strangers as familiar people, in contrast to Capgras syndrome . The person believes that a persecutor can change their appearance and identifies them in different individuals they know, such as neighbours, doctors, or attendants. It was first identified in 1927 and named after

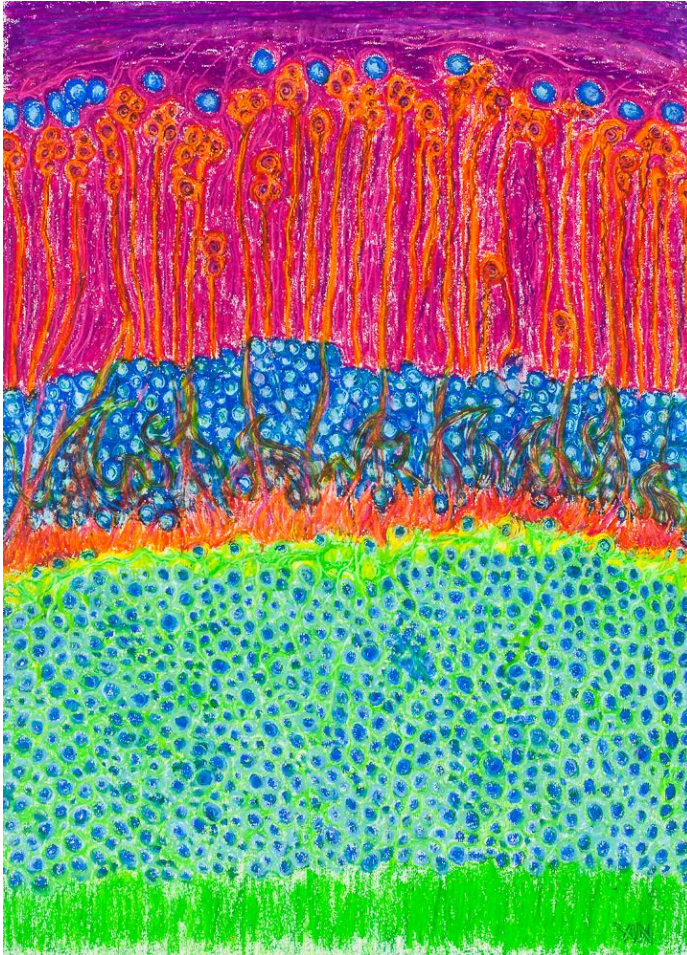
	Italian actor Leopoldo Fregoli, known for his skill in altering his appearance.
Ganser syndrome	A rare dissociative syndrome characterised by nonsensical or incorrect answers to questions, along with other dissociative symptoms such as a fugue, amnesia, or conversion disorder. Typically, the person gives “appropriate answers to questions, which are incorrect but related. It is sometimes seen in prison inmates and may be perceived as an attempt to gain leniency. It was first identified by Sigbert Ganser, who described impaired consciousness and distorted communication, particularly in the form of approximate answers. This syndrome is often classified as a dissociative disorder rather than malingering.
Gilles de la Tourette’s syndrome (GTS)	A childhood-onset neuropsychiatric movement disorder characterised by multiple motor tics and one or more vocal/phonic tics lasting more than a year. Tics are rapid, repetitive, uncontrolled movements or vocalisations that can be temporarily suppressed but reappear during periods of inactivity or relaxation. The syndrome is sometimes associated with inappropriate vocalisation or socially embarrassing actions.
Koro (also known as shrinking penis or genital retraction syndrome)	A culture-bound delusional disorder where individuals strongly believe that their sex organs are retracting and will disappear, despite no actual physical changes. It causes immense anxiety and likely arises because of poor understanding of anatomy. The term “koro” comes from Malay and refers to the resemblance of a turtle retracting its head into its shell.
Munchausen syndrome	Individuals deliberately fake or induce symptoms of illness in themselves and the syndrome is best regarded as a variant of pathological lying. Named after Baron Munchausen, known for his exaggerated tales, people with this syndrome lie about their symptoms, manipulate test results, self-inflict harm, and seek medical attention from multiple healthcare providers. Unlike hypochondriasis (genuine belief in being ill) or malingering (feigning illness for personal gain), individuals with Munchausen’s know they are fabricating symptoms and engage in manipulative behaviour without obvious benefits, often undergoing unnecessary medical procedures.
Munchausen by proxy	An individual, typically a caregiver or parent, deliberately induces or falsely reports symptoms in a dependent, such as a

	child. The person undertaking this behaviour aims to draw attention to themselves, and their actions often lead to unnecessary medical interventions for the dependent. It is a form of abuse that requires recognition and intervention to safeguard the well-being of the dependent individual.
Othello syndrome (delusional jealousy, morbid jealousy)	A form of paranoid delusional jealousy where an individual firmly believes, without evidence, that their partner is being unfaithful. It leads to obsessive thoughts and preoccupation with the imagined infidelity, causing significant distress and relationship difficulties. The term “Othello Syndrome” originates from Shakespeare’s play Othello, where the character mistakenly believes his wife is unfaithful and tragically kills her. However, there is ongoing debate as to whether Othello was genuinely deluded or deceived about his wife’s infidelity, questioning his representation of the syndrome.

Further Reading

- Casey, P., Casey, P. R., & Kelly, B. (2019). *Fish's clinical psychopathology: signs and symptoms in psychiatry*. Cambridge University Press.
- Oyeboode, F. (2022). *Sims' Symptoms in the Mind: Textbook of Descriptive Psychopathology E-Book: Sims' Symptoms in the Mind: Textbook of Descriptive Psychopathology E-Book*. Elsevier Health Sciences.

RESEARCH AND THEORETICAL MODELS



Artist: Anne Naylor
Title: *Landscape of the Brain*
Medium: Oil Pastel on Paper
Dimensions: 550 x 400 mm

Chapter 10: Phenomenology - A Philosophical Perspective

Danielle Winch

Specialty of Psychiatry, The University of Sydney, Australia.

“It has often been emphasised that psychiatry is still an ‘expertise’ and has not yet reached the status of a science . . . there is no sharp line of demarcation between the art and science of psychiatry. Science is continually extending into the field of clinical art but the latter always remains indispensable in its own right”

Jaspers (1963)

Introduction

It has been more than a century since Jaspers lamented psychiatry’s thwarted attempt to reach the scientific empiricism that governs the rest of medicine. In that time, scientific methods of inquiry have failed to yield robust disease models for most mental disorders. A model of disease requires that aetiology and mechanism be established, and it is from that foundation that we develop diagnostic tests and disease-modifying treatments. Psychiatry has almost no models of disease or diagnostic tests, making diagnosis a more challenging and subjective process. Nevertheless, psychiatrists must still assist those with mental disorder and find ways to ease distress.

When no disease model exists, medicine relies on syndromes to make a diagnosis. Diagnosis in psychiatry is therefore almost exclusively syndromic. A syndrome is defined as “a constellation of symptoms that are unique as a group” (Casey and Kelly, 2007: 1). When the aetiology of a syndrome is discovered it can be called a disease (Casey and Kelly, 2007). Diseases can be reproduced and proven in accordance with the laws of science, which seek to explain by establishing causal connections. Once explained, diseases can be classified within medicine (nosology). Without causal connections, it is not possible for us to explain psychiatric syndromes. When explanation is not possible, the goal of inquiry must be to *understand* by establishing meaning (Jaspers, 1963). If the goal is different, so must the methodology, and so psychiatry has different methods of inquiry to elicit signs and symptoms and make meaning from them. Phenomenology is one such method

of inquiry. It is a method adapted from a branch of philosophy. In trying to understand the nature of the human mind, psychiatry has historically drawn on philosophy as often as science as a way of constructing meaning. Phenomenology seeks to explore and describe the lived experience of patients, in order to understand what may not be readily explainable. In this chapter we will look carefully at phenomenology as a key method of inquiry in psychiatry and how it relates to the study of psychopathology.

Phenomenology as a Philosophy

Put simply, phenomenology is the philosophy of experience. It values lived experience as a tool for interrogating the nature and meaning of existence. It rejects the notion that theories or beliefs are required for the construction of meaning. Instead, it positions itself as a process; a way of doing philosophy (Broome et al., 2013: 1). The summary provided in this chapter is an intentional oversimplification of complex ideas, designed to ensure ease of accessibility for those unfamiliar with the concepts and branches of philosophy.

The structures of experience in phenomenology are called phenomena and can exist in the external environment or the inner world. The modern founder of phenomenology, Edmund Husserl, sought to describe these structures in a way that avoids prejudice and sets aside (“brackets”) prior knowledge/beliefs, focusing on the things themselves (de Monticelli, 2019). The goal of engaging with phenomena experientially, rather than analytically, is to access the essential qualities or “essence” of the phenomenon (*eidōs*) (Broome et al., 2013). This is a form of knowledge referred to as *a priori*, meaning that prior knowledge of the phenomenon is not required for its essence to be intuited. For example, to experience the essence of the colour green – to know what “green-ness” is – one does not need to know that it is a blend of blue and yellow. One needs only to have had the experience of seeing the colour green. Through that experience one can both recognise greenness and imagine an infinite “horizon” of variations in the colour green by extrapolating its greenness (Broome et al., 2013).

Martin Heidegger disputed Husserl’s idea that all knowledge can be suspended during (or “bracketed” out of) encounters with phenomena because sometimes the essence of a phenomenon is socially constructed. Such phenomena are experienced as a codified pattern in which the interpretation forms part of the experiential encounter (Broome et al., 2013). For example, when I walk into my clinic room I immediately take in the image of the desk and know it to be the place designated for me to sit and complete my work. I cannot put that interpretation to the side and return to a pure experience of a wooden surface with four legs and some draws. This is also the case when one encounters phenomena that are intended to

convey meaning by their very existence, for example the orange light that blinks on the side of a car that is about to turn (Broome et al., 2013). Heidegger called such signalling phenomena “signs”. Noting this process of reflexive interpretation of phenomena as an essential part of a human being’s experience in the world, Heidegger began to think of human beings as inherently interpretive creatures, driven to make sense of their worlds through meaning. He felt that part of the inescapable drive of interpretation was the fundamentally affective nature of human experience. We give meaning to experience because we are affected by it (Rosfort, 2019). Heidegger’s work moved away from Husserl’s epistemological phenomenology (to understand the nature of knowledge) towards a more ontological phenomenology (to understand the nature of being in the world).

Several years before Heidegger, Max Scheler, wrote extensively about human beings as “signs” or signalling phenomena. For Scheler, the human being is a quintessentially emotional entity with the capability to imagine or imbibe the mental lives of others. Scheler also identified emotion as core to the nature of human beings as phenomena, such that they cannot be reduced to bland concrete parts without their essence being lost (Broome et al., 2013). For example, it is not possible for me to see my companion’s eyes without seeing that they are looking at me and without seeing the emotion conveyed in the gaze. The sense-data in the human body allow perception of emotional states. A sudden blush in the cheeks conveys shame, a frown on the face, sadness (Frings and Scheler, 2012; Broome et al., 2013). These stigmata are part of the emotions they express and are the medium that allows communication of feelings between people (Dillard-Wright, 2007).

Scheler also delineated types of feeling experienced by human beings (Broome et al., 2013), including revisions of the hierarchy of feelings posited by Aristotle (Frings, 1974):

- Sensible feeling-states that related to an object: pain, smelling a scent
- Intentional feelings that relate to those feeling-states: suffer, endure, enjoy
- Vital feelings that relate to the life force of the being: weakness, vigour
- Spiritual-type feelings: despair, conscience, peace
- Feeling of the emotional characteristics of the atmosphere: serenity of a lake
- Feeling of values: agreeable, good

Given such detailed attention to emotions as constituent parts of human being and their communications with others, Scheler’s work is perhaps the most relevant to phenomenology as it exists within psychiatry.

Phenomenology in Psychiatry

The Object of Study in Psychiatry (Psychopathology)

Abnormal mental phenomena are the essential objects of study in psychiatry (Parnas et al., 2013). We are interested in how and why experiences might deviate from normal psychic experience. Our disorders, those of experience and expression, are organised under an umbrella of psychopathology. Psychopathology is the systematic study of abnormal experience, cognition and behaviour. A basic understanding of psychopathology is important because:

- It underpins our understanding of assessment and diagnosis in psychiatry.
- It forms the basis of our professional lexicon (how clinicians communicate).
- It is how we monitor treatment and progress.
- It underpins how we conduct research and construct diagnostic syndromes.

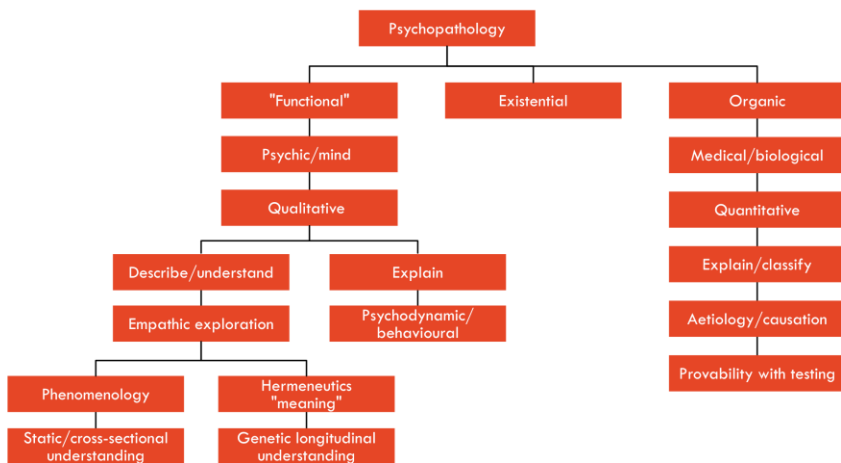


Figure 10.1. Organisation of psychopathology.

Phenomenology was appropriated by psychiatry to be deployed as a tool for studying psychopathology, particularly in the area of descriptive psychopathology, which seeks to describe and understand mental phenomena (Oyebode, 2023: 3). Mental phenomena could be any type of internal experience, including but not limited to, thoughts, affects, reactions, associations, perceptions.

Key Thinkers

Karl Jaspers

It is Karl Jaspers (1883-1969) who is credited with bringing the phenomenological method to the practice of psychiatry. Jaspers first described the dichotomy of understanding versus explaining as a way of conceptualising the divergence of psychiatry from the epistemology (knowledge) of the natural sciences. His introduction of the phenomenological method was his attempt to devise a mechanism for achieving understanding in psychiatric practice.

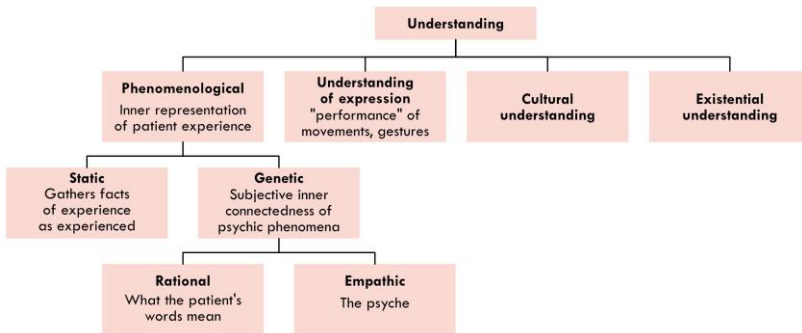
Jaspers was unapologetic about psychiatry's epistemological uniqueness within medicine. He believed there was human value in the subjective truth of lived experience. He rejected the idea that objective truths were more valid or worthy and saw the difference between objective and subjective knowledge as qualitative, not quantitative. It was therefore valuable to have a rigorous methodology for building subjective knowledge and developing shared understanding between psychiatrists and patients. Jaspers adapted Husserl's method to the practice of psychiatry and produced the first comprehensive description of mental disorders according to their essential qualities as phenomena (Jaspers, 1963). He identified three groups of phenomena accessible to the psychiatrist (Jaspers, 1968):

- Those known to us from our own lived experience.
- Those understood as exaggerations or diminutions of our lived experience.
- Those inaccessible by empathic understanding to which we can only get close through metaphor/analogy. Jaspers believed our perception of such phenomena arose through the “shock ... of the incomprehensible” (Jaspers, 1968: 1318)

Jaspers posited that up to four modes of understanding could be applied to the exploration of psychic phenomena (Figure 10.2). Cultural and existential types of understanding are outside the scope of this chapter. Neither will we discuss the understanding of expression in detail, as this will be discussed in sections on the mental state examination. Jaspers acknowledged the need for objective observation and description of the mechanical elements of behaviour - because behaviour expresses aspects of internal phenomena - but he prioritized direct exploration of the patient's inner world over the inferences made through behavioural observation (Parnas et al., 2013). We will therefore turn our attention to the phenomenological

modes of understanding that directly explore the psyche: static and genetic understanding.

Figure 10.2. Jaspers' organisation of understanding as described in General Psychopathology Volume One, parts 1 & 2.



Static understanding is cross-sectional. It describes and characterises a patient's internal experiences to create a landscape of the inner world at that time. The more precisely this is done and the more experiences that are examined, the more cohesive the representation becomes in the mind of the psychiatrist. Genetic understanding introduces a more analytic exploration of how one psychic experience arises from another. It requires empathic immersion into "the psychic situation" of the patient (Jaspers, 1963: 301) because the genesis and evolution of psychiatric symptoms does not proceed in the linear manner of the physical world (Dörr, 2013). Psychopathological processes can have an intrinsic logic/progression. It is important to understand how the patient makes meaning from connections, rather than impose our own meaning on them.

Eugène Minkowski

Eugène Minkowski's (1885-1972) method of phenomenological psychiatry runs somewhat antithetically to the medical model. He saw patients with mental disorder as "different" rather than "diseased" and believed that psychiatry failed to move from syndrome to disease because there was a whole living entity standing between the two (Broome et al., 2013). Minkowski's psychopathological focus was therefore the basic disturbances affecting human the being as a whole. He sought to examine how the structures of disorders influence the structures of the psyche. His method is often referred to as structural phenomenology for this reason.

In Minkowski's model of psychopathology, an individual is endowed with a "*morbid personality*" that intersects with something he calls the "*generating disorder*" (Broome et al., 2013; Urfer-Parnas, 2019). This intersection between personality

and disorder creates certain conditions from which phenomena (symptoms) materialise. Simply put, he posits that certain personality structures are prone to certain disorders.

Minkowski believed that the study of psychopathology should focus on establishing the underlying generating disorders, rather than on the grouping of symptoms. He did not believe that symptom clusters could ever be definitive in psychiatry because symptoms are only the patient's way of expressing their generating disorder and patients have only a few modes of expression at their disposal for conveying their lived experience. For example, somatic/hypochondriacal preoccupations can occur in schizophrenia and anxious depression but are phenomenologically different in each disorder. The generating disorder should be defined by its essence rather than variable expressions of it. In the case of schizophrenia, for example, the generating disorder is a loss of vital connection with reality (Urfer-Parnas, 2019). This intersects with the autistic manifestations of the patient's morbid personality to produce psychotic symptoms.

In this model, symptoms do not relate to brain functions but indicate modification of the structure of psychic life, which should be interpreted using a "*phenomenopsychopathological attitude*" (Broome et al., 2013). Minkowski believed that a hallucination should not be reduced to a disorder of perception, nor should a delusion be reduced to a disorder of judgement (lest judgement become nothing more than something that stops us having delusions). Instead, one should consider delusions a consequence of aberrant salience arising from a disorder in the "*natural flux of life*" (Broome et al., 2013). Aberrant salience occurs when a patient experiences a neutral environmental stimulus as unduly important. This experience is inexplicable and leads to questions that cannot be answered. The delusion creates an answer to the unanswerable question and removes the anxiety associated with the inexplicable. The role of the delusion in this case is to explain an inconsistency that threatens psychic unity. Psychic unity is essential for a cohesive experience of existence and is protected at all costs.

Ludwig Binswanger

Ludwig Binswanger (1881-1966) developed Heidegger's notion of *being-in-the-world* into a process of existential analysis through which one might appreciate the patient's world. Existential analysis examines the "*forms and configurations of existence*" (Broome et al., 2013: 117), or the nature of one's world, in so far as one exists within the world.

Existential analysis proceeds from the premise that there is a common or shared world, which can contentiously be called reality, and one's own subjective

personal world. Its use in psychiatry depends on the assumption that the world of the patient with mental illness differs from the collective experience of the shared world. Binswanger believed that inferences can be drawn about a patient's world-design or world-content from the language expression used to describe their lived experience. If we were to look at spatial and temporal domains of description, a temporal description of urgency and a spatial description of crowdedness might help us to understand the frantic and intrusive nature of patient's anxiety as it relates to perceived threat in their world (Broome et al., 2013).

Binswanger hypothesised that anxiety arose out of threat to the patient's world structure. He identified a relationship in which a patient was conditioned through their life experience to have a world structure with certain vulnerabilities to anxiety. Threats to those points of vulnerability then produce a reliably corresponding anxiety. A lack of food security growing up might cause a patient to hoard food, for example, and then a threat to the hoard activates the same anxiety that caused the hoarding in the first place. The core aspect of the experiential world in each case is a feeling of not having enough or being left empty.

Binswanger is quick to say that his method is not itself psychopathology, but a tool for communication between the world of illness and ours, which must then undergo psychopathological translation. One could also argue its value as a tool for experiential empathic understanding.

Kurt Schneider

Kurt Schneider (1887-1967) built on the descriptive classifications of Emil Kraepelin and Eugen Bleuler in their work distinguishing schizophrenia (*dementia praecox*) and affective psychoses. While Schneider valued the phenomenological attitude as a tool for research and understanding, his focus was establishing psychiatric nosology as a spectrum of “psychosyndromes” that could be classified even if they could not be defined as discrete entities (Jablensky, 2015).

Schneider was the first person to try and operationalise the work of psychopathologists into syndromes of characteristic symptoms. He is best known for creating a nosological classification for schizophrenia based on what he termed “first rank symptoms” (Jansson, 2019; Soares-Weiser et al., 2015). Schneider's first rank symptoms (Figure 10.3) formed the diagnostic criteria for schizophrenia in the International Classification of Diseases (ICD) until their omission in the most recent iteration (ICD-11). While first rank symptoms were thought to be pathognomonic for schizophrenia for decades, the prevailing contemporary understanding is that these symptoms occur not infrequently in other types of psychosis (Jansson, 2019) and cannot offer diagnostic fidelity.

Table 10. 1 Summary of Schneider’s first rank symptoms, adapted from Soares-Weiser et al. (2015).

Schneiderian First Rank Symptoms		
Auditory Hallucinations	Thought echo	Voices speaking thoughts aloud
	Running commentary	Voices commenting on every thought/action
	Third person	Multiple voices discussing the patient
Thought Manipulation	Thought insertion	Outside agent putting thoughts in the head
	Thought withdrawal	Outside agent removing thoughts from the head
	Thought broadcasting	Thoughts leaving the head and becoming common/shared property
Passivity	Motor passivity	Actions controlled by outside agent
	Affective/sensation passivity	Feelings controlled by outside agent
Delusional Perception		Sudden false belief about a true perceived stimulus

Hermeneutics

A phenomenological methodology cannot stand alone in the study of psychopathology without the capacity to interpret the phenomena. Hermeneutics is the philosophy of interpretation or “meaning making”. While it originally referred to the interpretation of texts, it is now applied more generally to language and communication. The history of hermeneutics is extensively intertwined with phenomenology given their mutual interest in human experience. Phenomenology deals with *how* we experience things, while hermeneutics deals with *what* we experience and *why* we experience things in the way we do (Rosfort, 2019). Hermeneutic phenomenology is a branch of philosophy that seeks to unite these elements to achieve a more holistic understanding of human experience.

When it comes to psychopathology and the practice of psychiatry, it is difficult to conceive of a phenomenological methodology that does not utilise hermeneutics. How can we make sense of mental suffering without understanding *what* a person experiences, *how* it manifests and *why* the suffering is occurring? While Heidegger brought hermeneutics to the phenomenological method in philosophy,

Jaspers was quick to identify meaning as a vital aspect of understanding in psychopathology, because every understanding is a form of interpretation (Dörr, 2013). In fact, Jaspers' notion of genetic understanding is driven by the meaning that connects internal experiences and ideas. The symptoms that comprise psychiatric syndromes are also linked through meaning, rather than through a demonstrated shared causality, so interpreting them should seek to achieve meaningful, rather than aetiological understanding (Rosfort, 2019). Understanding a delusion from within, as a series of faulty interpretations, has greater empathic value than a causal explanation of neurotransmitter dysfunction (Dörr, 2013).

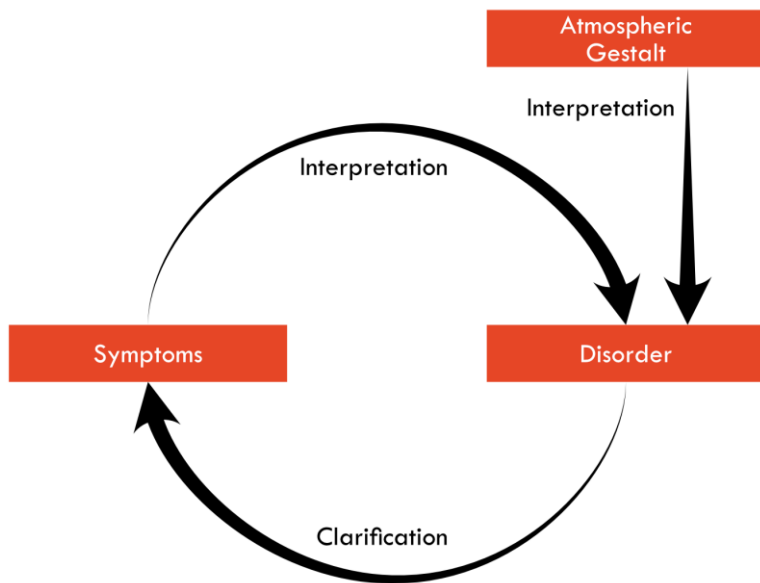


Figure 10.3. Hermeneutic circles in psychiatric assessment.

Phenomenology and hermeneutics work in tandem in all aspects of psychiatric assessment. While signs and symptoms may be observed as phenomena, they are described and discussed using language. The patient's description of their symptoms is an interpretation of their lived experience. The patient questions how best to meaningfully express themselves and the psychiatrist clarifies the patient's meaning in order to make their own interpretation of the patient's lived experience. The result is a hermeneutic co-construction of mental symptoms (Stanghellini and Aragona, 2016). Symptoms are interpreted by the psychiatrist with reference to a diagnostic paradigm, which can then direct further clarifying questions. This is a rough approximation of what is known as a "hermeneutic circle": an expanding cycle of reference between part and whole.

A psychiatrist adopting a phenomenological and hermeneutic attitude may become immediately attuned to an atmospheric gestalt in the patient's embodied presentation, which may invite an intuitive reference to a "whole" diagnosis before any discussion of symptoms ("parts"). For example, a patient presenting with a downcast gaze, a look of dysphoric consternation on the brow and cadaverization of the body invites an immediate interpretation of melancholia. The assessment then proceeds to clarify the truth of that interpretation by exploring its various "parts". While the psychiatrist is attuned to a gestalt in this case, the phenomenological experience of the encounter shares similarities with an eidetic quality (*eidos*).

Phenomenology in Clinical Practice

"Psychology and psychopathology really face a double task: that of knowing the person as the centre of his world and that of knowing the world of which he is the focal centre".

Spiegelberg (1975: 39)

One of the challenges of clinical practice in psychiatry is attempting to understand things that are not directly perceptible or accessible to us. We are trying to imagine phenomena that only the patient experiences directly. In many ways it is a phenomenology of vicarious experience (Spiegelberg, 1975). Accessing, exploring, and describing such phenomena accurately, and understanding lived experience is the true craft skill of the psychiatrist. While the data may be subjective, the experienced psychiatrist can achieve precision in their craft through a variety of tools.

Through repeated application of phenomenology to a multitude of clinical cases, one may hope to arrive at something analogous to the *eidos*, or phenomenological essences of a given disorder (Dörr, 2013). Much like the *eidos* of green can be experienced without the knowledge that it is a blend of blue and yellow, a clinician who is practiced in phenomenology could know the *eidos* of mental disorders, even without the knowledge of the diagnostic criteria. They can intuitively connect to the essence of melancholia, the evolution of delusion or the expansiveness of mania. We will now examine the application of phenomenological tools in the clinical practice of psychiatry.

Empathy

Empathy is not simply being kind and compassionate. Nor is it feeling the feelings of the patient (sympathy). It is the capacity to genuinely imagine someone else's experience, using your own cognitive and emotional experiences as a point of

reference. Empathy requires the psychiatrist to adopt the perspective of the patient and it has been suggested that a truly phenomenological approach to empathy might require transposal of the clinician into the place of the patient at that specific place and time (Spiegelberg, 1975).

Empathic precision requires cycles of clarification in which the psychiatrist's understanding of psychic experience is fed back to the patient to check accuracy and invite further exploration of discrepancy. True empathic exploration and understanding is therefore a cooperative encounter (Spiegelberg, 1975). Jaspers believed that a shared truth discovered through empathic clarification has greater capacity for achieving mutual understanding than simply informing a patient they have a particular illness.

Diagnosis

A lack of aetiological disease models doesn't obviate the need for a diagnostic classification in psychiatry. Psychiatric diagnoses currently exist as "typologies of human experience and behaviour" (Parnas and Zahavi, 2002: 137). For such typologies to have utility for research, treatment and prognosis, their classification would ideally have clearly delineated boundaries between disorders and between each disorder and "normal". Phenomenology attempts this through its pursuit of the essential qualities of a disorder. Jaspers felt this could be achieved through immersion in the behaviour and expression of the patient, questioning by the psychiatrist, and spontaneous accounts of experience by patients (Spiegelberg, 1975). Through our empathic connection with these data, we achieve an understanding of the lived experience of the patient. Elements of this lived experience are meaningfully connected to form a cohesive whole, or, organising Gestalt (Parnas and Zahavi, 2002: 157). The psychiatrist then subjects this Gestalt to a typification process (Parnas and Bovet, 2014: 205; Parnas and Zahavi, 2002: 156) that references the patient's experience against prototypic lived states or essential types. Here the psychiatrist asks to what extent the essential elements of the patient's experience resemble the essential elements of a given disorder. Psychiatry's methods of diagnosis have moved incrementally distant from this phenomenological ideal since their inception in the early Twentieth Century.

Operational Psychiatry

For most of this chapter, we have discussed very specific and technical definitions of phenomenology as it pertains to philosophy and psychiatry in the modern era. Unfortunately, over recent decades, the meaning of phenomenology in clinical practice has been eroded to a reductive and unrigorous definition approximating signs, symptoms, and underlying thoughts and feelings (Parnas and

Zahavi, 2002; Andreasen, 2007). Clinical assessment has suffered a similar decline in precision, as psychiatry has been progressively reduced to a set of symptom checklists. This process is referred to as the operationalisation of psychiatry (Parnas and Bovet, 2014; Parnas et al., 2013; Parnas and Zahavi, 2002) and some commentators have suggested that it has heralded the “death of phenomenology” (Andreasen, 2007). Operational diagnosis is now the standard methodology in psychiatry, and it stands in opposition to the phenomenological tradition. The benefits and products of operational psychiatry will be discussed in sections relating to classification. It is the role of this chapter to explain the shift in approach and make the case for phenomenology.

How Did It Happen?

Regional differences in the practice of psychiatry in the Twentieth Century created divergences in ethos between Europe and America. Europe continued its nosological pursuits, while the spread of psychoanalysis in America saw diagnosis largely abandoned as therapeutically insignificant (Andreasen, 2007). Uptake of ICD after its publication in 1956 was low and studies reporting overdiagnosis of schizophrenia in America began to appear (Kendell, 1975; Parnas et al., 2013; Andreasen, 2007). In addition, a controversial study was published in which participants feigned psychotic symptoms to gain psychiatric admission and were discharged (sometimes after weeks) with a diagnosis of schizophrenia, despite behaving in an entirely normal manner after admission (Rosenhan, 1973). Even though questions were raised about the integrity of the latter study decades later (Cahalan, 2020), at the time of its publication the ignominy of haphazard diagnostic practices became inescapable.

The (often accidental) discovery of effective treatments for psychiatric illness, which occurred around the same time, also renewed interest in nosology, especially given the persistent lack of aetiological models of disease. In America, this renewed interest led to the development and publications of a new diagnostic approach in the form of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III). DSM-III was ideologically conceived to create shared syndromic diagnoses that would foster research and clinical consistency.

The Problems of the DSM

Philosophical Problems

DSM intended to operationalise diagnosis in psychiatry. Psychiatry borrowed the term “operational” from physics but appropriated it in a way that arguably nullifies its scientific and epistemological meaning. In physics, an

operational definition defines something by the procedures through which it is determined (Parnas and Bovet, 2014). An approximate example within medicine would be the definition of intelligence as what is measured in an IQ-test. When it was proposed for use in psychiatry, it included the explicit assumption that our observation of the psychiatric object was the defining “operation”. The problem inherent in this proposal is that we are dealing with the experiential word of a whole person, which cannot be defined only through its observation by a psychiatrist. It takes psychiatry in a decidedly behaviourist direction, suggesting that we can understand psychiatric disorders simply through observation of behaviour, without reference to inner worlds (Parnas and Bovet, 2014: 196). It also removes vast domains of human experience that are not directly observable from diagnostic consideration (self-experience, identity, interpretation of experience). The only applicable definition of “operational” then becomes a rudimentary lay definition: “ready for use” (Parnas and Bovet, 2014: 200). In many ways, this is exactly what DSM has become: a ready-for-use system for counting observable signs and symptoms, using common-sense, lay language, at the lowest order of inference (Parnas and Bovet, 2014: 199). It empowers clinicians, even those with minimal training, to make diagnoses with naïve confidence, based on the presence or absence of symptoms from a designated list. This accessible, common-sensical and “atheoretical” position was intended as a noble endeavour to further research and diagnostic consistency and the problems I have mentioned were an unforeseen consequence (Andreasen, 2007).

Practical Problems

The first issue to mention with DSM is the sacrificing of validity for the sake of reliability (Andreasen, 2007; Parnas et al., 2013; Parnas and Zahavi, 2002; Parnas and Bovet, 2014). Reliability was felt to be paramount for research, training, and clinical communication at the time (Andreasen, 2007). Reliability is based on reproducibility, and this was achieved through the creation of standardised lists of symptoms, from which diagnosis is achieved by reaching a threshold quantity. The problem with this is the symptoms themselves don’t confer the essential meaning of the condition (Parnas and Bovet, 2014). If we take depression as our example, the diagnosis requires five symptoms from a list of nine, which can occur in hundreds of combinations. This raised numerous questions. How essential can any of these features be if they can be omitted from diagnosis? Which combination of five features better reflects what it is to be depressed? What does it mean if you only have four symptoms from the list but have symptoms that are not on the list? How many symptoms could you have from the list but still be experiencing a “normal” form of distress in response to life events? With many symptoms of depression corresponding to physiological features of the human stress response, how does depression separate from normal responses to stress? With so many questions raised,

it seems clear that a symptom-based method doesn't get us closer to the essence of what it is to be depressed.

The second issue to highlight is the progressive deskilling of psychiatrists in the study of psychopathology. DSM has become more of a textbook for training than seminal psychopathological texts and training in clinical assessment (Andreasen, 2007) has been largely reduced to a box-ticking exercise. The ability to explore psychic phenomena from within and the capacity to take in an atmospheric gestalt in an analytically attuned manner is diminishing. This leads on to the third problem with DSM: its dehumanizing effect on psychiatry (Andreasen, 2007). It places the disorder at the centre of assessment, rather than the patient. While this may be an unconscious process for the clinician, it is antithetical to the notion of patient-centred care that has become a central tenet of the practice of medicine. It is for all of the above reasons that there has been a resounding call for a return to phenomenology and descriptive psychopathology over the last two decades.

Further Reading

Berrios, G. E. (1996). *The history of mental symptoms: Descriptive psychopathology since the nineteenth century*. Cambridge University Press.

Chapter 11: Psychiatric Epidemiology

Brian O'Toole

Brain and Mind Centre, The University of Sydney, Australia, and Specialty of Psychiatry, The University of Sydney, Australia.

Introduction

According to the Dictionary of Epidemiology (Porta, 2014) the science of epidemiology is the study of the distribution and determinants of disease in populations. The word itself comes from the Greek words “epi”, which means *upon* and “demos”, which means *the people*. The term was first used by Homer but its medical meaning was conferred by Hippocrates (Martin and Martin-Granel, 2006). It invoked the old superstitious notion of the things that vengeful gods send to afflict the people, like fires, flood, famine, frogs, plagues, and pestilence. In the context of mental health, psychiatric epidemiology refers to psychiatric illnesses in humans and in a treatment context the term “clinical epidemiology” has been coined to refer to the study of the distribution and determinants of diseases encountered in the clinic, but the essential scientific underpinning of this variant differs only from population epidemiology in its focus; the scientific methodology remains the same although the techniques may vary with each research question. And the principles apply not only to noxious exposures: for example, risk and protective factors, such as having a positive and warm emotional environment while growing up, can ameliorate a good deal of later emergent psychiatric disorders.

Humans are possibly the most complex and least understood of scientific fields of enquiry, particularly our psyche. As we know now, the complexity of this field encompasses genes, upbringing, emotions, cognitions, personalities, and behaviour, and is an exciting and developing field in Australia. Epidemiology is devoted to uncovering the causal relations between *exposure* to something and some consequent *outcome*, for example cannabis consumption and psychotic disorder, or interpersonal domestic violence and depression. If a causal pathway from a particular exposure to a particular disorder can be shown, then this opens up the possibility of interventions that may prevent the disorder from occurring. Epidemiology has evolved from roots in sociology, psychology, statistics and medicine to develop

reliable methods to pursue an understanding of the human condition. Any practical scientific study of these things has to rely on data that come from smaller samples that, hopefully, represent their parent populations. And this is where the trouble starts. There are many, many threats to the validity of clinical epidemiological studies, and each has a bearing on the process of statistical inference; that is, the process of using statistics to make inferences about human or biological characteristics that apply beyond the sample of data results to hand. Statistical inference is the bedrock of epidemiological research.

The statistic that tells us whether we have a significant association between the exposure of interest and the outcome of interest rests on a set of assumptions that must stringently apply. These rest on only two fundamental features: the assumptions that are necessary about the population members (and hence the sample) and the assumptions that are necessary about the parameter (statistic) of interest. These twin features lead to the source of the three possible threats to causal inference. Before outlining these, it is important to understand how epidemiological studies are designed, because the analysis – and therefore the conclusion – depends on how the study is constructed.

Study Designs

The main goal of medical research is to explore the possible causal associations between an Exposure (**E**) of some kind of agent and the subsequent development of a Disease (**D**) of some kind. In the simplest case, we can think of two basic groups of people, in one of whom some people are Exposed to a causal agent and some people are not Exposed ($\bar{\mathbf{E}}$), and some who develop Disease (**D+ve**) and some who do not (**D-ve**).

A simple example would be a study where a group of people who have been diagnosed with major depressive disorder and a group of people who have not been so diagnosed are compared to see whether they were admitted to the emergency department (ED) with suspected suicidal attempt. In this simple scenario we can construct a 2x2 table that shows this relationship:

	D +ve	D -ve	<i>Total</i>
E	<i>a</i>	<i>b</i>	<i>a + b</i>
$\bar{\mathbf{E}}$	<i>c</i>	<i>d</i>	<i>c + d</i>
<i>Total</i>	<i>a + c</i>	<i>b + d</i>	<i>N = a+b+c+d</i>

E = Depressed, $\bar{\mathbf{E}}$ = Not depressed, **D +ve** = Admitted to ED, **D -ve** = Not admitted to ED. (Those who remember their Algebra from high school will

recognise that a , b , c , and d are the numbers of people in each cell of the table, and N is the total number of people in the study.)

There are not too many ways to design an epidemiological study. Life can be simple. The most obvious way to begin is to select a group of people who are already exposed to something and a group of people who are not exposed. Broadly speaking, this strategy of *selection based on exposure* gives a *Cohort Study* design. Under these circumstances, selection sweeps across the rows of the table above, where a total of $(a + b)$ people have been diagnosed with major depressive disorder and a total of $(c + d)$ people are not so diagnosed; $(a + c)$ are admitted to ED and $(b + d)$ have not been admitted. The numbers $(a + b)$ and $(c + d)$ are fixed by design, while the numbers $(a + c)$ and $(b + d)$ depend on the outcome of the study assessments.

Of course, it is sometimes feasible to conduct an *experiment*, where an experimenter actually *causes* the exposure. This is common in lab-based and therapeutic or drug evaluation studies. When the exposure is *randomly allocated* to the exposed and unexposed groups, this broadly defines a *Randomised Controlled Trial* (RCT). Randomisation is used to distribute any important errors randomly across the two groups in order to control for potential confounding. It also gives control of exposure to the experimenter. Of course, it is not always possible to conduct an RCT, perhaps because of legal or moral or practical considerations, in which case we have to resort to other types of studies to provide the best available evidence.

Difficulties that adhere to cohort studies are often because it is hard to assemble a comparable cohort, or perhaps because the disorder is rare and thus less likely to be detected in a cohort study (unless the study was huge). Another consideration is the location of the researcher – a clinical epidemiologist needs access to a clinic and clinicians (and clinicians need access to an epidemiologist and statistician!). It is understandable that much of medical research is carried out on patients in tertiary referral teaching hospitals, often with captive subjects/patients. This is often the case with diseases such as the variety of cancers that are individually rare (such as cervical or prostate or pancreatic) or people with schizophrenia because it would be tiring and fruitless to assemble a community-based cohort to try and detect them efficiently. In this instance, study designs can begin by selecting people (cases) who already have the disorder and then select a set of *control* people who do not. This strategy of *selection based on disease outcome* broadly defines a *Case-Control Study*. Under these circumstances selection begins from the top of the table and sweeps downwards, where a total of $(a + c)$ cases has been selected and a total of $(b + d)$ controls has been selected to compare with them. (Importantly, in this case, the marginal totals $(a + b)$ and $(c + d)$ have no meaning.)

Another strategy for conducting epidemiological studies that does not rely on selection by exposure or selection by disease, is simply to select subjects based on some other characteristic. An example of this strategy design would be a study that took all consecutive patients admitted to Westmead Hospital and then determine how many of them had the exposure of interest (e.g., how many were victims of interpersonal violence) and how many developed the outcome of interest (e.g., major depressive illness). This general strategy broadly defines a *Cross-Sectional Study*, and is the method commonly used for community and social surveys. Cross sectional studies are very useful in determining the extent of a particular problem, for example the Australian Bureau of Statistics' National Surveys of Mental Health and Wellbeing, or the WHO World Mental Health Surveys that describe the prevalence in the community of a range of non-psychotic mental health diagnoses.

Finally, some studies in clinical epidemiology restrict themselves to studies of cases only; in this situation, *selection is based on disease outcome* but no control group enters the picture. This design is only powerful if the exposure and the outcome are so clearly related that anybody could see them (for example, prescribing the drug thalidomide for morning sickness and seeing the incidence of birth defects).

This simple explication of available study designs reveals only a few possible designs, but the world is more complicated than that since there are many other more subtle types of designs that expand this rubric (Catts et al., 2010). Moreover, hybrid designs can be used, for example a longitudinal study of cases with a comparison control group drawn from the community if, when followed up over time, would constitute a *case-cohort* study, incorporating features of both case-control and cohort studies.

In the accepted wisdom that prevails in Epi-Land, study designs can be ranked based on the quality of the scientific evidence; for example, the Cochrane Collaboration limits its attention to reviews of RCTs as the only reliable solid evidence acceptable to the Evidence-Based Medicine devotees. This is, of course, controversial. Each of the possible study designs are useful for different research questions. A case series is useful to define a particular disease and may involve qualitative analysis to discern the essential features of a particular disorder and discriminate it from other variants or subtypes. These studies are the means of differentiating diagnoses, where, for example, schizophrenia can be distinguishable from schizophreniform disorder, anxiety can be distinguished from depression in spite of symptom overlap, and Bipolar I can be distinguished from Bipolar II. Once the disorder has been defined, cross-sectional studies are useful to estimate the size of the problem. For example, the regular National Surveys of Mental Health and Wellbeing (NSMHWB) that are conducted by the Australian Bureau of Statistics are there precisely to determine the prevalence of psychiatric disorders in the Australian

population. But these do not provide a basis for analysis of causes or risk factors, except in a limited descriptive sense. Case-control studies are very useful for assessing the risk factors for disorders, particularly disorders that have low prevalence in population studies like the NSMHWB. A prime example of this are the psychotic disorders, since a very large population sample would be needed to provide a large enough sample to give accurate estimates for such low prevalence disorders (O'Toole, 2000).

Cohort studies permit the examination of outcomes over time, for example the effectiveness of treatment of first episode psychosis, where people who present to hospital are followed to determine the proportion whose condition improves over time. Some cohort studies can continue for many years, such as the famous Dunedin Study (Koenen et al., 2007) that enrolled all children born in a one-year period 1972-73 in the New Zealand city of Dunedin and followed them for more than 36 years – with an enviable 96% response rate!

Finally, experimental studies such as randomised controlled trials are useful for evaluating treatments that are randomly allocated to individuals to compare treated and untreated individuals. While it is often cited that RCTs provide the best evidence – for treatments – they cannot be used where, for example, the exposure itself cannot be administered at random, or indeed at all, in situations where the exposure is toxic (think child sexual abuse, combat trauma). A well-conducted cohort study has the potential to provide excellent evidence, as does a well-conducted case-control study. But the physical abnormalities associated with Thalidomide during pregnancy were discovered from a case series. And Semmelweis discovered the cause of puerperal fever in childbirth by washing his hands a few times and not washing his hands a few times and noting the death rate! His colleagues treated him like a germ despite the fact that he had just put his finger on a prime mechanism of disease transmission. But remember, there has never been an RCT reported of the effectiveness of parachutes (Smith and Pell, 2003).

Incidence and Prevalence

All illness starts with an event, like the lighting of a fuse to a bomb. The event might be the experience of interpersonal violence, or it could be just being conceived (in cases such as genetic disorders). At some point in time, the individual is disease-free, but after that time they are not (i.e., they have been struck down with or acquired the illness or condition). When cases appear for the first time, they are called *incident* cases. For example, the number of new cases of severe depression presenting at Emergency Department in a specified time period would constitute a class of incident cases of depression. The concept behind incidence is that incident cases are *new* cases of disease. In epidemiologic studies it is often preferable to study

incident cases because there is no other factor such as prior treatment or diagnoses to complicate matters.

Prevalence, on the other hand, refers to the number of existing cases of a particular disease at a particular point in time. For example, the number of patients who are known to have anxiety disorder in New South Wales in June 2009 would constitute a prevalence. A special example is *lifetime prevalences* (also described by epidemiologists as cumulative incidence), which is the number of people who experience the disease over their lifetime up to the present. Psychiatric epidemiological assessment routinely records the onset and recency of symptoms to give prevalence over a lifetime (or cumulative incidence), one-year prevalence, six-and- one month prevalence, and so on. The important feature of the term prevalence is that it designates a state of nature at a particular point in time and place. In contrast, incidence is a change in the state of nature (Miettinen, 2007).

Errors in Studies

There are only three fundamental errors that can occur when using samples to draw inferences about larger populations: one can either study a biased set of subjects, or use measures that are biased or inaccurate, or something else creeps in to bias the observed relationships. These errors are termed selection errors, measurement errors, and confounding errors. Although there are only three fundamental types of errors there are many, many ways to make them.

The first error that can vitiate the results of an otherwise well-conducted study, is the issue of the unit on which the data are based. Assembling a set of subjects that do not allow a fair test of the hypothesis or theory that a researcher is trying to grapple with will lead to *selection errors*. Using only people who come to Emergency Department with first episode psychosis will not guarantee a random sample of people with psychosis. A bias is automatically introduced if there are discrepancies between the population of people that you want to make inferences about and the sample of people that you actually collect data from. Every study needs to ask fundamental questions: *Whom do I want to talk about?* That is, in statistical parlance, *what is the unit of observation?* And, as well, there is the critical supplementary question: *How will I gather a suitable sample of them to study?*

The Process of Subject Selection

Conceptually, selection begins with specification of the people you want to draw inferences about; they are conceived in broad terms like “doctors”, “women”, “people with major depressive illness”. At this level, they are termed the *reference population*. Variation away from the reference population results in a threat to the

external validity of the study, where the results of the study may not be applicable (generalisable) to the parent population that the researchers intended it to be. For example, a survey of AMA members cannot be representative of the total population of doctors simply because not all doctors belong to the AMA, so that findings from a survey of AMA members might not be applicable to – i.e. generalisable to – all doctors.

Next, the study must then proceed to determine who is available and eligible for selection. This requires the construction of the *sample frame*. It sets the eligibility criteria for inclusion and exclusion. Examples of sample frames could be things like a register of GPs in the Nepean Division of General Practice, or patients at Orygen in Melbourne, or patients in specific psychiatric wards. Sample frames are tools that allow study subjects or units of observation to be accessed and selected for study and are a practical way of determining who is eligible for study; it is critical that they be representative of the reference population, otherwise bias creeps in.

Once the sample frame has been identified and constructed there has to be a mechanism for selection. Strictly speaking, to be able to use inferential statistics the sample must be selected as a *probability sample*, where the probability of selection is known. Just taking any higgledy-piggledy convenience sample that was assembled and close by (e.g., captive patients in a ward) and expecting to apply the results to a general population is sadly misguided. It may be appropriate if you only want to confine your conclusions to the ward but not if you want to go beyond and generalise to other people. The determination of who is actually selected and with what probability is a critical element in the chain of decisions from study conception to publication of scientifically sound findings. Probability sampling requires that each potential individual for study has a known and non-zero chance of selection. Non-probability samples include haphazard methods like volunteering, purposive sampling, internet surveys, and general convenience samples. All of these other methods have no probability basis and therefore confer no validity in statistical analysis. Beware.

Once the sample frame and the sampling scheme have been determined, the last step is to ensure study subjects respond. In human studies, subjects can be lost to follow-up by, for example, dropping out of studies or failing to comply with medication regimens after selection and data collection. Examples abound where, for instance, people who have depression in the first wave of a longitudinal study fail to respond in a second wave, or even fail to respond in a first wave. In all types of study, it should be necessary to follow and document the path from the identification and selection of study subjects through to the actual gathering of complete data from them. Any deviation in representativeness of subjects from the reference population along this path will inevitably introduce errors and consequent bias when inferences

are drawn from the data analysis. The Australian Bureau of Statistics and the World Health Organisation do not use non-probability methods and consequently their data is much more accurate. In the face of non-response from the selected subjects, sometimes it is possible to quantify and adjust for the differences between the selected and responding subjects (Lynch et al., 1993; O'Toole et al., 1996) in cases where information is available on the whole sample.

Measurement Errors

It is axiomatic that every measure taken on an individual carries with it an element of uncertainty because classification is never perfect. Thus, the second error that can creep into epidemiological studies is that of *measurement error*. The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM), now in its fifth edition (published in 2013 with a text revision in 2022), was developed to standardise psychiatric diagnostic criteria across the United States (and the world), alongside the World Health Organisation's International Classification of Disease (ICD), now in its 11th edition. Unfortunately, DSM and ICD do not always agree on their definitions; while DSM-III introduced the diagnosis of posttraumatic stress disorder (PTSD) in 1980 it took ICD a further 12 years to add it to their lexicon, and the diagnostic criteria for PTSD have changed five times (Spitzer et al., 2007).

Since the 1980s clinician-scientists have developed standardised interviews for assessing population mental health; such interviews as the Diagnostic Interview Schedule (DIS) (Robins, 1981), the Structured Clinical Interview for DSM (SCID) (Spitzer et al 1987) now in its 5th edition, and the World Health Organisation's Composite International Diagnostic Interview (CIDI) (WHO 1997) have been developed to provide standardised diagnoses in epidemiological studies; the CIDI in particular has been developed as a computerised interview that can be administered by lay interviewers and indeed by computer assisted self-completion without a clinician or interviewer present. The CIDI algorithms yield diagnoses that are mapped both onto DSM and ICD systems and are the bedrock of the WHO World Mental Health Surveys. Further developments have led to a plethora of standardised instruments that focus on specific disorders, such as alcohol use disorders, sleep disorders (Taylor et al., 2018), PTSD (Weathers et al., 2018), and many others. If psychiatric studies use these standardised methods, then this directly permits comparison with other studies. In Australia, using the same CIDI that the ABS used in the National Survey of Mental Health and Wellbeing has enabled studies comparing the relative prevalence of disorders in peoples as diverse as Australian Vietnam veterans (O'Toole et al., 2009) and Aboriginal and Torres Strait Islander Australians (Nasir et al., 2018).

Errors in the diagnostic rubric and changes in diagnostic measurement can be disastrous in clinical studies. Accurate diagnosis is essential for studies that seek to find risk factors or markers for disease, for example, genomic studies of schizophrenia must rely on accurate diagnosis. Unfortunately, medical diagnosis is rarely 100% accurate, so errors in diagnostic status have to be accepted as part of the territory for a clinical epidemiologist (and statistician). In some cases, these errors can be specific to the population of study subjects assembled where, for example, cultural or language features may lead to misclassification of study subjects. For example, application of diagnostic and symptom criteria developed for white, western nations, may misclassify Aboriginal and Torres Strait Islander Australians at-risk for psychosis (Hamilton, 2008).

Confounding

The third error that operates to cast doubt on research findings is that of *confounding*, from the Latin *confundere*, to mix together. Literally, when this is operating, the statistical results that have been observed could be accounted for by some other, perhaps undetected or unmeasured variable or relationship. There is an example in a study of cancer and schizophrenia. It is well known that a diagnosis of schizophrenia increases the risk for a number of physical ailments, such as obesity (with its accompanying increased cancer and mortality risk), poorer access to medical care, increased risk of suicide, and lower life expectancy. However, studies of cancer incidence in schizophrenia have been highly various, with some studies (Lichtermann et al., 2001) showing increased rates of cancer and others (Dalton et al., 2005) showing no increased risk and others still (Grinshpoon et al., 2005) showing a reduced risk. Given the increased risk factor load, such as very high smoking rates, the discrepancy between observed and expected cancer rates was acknowledged as an “epidemiological puzzle” (Jablensky and Lawrence, 2001). However, many previous studies comparing people with schizophrenia with the general population had failed to account for a prime risk factor for cancer: smoking. In a highly cited meta-analysis (Catts et al., 2008) of studies that used population register-based studies where the incidence of cancer in patients and their first-degree relatives was reported, when background population rates of smoking were adjusted for it was found that schizophrenia carried with it a *reduced* risk of cancer, not only in the primary sufferers but also in their first degree relatives. This has implications for genetic theories of cancer vulnerability in people with schizophrenia. It appears that smoking may have been an unacknowledged and unmeasured confounder of the relationship between schizophrenia and the incidence of cancer in previous studies.

Confounding occurs where a variable that is associated with exposure groups is also associated with the outcome. Unfortunately, confounding is not detectable using statistical significance testing, or by obtaining ever larger samples.

This is because statistical significance is a phenomenon determined by sample size - the larger the n the more likely there is to be a significant result – but confounding is a *state of nature*, a property of the subjects assembled for study, and independent of sample size. In strictly statistical terms, confounding is the detection of interaction among study variables, and is assessed in terms of multicollinearity, that is, the extent to which two variables share statistical variance.

Identification of potential confounders is totally dependent upon the state of knowledge at the time. Confounding appears wherever it is not wanted and may be mistaken for something else, such as an effect modifier, like where age modifies the prevalence of dementia. It may also be evidence of a variable that is on the causal pathway between an exposure and a disease, such as a poorer environment while growing up. From a statistical viewpoint, a significant interaction effect between an exposure variable, a potentially confounding third variable, and an outcome would be evidence of any of these situations: the statistics cannot discriminate. This puts the onus firmly on the researcher to interpret the finding in light of the known and available evidence: the statistic itself is neutral. The history of the advancement of knowledge in epidemiology is isomorphic with the discovery, recognition and control of confounding in causal inference. And it is comforting to know that there are only three kinds of errors or biases that can creep into clinical epidemiological studies.

Critically Judging Research

What follows in Table 11.1 is a guide to appraising studies that is the culmination of the information contained above. Using this guide will help to appraise research papers and research studies – maybe even your own – in terms of the underlying basis of the statistical inferences that are used to support (or demolish) causal inferences drawn from research. It's a complex business. But it is the framework used by journal editors and scientific peer reviewers. And commended to those who want to ensure their practice is on safe grounds.

Table 11.1. Critical Appraisal in Clinical Epidemiology

Critical Question	Is There a Problem?	Does it Threaten Validity?
1. What is the research question?	Is it the impact of an intervention, a question of causality, or the magnitude of a problem?	Is it really worth doing?
2. What is the study type?	Is the type of study appropriate to the research question?	If not, how useful are the results going to be?
3. What are the outcome factors (dependent variables) and how are they measured?	Are all relevant outcomes assessed? Is there measurement error? Or differential measurement error?	How important are the omitted outcomes? Is differential or non-differential measurement error an important source of bias?
4. What are the study factors (exposures, or independent variables) and how are they measured?	Is there measurement error? Or differential measurement error?	What effect does measurement error have on the outcome of the study?
5. What is the reference population? The source population? The actual sample? Where did they come from?	Are there selection errors? Was the sample selected as a probability sample? What is the response rate? Is drop-out a problem?	Does this threaten the generalisability (external validity) of the study?
6. In an experiment, how were subjects assigned to treatment? In a follow up study, how many were lost to follow up?	Is this explicit? Were any adjustments made for potential response bias?	Does this threaten the internal validity of the study?
7. What important confounding variables were considered?	Are potential confounders identified and controlled for?	Is confounding an important source of bias?
8. Are statistical tests used?	Are they appropriate to the level of measurement and the number of tests used?	Is it reasonable that significant results have not been obtained by chance?
9. Are confidence intervals given for all major outcomes?	Are they appropriate to the level of measurement and the number of tests used?	Are the results clinically significant?
10. If the results are negative, is a power analysis available?	Was the sample size sufficient to detect a clinically significant result?	Is the study useful, or is the result inconclusive?
11. What conclusions did the author (or you) draw about the research question? Are new hypotheses generated from the study?		Do you believe them? If so, why? If not, why not?

Further Reading

Das-Munshi, J., Ford, T., & Hotopf, M. (2020). Psychiatric epidemiology: Looking to the future. *Practical Psychiatric Epidemiology*, 425.

Lash, T.L., VanderWeele, T.J., Haneouse S., & Rothman, K. (2021) *Modern Epidemiology*, Wolters Kluwer Health.

Chapter 12: Research Methods in Psychiatry

Rebecca Koncz

Specialty of Psychiatry, The University of Sydney, Australia, and Sydney Local Health District Mental Health Services, Australia.

and

Nerissa Soh

Specialty of Psychiatry, The University of Sydney, Australia.

Introduction

It is an unfortunate falsehood held by some doctors in training that education in basic research methods may serve little more than a pathway to complete your (potentially tedious) “evidence-based medicine” tasks. To the contrary, research is a fascinating and rewarding aspect of psychiatry that enables us to think outside of the box about complex and interesting diagnostic and management conundrums. If understood as intended, the principles of basic research methodology provide an important foundation for interpreting and delivering sound clinical care to your patients well into the twilight of your medical, psychiatric, or (dare we say) surgical careers.

Moreover, questionable research practices unfortunately run rife throughout the scientific community. Whilst controversial, it is estimated that up to one-third of scientists admit to questionable research practices, and this may well be an underestimate (Fanelli, 2009). Questionable research practices vary in nature, from examples such as selective reporting of results and overstating their implications (Gerrits et al., 2019), to less common although more serious scientific misconduct, which includes fabrication, falsification or plagiarism (Bauchner et al., 2018). Therefore, it is the responsibility of us as clinicians and researchers to uphold rigorous ethical and practical standards when interpreting and conducting research to ensure we do not fall prey to such malpractice.

This chapter will endeavour to provide a brief, but practical, overview of the principles of research methodology used in psychiatry. This includes an introduction to study types, common terminology, as well as basic statistical methods as a foundation to interpreting study results and applying them to your patients.

Study Types

When reading the abstract of a paper, begin by asking yourself “What type of study is this?”

Research studies may broadly be considered qualitative, quantitative, or a mixed method approach – that is, a combination of the two. The most common qualitative methods include participant observation (typically in their natural environment), in-depth or semi-structured interviews and focus groups. These types of studies offer insights into one’s experience, perceptions and social concepts (Hammarberg et al., 2016) by describing, interpreting and developing theories from observations as they occur in natural, and not experimental, conditions. Although there is some debate as to the methodological standards to be expected of qualitative research, the current recommendations are outlined in the “Standards for Reporting Qualitative Research” (O’Brien et al., 2014).

Quantitative methods, on the other hand, typically enable a researcher to test a particular hypothesis using numerical or pre-defined categorical data. Quantitative methods describe, explain, predict, or control variables of interest. These studies may be observational – that is, without specific intervention from the researcher – or experimental (which may also be referred to as interventional). Key features to look for when reading a peer-reviewed study, whatever the study type, include:

1. Is there a clear description of the research question, aim, hypotheses or purpose of the study?
2. Is there a clear description of the participants of the study? Depending on the study type, this may be, for example, the profile of a patient (a case report), the demographic profile of a population (a cohort population) or the profile of a defined group (a randomised control trial; RCT, a comparative study, or a single arm study).
3. Is there a clear description of how the participants were recruited? Was ethics approval required and if yes, was it sought?

4. Is there a clear description of the study method? This may range from a very detailed trial protocol for an RCT, to a registered systematic review protocol presenting how a literature search was conducted. Validity and reliability of measurement methods (assays, psychometric tools) would be presented here. If there are statistical results presented in the study, is the statistical methodology used explained?
5. Are the results that are presented relevant to the methods and to the aims of the study?
6. Does the discussion interpret the results to answer the aims of the study? This is where nuance of the study's findings and its relevance to previous research are presented.
7. Does the study explain its limitations? A study's limitations often feed into potential risk of bias. These may include a small sample size, the limited profile or recruitment of a study sample rendering it unsuitable for generalization to the rest of the population, study methods that are not validated or not validated in the study population, low retention rates of study participants, or too few participants to provide sufficient statistical power to make a conclusion. In the case of systematic reviews, limitations may relate to the languages studies were published in, the inability to access unpublished data, or the low quality (low certainty) of the available evidence.
8. Did the authors state their conflicts of interest? This often relates to financial conflicts of interest, or that the study is sponsored by a commercial body.

A brief summary of the types of quantitative studies follows.

Observational Studies: Descriptive and Analytical

Descriptive studies, as the name suggests, systematically describe aspects of a variable of interest in a particular population. Data collection is commonly 'naturalistic' - that is, occurs in a natural environment without interference from the experiment - and is not typically hypothesis driven. Descriptive studies do not attempt to analyse the link (correlational, causal or otherwise) between an exposure and an outcome (Beaglehole et al., 1993), but may define the prevalence. *Prevalence* is the frequency of a particular characteristic, condition or disease process in a defined population within a specific time period (Adams and McGuire, 2022). It is numerically defined as:

$$\text{Prevalence} = \frac{\text{Number of cases in a population at one time point}}{\text{Total population at the same time point}}$$

An example of a descriptive study in psychiatry could be the prevalence of alcohol use in adults with severe mental illness from 1950 to 2000.

The *case study* is a particular type of descriptive study that provides an in-depth, detailed description about a person, group or condition. This is particularly helpful when there is limited or no pre-existing literature on the given subject, such as when describing rare phenomena.

Analytical observational studies include *cross-sectional*, *case-control* and *cohort studies*. *Cross-sectional studies* investigate the association between a potential risk factor for an illness, and the outcome. In a cross-sectional study, all variables (exposure, outcome and confounding variables) are measured simultaneously. On the one hand, these studies are relatively time-efficient, easy to conduct and like descriptive studies, are useful for analysing the prevalence of a condition. However, it is not possible to make conclusions about the association between the risk factor and outcome.

Case-control studies require a group with the disease (the “cases”) within a given population to be identified, and then compares this group to a suitable group without the disease (the “controls”). It is a type of longitudinal study in which the investigator retrospectively assesses the “exposure” (or risk factor) for the given disease between the cases and controls. Case-control studies therefore estimate the strength of an association between a disease condition and the risk factor(s) under question. The control group should be comparable based on other demographic (such as age, sex, source of recruitment) and clinical variables, particularly the presumed distribution of the exposure, to minimise the risk of selection bias (Lewis and Pelosi, 1990).

Case-control studies are commonly analysed using an “odds ratio” (OR), which is the measure of the disease in the exposed compared to the rate of the disease in the unexposed. Mathematically, this looks like:

	Cases	Controls	Total
Exposed	A	B	A+B
Unexposed	C	D	C+D
Total	A+C	B+D	

$$\text{Odds ratio} = \frac{A/B}{C/D} = \frac{AD}{BC}$$

One advantage of this study type is its use in uncommon illnesses, such as schizophrenia. An example of a large case-control study is to assess parental age and the risk of schizophrenia (Byrne et al., 2003).

Cohort studies begin by defining the population (cohort) without the disease. The cohort is selected based on exposure to a particular risk factor, and then followed up longitudinally to see who develops the disease or outcome. The exposure may be binary (i.e. exposed or not) or graded (e.g. minimal, moderate, extensive), the latter of which may provide evidence for a dose-response relationship (Prince et al., 2003). Given the temporal sequencing of events, and that participants are chosen based on their exposure (not an outcome, like a case-control study), the cohort study is preferential to provide information on causality and/or risk of developing a disease. Other advantages of this study type are its capacity to examine both rare exposures and multiple outcomes in one study. However, cohort studies may require an extensive follow-up duration to obtain data on a given outcome that occurs many years after an exposure, which therefore may be particularly costly. Cohort studies are useful to determine the *incidence* of a condition. *Incidence* is the number of *new* cases of a disease or condition in a defined population within a specific time period (Centers for Disease Control and Prevention, 2012).

Finally, it should be noted that guidelines have been developed to improve the quality of reporting observational trials. The STROBE – Strengthening the Reporting of Observational Studies in Epidemiology – statement is recognised internationally and provides a checklist to this effect (Vandenbroucke et al., 2007).

Interventional Studies – Randomised Controlled Trials

The *randomised controlled trial* (RCT) is widely considered to be the gold standard for evaluating treatment efficacy and safety. Subjects are randomly assigned to one of two groups: the ‘experimental’ group – that is, the group to receive the treatment under investigation – or the other ‘control’ (or comparator) group. The randomisation process should distribute any potentially confounding factors equally between groups, to minimise selection and/or observer bias, so that any difference in outcome can be attributed to the treatment itself (Kendall, 2003).

As with all studies, there should be a clearly defined hypothesis *a priori*, which will in turn define the intervention, population and primary outcome of the study. “PICO” is a commonly used acronym to assist in defining the study question. For example:

- Population, for example, adults with schizophrenia
- Intervention, for example, daily treatment with olanzapine
- Comparator, for example, compared to placebo
- Outcome, for example, reduce the positive symptoms of psychosis?

Effective randomisation includes the process of allocation concealment, and the subject and investigator should remain “blind” to group allocation for the duration of the study. If both the investigator and participant do not know who is allocated to each group, it is referred to as “double-blinded”. Apart from the different intervention, both groups should be treated identically, so that any difference in outcome can be confidently attributed to the intervention itself (Akobeng, 2005). Quality control in reporting of RCT protocols and results is of utmost importance. ‘CONSORT’ - Consolidated Standards of Reporting Trials’ (Schulz et al., 2010) remains the best practice guideline for reporting RCTs.

Systematic Reviews

A systematic review is a review that uses clear and systematic methods to identify all empirical evidence (primary studies) in order to answer a specific research question. The method aims to reduce bias and thus result in more reliable conclusions for informing decision-making. Systematic reviews commence with a clear and detailed protocol that presents an *a priori* methodological approach (Moher et al., 2015). Protocols for systematic reviews may be registered at the international Prospective Register Of Systematic Reviews PROSPERO.

Systematic review protocols include clear and explicit research questions (see PICO criteria) that the final review aims to answer, proposed search strategies including search terms, the databases and data sources that will be searched, inclusion and exclusion criteria for the empirical studies that are identified, how the risk of bias of empirical studies will be assessed and how the included data will be synthesised. The Preferred Reporting Items for Systematic reviews and Meta-Analysis (PRISMA) (Moher et al., 2015) is a checklist which many peer-reviewed journals require a systematic review to meet and include to be accepted for publication.

A published systematic review will clearly articulate the research question it aims to answer, present the search strategy and data sources that were searched,

the inclusion/exclusion criteria for empirical studies and the assessment tools used to assess the empirical studies' risk of bias. The inclusion/exclusion criteria will often be detailed for each of the PICO criteria, as well as study type (e.g., RCTs, cohort studies, case series), publication date limits and language limits. The dates that the database searches were conducted should also be stated. The total number of unique studies and publications retrieved is stated along with the number excluded and the reasons for exclusion reflecting the inclusion/exclusion criteria. The number of empirical studies retained is stated and often the number that are included in quantitative analyses (that is, a meta-analysis) and qualitative analyses.

As systematic reviews formally assess the risk of bias of their included empirical studies and as well as their own methodology, they allow for judgements to be made as to how reliable or robust their conclusions are. Risks of bias include publication bias or non-reporting bias, such as non-published studies not being canvassed. Other risks of bias assessed by tools (e.g., Cochrane's Risk of Bias tool for RCTs or for Non-Randomised Studies of Intervention (ROBINS-I)) include whether or not participants and assessors were blinded to the intervention, selective reporting of outcomes and in the case of RCTs, whether or not the participants were truly randomized in the selection process. Authors' conflicts of interest should also be presented and are usually incorporated into studies' risk of bias assessments.

Meta-Analysis

A meta-analysis is when the results of individual empirical studies are combined to give a summary statistic. The basis of a meta-analysis is a systematic review, which identifies the empirical evidence that will be combined to give the overall statistic. Not every empirical study included in a systematic review will be included in a meta-analysis, as meta-analyses require the set of empirical studies to use similar methods for assessing outcomes.

Generally, systematic reviews and meta-analyses do not require ethics approval, as they assess primary studies that have already received approval and are not directly collecting data from participants or using participants' tissues.

Hierarchy of Evidence

Study design influences the quality of a study's evidence (Schünemann, 2013). For studies of interventions, the strongest level of evidence (and thus with the least bias) is a systematic review of RCTs, followed by RCTs, then non-randomised comparative studies (cohort studies, case controls studies) and then case series or case reports (Coleman et al., 2009). RCTs generally provide stronger

evidence than observational studies, which in turn provide stronger evidence than uncontrolled case series (Schünemann, 2013).

However, it should be noted that a well-conducted cohort study may yield better quality evidence than a poorly conducted RCT. Further, some topics do not lend themselves to RCT methodology, including for ethical reasons. The quality of the evidence is reduced if there is bias (see Systematic Reviews). With regard to diagnostic accuracy, high quality evidence is derived from cross-sectional or cohort studies in patients with diagnostic uncertainty and studies that directly compare test results with an appropriate reference standard (Schünemann, 2013).

Measurement Concepts

Reliability and Validity

Reliability and validity are concepts used to evaluate the quality of measurement (Adams and McGuire, 2022). *Reliability* refers to the consistency of measurement across time and between different observers. Reliability can be assessed in several ways:

1. Internal consistency: how consistently each item on a scale measures the same construct.
2. Inter-rater reliability: the extent of agreement between scores provided by different raters.
3. Test-retest reliability: how consistently the scores are measured over time.

Validity refers to the accuracy of findings or measures, that is, how well a tool measures what it is supposed to measure. Validity can be assessed using different dimensions. Some examples of these include:

- Face validity: does the tool appear to measure what it purports to?
- Construct validity: does the tool reflect the characteristics of the hypothetical construct we want to measure? An example may be a scale to measure depression severity, based on relevant existing knowledge and criteria for the concept of depression.
- Content validity: does the tool include all aspects that are relevant to the construct?
- Criterion-related validity: how well does the test sub-score correlate with or perform against an independent measure or standard (concurrent validity) or future standard (predictive validity)?

Diagnostic Studies

A diagnostic test is a procedure used to determine if a patient or study participant is likely to have a particular condition. In psychiatry, diagnostic tests are relatively rare as we are yet to identify specific, quantifiable biomarkers to determine the presence of a primary psychiatric illness. However, in Alzheimer's disease for example, advances in positron emission tomography (PET) imaging technology have enabled us to visualise core pathological features *in vivo*, such as β -amyloid and tau. Such diagnostic tests are usually compared to a 'gold standard', which for Alzheimer's disease has historically only been confirmed at post-mortem (Peacock and Peacock, 2011). For a diagnostic test, four measures are commonly used to describe the adequacy and utility of the test (Trevethan, 2017):

1. Sensitivity: The ability of a test to detect all people who have a condition (i.e. the 'true positives'). Mathematically, this looks like:

$$\text{Sensitivity} = \frac{\text{True positives}}{\text{True positives} + \text{False negatives}}$$

2. Specificity: The ability of a test to detect all people who do not have a condition (i.e. the 'true negatives'). This is equivalent to:

$$\text{Specificity} = \frac{\text{True negatives}}{\text{True negatives} + \text{False positives}}$$

3. Positive predictive value (PPV): The probability that an individual with a positive test result will truly have the condition. Mathematically, this looks like:

$$\text{Positive predictive value} = \frac{\text{True positives}}{\text{True positives} + \text{False positives}}$$

4. Negative predictive value (NPV): The likelihood that a person with a negative test result will truly not have the condition. Mathematically, this looks like:

$$\text{Negative predictive value} = \frac{\text{True negatives}}{\text{True negatives} + \text{False negatives}}$$

Risky Business

An important concept in psychiatric research is “risk”. This may apply to the probability of acquiring a psychiatric disorder following exposure to a particular risk factor (for example, the risk of developing schizophrenia after exposure to regular cannabis use), or the risk of a particular event following exposure to treatment (for example, the risk of elderly adults developing a stroke following the use of antipsychotic medication). Statistically speaking, the two commonly cited types of risk include:

1. Absolute risk: This is the probability of an event occurring over a period of time (Boyce and Hadzi-Pavlovic, 2011). An example may be the risk of developing late life Alzheimer’s disease following treatment for mid-life hypertension.
2. Relative risk: This is a proportional measure of risk between two groups which differ by some factor, such as a demographic or clinical factors, including exposure to a particular risk or treatment. For example, if the lifetime risk of developing autism spectrum disorder is 43 per 1000 males and 11 per 1000 females (Maenner, 2021), the relative risk is $0.043/0.011 = 3.9$ in males compared to females.

Number Needed to Treat and Number Needed to Harm

Number needed to treat (NNT) is defined as the expected number of people who need to receive an intervention instead of a comparator therapy in order for one additional person to achieve or avoid an event or outcome within a specified timeframe. For example, a NNT of 10 indicates for every 10 participants given the intervention treatment, one additional person will achieve or avoid an event compared to when the comparator treatment is given. Mathematically, NNT is calculated from risk differences (RDs) and is represented by the formula below (Schünemann et al., 2022).

$$\text{NNT} = \frac{1}{\text{absolute value of risk difference}} = \frac{1}{|\text{RD}|}$$

NNTs can also be calculated from risk ratios (RR) and from odds ratios and assumed control risks (ORs and ACRs). NNTs are expressed as positive whole numbers and if the calculated values have decimals, they are always rounded up.

You may also come across the term “number needed to harm” (NNH), which implies that the intervention treatment results in an unfavourable event. It is not recommended that this term be used and instead, be replaced with the terms “number needed to treat for an additional beneficial outcome” or “number needed to treat for an additional harmful outcome” (Schünemann et al., 2022).

Other Concepts in Basic Statistics

Effect size refers to the magnitude of the effect of a variable. This is commonly reported using the statistic, Cohen’s *d*, whereby it is widely accepted that a value of 0.2 indicates a small effect size, 0.5 indicates a medium effect size, and 0.8 and above represents a strong effect size (Adams and McGuire, 2022).

A *confidence interval* describes the degree of certainty with which the true value of the statistical test lies within a given range. A 95% confidence interval indicates that we are 95% certain that the true value lies within the range. Smaller confidence intervals therefore indicate a more precise estimate of the mean value (Riffenburgh, 2012).

Statistical significance is the likelihood that the relationship between two variables is not due to chance. This is typically determined by hypothesis testing. The null hypothesis states that any observed differences between groups is entirely due to chance (Beaglehole et al., 1993). The *p-value* is then used to calculate the likelihood that the null hypothesis is either accepted or rejected; the larger the *p-value*, the greater the probability that the null hypothesis is accepted – that is, observed differences between groups are likely due to chance and not true between-group differences. It is customary practice to accept 0.05 or 0.01 as significance levels.

Although widely used, much confusion and debate shrouds the misinterpretation and over-reliance on *p-values*. The *p-value* itself is a ‘probability statement about the observed sample in the context of a hypothesis’ and should be augmented with additional statistical information (such as effect size) to avoid misinterpretation (Altman and Krzywinski, 2017). There has even been a push from within the scientific community for the ‘entire concept of statistical significance to be abandoned’ - not an eradication of *p-values* or confidence intervals, but no longer treated categorically as either proving or disproving the null hypothesis (Amrhein et

al., 2019). As Amrhein et al. (2019) argue, this oversimplification can lead to brash assumptions, and incorrect interpretations, including that non-significance equates to no effect. Caution is warranted when interpreting a p-value, large or small, and weight given to other factors such as study design and data quality.

Finally, it should be noted that statistical significance does not necessarily equate to clinical significance. For example, a statistically significant 2-point increase on a scale measuring depressive symptoms may not be clinically relevant. One must always consider the clinical application, which includes the generalizability and limitations of research to relevant patient populations when interpreting the results of any study.

Ethics

Research ethics may be defined as ‘the ethics of the planning, conduct, and reporting of research’ and should at a minimum protect interests of the public, the research participants (both human and animal) and the researchers conducting the studies (Resources for Research Ethics Education, 1999-2016). The World Medical Association’s Declaration of Helsinki (The World Medical Association, 2013) is ‘a statement of ethical principles for medical research involving human subjects, including research on identifiable human material and data’. It comprises general principles that places the patient’s (participant’s) health and wellbeing at the forefront when conducting research, as well as principles related to risks, burdens and benefits of the research, groups and individuals that are vulnerable, the privacy and confidentiality of research participants, informed consent, post-trial provisions for participants and communication of results. Particularly pertinent to research in psychiatry are the principles relating to research where participants are mentally incapable of giving informed consent, and the principle that research in a vulnerable group is only justified if it serves the needs or priorities of the vulnerable group and benefits them, and thus the research cannot be conducted in a non-vulnerable group.

In order to be published in peer reviewed journals, researchers usually must declare that the study was conducted in line with ethics approval, or that ethics approval was not required, for example in a literature review or a quality assurance project. For example, the Australian and New Zealand Journal of Psychiatry (ANZJP) requires research conducted in human participants to adhere to the Declaration of Helsinki. Further, ANZJP requires that studies with human (or animal) participants must state in the methods section that approval was provided by the relevant Ethics Committee or Institutional Review Board, or that the approval was waived.

The values and principles of ethical research in humans comprise (The Australian Research Council and Universities Australia, Updated 2018):

1. Respect for human beings and valuing their autonomy, including protecting and empowering those with reduced or no autonomy.
2. That the research has merit and integrity, to ethically justify being conducted with human participants.
3. That the research is just, where the benefits and burdens of research are fairly distributed and there is 'fair treatment' in participant recruitment.
4. Beneficence is practised by researchers, by considering and assessing risks of harm and potential benefits of the research to participants and the wider community.

Research with merit is characterized by its capacity to enhance knowledge, social welfare, and individual wellbeing, while also advancing researchers' proficiency. Integrity in research is upheld by scholars dedicated to the pursuit of knowledge, adhering to ethical principles, and conducting research with honesty. When research maintains integrity, both favourable and unfavourable outcomes are transparently communicated, facilitating scrutiny, and enabling the findings to enrich public knowledge and comprehension. Human research ethics protects both the researcher and the participant, ensuring that the research benefits outweigh any negative effects of the study, that the research does not waste resources. A study should not deviate from the ethically approved protocol unless approval is sought and granted by the ethics committee for the amendments. In addition, ethics approval usually requires researchers to submit regular progress and safety reports.

Concluding Remarks

Whilst this chapter is not an exhaustive deep dive into research methodology, it provided an overview of common study types and statistical concepts. Further reading is recommended. Nevertheless, this has hopefully imparted a foundation to understand and define basic principles in psychiatric research to enable critical evaluation of the literature, to ultimately improve the standard of clinical care provided to patients.

Further Reading

Hammarberg, K., Kirkman, M., & De Lacey, S. (2016). Qualitative research methods: when to use them and how to judge them. *Human Reproduction*, 31(3), 498-501.

National Health and Medical Research Council (2009). NMHRC levels of evidence and grades for recommendations for developers of guidelines. Available at:

[https://edit.nhmrc.gov.au/sites/default/files/images/NHMRC%20Levels%20and%20Grades%20\(2009\).pdf](https://edit.nhmrc.gov.au/sites/default/files/images/NHMRC%20Levels%20and%20Grades%20(2009).pdf)

Chapter 13: Attachment

Loyola McLean

Westmead Psychotherapy Program, Western Sydney Local Health District, and Specialty of Psychiatry, The University of Sydney, Australia.

Background

Attachment theory was developed initially by John Bowlby, a British child psychiatrist and psychoanalyst, in the mid-20th century in an effort to explain two of his clinical observations: (1) disruptions in the mother-child relationship appear to be a precursor to later psychopathology; (2) children express distress, anxiety or anger on separation from parents (Bowlby, 1969; Cassidy and Shaver, 1999). Bowlby's observations and thinking were occurring at a time of profound challenges people were facing post WWII. For example, the sequelae of separation of children from their parents, often for reasons of safety, but then family disruption from loss and trauma. It was in this context that the WHO requested a formal report from him (Bowlby, 1951).

The psychoanalytic and social learning theories of the time focused on the feeding relationship between mother and infant, without appearing to account for the data from ethology that bonds seemed to form without this feeding. There was also a tendency to focus on the intrapsychic experience of the child and not consider the important relational interplay between caregiver/s and the child. The search to develop the theory led Bowlby down a sophisticated path, over a number of years, of synthesizing information from various disciplines such as evolutionary biology, ethology (Lorenz's work was foundational), cognitive science (including learning theory and the work of Piaget), developmental psychology, and control systems/cybernetics as well as psychoanalysis (Bowlby, 1951; Bowlby, 1969; Bowlby and Bowlby, 2012). This integration of knowledge and ideas continued to develop in collaboration with others including Mary Ainsworth to arrive at the notion of a motivational and behavioural system that supported the development of a bond between infant and caregiver, conferring evolutionary advantage. This advantage was multi-factorial, promoting protection, *feeding, development, self- and social regulation, and learning*. Although having its evolutionary origin in the imprinting of birds, it was phylogenetically preserved and developed in mammals, primates and humans and is a central system for human development and our

capacity to manage stress as individuals and as a species interconnecting with many other biopsychosocial systems (Cassidy and Shaver, 1999).

Evidence across a number of domains and types, including observational, epidemiological, experimental and longitudinal research, including research that spans several generations of families, has then continued to accrue and led to the idea of a Biopsychosocial Organizing System, that plays an organising role in neurological, psychological, psychophysiological and social development with important “cross-talk” with other systems. This has been termed the Attachment System, and disruptions within this system can impact normal attachment and may be risk factors for illness and disorders. Conversely, the development of a *secure attachment* can be protective and support resilience (Maunder and Hunter, 2001; Stovall-McClough and Dozier, 2016).

In the context of medicine, a biopsychosocial model of development and psychophysiological development is understandably complex. However, as attachment speaks to how personal and relational experience shapes individual homeostasis and stress regulation strategies, which are optimally social, attachment is contributing to models around symptom, syndrome and illness development, in psychiatric disorders and in medical disorders, including psychoneuroimmunology, and can contribute to frameworks and therapies for prevention and treatment (Kozłowska et al., 2015a; Maunder and Hunter, 2001).

We now understand that nature and nurture are thoroughly intertwined, and that attachment and parenting interact with genetics and epigenetics. Therefore, the transgenerational effects of protective care behaviour as well as the challenges of stress, loss and trauma are important considerations.

The Infant’s Need for Care and Protection

The fundamental need for a system of Attachment is the fact that human infants will not survive without protection, safety, care and connection with others. There is a period of inevitable dependence during development. Functions as basic as temperature regulation, sleep, emotion regulation, and the functioning of the autonomic nervous system are not fully “programmed” at birth, but rather require the attuned care of another for optimal development of networks of regulation and safe exposure to experience. Attachment experiences are thought to lay down both “an internal working model” of human relationships (discussed further in section below) and social regulation but also connect up many stress response systems, like the autonomic nervous system. This developmental role of the attachment system and relationship for establishing self-regulation and social regulation has been increasingly researched and recognised.

Bowlby

Bowlby formally studied the attachment system through children's experiences of separation and loss, demonstrating that infants are inherently motivated to attach to their parents/carers. This was in conversation with the work of James and Joyce Robertson, who had filmed the way children responded to parental separations for hospital admissions and then filmed responses of children, separated from parents and in foster care (Kennedy, 1969; Robertson and Bowlby, 1952). The strong emotional response to separation included distress, sadness, anger and, when it went beyond a certain period, lead to despair (Cassidy and Shaver, 1999). Once established, the system is activated when separation or stressors become too great, and proximity to the caregiver is increased: the infant cries, calls and reaches out, signalling for help. Once safety and soothing are re-established the bids for attention should cease.

Emotional Role

Affect, emotions and feeling are closely associated with the attachment system, which is an interpersonal motivational and behavioural system. Affect regulation is an outcome of the system: a child expresses distress or anger at separation and is soothed by parental approach and attention. We note the joyous forming of a bond, the delight shared between the parties in an attached couple, grief at the loss of the bond and the anxiety or angry protest aroused at threatened loss. These feelings can also form a regulatory function within the attachment relationship: "Many of the most intense emotions arise during the formation, the maintenance, the disruption, and the renewal of attachment relationships." Differences in patterns of affect regulation are seen across the different attachment patterns described below.

A flexible and balanced psychophysiological regulation depends on developmental experiences of *Affective Attunement*. Through empathy, the caregiver recognizes the baby's emotion and attunes to it: (re)-establishing safety and comfort; soothing; reflecting and validating experiences and emotions; modulating affect and body states both up and down, as needed. The down-regulated quiet child may need amplification, the up-regulated labile or anxious child needs settling. Without this, children need to develop strategies to maximise their own safety and comfort, often at a psychophysiological cost. Megan Gunnar and team's research shows that the seemingly "independent" infant, who has an "avoidant" attachment strategy shows raised markers of stress in the hypothalamic-pituitary-adrenal (HPA) axis during parental separation (Hertsgaard et al., 1995) and up-regulation in children with non-normative attachment has been observed in stress system disorders such as

Functional Neurological Disorder (Kozłowska et al., 2015a; Maunder and Hunter, 2021).

The early relational experience of care, comfort and safety allows the child to develop flexible regulation of the internal environment and a balance between safe rest, soothing and safe exploration. The attuned parent/caregiver also knows how to scaffold new experience for the child to foster optimal learning through play and exploration and to recognise when a child is blocked or limited, requiring more help to negotiate the next stage of development. This experience of regulation within the dyad from early in infancy is the beginning of the emergent capacities for *rest, play, love and work*.

Cognitive and Emotional Schema

Bowlby proposed cognitive components to the attachment system. These consisted of mental representations of the attachment figure, the self, and the environment that develop out of experience, not just phantasy. However, due to individual differences in managing, organizing and representing experience, seemingly similar experience can still result in different states of mind. These internal working models of relationships are powerful and predictive cognitive and emotional schema or templates underlying expectations about relationships (Cassidy and Shaver, 1999). We expect help from others to be available when needed if we have experienced responsive and sensitive help in the past. If acquired early or regularly enough, this experience is encoded as procedural memory, the “*way things are*”, and so can be out of immediate conscious awareness but implicitly known and expected.

These templates, also referred to as schemata, are now considered to be seen in relationships beyond those with early caregivers as predictors of the likelihood of receiving help and mapping learned experience of the behaviours that will lead to care-giving from others. This can be seen in the *transference-countertransference (the unconscious aspects of relationships)* in medicine and psychiatry, and the patient’s expectations of help versus harm. This underpins the potential consideration of attachment when dealing with patients as it may relate to their conscious and unconscious expectations of help and care and their pattern/s of self-regulation (Maunder and Hunter, 2001; Maunder and Hunter, 2009).

The Secure Base

The attachment system interacts with other systems, such as the fear system and the exploration system. There is an important relationship between the developmental need and drive of the infant to *explore* the environment in order to

develop knowledge and skills and the need and drive to return to the carer, as “safe haven”, for comfort and safety. The smooth working of these two drives is termed “*the secure base*” of the attachment figure, from which the infant explores. Ainsworth pointed out that equilibrium between proximity seeking and the need to explore is important for development and survival: a child who is safe and settled enough to explore and play, knowing they will be met with soothing and help if they encounter trouble, will optimise appropriate safe exploration for development (Ainsworth, 1972). An attuned parent will encourage and regulate this safe exploration and the child’s return for reasons of needing safety, comfort or emotional and cognitive processing or restoration (Ainsworth, 1964; Ainsworth, 1972).

Deviations from the *secure* organization of attachment lead to *insecurity* of several types. Children who have come to learn that they need to rely on their own self-soothing, favour exploration over comfort and proximity to the care-giver and are described as having an *anxious avoidant* attachment; others who are difficult to settle back to exploration after stress are often fussy and clingy, and so seek but also resist proximity and comfort and are termed to have an *anxious ambivalent* attachment. One good way of seeing this in action is to look at the internet for the videos of the Strange Situation Procedure – an experimental procedure that assesses the nature and quality of the attachment relationship in infants (Ainsworth et al., 1978). The adult types of insecure attachment are dismissing, preoccupied and unresolved. Accounts of the various childhood and adult attachment classifications help clinicians “see” the way patients exhibit these (Main, 2000; Hesse and Main, 2000; Hesse, 2008; Maunder and Hunter, 2009).

The *fear system* or *fear cascade* also interacts powerfully (Kozłowska et al., 2015c) with the Attachment System. Fear management and protection is a goal of the attachment system. Humans are mammals with a social nervous system that has built on the simple autonomic nervous system of reptiles and offers not just the basic options of sympathetic arousal and parasympathetic withdrawal but a higher order opportunity for social soothing, networked with facial expression, gaze and voice (Porges, 2011) – we have a social nervous system, where optimal regulation is social. The attachment system initially links this opportunity to be soothed and protected socially to one or more specific attachment figures and scaffolds the template that expects help and soothing from these carers. As the internal working model develops and generalizes the child comes to expect a kind of relationship from others more broadly. In a clinical setting this then includes the expectations of help or harm from clinical carers, and in psychiatric practice a large number of presenting patients have had childhood experiences where the parent may be a source of fear, either through actual direct frightening acts or because care-givers are themselves frightened or helpless. Even these *frightened* parental states are experienced as *frightening* by a child. Children need the parent to be the “strong, wise, soothing”

one, not a source of fear. This places the child in a powerful dilemma, a confrontation between systems: the care-giver who should be comforting is *frightening/frightened* and research suggests this *disorganizes* the normal organization of the child's attachment strategies and cognitive and emotional processes. These are powerful moments of psychophysiological disorganization, associated with dissociation and appear to have a strong relationship with psychopathology (Hesse and Main, 2000; Main, 2000).

Clinical Strategies

Bringing Forward the Templates

Explicit use of attachment theory with patients and carers can foster the therapeutic alliance, helping to better conceptualize formulation, diagnosis, and management goals and then to tailor the management plan accordingly. By applying the principles of attachment, we can *listen* at interview as the patient relates the account of the development of the symptoms, for example, depression, as they saw it, and then we could enquire about early and current relationships, considering experiences of help, comfort, safety and fear. We listen closely to the information provided, both for *content* and *form*, listening for the *organization* of the account (whether *secure*, *dismissing* or *preoccupied*) and any moments of disorganization or dissociation (*unresolved loss or trauma*). These patterns of reasoning, language and behaviour have been elucidated in attachment research, including the Adult Attachment Interview (Hesse, 2008). In psychiatric samples we can also see *low coherence* or *competing strategies* likely based on childhood *disorganization* (Speranza et al., 2017).

If information relevant to medical health is considered as related to self-regulation, for example sleep, diet and exercise, the *biopsychosocial* information could then be tentatively integrated collaboratively with the patient, getting them to reflect on how they organize themselves and their relationships and any challenges/stressors/changes. Feedback can be offered, or questions raised where inconsistencies or gaps might suggest a slightly different reading. In this way a collaborative formulation can be reached with a more transparent way of discussing the older ways of organizing oneself, rooted in childhood or other experiences and so beginning to discuss things that are not immediately in awareness. The formulation can then be more collaboratively created. Treatment can then be considered in the light of that information and a more collaborative contract for treatment made.

Applications: General, Medical, and Psychiatric

In illness of self or those close to us, and at other times of stress (e.g., childbirth) the attachment system may be activated. Those with a secure state of mind are going to be comfortable to be appropriately help-seeking as per the classic and normal “sick role” and, unless disrupted by trauma or loss, comfortably relinquish care as recovery proceeds (Parsons, 1951). The attachment state/s of mind can shape the clinical coping and interaction (See Table 13.1). Those who have a dismissing or avoidant strategy can be avoidant of care due to a need to be “independent” (here meaning an avoidance of dependence) whereas those who are ambivalent may require/ demand more reassurance or be *angry/blaming* or perhaps *passive* in their recovery. These are not the only possibilities, but some exploration of attitudes to care can often reveal how early experience has shaped them and help shape our therapeutic alliance. Those who are *disoriented/disorganized/dissociated* due to active unresolved loss or trauma, or *chronic complex trauma* may show unusual states of *immobility/ fragmentation/ disconnection* at assessment or during treatment, which will need attending to both directly and via general principles from *trauma-informed care*: a health wide approach that acknowledges stressful and traumatic experiences and their sequelae and the need for attuned responsiveness by clinicians and health systems.

Table 13.1. Orientation towards health care and sick role by attachment state of mind.

Attachment state of mind	Interactional quality	Impact on Sick role
Secure	Collaborative	Appropriate Sick role
Dismissing	Avoiding dependence	Avoids or resists health care
Preoccupied	Overly dependent	Clings to care/ blaming/ passive
Unresolved or complex traumatic attachment	Fear, fragmentation and disconnection	Traumatic or loss experience gets in the way of collaborative treatment

Applications to the Clinician’s/ Team’s Response to the Patient (Countertransference)

The theory facilitates understanding likely difficulties that may arise in patient-clinician interactions. For example, clinicians /teams with a more avoidant/dismissing strategy or culture will tend to label even normal help-seeking behaviour as “clingy” or overly dependent and will tend to validate stoical or

minimizing responses to illness as “coping”. Similarly, they will tend to label the ambivalent strategy “crazy/disordered”. A reflective clinical practice can consider where we currently sit in the spectrum of attachment states of mind and is useful at the personal clinician and team level. Understanding how I and/or my team are organising to manage stress/ trauma/ loss will help me/us understand reactions and fluctuations in responsivity to patients and situations. For example, a team that has experienced a patient’s recent suicide will be shocked, numb or perhaps hypervigilant and grieving for a time in a way that may influence interactions with other patients. The team and its members will need to resolve *losses and traumas*. This approach dovetails with a reflective and supervisory approach to team supervision and trauma-informed care (Maunder and Hunter, 2001; Maunder and Hunter, 2009).

Summary of Conceptual Overview

So, in summary, attachment theory can provide a general understanding of human beings under stress. It affirms that we never outgrow our healthy need to be dependent at times, especially during stress and illness and that we may need attachment relationships throughout our lifespan. It articulates that healthy, flexible, personal development occurs optimally from a secure base as does healthy grieving and recovery from substantial stress, loss, illness and trauma. It maps out the framework that early attachment relationships are strong determinants of physical, emotional, cognitive and social development. It directs attention to the specific current organization of the stress response for the individual, dyad or family and offers points for intervention to help the individual or system regain a healthy homeostasis and recover.

Clinical Approach

The patient-doctor, client-clinician interaction should basically be functioning as if it is a secure relationship, where both parties feel safe enough and fear and strong emotion is manageable between them, and the process is collaborative. Clearly new caring relationships cannot be fully developed attachment relationships, as bonds take time. However, when both parties bring the capacity for security and the clinician can foster this, then those templates can be accessed and a secure enough working relationship co-constructed. The corollary will be that the doctor is useful in helping the patient manage their unmanageable feelings and fears. However, the patient’s and the clinician’s attachment states of mind will powerfully influence the kind of interaction. This is the heartland of *transference-countertransference*, where experiences that are not immediately conscious shape our feelings, thoughts, behaviours and relationships.

The *assessment* of a patient is then conducted with the following strategy: to explicitly explore the different possible attachment patterns and collaboratively identify ongoing patterns and the impact of stress, loss and/or trauma in the story of the presentation. Exploring past and current state(s) of mind with respect to attachments will help formulate the presentation – why this patient presents with these symptoms at this time, due to stress regulation being insufficient for the current challenges of stress/loss/trauma and perhaps associated with a breakdown in organization due to being traumatically overwhelmed. It can then help differential diagnosis and offer possible areas for intervention/management, attending to symptoms in a patient-centred way, while encouraging better social and personal regulation. Sometimes a presentation to health care does mean that a person may need to shift their coping strategies, and this might entail some supportive or other psychotherapy.

Given that in a psychiatric population many patients are *unresolved with respect to loss or trauma* or have more incoherent attachment strategies at point of presentation it becomes crucial to spot those who have unusual states of *disorganization* or *dissociation* and states of unregulated fear. For work on this see the work on the Fear Cascade (Kozłowska et al., 2015c).

Applications – General, Medical, and Common Problems

Normal Bereavement and Significant Trauma

In adults, bereavements and traumas will often lead to a state of temporary disorganization (usually at least 12 months for the loss of a close relationship). For bereavement this normally resolves, especially if normal responses to grieving are validated. The process is not dissimilar for trauma. The person may resolve the loss/trauma or will dismiss it or continue to be preoccupied by it or remain disorganized (*unresolved*). However, there is growing evidence that unresolved losses and traumas are associated with psychiatric conditions of significance that have a degenerative effect on mental life: complicated grief and chronic PTSD (see Chapter 20) (Stovall-McClough and Dozier, 2016). Therapeutic work is then focused on *resolving* the loss or trauma after *safety* and *stabilization* are established and involving *integrating* the experience, *cognitively*, *emotionally* and *procedurally*.

Other Important Consequences

Children born to parents who have not resolved loss or trauma are at risk of insecure and potentially disorganized attachments. The way a parent's own

disorganized state of mind affects the interaction with the child and the child's organization described above is one of the pathways to childhood and family distress and *transgenerational transmission* of psychopathology. This indicates the importance of perinatal and family support for at-risk families and communities (Hesse and Main, 2000; Slade et al., 2020).

Applications and the Attachment Meanings of Treatment

Treatments will have relational meanings and are likely to *elicit the background attachment template* as the template is activated by stress (Bowlby, 1969). For example, with medication, some may enact their attachment pattern as a side effect sensitivity, and patterns of adherence (Maunder and Hunter, 2001; Maunder and Hunter, 2009). A recent systematic review has highlighted different patterns of engagement for this with dismissing/anxious-avoidant attachment strategies versus preoccupied/anxious-ambivalent (Adams et al., 2018). Surgery has a powerful implicit requisite of safety and trust of others and systems, whereas some patients including many psychiatric patients have never had safe, trustworthy relationships and experiences of care. This is part of the reason for our current focus on *trauma-informed care*. It helps us understand the central role of creating trust and collaboration with patients in order to deliver the health care they need.

Psychodynamic psychotherapy and therapies such as schema therapy explicitly aim to set up new templates of relationship while necessarily mobilizing the old attachment templates set up in childhood. One goal is then to move patients from a disorganized or insecure template towards a more secure state of mind and better interpersonal and self-regulation (McLean, 2012; McLean and Korner, 2014).

Conclusion

Working with an awareness of attachment allows us to understand how our patients have come to organize their coping responses and helps us to approach construction of our therapeutic alliance as founded on the idea of *secure base*, and the challenges to that. From early in assessment and treatment we can recognize and address the breakdown of organized coping strategies and the *disorganizing role of overwhelming stress, loss and trauma*. It can help us think about interpersonal and self-regulation and patient attitudes to treatment based on past experiences around help, shaping current attitudes to medication, surgery, rehabilitation and psychotherapy. It also helps our reflective practice if we can acknowledge our own ways of organizing our own responses to stress and consider where this interacts with the patient/family we are treating in the *transference-countertransference*. We are also supported by evidence to argue for the public health importance of good care of

families perinatally and in at-risk situations of mounting stress, loss and trauma, so that adults and parents are supported and children and young people have the chance of safely, optimally developing, and to ameliorate the transgenerational transmission of loss and trauma.

Further Reading

- Cassidy J. The Nature of the Child's Ties. In: Cassidy, J., & Shaver, P. R. (Eds.). (1999). *Handbook of attachment: Theory, research, and clinical applications*. Rough Guides. p. 30-70.
- Erik, H. The Adult Attachment Interview: Protocol, Method of Analysis and Empirical Studies. In: Cassidy, J., & Shaver, P. R. (Eds.). (2008). *Handbook of attachment: Theory, research, and clinical applications*. Rough Guides. P. 552-598.
- Hesse, E., & Main, M. (2000). Disorganized infant, child, and adult attachment: Collapse in behavioral and attentional strategies. *Journal of the American Psychoanalytic Association*, 48(4), 1097-1127.
- Main, M. (2000). The organized categories of infant, child, and adult attachment: Flexible vs. inflexible attention under attachment-related stress. *Journal of the American Psychoanalytic Association*, 48(4), 1055-1096.
- Maunder, R. G., & Hunter, J. J. (2001). Attachment and psychosomatic medicine: developmental contributions to stress and disease. *Psychosomatic Medicine*, 63(4), 556-567.

Chapter 14: Neurobiological Models in Psychiatry

Anthony Harris

Specialty of Psychiatry, Sydney Medical School, The University of Sydney, Australia, Westmead Institute for Medical Research, Australia, and Western Sydney Local Health District Mental Health Services, Australia.

“I want you to take note that in this machine all these functions follow naturally from the disposition of its organs alone, no more and no less than those of a clock or automaton follow from that of its counterweights and wheels”

Descartes (1662; 1998)

“Psychological Diseases are diseases of the brain... insanity itself is only a symptom”

*Griesinger from Pathologie und Therapie
der psychischen Krankheiten (1845) cited in Porter (1997)*

*“If mental disorders are brain disorders, then it follows logically that the basic sciences
of psychiatry must include neuroscience and genomics”*

Tom Insel (Insel and Quirion, 2005)

Introduction

The idea that psychiatric diseases are brain diseases has been a driving force in much of the conceptualisation of mental illness and its treatment. It has been held in counterpoise in the Western tradition by psychological or psychosocial conceptualisations of disease, the dominance of one particular conception ebbing and flowing. Advances in neuroscience have led to enthusiasm in the search for a neurobiological model of mental illness. However, no neurobiological model has been found that fully explains the syndromes treated by psychiatrists. And there is frustration at the lack of translation of new discoveries into effective treatments.

Neurobiological models of mental illness seek to understand psychiatric illness primarily or only in physical terms formed from the basic scientific knowledge

around brain function: anatomy, genetics, proteomics, cellular mechanisms, neurotransmitters, physiology and connectivity. The models differ in their scale and ambitions from those that aim to describe a restricted or defined dysfunction (e.g., how might an auditory hallucination arise) as against a global model of brain function and disease. These models reference existing conceptualisations of how our brain works. These have tended to project the most complex technology of the day onto the problem of how the brain functions. In Descartes' era the metaphor of the watch in all its mechanical complexity was a compelling model of brain function (Draaisma, 2000). More recent iterations have built up models based upon the biophysical properties of neurons linked into a comprehensive computational model of the brain using electroencephalography and the underlying connectome of the brain (Shine et al., 2021). These models track the development of seizure activity convincingly but are less compelling when explaining mental illness.

In its most basic form, the biomedical model of disease, “assumes disease to be fully accounted for by deviations from the norm of measurable biological (somatic) variables” (Engel, 1977). Likewise, the symptoms and signs of disease can also be reduced to parts that can be analysed using the scientific method and be understood to be caused by a change in the functioning or anatomy of the body. Treatment of that change, by an expert such as a psychiatrist, should be able to fix the problem. Disease comes about in the individual, and the patient bears little responsibility for their disease but is expected to cooperate with treatment. In return, their illness is recognised and they have special rights to our care and compassion (Wade and Halligan, 2004). This simple model initially appeared to be of use in psychiatry with the identification of the effects of syphilis on the brain and the identification of the psychiatric and neurological manifestations of that disease. However, it has not coped well with the complexity of mental illness. It failed to take into account social, environmental and psychological factors, ignored prevention and presented illness as a neutral objective scientific process, which it most certainly is not. It has been caricatured as “mindless” psychiatry in opposition to the “brainless” modes of explanation that prioritise psychological models at the expense of any consideration of the body (Eisenberg, 1986).

The weakness of this casuistry can be seen from attempts to understand the aetiology of schizophrenia. The similarity of delusions and hallucinations to the effects of other illnesses or psychedelic drugs has suggested to many that this may be part of the explanation for positive psychotic phenomena in schizophrenia. The identification of an unusual catecholamine in the urine of people with schizophrenia, identified via a “pink spot” on urine chromatography (Friedhoff and Winkle, 1962), was initially thought to be similar to or a metabolite of mescaline and hence it was proposed that schizophrenia was due to an inborn error of metabolism that caused the production of an endogenous psychomimetic substance. Further investigation

of the metabolite found it to be related to the heavy tea consumption of institutionalised people and nothing related to the cause of their illness (Stabenau et al., 1970). Although this can be characterised as a naïve attempt to understand a complex illness, it also underlines the limitations of a reductionist neurobiological approach to test a hypothesis and find it wanting.

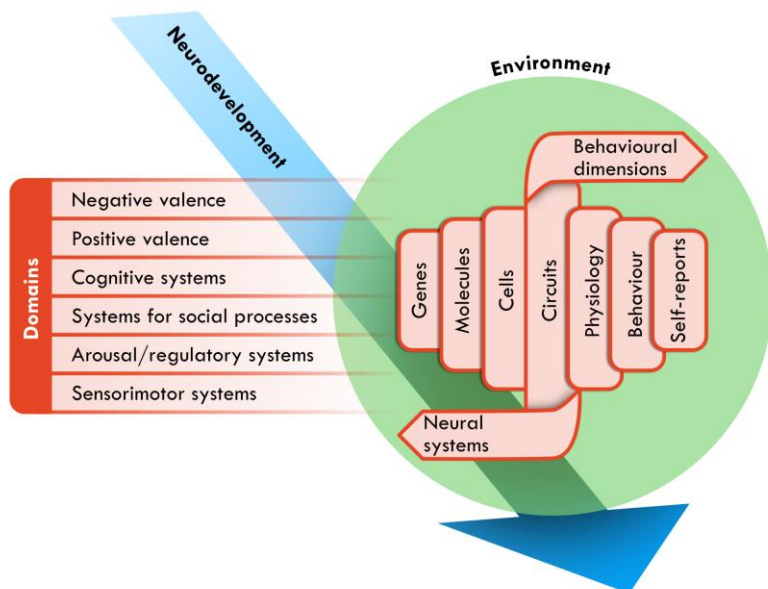


Figure 14.1. RDoC Framework for the understanding of mental illness (Cuthbert, 2020)

One of the hallmarks of the neurobiological models of disease is the expectation that function, and disorder will be identified through close examination of the constituent parts. These constituent parts are ultimately related to physical causes which invite physical interventions or treatments (Rocca and Anjum, 2020). A recent application of a neurobiological approach has been the Research Domain Criteria (RDoC) project (see Figure 14.1) (Insel et al., 2010). This considers cognitive and psychological dimensions as emergent properties of complex systems that become manifest as the system evolves and functions. These dimensions are not necessarily predictable on the basis of the bottom-up functioning of cells, and require a meeting of the bottom-up neurobiology with an understanding of top down hierarchical groupings that represent cognitive constructs (Insel et al., 2010). The RDoC model has eschewed conventional psychiatric diagnosis because of the lack of alignment of diagnoses with clear neuropathology, physiology or genetics. However, it has not provided an alternate way of understanding health as against illness, or consider disability, all essential issues in describing illness. It may have

reified measures which have as little scientific basis as the diagnoses they are replacing and mistake similarity in test performance for an underlying unity of causation – something which is unlikely (Weinberger et al., 2015). It also still underappreciates the significant effect of social forces in the development of illness (Marmot, 2015) and the bidirectional effect of psychosocial forces on the brain (Teicher et al., 2016). Finally, change on a test is not the same as illness. Without an understanding of disability caused, and the social context of the problem it causes, the change in the RDoC domain cannot be equated with the end effects known as disease and illness.

In addition to the difficulty that we are having understanding the complexity of the brain, all models of mental illness have to tussle with some basic issues around the characterisation of the disorders – equifinality, multifinality and multicausality (Cicchetti and Rogosch, 1996). Equifinality is the arrival at a common endpoint from many different causes. A significant depression can be seen as the outcome of loss, early life stress, influenced by social position, or as the sequelae of endocrine illness, brain lesions or genetic loading. Any single explanation of a mental illness may have validity for an individual and contribute to an understanding of the disease but cannot alone be equated with the disease. Current psychiatric practice would emphasise the complexity of causation and explain illness through the lens of multicausality, but leave it unclear as to how to proportion the many causes or how to measure them. Multifinality recognises that a single factor, such as early life stress, the effects of which can be measured biologically (Chu et al., 2019), or a gene that may have different effects depending upon the stage of development or the environment in which it is being expressed, can plausibly lead to a range of psychiatric illnesses, and that it is difficult to know which one will, if any, it result in. These factors, though seen in other complex systems are basic to the understanding of mental illness and make simple biomedical models unviable.

Neurobiological models of mental illness have provided important insights into the possible causes of mental illness but have not given rise to new treatments. Rather they have often been developed in response to the need to explain the effectiveness of those treatments, for example, the dopaminergic hypothesis of schizophrenia arose out of the observation that all effective antipsychotic medications were dopamine receptor blockers. Attempts to place mental illness onto a basic science platform, such as RDoC, although of heuristic value, have not shifted our understanding of nosology or treatment. Although individual diseases may be shelled out of the general syndromes that is treated by a breakthrough in understanding such as was seen with neurosyphilis, an understanding of the aetiology of the majority of mental illnesses has not yielded to a neurobiological explanation, rather, understanding is best achieved through a synthesis of biological with psychosocial factors seen in a developmental context. This complexity makes

psychiatry difficult, but this integration of psychosocial and developmental factors allows for a better conceptualisation of an illness in an individual and personalisation of their treatment plan.

Further Reading

Insel, T. R., & Cuthbert, B. N. (2015). Brain disorders? precisely. *Science*, 348(6234), 499-500.

CLINICAL DISORDERS



Artist: Anne Naylor

Title: *Uncharted Waters*

Medium: Acrylic Paint on Canvas

Dimensions: 600 x 760 mm

Chapter 15: Schizophrenia

Anthony Harris

Specialty of Psychiatry, Sydney Medical School, The University of Sydney, Australia, Westmead Institute for Medical Research, Australia, and Western Sydney Local Health District Mental Health Services, Australia.

Introduction

Schizophrenia is the most common of the group of psychotic illnesses that include schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, bipolar disorder and major depression with psychotic features. In most of our public hospitals people admitted with psychotic disorders form a significant proportion of the total number of admissions and the personal and societal cost of their care is high and increasing in both high and low income countries (Solmi et al., 2023). Schizophrenia is also a highly stigmatised illness, the effect of which has contributed to the under-resourcing of mental health services and the inadequacy of treatments available for people with schizophrenia when they present for the treatment of their physical illnesses.

Epidemiology

Schizophrenia is an uncommon but chronic disorder with a lifetime prevalence of 7.2/1000 (95% CI 3.1 – 27.1) (Saha et al., 2005). The risk of having the disorder is increased by being male, living in an urban environment or migrating into a community that is subject to discrimination. It is an illness with an onset mostly in young adulthood with 64.8% of people having the onset of the illness before the age of 25 years. As you can see from Figure 15.1 it is rare to see schizophrenia in people younger than 15 years, and in those people developing schizophrenia over the age of 30 there is a slight preponderance of women. The onset of the illness mostly occurs over a one to six month timespan. The period prior to presentation is usually marked by a decrease in functioning and withdrawal from family and friends. This period of time is known as the prodrome of the illness. Recent improvements in early intervention services have allowed for a proportion of these individuals to be identified before the clear onset of their full psychotic illness. These individuals are said to have an “at risk mental state”. Intervention at this stage may contribute to a decrease in the number of people who transitioned to a full psychotic illness.

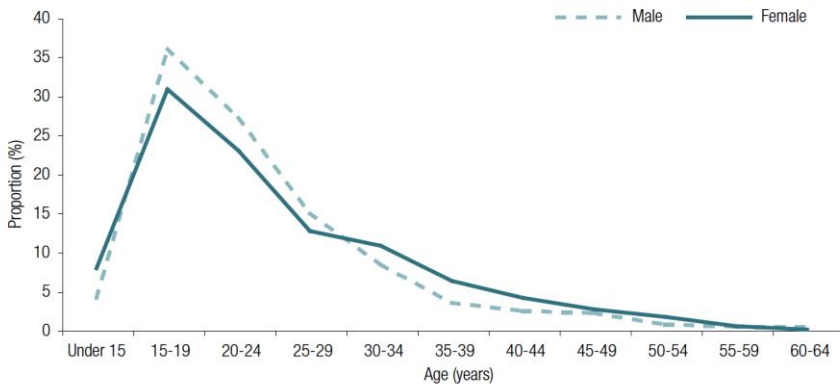


Figure 15.1. Age of onset of psychosis by sex (Morgan et al., 2012a).

Aetiology

Although there is no clear pathophysiology for schizophrenia at present, we know much about the aetiology of the disease. It is genetically based. If your monozygotic twin has schizophrenia your morbid risk of also getting the disease ranges from 41-68%. If, on the other hand, your dizygotic twin has schizophrenia then that risk is decreased to between 0-28% (Cardno and Gottesman, 2000). This information underlines the importance of the genetic contribution to schizophrenia but also indicates a considerable environmental or non-inheritable contribution to the disorder.

Large scale Genome-Wide Association Studies (GWAS) have now identified 287 distinct genetic loci that are associated with schizophrenia (Trubetskoy et al., 2022). These genes, individually of low effect size, code for both presynaptic and postsynaptic functions, in a number of cell types, including interneurons, pyramidal and spiky neurons, and in multiple brain regions. A small proportion of schizophrenia is associated with *de novo* or rare copy number variants that have a large effect and are sometimes a part of other neurodevelopmental disorders such as velo-cardio-facial syndrome. These individuals have a higher incidence of other neurodevelopmental disorders such as developmental delay (Rees et al., 2015).

Schizophrenia was thought to be a disorder of excess dopaminergic activity on the basis that (1) all effective antipsychotic medications block or partially agonise dopamine D2 receptors, (2) the psychotogenic effect of dopamine agonists such as amphetamines or dopamine precursors such as levodopa, and, (3) an increase in presynaptic dopamine synthesis and release in schizophrenia (Howes et al., 2017).

However, other strands of evidence clearly implicate the glutamatergic system and in particular the N-methyl-D-aspartate (NMDA) receptor. This is strongly supported by the GWAS evidence that has identified numerous genes important to glutamatergic and NMDA function. Substances such as ketamine and phencyclidine are also potent glutamatergic agents acting at the NMDA receptor. Intoxication with these substances mimic the positive symptoms of psychosis. However, medications that act via the glutamatergic system have yet to effectively treat schizophrenia in randomised controlled trials.

Substance abuse is frequently implicated in the onset or relapse of psychosis (see Chapter 29). There is a complex association of substances such as cannabis or amphetamines with psychotic symptoms during the phase of intoxication, but also during withdrawal and subsequently with persistent psychotic syndromes. Chronic abuse of substances such as amphetamines or cannabis can result in a chronic psychotic state that is phenomenologically identical to schizophrenia. However, this is by no means inevitable and the majority of people who abuse these substances do not develop schizophrenia. A gene x environment relationship has been identified for cannabis use and an increased risk of the development of schizophrenia in individuals with a specific single nucleotide polymorphism (SNP) – either in the catechol-O-methyltransferase (COMT) gene or the AKT1 gene (Caspi et al., 2005; Di Forti et al., 2012).

Early life stress (ELS) is a significant risk for most psychiatric illnesses and schizophrenia is no exception. These early life stressors can be biological factors that interfere with the normal neurodevelopment of the foetus such as maternal infection, malnutrition such as experienced in the Dutch hunger winter of 1944 or obstetric complications. ELS caused by physical abuse, sexual abuse or neglect during childhood is also associated with schizophrenia and other psychotic disorders (Bailey et al., 2018). The effect of interpersonal stress is probably one of the factors contributing to the increased rate of schizophrenia seen in migrant populations from marginalised communities such as the Afro-Caribbean community in Britain.

These biological, psychological and social factors have a measurable effect upon the brain. Neuroimaging studies have identified widespread reductions in cortical grey and white matter and increases in lateral and 3rd ventricle size in people with schizophrenia (Brugger and Howes, 2017). On a microscopic scale, the cortical grey matter is thinner, with a loss of volume particularly in laminae 2 and 3. There is no decrease in the number of pyramidal cells, but a loss of the neurophil between the cells caused by a decrease in cell arborization – a reduction in cell dendrites and axons. Aberrant cell migration is also seen. There may be a reduction in oligodendrocytes and in white matter volume. Unfortunately, none of these changes are pathognomonic of schizophrenia (Harrison, 1999).

The effect of all this is to disrupt the normal network of connectivity in the brain. This subtle disruption to cortical connectivity is thought to be at the heart of the typical symptoms of schizophrenia such as hallucinations and delusions as well as the loss of function seen with the negative and cognitive symptoms. A useful model of schizophrenia is that of a genetically based neurodevelopmental disorder which requires multiple additional triggers before the actual disorder is manifest. These triggers are diverse and may be an obstetric problem at birth or maternal infection during the first or second trimester. These individuals are then further compromised (or protected) by the quality of their early childhood upbringing and their history of substance use. This model makes sense of the multiple aetiological strands of the illness and, importantly, provides avenues for intervention.

Symptomatology

The signs and symptoms of schizophrenia are diverse, and it is possible for two individuals to both be diagnosed with this illness but share few symptoms. A five-domain model (see Table 15.1) of the signs and symptoms of schizophrenia helps with understanding the management of the disorder and will be used here.

Table 15.1. Five domain model of the signs and symptoms of schizophrenia.

Positive	Negative	Disorganisation	Mood	Cognitive
Hallucinations (e.g., hearing voices)	Lack of motivation Anhedonia	Disorganised thinking and speech	Depression Anxiety	Impaired attention and concentration
Delusions (e.g., persecutory, bizarre, grandiose)	Blunted affect Reduced speech content and output Social withdrawal	Disorganised behaviour Aggression Hostility		Reduced mental flexibility Impaired Memory Impaired Social cognition

The positive symptoms are hallucinations and delusions. A hallucination is a perception in the absence of any external stimulus. They may be perceived in any one of our senses (auditory, visual, olfactory, gustatory, somatosensory e.g., feeling something touch their skin, as well as interoception e.g., feeling something living inside of the person). Although auditory hallucinations are typical of schizophrenia -

experienced in approximately 80% of people with schizophrenia - they are not pathognomonic as they also occur in other pathological states such as dementia, mania or alcoholic hallucinosis. The voices are commonly experienced as originating outside of the person, frequently derogatory or critical and often male. The volume can vary from the softest whisper to shouts and the content can vary from conversation or a voice describing what the person is doing ("She is now lifting a cup") to voices talking about, criticising or commanding the patient. They can echo or speak the person's thoughts out loud. They can persist all day or only occur fleetingly.

Delusions are false unshakeable beliefs out of keeping with the person's cultural and educational background. These beliefs are very varied but commonly include delusions of persecution (that someone is trying to harm them, e.g., the CIA or KGB), of reference – that experiences or sensations in the outside world pertain to the individual (the TV is talking about them); of passivity – that their own thoughts, feeling and actions are taken over by an external agency; of thought alienation – that their own thoughts are being controlled or broadcast out loud, or are being inserted into their mind; of misidentification – that familiar people have been replaced by others (Capgras syndrome). Many other delusional beliefs are possible.

The negative symptoms of schizophrenia describe a loss of function. These include blunted affect in which the emotional expressivity of the person is lost; anhedonia, when the ability to experience emotions, both good or bad, is lost; amotivation, when personal drive or motivation is diminished or lost; asociality, when the wish to be with others is decreased or lost; and alogia where there is a loss of the richness, complexity and/or quantity of thought, leading to a poverty of expression or content of thought.

Disorganisation describes how an individual's behaviour and thought process can be fractured and disorganised. This can be seen in the increasingly chaotic behaviour of people with psychosis which can put them on the streets or renders their homes filthy. It also is associated with an increase in aggression and hostility. Schizophrenia is associated with an increased rate of aggression towards other people particularly in the early stages of the illness. This is exacerbated by substance use. The disorganisation seen in behaviour is also seen in thought processes. Formal thought disorder describes the fragmentation of the coherence of thought as expressed in speech or writing. This can range from minor changes, such as unusual syntax, to the complete breakdown in the expression of thought in word salad, where words are jumbled together in a seemingly meaningless combination.

Schizophrenia is also associated with cognitive problems in both the neurocognitive (attention, concentration, speed of processing, memory and executive functioning) and social cognitive domains (emotion recognition, social knowledge and perception, theory of mind and attributional bias) (Heinrichs and Zakzanis, 1998; Savla et al., 2013). These deficits become apparent during the prodrome when there is frequently a deterioration in the level of cognitive functioning prior to the onset of the first psychotic episode. Cognitive functioning thereafter tends to remain stable despite increasing chronicity. The negative symptoms and the cognitive symptoms of schizophrenia are responsible for the bulk of the loss of function in individuals in our community.

People with schizophrenia have high rates of both anxiety and depression. The anxiety that people with schizophrenia suffer from may arise independently of the psychotic illness causing the full array of anxiety disorders, for example social anxiety disorder, or it can arise secondary to psychotic symptoms. For example, someone with persecutory delusions can feel intensely anxious and under scrutiny as part of their delusional thoughts. Treatment will differ according to if the anxiety is primary or secondary to other psychotic symptoms. People with schizophrenia also have a high rate of post-traumatic stress disorder.

Depression is also common in people with schizophrenia. It can occur as part of the prodrome of schizophrenia, or in response to the stress of having a psychotic illness, or independently as a comorbid condition. Suicide unfortunately ends the lives of approximately 5.6% (95% CI 3.7 – 8.5%) of people with schizophrenia (Palmer et al., 2005) and this tends to occur most frequently in the first one to two years after the diagnosis of the disorder.

A loss of insight, into the idea that the signs and symptoms of schizophrenia are part of an illness is frequent, with between 30-55% of people with schizophrenia having a decrease or lack of awareness of certain symptoms or their level of functioning (Sevy et al., 2004). Finally, schizophrenia is associated with a wide range of movement disorders. Some, such as catatonia, cause changes in the control of movement with people displaying mutism, posturing, negativism, staring, rigidity, and echophenomena (Taylor and Fink, 2003). People with schizophrenia not uncommonly have other movement disorders e.g. tardive dyskinesia, most frequently as an adverse effect of treatment but also sometimes developing prior to the start of treatment.

Diagnosis

Diagnosis requires the clinician to consider symptom profile, duration of symptoms and effect upon function. It requires a full assessment and history

including mental state examination and physical examination (see Table 15.2). There is a considerable differential diagnosis that needs to be considered. Schizophrenia usually presents after a prodrome of between one to six months with a clinical picture of positive, negative and disorganisation symptoms in the context of a deterioration in the level of functioning. The presentation to services is usually precipitated by an exacerbation of the positive symptoms. Occasionally, the negative symptoms of the disease predominate, and the person presents with a picture of social withdrawal, amotivation and severe functional decline.

Table 15.2. Differential diagnosis for psychotic disorders in adults.

Disorder	Typical Symptoms	Duration	Context	Function
At risk mental state (ARMS)	Positive	Attenuated or Fleeting		some
Brief Psychotic Disorder	Positive	< 4 weeks		some
Schizophreniform	Positive, negative	< 6 months		some
Schizoaffective	Positive, negative & mood	> 2 weeks		
Schizophrenia	Positive, negative & cognitive	> 6 months		marked
Substance Induced Psychotic Disorder	Positive	> 2 weeks	Substance Use	varied
Bipolar Disorder	Mood	> 1 week	History of moods symptoms	varied
MDD with psychotic features	Mood	> 2 weeks	History of moods symptoms	
Autoimmune encephalitis	Positive	Rapid onset	Neurological symptoms.	marked
Autistic Spectrum Disorder	Social and communicative	Lifelong	Childhood onset	yes
Schizoid Personality Disorder	Social	Lifelong	Adolescence	

Investigations

Investigations play a two-fold role in the assessment of people with schizophrenia. Firstly, to exclude possible neurological or other organic causes of psychotic illness such as autoimmune encephalitis, complex partial seizures, or thyroid disease. These are relatively rare. Secondly, they serve to provide a baseline for subsequent pharmacological treatment which can cause significant physical morbidity. This includes basic anthropometric information such as cardiovascular parameters and body mass index as well as a range of investigations (see Table 15.3).

Table 15.3. Adapted from the RANZCP Clinical Guidelines for the management of Schizophrenia (Galletly et al., 2016).

Investigation	Baseline	6 monthly	Indication
Physical examination	✓	✓	Monitor physical health, movement disorders
BP, weight, BMI, waist circumference	✓	✓	Monitor weight, metabolic syndrome
Full Blood Count	✓	✓	Blood dyscrasias
Electrolytes, Liver Function Tests	✓	✓	Renal/hepatic dysfunction
Fasting glucose, blood lipids, HbA _{1c}	✓	✓	Metabolic state
Prolactin	✓	✓	Hyperprolactinaemia
Thyroid Function Test	✓	-	Thyroid disease
Anti-NMDA, GAD	✓	-	Autoimmune encephalitis
Urine drug screen	✓	If indicated	Substance abuse
Electrocardiogram	✓	✓	Cardiac arrhythmias, QTc
Electroencephalogram	If indicated	-	Seizure activity
MRI/CT - head	✓	-	Brain pathology
Psychometric testing	✓	-	Cognitive deficits
Blood borne virus	If indicated	-	HIV, liver pathology
Sexually transmitted disease	If indicated		Syphilis, other STD
Pregnancy test	If indicated		Teratogenetic risk

Note: BP = Blood Pressure; BMI = Body Mass Index; HbA_{1c} = Hemoglobin A1c; NMDA = N-Methyl-D-Aspartate (receptor); GAD = Glutamic Acid Decarboxylase; MRI = Magnetic Resonance Imaging; CT = Computed Tomography; STD = Sexually Transmitted Disease; HIV = Human Immunodeficiency Virus.

Management

The biopsychosocial aetiology and broad impact of schizophrenia needs to be managed in a way that addresses the complexity of the disorder. This requires both a pharmacological and psychosocial approach to treatment. Guidelines for the

management of schizophrenia (Galletly et al., 2016) and first episode psychosis (Early Psychosis Guidelines Writing Group and EPPIC National Support Program, 2016) are available and are both excellent resources. Early intervention in psychosis, an approach developed in Australia, reduces the number of hospitalisations and improves functioning and quality of life of young people with psychosis (Correll et al., 2018). Nonetheless, chronicity and disability remain a significant problem and the basic course of the disorder has not been altered.

Pharmacological Management

At initial presentation, it is reasonable to use a benzodiazepine such as diazepam or lorazepam to provide sedation and anxiolysis. However, if the key symptoms of psychosis continue then initiating an antipsychotic medication with a low adverse effect profile and an evidence base in first episode psychosis is recommended (see Figure 15.2) (Galletly et al., 2016). If the clinical situation allows, a slow but steady increase in medication dose over a 2-4 week period is done until there is a significant reduction in symptoms. Continued use of benzodiazepines, to be tapered and discontinued later, can assist in calming the person and helping with sleep. Non-response should prompt a review of other possible aetiological causes and treatment adherence, as around 80% of first episode schizophrenia will respond (Zhu et al., 2017).

Unfortunately, the majority of people with a first episode of schizophrenia will relapse, though this is usually due to ceasing antipsychotic medication. With each relapse, treatment response decreases (Bowtell et al., 2018). Changing to another antipsychotic with a different receptor activity profile is reasonable, along with the use of a long-acting injectable form of medication if available. If three trials of medication have failed to help the person with schizophrenia, or if significant problems with movement disorder or aggression occurs, a trial of clozapine is recommended.

Antipsychotic Adverse Effects

There are many antipsychotic medications available, all with differing profiles of adverse effects (see Table 15.4). As schizophrenia usually warrants long term treatment, it is important to anticipate possible problems and promote the long-term health of the individual from the time of first treatment with education, exercise and diet. Common adverse effects (Huhn et al., 2019) of medication include weight gain (particularly chlorpromazine, olanzapine, clozapine and quetiapine), movement disorders such as dystonia, parkinsonism, akathisia and tardive dyskinesia (chlorpromazine, haloperidol, zuclopenthixol), raised prolactin (haloperidol, amisulpride, risperidone, paliperidone), sedation (clozapine,

olanzapine, chlorpromazine, quetiapine) and QTc changes (amisulpride and ziprasidone).

Rarer or unusual adverse effects such as blood dyscrasias, neuroleptic malignant syndrome, phototoxicity or the propensity to water intoxication need to be checked for. However, the use of antipsychotics with a heavier burden of adverse effects, may be required because of a poor response to first-line medications.

Clozapine is a highly effective medication that has been relegated to the third line because of the range and severity of adverse effects. Of most concern is its propensity to cause agranulocytosis in approximately 1 to 2% of people taking it. Because of this, a compulsory monitoring system has been developed to ensure that all people on clozapine have regular full blood counts. In addition, clozapine can cause myocarditis and cardiomyopathy requiring close cardiac monitoring (initially cardiac enzymes and ECG, followed by regular ECG and echocardiogram). Clozapine's other adverse effects are numerous and can be dangerous as well as inconvenient, for example severe constipation.

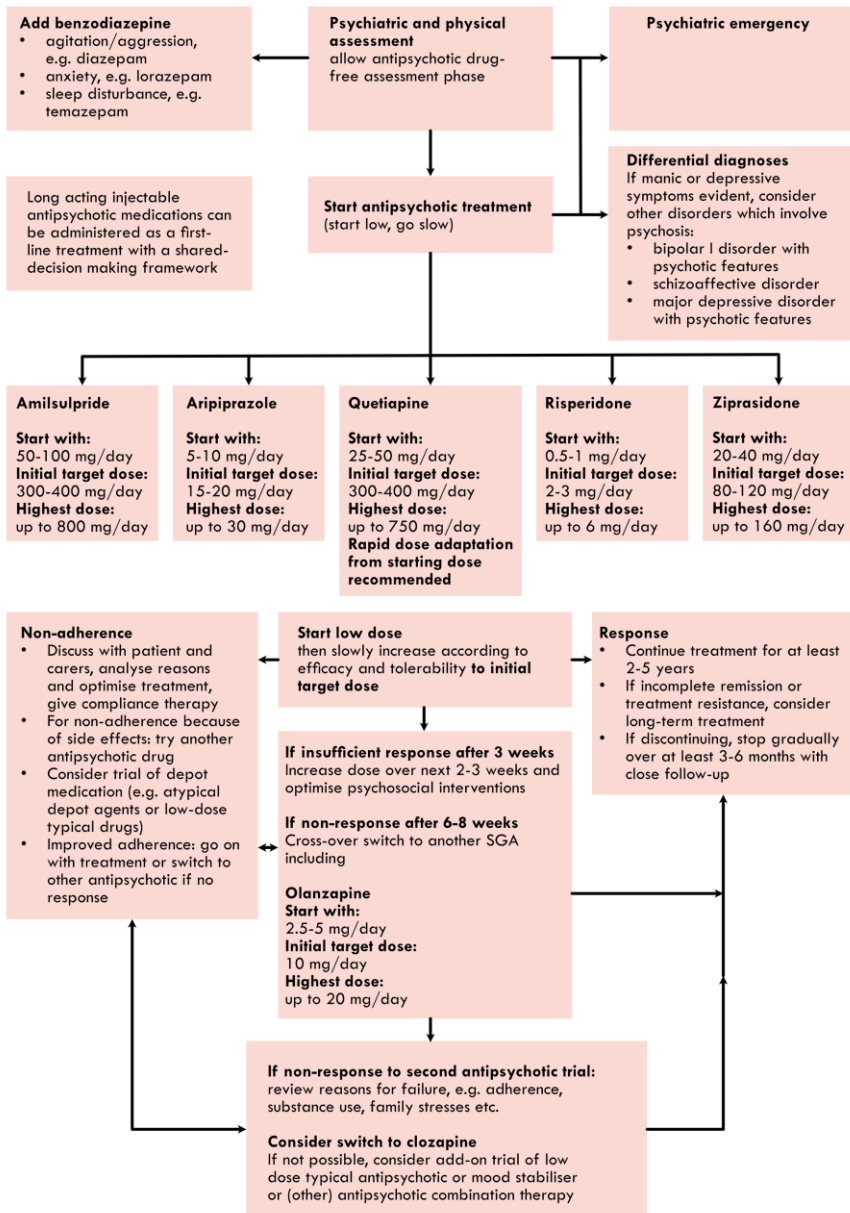


Figure 15.2. From Galletly et al. (2016). Treatment algorithm for first episode schizophrenia.

Psychosocial Management

Psychosocial management describes a range of interventions that aim to engage people in treatment, educate them about the symptoms and problems caused by the disorder, provide them with the cognitive and behavioural tools to tackle their disabilities, and monitor their progress. These treatments are best provided by a multidisciplinary team, including peer workers.

Treatment is built around a case manager whose role it is to remain in close contact with the person with schizophrenia and help coordinate the many aspects of care. This can be provided assertively for people with a record of multiple hospitalisations and difficulty in engagement so as to reduce readmission rates (Burns et al., 2007). Accurate information is essential in an area such as mental health and psychoeducation provides information about aetiology, treatment and the mental health care system. This lowers hospitalisation and improves satisfaction (Xia et al., 2011). Schizophrenia is corrosive of family ties. Many families benefit from family therapy that aims to educate, provide problems solving skills and reduce miscommunication and the resultant arguments or family dysfunction (Bighelli et al., 2021). Cognitive behavioural therapy (see Chapter 33) can be used to challenge pathological thoughts and perceptions such as hallucinations and delusions. It has also been used to improve medication adherence (Bighelli et al., 2021; Gray et al., 2016).

Table 15.4. Antipsychotic Medication Adverse effects.

	Sedation	Weight Gain	EPSE	Anti - cholinergic	Prolactin	QTc
Older D2 antagonists						
Chlorpromazine	+++	+++	++	+++	+++	++
Flupentixol	+	++	++	++	+++	+
Haloperidol	+	++	++ +	+	+++	++
Zuclopenthixol	+++	++	++	++	+++	?
D2 -5HT _{2a} antagonists						
Asenapine	++	++	+	-	+/-	+
Clozapine	++++	+++	0	XXX	0	+
Lurasidone	++	+	+	-	+	-
Olanzapine	+++	+++	+	++	+	+
Paliperidone	+	++	+	-	+++	+
Quetiapine	++	++	+	-	+	++
Risperidone	+	++	++	-	+++	+
Ziprasidone	+	+/-	+	-	0	++
Benzamide antipsychotic						
Amisulpride	+	+	++	-	+++	++
D2 partial agonists						
Aripiprazole	+/-	+/-	+	-	0	+
Brexipiprazole	+/-	+/-	+	-	+/-	-
Cariprazine	+	+	+	-	-	-

Note: EPSE – extrapyramidal side effects, for example parkinsonism, rigidity, tremor, dystonia and propensity to tardive dyskinesia; anticholinergic – dry mouth, blurry eyes, dilated pupils, constipation, urinary hesitancy or retention, decreased sweating; prolactin – hyperprolactinaemia causing breast tenderness or enlargement, galactorrhoea and menstrual irregularity, long term risk of osteoporosis; QTc – corrected QT interval, indication of risk of torsades de pointes cardiac rhythm; XXX – hypersalivation despite high anticholinergic effects.

An important goal for many is to return to work (Morgan et al., 2011). The most successful approach for this in people with schizophrenia is supported employment – more specifically known as the Individual Placement and Support model - that places people directly in employment and supports them in the workplace (Modini et al., 2016). Cognitive Remediation Therapy, a therapy that

targets the significant cognitive dysfunction that is part of psychotic illnesses, improves both neurocognition and social cognition. This flows on to an improvement in community functioning, especially when combined with other psychosocial interventions e.g. supported employment (Vita et al., 2021).

Anxiety and Depression

Anxiety and depression form one of the domains of symptomatology and there are increased rates of both major depression (see Chapter 16) and anxiety disorders (see Chapter 18) in people with schizophrenia compared to the rest of the community (Buckley et al., 2009). Suicide ends the life of approximately 5% of people with schizophrenia (Palmer et al., 2005). Diagnosis of these disorders is frequently overshadowed by psychotic symptoms. Treatment with CBT (Chapter 33), pharmacotherapy (Chapter 35) or both is appropriate and effective.

Physical Health

People with schizophrenia die on average 14.5 years earlier than the rest of the community (Hjorthøj et al., 2017). This decrease in life expectancy is not just due to suicide and accident, rather it relates to a broad range of chronic medical conditions including infections, cardiovascular, respiratory, endocrine and substance use (Laursen et al., 2019). There are a number of reasons for this. Adverse effects of psychotropic drugs contribute to an increase weight and the frequency of metabolic syndrome, though overall treatment adherence is associated with a lower morbidity and mortality rate (Taipale et al., 2020). There is a higher rate of substance abuse including nicotine in people with schizophrenia (Firth et al., 2019). Lifestyles are less healthy with less sleep, exercise and a poorer diet (Morgan et al., 2011; Firth et al., 2019). However, despite coming into contact with medical practitioners more often than the rest of the community, they are less likely to have routine investigations, diagnostic procedures or interventions for illnesses such as cardiovascular disease when in care, which reflects physician behaviour and stigma (Mitchell et al., 2012; Solmi et al., 2021). Ironically, lifestyle interventions such as diet and exercise are effective for people with schizophrenia (Teasdale et al., 2017) and they adhere to treatments for their physical illnesses just as well as the rest of the community (Owen-Smith et al., 2016), they are just not tried.

Outcome

The majority of people with schizophrenia have a reasonable outcome with symptomatic improvement but a less certain functional recovery. Recent work suggests that multimodal treatment and early intervention improves outcome (Lally et al., 2017). However this is yet to change long term outcome which has remained

stubbornly poor with only 13.5% of people with schizophrenia recovering when recovery is defined as a remission of symptoms and a return of function in social, occupational and educational domains (Jääskeläinen et al., 2012). This is almost certainly contributed to by the demands of our developed world as outcome is better in developing countries. Poor treatment adherence and substance use often leads to relapse and the development of chronicity. Also, around 20% of people with schizophrenia never respond to treatment even at their first presentation to services (Siskind et al., 2022). These individuals are more likely to be male, with poor education and a long duration of undiagnosed psychosis (Morgan et al., 2021). This clinical picture, along with strong consumer advocacy, has led to the adoption of recovery-led practice models of care that focus less on symptomatic remission and more on the development of a meaningful and contributing life (Australian Health Ministers' Advisory Council, 2013).

Conclusion

Schizophrenia is the most common of the psychotic illnesses. It develops early in adult life disabling the majority who have it, isolating them from family and friends and interrupting relationships, education and employment. We have discovered much about its underlying causes, however this has not significantly altered outcome. Management requires a multidisciplinary team with both pharmacological and psychosocial interventions. Finally, schizophrenia contributes to an early death due to physical illness and poor care. Neglect based upon stigma and marginalisation continue to affect those who have the disorder.

Further Reading

- Galletly, C., Castle, D., Dark, F., Humberstone, V., Jablensky, A., Killackey, E., ... & Tran, N. (2016). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the management of schizophrenia and related disorders. *Australian & New Zealand Journal of Psychiatry*, 50(5), 410-472.
- McCutcheon, R. A., Abi-Dargham, A., & Howes, O. D. (2019). Schizophrenia, dopamine and the striatum: from biology to symptoms. *Trends in Neurosciences*, 42(3), 205-220.

Chapter 16: Depressive Disorders

Gin S. Malhi

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia, Visiting Professor, Department of Psychiatry, The University of Oxford, UK, and Oxford Uehiro Centre for Practical Ethics, Faculty of Philosophy, The University of Oxford, Oxford, UK.

Erica Bell

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

and

Philip Boyce

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia, Westmead Institute of Medical Research, Australia, and Faculty of Health, Charles Darwin University, Australia.

Introduction

Depression is a normal human emotion; we can all feel depressed or experience a low mood when confronted with adverse experiences, such as the loss of a relationship or an occupation. Generally, such low mood is transient and is not considered to be a ‘disorder’ per se. However, when a low mood is accompanied by other symptoms, is persistent (lasting over 2 weeks) and leads to some functional impairment, a clinically significant depressive disorder can be diagnosed.

DSM-5-TR refers to clinically significant depression as major depressive disorder (American Psychiatric Association, 2022). It is one of the most common psychiatric disorders (see *Epidemiology*) and is treatable. However, depression is often not identified or recognised, and effective treatments are delayed or missed altogether. This is especially problematic, as depression has widespread and significant impacts on the individual affected, their family, relationships and the wider community (see Table 16.1).

Table 16.1. Impact of depression.

Individual	Suffering, distress, and poor quality of life
	Increased risk of suicide
	Impaired work performance
	Lower income
	Poor academic achievement
	Adverse impact on medical problems (worsened illness course, increased disability, and increased mortality)
	Increased all-cause mortality
Social network	Reduced wellbeing of families and friends
	Disturbed inter-personal relationships
Community	Absenteeism and reduced work productivity
	Increased medical care costs

Epidemiology

Depression is one of the leading causes of disease burden worldwide because of prolonged morbidity and functional impairment, as such it is an important public health issue (Vos et al., 2020). There have been considerable efforts globally to raise awareness about depression and to destigmatise both the diagnosis and its treatment. However, globally, the 12-month prevalence of major depressive disorder (MDD), remains high (7.2%) and the prevalence of MDD over a lifetime is approximately double this (7.5-13.6%) (McGrath et al., 2023; Lim et al., 2018). Depression is found in all cultures, across all countries, and is twice as common in women as men - in contrast to bipolar disorder (Kuehner, 2017; McGrath et al., 2023). In Australia, approximately one in five people will suffer from an episode of depression in their lifetime (Australian Institute of Health and Welfare, 2023) and many will attempt suicide (Australian Institute of Health and Welfare, 2022c).

Aetiology

The aetiology of depression is multifactorial. Both societal and environmental factors play a significant role in the development of depression, as well as contributions from biological and genetic factors. These influences interact

via epigenetic mechanisms and therefore while the illness is familial, its incidence in a particular individual is difficult to predict.

One explanation for depression has been the serotonin hypothesis (Moncrieff et al., 2022). This emerged from the serendipitous discovery the monoamine oxidase inhibitors (MAOIs) lead to remission of depressive symptoms in the 1950s, and the subsequent finding that the tricyclic antidepressants are effective (Malhi and Mann, 2018). The drugs have an effect on transmission of monoamines (serotonin and noradrenaline) in the brain. The subsequent development of drugs that target serotonin receptors (the SSRIs) lead to the serotonin hypothesis; that depression was the result of defective central serotonin transmission. This hypothesis has been used as an explanation to patients as a rationale for the prescription of an antidepressant. However, this model has now been superseded by a number of other hypotheses that relate to stress (the HPA axis), genetic and epigenetic phenomena, immunological causes, and the role of trauma.

The development of depression is perhaps best understood using a stress diathesis model; a depressive episode is triggered by an adverse life event (stress) in those that have a vulnerability (diathesis) to developing depression. As mentioned above, the vulnerability may be biological (genetic), social (here social inequality is a key factor) or a variety of psychological vulnerabilities (such as low self-esteem or having experienced adverse childhood experiences).

Onset

Most commonly depression presents in primary care, manifesting initially from mid-adolescence through to the fourth and fifth decade of life. The illness is therefore not uncommon in the third decade of life (twenties), but a significant number of individuals will experience their first episode of depression before they turn 20 (Solmi et al., 2022). The onset of depression can be insidious but can also be sudden when triggered by significant life events.

Depression in Primary Care

As noted above, people first present with depression in primary care. Indeed, depression is one of the most common reasons for which people seek a GP appointment. However, sometimes the person presents with another illness or with somatic symptoms associated with depression as their primary complaint. It is estimated that over 50% of people with depression presenting in primary care do not have the illness recognised by their GP, thereby delaying or missing out on

appropriate treatment. Furthermore, of those that have their depression identified, only 50% will be adequately treated (Marx et al., 2023).

Diagnosis

A depressed mood is a normal human experience, something that can happen to all people when an adverse experience happens such as the breakup of a relationship, the loss of a job, or a bereavement. What differentiates this normal human emotion from a depressive illness is the presence of additional symptoms, the persistence of the low mood and the duration it has been present for. The incidence and severity of depressive symptoms varies. The key symptoms that are used to define depression are shown in Table 16.2 (see also, Figure 17.1 within Chapter 17: Bipolar Disorder), 5 symptoms are required to be present to qualify for the diagnosis. When present concurrently these symptoms constitute a depressive syndrome, and if present on most days (persistence) for a period of at least two weeks (duration) they then qualify for designation as a depressive episode. Depressive episodes are the basis of major depressive disorder which is often a lifelong recurrent disorder.

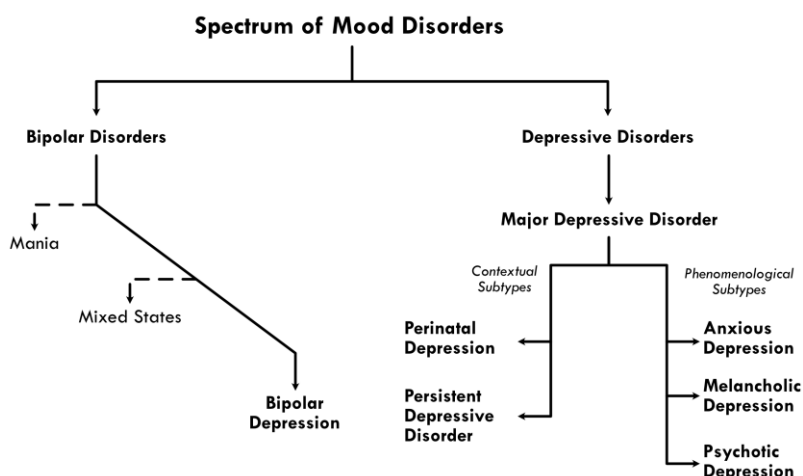


Figure 16.1. Putative types of depressive disorders. This schematic organises some of the various 'types' of depression. It is important to note that this is not comprehensive and that only recognised examples of depressive disorders that are commonly encountered in clinical practice are shown.

It is important to note that major depressive disorder is an umbrella term that encompasses different 'types' of depression. That is to say, depressive syndromes that vary in terms of their prevailing symptoms or distinguishing features

such as context within which the depression has manifested. In the past, depression was subdivided into 2 types; one with a ‘biological underpinning’ called endogenous depression (or melancholia) and the other, arising from psychosocial factors. The validity of this binary distinction has not been confirmed, and currently it is recognised that depression is a heterogeneous disorder. The key types of depression are listed within Figure 16.1, but it is important to note that these types are purely based on phenomenology and are not discrete, separate kinds of depression.

Clinically and in research, the diagnosis of depression is therefore regarded categorically even though individual symptoms are dimensional, and the illness (major depressive disorder) exists on a spectrum of mood disorders. Therefore, in practice, qualification for a diagnosis of major depressive disorder also requires a degree of distress and functional impairment that distinguishes it from normalcy. Further specification of depressive disorders is based on severity (mild, moderate, and severe) and syndromal subtypes (e.g., melancholic, psychotic) – and both specifiers may be used to refine management.

In this context, the Activity, Cognition and Emotion (ACE) model (Malhi et al., 2018b) is useful as it underscores both the dimensional nature of depressive symptoms and the need for adopting a longitudinal perspective when assessing mood disorders (see Bipolar chapter for further detail). In practice, an emphasis on careful assessment is critical as there are no biomarkers that can supplant clinical acumen.

Diagnostic criteria for major depressive disorder

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

1. Depressed mood*.
2. Markedly diminished interest or pleasure in all, or almost all, activities (anhedonia)*.
3. Significant changes in weight or appetite (either increased or decreased).
4. Significant changes in sleep (insomnia or hypersomnia).
5. Psychomotor agitation or retardation (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional).
8. Diminished ability to think or concentrate, or indecisiveness.
9. Recurrent thoughts of death (not just fear of dying); recurrent suicidal ideation without a specific plan; a specific suicide plan; or a suicide attempt.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or another medical condition.

D. At least one major depressive episode is not better explained by a psychotic disorder.

E. There has never been a manic episode or a hypomanic episode.

* Key symptoms, either depressed mood or anhedonia must be present.

Course and Impact

The course of depression is equally varied as is its diagnosis, but, by and large, it is an episodic disorder and patients tend to feel reasonably well between episodes. However, it is important to note once depression has manifested it is never cured *per se* and that an underlying vulnerability for depression remains lifelong.

Typically, untreated episodes of depression last several months but can extend to years, and even with successful treatment complete recovery can take up to a year. Even so, following recovery, the likelihood of recurrence remains high, and up to 50% of patients with depression will experience further episodes of

depression during their lifetime. The likelihood of a favourable outcome reduces with age and with older age of onset. Further, with each episode the probability of further recurrences increases. Thus, overall, roughly 50% of those experiencing a major depressive episode will recover within 6 months but more than a quarter will remain unwell and develop a chronic depressive disorder (Angst et al., 2009; Boschloo et al., 2014; Hengartner, 2020). This is partly why depression confers a high global burden of disease, and why by 2030 it will be the leading cause of burden of disease worldwide (World Health Organization, 2011).

Management of Depression

Diagnosis and Formulation

Taking a thorough history, ideally over a period of time, which allows the observation of symptoms, and the gathering of collateral history is the key to making an accurate diagnosis and fully understanding depression. To make sense of the illness, a complete picture of the person's life is needed. So, after assessing the current symptoms (*presenting complaint*) and how these have emerged (*history of presenting complaint*) and reviewing the course of the illness (*past psychiatric history*), it is important to put this in context, with respect to the development of the individual, their relationships, and roles. The components of taking a clinical history are shown in Figure 16.2.

Understanding the nature of depression, how it has emerged, and how it can be best treated, is essential for patient engagement and for ensuring optimal outcomes. It helps develop a comprehensive and effective management plan. Once a clinical picture of the current depressive episode and its clinical context is formed, it is possible to consider the degree to which its aetiology and pathogenesis is driven by biological and/or psychological factors in combination with personal, social and lifestyle determinants. This informs treatment.

In most cases, and especially where there are many psychosocial contributory factors to the depressive illness, the initial steps of management should involve *Actions* (see Figure 16.3). Note, treatment with medications is not always necessary and is considered later under *Choices* and *Alternatives* (Malhi et al., 2021b).

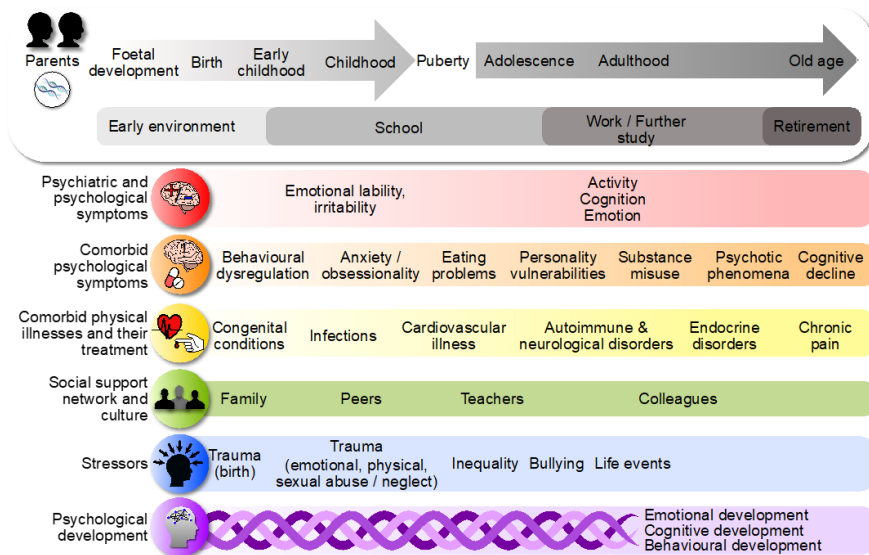


Figure 16.2. Framework for assessing clinical factors that contribute to depression, adapted from Malhi et al. (2020b). The growth and development of an individual is depicted at the top of the figure and this 'life-line' provides a timeframe for changes across several domains. The domains are shown on the left and the key aspects that should be considered and assessed are depicted according to stage of life.

In many cases of depression, changes to lifestyle, the removal of stressors and the use of evidence-based psychological interventions alongside psychoeducation, are sufficient to manage uncomplicated depressive symptoms. In patients where pharmacotherapy is needed from the outset, because of symptom severity or patient preference, it is important to ensure that *Actions* have been undertaken (Malhi et al., 2021b). This is because psychological therapies alongside pharmacotherapy enhance outcome, and adverse social and lifestyle factors often maintain depression. Furthermore, many of the *Actions* are likely to optimise the pharmacotherapeutic response and therefore it is essential that they are given full consideration at the outset of management and subsequently maintained throughout.

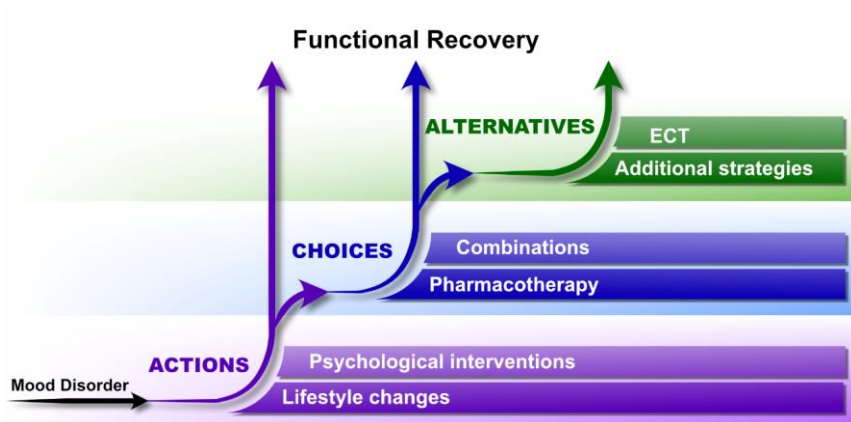


Figure 16.3. Actions, Choices, and Alternatives framework for managing mood disorders, adapted from Malhi et al. (2020b). This framework includes 3 components. 1) *Actions* – form the basic foundation of management and should be instituted whenever possible. They include lifestyle changes and psychological interventions. 2) *Choices* – involve those pharmacotherapeutic options that are recommended and should be trialled initially. 3) *Alternatives* – include complex medication strategies and physical treatments such as ECT. Functional recovery can be achieved at any point, and the aim should be to achieve this as quickly and effectively as possible.

Actions

In the management of depression, *Actions* involve the development of good habits, gaining knowledge, and measuring and facilitating the development of new coping skills. These are outlined in Figure 16.4, and amongst these, note that evidence-based psychological interventions require delivery by trained clinicians, typically psychiatrists, psychologists, and primary care physicians (general practitioners) with an interest in psychological therapies.



Figure 16.4. Actions for managing depression, adapted from Malhi et al. (2020b). There are three groups of Actions that can facilitate functional recovery. The first are those the patient needs to institute, such as lifestyle changes. These include sleep hygiene, maintaining a healthy diet and taking regular exercise. The second are those the patient needs to address as a priority – although they may require assistance – such as limiting drinking and the cessation of smoking. The third are those that need to be implemented. These include psychoeducation and psychological treatment. In addition, it is important to consider the social needs of the individual, and how these are impacting their depressive illness, and assist with these if possible (e.g., referring to social worker). Finally, the assessment (measurement) of clinical symptoms, and especially those related to any risks, and an appraisal of overall outcome are important.

Psychological Treatments

Several evidence-based psychological treatments have substantive support for acute major depressive episodes, with emerging evidence of longer-term benefits. Therapies, such as cognitive behavioural therapy (CBT), Interpersonal Therapy (IPT), Problem-solving therapy (PST), Behavioural activation therapy (BAT), Nondirective supportive therapy, and Short-term psychodynamic psychotherapy (STPP) have been found to be effective. The UK NICE guideline that considered cost and access alongside effectiveness ranked interventions for major depressive disorder having divided the depressive spectrum into less and more severe (NICE, 2022) (see Figure 16.5). Notably, it ranked group interventions as being better for less severe depression and counselling (individual supportive therapy) as being better than STPP (Malhi et al., 2022). But overall, CBT is the primary recommended approach for acute MDD.

Treatment options listed in order of recommended use for:

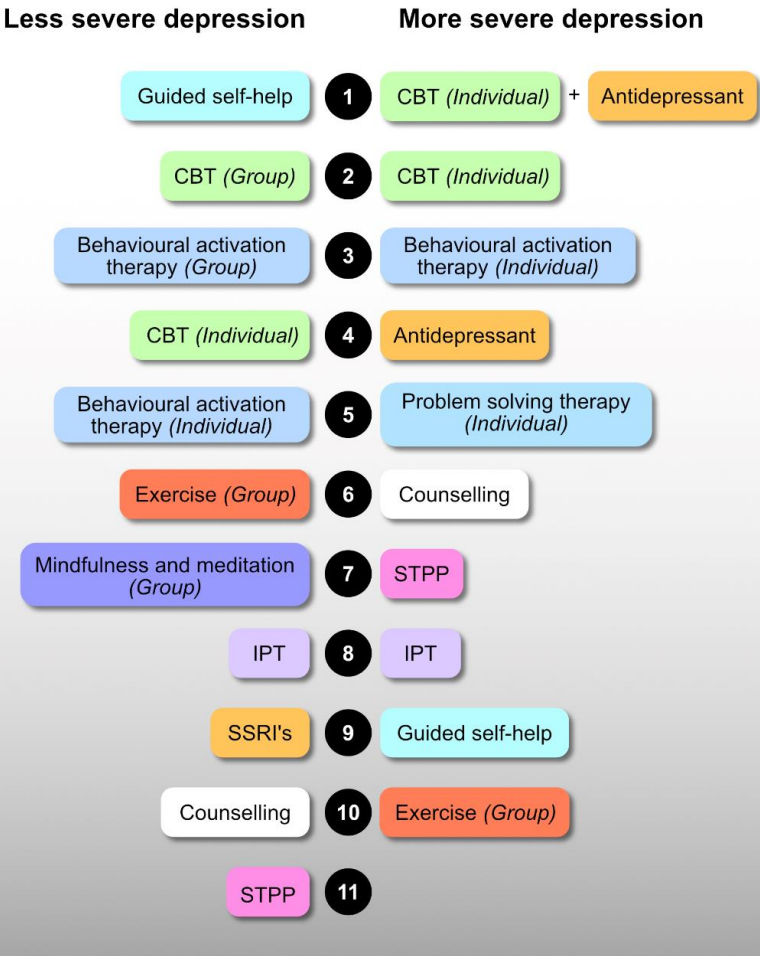


Figure 16.5. Schematic illustrating treatments ordered by recommended use according to the 2022 NICE guidelines, adapted from Malhi et al. (2023). Treatments are identified by separate colours, with mode of delivery shown in italics. In addition, SSRIs and the broader category of antidepressants are shown in the same colours. CBT = cognitive behavioural therapy; IPT = Interpersonal therapy; SSRI's = selective serotonin reuptake inhibitors.

Reflecting the rapidly changing delivery landscape (e.g., during and post-pandemic), the 2020 RANZCP guidelines include detailed consideration of the digital delivery of psychological therapies (interventions offered on computer,

tablet, or smart phone). Extant evidence suggests that digitally delivered interventions may have equivalent efficacy to in-person treatment for major depressive disorder but with significant cost benefits. This is particularly important in remote areas where there is limited access to mental health professionals. Even so, clinically, in-person treatment remains essential and is often preferred by both patients and clinicians, highlighting the relative strengths and weaknesses of in-person versus digital delivery for different patients and across various phases of management.

Choices

If the above *Actions* are insufficient and are unable to produce remission, and the patient is willing to consider medication, then an antidepressant should be prescribed. In some cases, an antidepressant may be prescribed from the outset, especially if this has been necessary in the past and this should be combined with actions. Hence, it is important to determine whether the patient has had previous treatment. When selecting an antidepressant, there are two main considerations: *efficacy* and *tolerability*¹.

Efficacy

All antidepressants have been shown to be better than placebo², but clinically there is a range of effects. The differential activity of antidepressants is evident both clinically and in research studies. Generally, medications with a broader spectrum of actions, such as the tricyclics (e.g., amitriptyline), appear to be more efficacious than those that are selective. However, agents with a broad mechanism of action are also more likely to have side effects that reduce compliance and effectiveness.

The efficacy of antidepressants is also dependent on depression severity, subtype, and the clinical profile of symptoms the person is experiencing. Therefore, efficacy should not be the only consideration when selecting an antidepressant.

The most commonly prescribed classes of antidepressants in Australia are the serotonin reuptake inhibitors (SSRIs), followed by the dual acting serotonin and noradrenaline reuptake inhibitors (SNRIs).

¹ Note: *effectiveness* is a combination of the two, and it is *not* synonymous with efficacy.

² Note: superiority over placebo is required for regulatory approval.

Table 16.2. Pharmacological treatment based on clinical profile. Considerations must also include efficacy and tolerability, depressive subtype and severity, past treatment history and responsiveness, and patient preference.

Key/Prominent Symptom(s)	Antidepressant
Anxiety	Escitalopram Venlafaxine
Cognitive difficulties (e.g., learning, memory, decision-making)	Vortioxetine
Fatigue	Bupropion
Melancholia (e.g., psychomotor slowing, diurnal mood variation)	Amitriptyline Venlafaxine
Pain	Amitriptyline
Sleep disturbances (e.g., Insomnia)	Agomelatine Mirtazapine

Tolerability

Tolerability is equally important as efficacy, and adherence to medication is a critical factor in treatment success. While antidepressants are frequently prescribed, up to 50% of people do not continue them (Sansone and Sansone, 2012). When commencing antidepressant therapy, patients should be advised of potential side-effects and asked to be vigilant and monitor not only their symptoms, but also how they react to their medication. The key side effects of the major classes of antidepressants are shown in Table 16.3. It is imperative that patients are informed of the time needed for an antidepressant to work (at least 2 weeks) and that they need to be taken regularly. They should be encouraged to report side effects promptly but not necessarily stop medication – unless the side effects are intolerable. Instead, they should seek prompt assistance and review by their doctor.

In practice, once antidepressant therapy has been initiated, it is useful to schedule a follow-up appointment within 2 weeks. Most side effects occur within the first week of treatment by which time steady state serum levels of most antidepressants will have been reached. A ‘start low’ and ‘go slow’ approach to dosing can be adopted for patients of advanced age, or those who have a history of poor medication tolerance or are anxious about taking medication.

The most common side-effects reported by patients include gastrointestinal symptoms (nausea and diarrhoea), sexual dysfunction, sedation, weight gain, anxiety, over-stimulation, and agitation (see Table 17.3). It is important to consider

potential interactions with other medications and sometimes the risk of suicide needs to be factored into prescription choice as some antidepressants are potentially more dangerous than others in high doses – as occurs in an overdose (e.g., tricyclic antidepressants).

Table 16.3. Side effects associated with common antidepressant classes, adapted from Malhi et al. (2015).

	Weight gain	Sexual dysfunction	CNS effect (e.g. sedation, fatigue, agitation)	Anticholinergic effect (e.g. tremor, dry mouth)	GI distress
SSRI	+	+++	++	++	++
NARI	++	+	+	+	++
NaSSA	++	++	+++	++	+
Melatonergic agonist	+	+	+	+	+
NDRI	++	+	++	++	+
SNRI	+	+++	++	++	++
TCA	++	+	+++	+++	+
MAOI*	+	+	+	+	+
SARI	+	+	+++	+	++

Note: + < 10%, ++ = 10-30%, +++ > 30%. SSRI = Selective Serotonin Reuptake Inhibitor; NARI = Norepinephrine and Dopamine Reuptake Inhibitor; NaSSA = Noradrenergic and Specific Serotonergic Antidepressant; Melatonergic agonist = Melatonergic Agonist; NDRI = Norepinephrine-Dopamine Reuptake Inhibitor; SNRI = Serotonin-Norepinephrine Reuptake Inhibitor; TCA = Tricyclic Antidepressant; MAOI = Monoamine Oxidase Inhibitor; SARI = Serotonin Antagonist and Reuptake Inhibitor.*Some combinations of MAOIs with other medications can cause serious side effects and on occasion be fatal. Therefore, contraindications and dietary restrictions must be reviewed and adhered to. See ‘Further reading’ section at the end of this chapter for additional details.

Selecting an Antidepressant

The actions of antidepressants should be tailored to the clinical profile of the patient. This requires an understanding of the pharmacological actions of different medications (see Table 16.3). Figure 16.6 illustrates the seven drugs recommended as *Choice* agents. Each of these has a slightly different pharmacological profile, which means they each have a unique effect as regards efficacy and tolerability. Together, the seven antidepressants provide a range of mechanisms of action. It is important to note that monoaminoxidase inhibition is *not* represented by these 7 agents. Inhibitors of this enzyme provide a broad spectrum of efficacy, but

because antidepressants in this class have many interactions, they are regarded as an *Alternative*, rather than a *Choice* antidepressant.



Figure 16.6. Pharmacotherapy Choices for the management of depression, adapted from Malhi et al. (2020b). The *Choice* antidepressants each have different mechanisms of action. Broadly, all of them have actions on monoaminergic systems, with some medications having additional effects. The different receptor-binding profile of each antidepressant means that there is some separation in terms of clinical effects – both efficacy and tolerability. Knowledge of the different mechanisms of action is important as it provides a basis for understanding the different clinical effects and profiles of these medications. The seven *Choice* antidepressants are depicted according to their relative efficacy and tolerability, but selection should also consider clinical profile, depressive subtype, illness severity, past treatment history, prior responsiveness, and patient preference.

Alternatives

There are now nearly 30 medications that have antidepressant properties and have an indication for the treatment of depression. Therefore, there are multiple choices and options available. However, in many cases, these interventions are either ineffective or insufficient on their own, and this means that additional, *Alternative* strategies have to be employed.

Chief amongst these alternatives is the combination of the various types of treatment that have been discussed. As has already been emphasised, the combination of psychological interventions with medications is most effective and preferable where possible. And ideally, this should occur in the context of lifestyle and habit-transforming *Actions*.

In some cases of depression these steps do not produce a satisfactory response, and the patient is unable to achieve remission and recovery. In these

instances, *Alternatives* are necessary. These are summarised in Figure 16.7 and can be remembered using the acronym *MIDAS*.

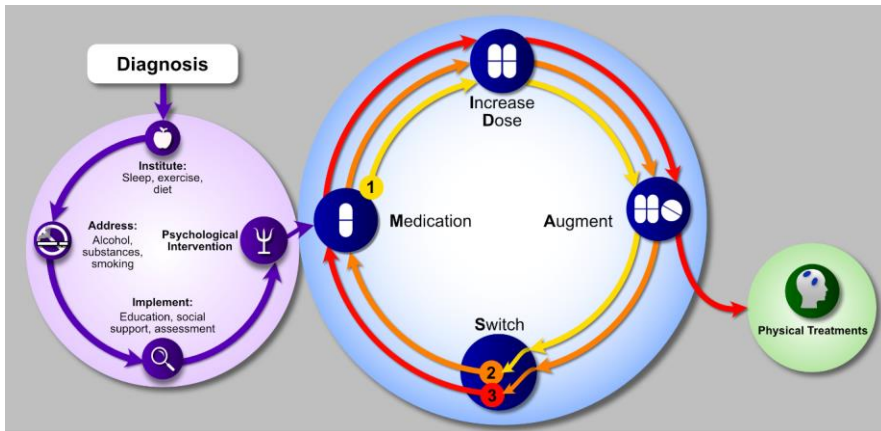


Figure 16.7. MIDAS approach, adapted from Malhi et al. (2020b). This figure provides an overview of the management of depression. Following accurate diagnosis and clinical formulation, *Actions* are instituted, and psychological interventions are administered on their own or alongside pharmacotherapy. *Medication* if needed is selected to suit the clinical profile of the depressive presentation as per the Choices available and according to effectiveness. The first antidepressant (1) is then prescribed and the dose of this can be increased (*Increase Dose, ID*) if necessary. If this is insufficient, then *Augmentation* can be considered. This involves the addition of an agent such as lithium, which augments the actions of the antidepressant. If this strategy is ineffective, then the antidepressant needs to be substituted. *Switching* the antidepressant should ideally involve shifting to another class altogether (e.g., a different *Choice* antidepressant medication), but switching within class is acceptable if the main reason for doing so is that the initial medication was not taken as prescribed (e.g., because of side effects or poor adherence). The MIDAS cycle should be repeated for as many of the *Choice* antidepressants as possible (and at least 3) before considering ECT. However, ECT may be administered much earlier in some cases, for example where depression is marked by psychotic symptoms or during pregnancy.

Physical Treatments

Many kinds of physical treatments have been developed and trialled for the treatment of depression. Most are still experimental, and none are as effective as electroconvulsive therapy (ECT; see Chapter 36). Figure 16.8 shows progression through several variations of ECT, in which the stimulus intensity and electrode

placement is altered. Note, with increasing efficacy, there is the increasing likelihood of transient cognitive side-effects. Therefore, in practice, whenever possible, brief, or ultra-brief pulse unilateral ECT should be trialled initially. ECT is especially useful in psychotic depression, and depression with melancholic features. ECT can also be administered as a matter of urgency where a patient is refusing oral medication and there is an urgent need for treatment because of inanition and dehydration. Importantly, ECT is safe in pregnancy, and may sometimes be the preferred choice.

Patients will need to give informed consent for ECT. If they do not have the capacity to provide this, or if they are an involuntary patient, then an order for involuntary treatment with ECT can be made under the appropriate Mental Health Act. Patients will need to have an anaesthetic review prior to the administration of ECT to ensure their fitness for an anaesthetic.

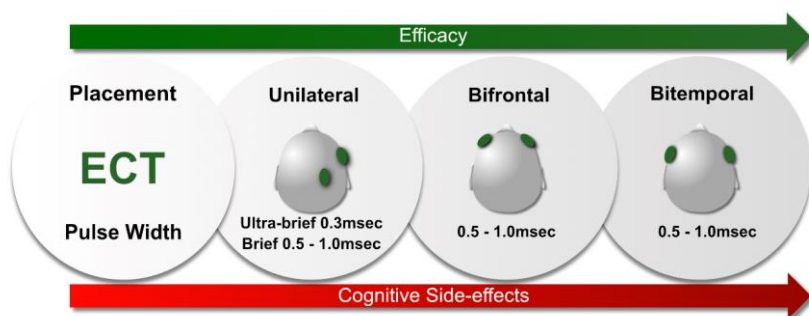


Figure 16.8. ECT in depression, adapted from Malhi et al. (2020b). ECT can be applied unilaterally or bilaterally, over frontal and temporal regions (bifrontal and bitemporal, respectively). The pulse-width can be varied from 0.3- 1.0 msec, allowing different amounts of stimulation to be applied. Efficacy increases from unilateral to bilateral ECT and with an increase in pulse-width from ultra-brief to brief. With increasing efficacy there is an increased likelihood of cognitive side-effects, which are usually transient.

Maintenance Treatment

For most episodes of depression, one or other of the many treatments available (lifestyle, psychological, pharmacological, and physical) is likely to be effective. It is also important to remember however, that episodes of depression are intrinsically recurrent, and therefore periods of acute illness will often remit spontaneously. But even in these instances persisting with treatment is important as it ensures continuing engagement of the individual and allows for the ongoing provision of care. In this regard, it is important not to limit the focus of management to the treatment of an acute episode of depression. The disorder's recurrent course,

the persistence of subsyndromal symptoms, and increased suicide risk suggest that continued monitoring is warranted. Indeed, it is during the maintenance phase of management that the focus of clinicians can fully shift to building resilience against future episodes and moving towards quality-of-life enhancement.

Treatment Responsivity

Not all patients will respond to their initial treatment (about 40-50% will respond to an initial antidepressant) with a further 20% responding to a second round of antidepressant (Rush et al., 2006). Many patients coming into hospital may not have responded to a number of rounds of treatments.

The lack of response to treatments in depression has traditionally been described as ‘treatment-resistant depression’ (TRD), and more recently as ‘difficult-to-treat depression’ (DTD). However, these concepts are problematic as they emphasize lack of response and focus management on poor outcomes and failure to respond. This negative approach blames the individual and raises the complex and largely insurmountable role of personality factors. An alternative framing – the *responsivity paradigm* – addresses these problems.

An unsatisfactory response is often the result of poor adherence to treatment as prescribed. This applies to lifestyle advice, psychological interventions, and medications. Many of the Actions, such as diet and exercise, are challenging and are difficult for everyone irrespective of whether they are suffering from depression. Psychological interventions often require motivation, structure, and organisation. Medications are often seen as stigmatising and may also be regarded as unnecessary or dependence-inducing, and so patients fail to take their medications as prescribed – changing doses for example and stopping and starting treatment as they deem appropriate. Side effects and a lack of understanding about the need to continue treatment, may often lead to non-adherence, underscoring the importance of psychoeducation when prescribing medication.

The *responsivity paradigm* advises that these issues should be acknowledged at the outset, and that it should be accepted that there are several different *channels* (pathways) of treatment that can be pursued. Further, that no one knows at the start of treatment which of the various treatments is most likely to work. Hence, it is understood that several different strategies will have to be trialled until the appropriate strategy is found.

Having this discussion at the beginning of management allows the provision of a series of strategies that encompass different kinds of treatments. The discussion makes it clear from the outset that successful treatment may take time, and that

management requires regular review and revision of the treatment strategy being employed.

Further, reviewing the diagnosis and formulation regularly will open many channels of responsivity, and as treatment progresses, different strategies can be trialled.

The examples shown in Figure 16.9 make it clear that in some instances, more than one kind of therapy may be needed, and that in some cases, initial treatment may ‘prime’ the effects of later treatments, thus facilitating overall efficacy. The key advantage of this approach is that it provides a positive mindset, both for the patient and clinician, and avoids repeated disappointments and discussions regarding lack of response. Instead, clinical improvement is monitored and discussed, and versatility is introduced into management from the outset.

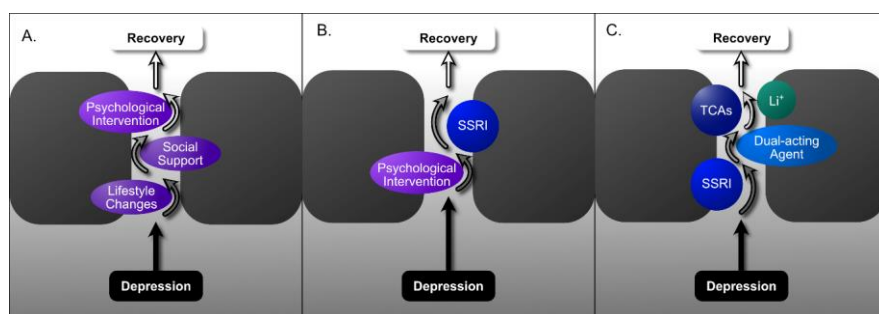


Figure 16.9. Responsivity Paradigm. The schematic above shows three channels that represent typical examples of pathways from depression to recovery. Each of the figures depicts different sets of treatments being used individually in sequence or in combination to achieve change. (A) Combination of lifestyle changes, social support, and psychological interventions (such as CBT) to facilitate recovery from depression. (B) Combination of psychological intervention and an SSRI to achieve recovery. (C) Sequencing of agents. First, an SSRI may be prescribed, but this only achieves a partial response and therefore it is suited to a dual-acting agent and then further supplanted by a broad-spectrum antidepressant such as a tricyclic (TCA). However, in this instance further treatment is necessary and an augmentation strategy (addition of lithium) is implemented to achieve recovery.

Conclusion

This chapter has outlined the foundational knowledge needed to address the most prototypical presentations of depression. However, in real-world practice, treating depression is rarely straightforward and often complicated by other factors such as atypical presentations, prior treatment history and psychological or

physiological comorbidities. For more detailed information regarding the nuances of treatment and the management of depression in clinical practice, see the 'Further reading' list at the end of this chapter.

Further Reading

- Malhi, G. S., & Mann, J. J. (2018). Depression. *The Lancet*, 392 (10161), 2299–2312.
- Malhi, G. S., Bell, E., Bassett, D., Boyce, P., Bryant, R., Hazell, P., ... & Murray, G. (2021). The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian & New Zealand Journal of Psychiatry*, 55(1), 7-117.
- Malhi, G. S., Bell, E., Singh, A. B., Bassett, D., Berk, M., Boyce, P., ... & Murray, G. (2020). The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders: major depression summary. *Bipolar Disorders*, 22(8), 788-804.
- Malhi, G. S., Bell, E., Bassett, D., Boyce, P., Hazell, P., Hopwood, M., ... & Murray, G. (2023). Positioning of psychodynamic psychotherapy in the treatment of depression: A comparison of the RANZCP 2020 and NICE 2022 guidelines. *Australasian Psychiatry*, 31(3), 322-325.
- Malhi, G. S., Bassett, D., Boyce, P., Bryant, R., Fitzgerald, P. B., Fritz, K., ... & Singh, A. B. (2015). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian & New Zealand Journal of Psychiatry*, 49(12), 1087-1206.
- NICE. (2022). Depression in adults: treatment and management. Vol NICE Guideline No. NG222. London, UK: National Institute for Health Care Excellence.

Chapter 17: Bipolar Disorder

Gin S. Malhi

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia, Visiting Professor, Department of Psychiatry, The University of Oxford, UK, and Oxford Uehiro Centre for Practical Ethics, Faculty of Philosophy, The University of Oxford, Oxford, UK.

Erica Bell

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

and

Philip Boyce

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia, Westmead Institute of Medical Research, Australia, and Faculty of Health, Charles Darwin University, Australia.

Introduction

Bipolar disorder is a recurrent mood disorder in which the individual usually experiences symptoms of both mania and depression. However, it is diagnosed and indeed defined based on symptoms of mania (McIntyre et al., 2020). Typically, these symptoms occur episodically and comprise elevated or expansive mood coupled with increased energy along with changes in behaviour and cognition. In addition, episodes of depression (low mood, anhedonia and diminished energy) are characteristic of bipolar disorder and episodes of illness are separated by periods of euthymia, in which patients describe their mood and overall functioning as near normal.

The illness confers a significant burden on the individual and those around them and behavioural changes can pose a considerable risk – especially when impulsive and lacking in insight. By the same token, increased energy and drive along with novel thinking can be potentially productive and even be regarded as beneficial to some extent, depending on the individual's role and the extent to which this phase of the illness can be contained. However, the disorder also carries with it a high risk of suicide.

As such, it has been speculated from time to time that bipolar disorder may be linked to creativity, and this is especially the case in artists who also suffered from bipolar disorder. For example, Robert Schuman and Vincent Van Gogh who had manic-depressive illness (the forerunner of bipolar disorder), were highly productive during their manic episodes, but it is not clear whether having manic symptoms was a key component of their artistic talent *per se*, or whether their increased productivity was simply a consequence of having more energy and time because of a diminished need for sleep.

The story of capturing bipolar disorder extends back almost two millennia to Aretaeus of Cappadocia, a practitioner in Rome who first connected the two poles of the illness noting ‘it appears to me that melancholy is the commencement and a part of mania’ (Aydemir and Malhi, 2007). In the centuries since, many others have made observations akin to this, but it was not until the late 19th century, when Emil Kraepelin definitively segregated the psychotic disorders based upon their longitudinal course that the forerunner of today’s bipolar disorder, namely manic-depressive illness, first took shape. He observed that some psychotic patients had a progressive and deteriorating course, and he labelled this as ‘dementia praecox’ (which subsumes modern-day schizophrenia). However, other patients had a more episodic course that culminated in a more favourable outcome, and he described this manic-depressive insanity (renamed subsequently as Manic-Depressive Illness).

Maintaining Kraepelin’s longitudinal perspective and emphasising recurrence, manic-depressive illness was used for most of the 20th century. However, in the 1980’s it was supplanted by the term bipolar disorder, which was introduced in DSM-III by the American Psychiatric Association (American Psychiatric Association, 1980) to give greater prominence to ‘polarity’ (i.e. manic and depressive poles of the illness) instead of cyclicity of trajectory over time. Subsequently bipolar disorder was subdivided into bipolar I and bipolar II based on whether patients experienced episodes of mania or only hypomania. In recent years this subtyping has been challenged.

The story of the treatment of bipolar disorder has run alongside the story of its nosology and classification. The treatment of bipolar disorder was revolutionised with the discovery that lithium had clear antimanic properties and a prophylactic effect on bipolar disorder, that prevented episodes of illness. Lithium salts were known to have a beneficial effect on health in general and in particular mood symptoms since the 19th century – especially through the work of Carl Lange (Schioldann, 2009), but it was an Australian psychiatrist, John Cade, who should be credited for the discovery its antimanic properties (Schioldann, 2013). Lithium is one of the most unique drugs in psychiatry with its specific effects on bipolar

disorder. It also has neuroprotective properties as well as being the only medication with anti-suicide properties (Malhi et al., 2018a).

Epidemiology

Bipolar Disorder affects approximately 2.5% of the population worldwide, or 1 in 40 people globally (McIntyre et al., 2020). It is a chronic, recurrent illness that usually first emerges in late adolescence and early adulthood and as such it confers a significant degree of lifelong dysfunction and disability (Yunxi et al., 2024). Unlike depression, bipolar disorder impacts men and women equally (Vöhringer and Perlis, 2016).

Aetiology

The aetiology of bipolar disorder is similar to that of depression (see Chapter 16), in that mood disorders are on the whole a product of a combination of genetic and environmental influences. Bipolar disorder has a strong genetic underpinning and a high heritability (approximately 70%), and an estimated 10 – 15% of first-degree relatives of people with bipolar disorder are likely to have a mood disorder (Gordovez and McMahon, 2020; McIntyre et al., 2020). But while genetic factors are important, psychosocial and psychological factors play an important role in the onset and, critically, the maintenance of the disorder and as such anticipating the illness remains a considerable challenge.

Onset

Bipolar disorder affects all ages from adolescence through to old age, with peak onset late teens and early adulthood (Mitchell et al., 2004; Vöhringer and Perlis, 2016). Although bipolar disorder is defined by the occurrence of mania (see Diagnosis below), the illness usually first presents as episodes of depression that are clinically identical to those that characterise major depression. It is only when mania manifests, that a diagnosis of bipolar disorder can be made. Thus, there is often a significant delay between illness onset and diagnosis (an average of approximately 8 years) (Keramatian et al., 2022; Fritz et al., 2017).

Diagnosis

The diagnosis of bipolar disorder is predicated on having mania, the symptoms of which can be regarded as the polar opposites of the symptoms of depression (Figure 17.1 below, and previous Depression chapter). A notable exception is irritability, which permeates both poles of the illness (Bell et al.,

2021a). The symptoms of bipolar depression are clinically indistinguishable from those of major depression, and it is this commonality that causes both diagnostic delay and treatment difficulties. It is important to note that whilst bipolar depression and major depression are clinically indistinguishable, the treatments effective in managing major depression are not equally effective in treating bipolar depression.

Diagnostic criteria for manic episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, lasting at least 1 week and present most of the day, nearly every day (or any duration if hospitalization is necessary).

B. During the period of mood disturbance and increased energy or activity, three (or more) of the following symptoms (four if the mood is only irritable) are present to a significant degree and represent a noticeable change from usual behaviour:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation (i.e., purposeless non-goal-directed activity).
7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The mood disturbance is sufficiently severe to cause marked impairment in social or occupational functioning or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

D. The episode is not attributable to the physiological effects of a substance or another medical condition.

The main classificatory systems, the Diagnostic and Statistical Manual of Mental Disorders, 5th edition text revision (DSM-5-TR) (American Psychiatric Association, 2022) and the International Classification of Diseases, 11th revision (ICD-11) (World Health Organization, 2019) recognise subtypes of bipolar disorder and draw a distinction between mania and milder forms. They divide bipolar disorder into two subtypes, mentioned earlier, the basis of which has been critically contended (Angst et al., 2019; Malhi et al., 2016a). Bipolar I disorder describes patients who experience a manic episode, whereas bipolar II is used to denote those

that experience manic *symptoms* but do not meet the criteria for an episode of mania. Hence, these ‘episodes’ are termed hypomania. In other words, hypomania is regarded as a less severe form of mania that typically has fewer symptoms and a shorter duration than mania.

The partitioning of bipolar disorder occurred more than 40 years ago and the debate concerning the existence of a bipolar II subtype centres on the fact that no clear differences have been found between bipolar I and II with regard to symptomatology, and there are no meaningful implications for management. Hence why, in terms of treatment, the RANZCP clinical practice guidelines for mood disorders consider only bipolar disorder and do not recognise any subtypes (Malhi et al., 2021b).

For the purposes of diagnosing bipolar disorder, the occurrence of manic symptoms is an essential requirement. Further, once an individual experiences these symptoms they are automatically diagnosed as having bipolar disorder even if they have previously been diagnosed as having major depression. Note, once bipolar disorder has been diagnosed subsequent episodes of depression are termed bipolar depression.

Mixed States

The co-occurrence of symptoms from both the manic and depressive poles of mood are categorised as mixed states, or mixed mood episodes. Mixed states are very heterogeneous, firstly because they may stem from a variety of causes, and secondly because of the different symptoms of mania and depression that can be variously combined to produce a myriad of presentations. Current taxonomies do not allow for the adequate capturing of mixed mood episodes, with DSM-5-TR utilising a ‘mixed features’ specifier, that can be ‘attached’ to an episode of either depression or mania (American Psychiatric Association, 2022).

Mixed episodes (or mixed features) are perhaps best understood by ‘deconstructing’ the range of mood symptoms from both poles of the illness and recognising that there can be various combinations of symptoms related to changes in activity, cognition, and emotion, as described in the Activity, Cognition and Emotion (ACE) Model (Malhi et al., 2018b).

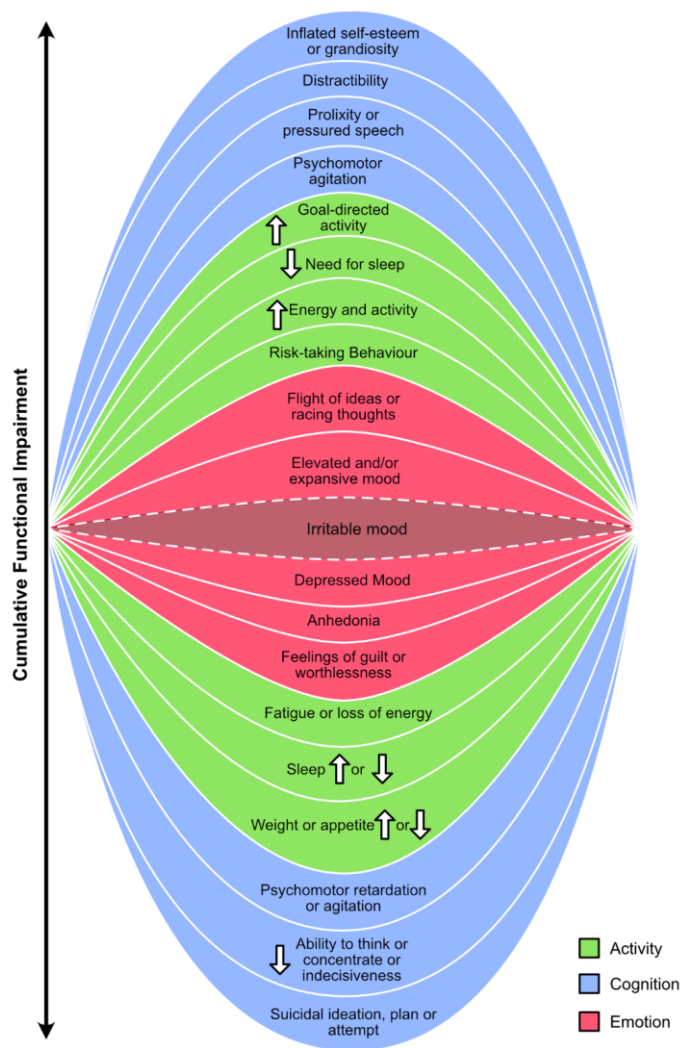


Figure 17.1. Symptoms of depression and mania in bipolar disorder, adapted from Malhi et al. (2021b). The symptoms that may occur in an episode of either mania or depression are shown as mirror images. These symptoms have been coloured according to the ACE domains (Green = activity; Blue = cognition; Red = emotion). For further detail, see ‘ACE Model’ section below. These symptoms, produce cumulative functional impairment but are not ranked in any particular order (i.e. inflated self-esteem is not more indicative of mania than elevated and/or expansive mood). Irritability (‘irritable mood’ within DSM-5-TR) is present in both mania and depression.

The existence of mixed states suggests that the view of bipolar disorder as a disorder of two states (mania and depression) is incomplete. Furthermore, the partitioning of mania into subtypes (I and II) that are separate categories of disorder is perhaps not the most accurate reflection of clinical reality. Consequently, in recent years, a dimensional perspective has gained ground and bipolar disorder is increasingly regarded as a spectrum with expressions of illness that span all manner of admixtures between mania and depression. This spectrum view of symptoms also accommodates expressions of major depression therefore connecting mood disorders (depressive and bipolar) and forming one set of contiguous illnesses. This dimensional view forms the basis for treatment of mood disorders.

The ACE Model

The Activity, Cognition and Emotion (ACE) Model provides a useful framework to conceptualise the symptoms of mania and depression in bipolar disorder and the symptoms of major depression. The ACE Model categorises manic and depressive symptoms according to the 3 domains of Activity, Cognition and Emotion, allowing for a more granular appraisal of symptoms and how these change over time. It is therefore best applied longitudinally as well as cross-sectionally (see Figures 17.2 and 17.3). Importantly, the architecture of the ACE model allows mixed states to be accommodated automatically and without invalidating either depression or mania. In practice, mixed mood presentations account for approximately one-third of mood episodes in patients with bipolar disorder.

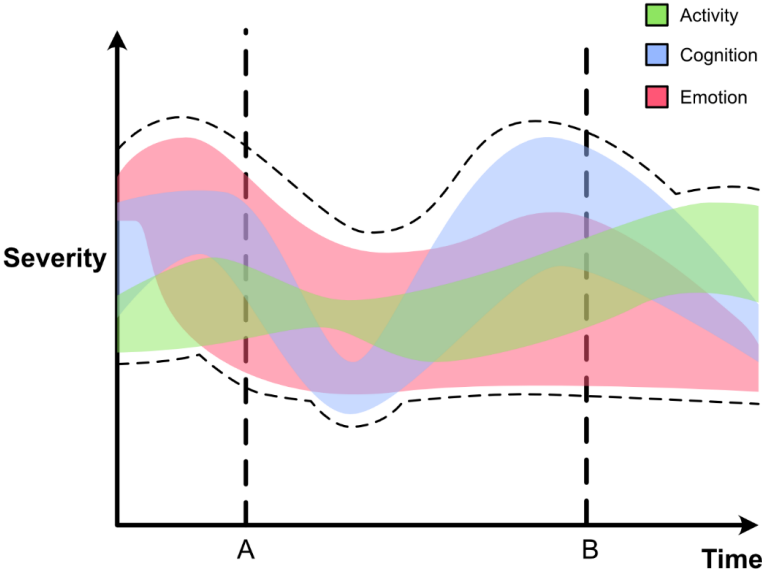


Figure 17.2. Change in components of ACE with time, adapted from Malhi et al. (2021b). The schematic shows the three Activity (green), Cognition (blue) and Emotion (red) that vary in severity over time. The overall range of severity (peak and trough) is shown with a black dashed line. At points A and B, the total severity appears to be the same. However, it is evident that individual domains vary differentially. For example, at point A it is emotion that is most prominent, followed by cognition, but later, at point B this order is reversed, and cognition becomes the major contributor to severity. Thus, by mapping domains and individual symptoms, rather than the syndrome as a whole, the ACE model permits a more granular examination of changes over time.

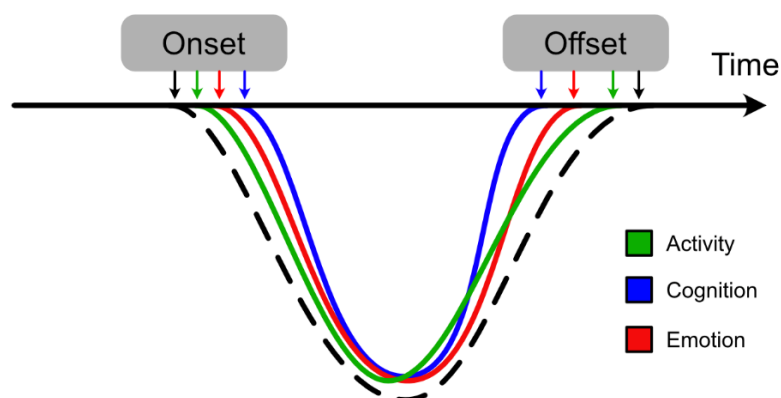


Figure 17.3. Differential timing in onset and offset of symptoms – illustrated by ACE, adapted from Malhi et al. (2021b). The black dashed line shows the overall change in symptoms with depression emerging gradually and resolving slowly. However, within this, it can be seen that changes in activity precede those of emotion and cognition and that in response to treatment or spontaneous remission, it is the cognitive symptoms that recover first followed by emotion, with activity taking the longest. This is important when monitoring the effects of treatments and also when communicating to patients what to anticipate as regards recovery. Often as initial symptoms remit, they may lead a patient to believe that they have recovered and may even prompt them to consider stopping treatment. But as is evident, some symptoms take considerably longer to remit and therefore it is important to emphasise to patients that they continue with treatment until they have regained full function.

Course and Impact

By its very nature, bipolar disorder is a recurrent illness, in which almost one-half of all individuals who have experienced a mood episode will have a further episode of illness. In most individuals, the depressed phase is more common than mania, with many patients spending much of their time depressed. However, even though the illness is termed bipolar disorder, approximately a third of episodes are likely to be an admixture of manic and depressive symptoms (mixed episodes).

As bipolar disorder typically emerges during adolescence and is associated with cognitive impairment (Chakrabarty et al., 2021) and brain tissue loss (Keramatian et al., 2021), it confers significant functional disability, particularly among young people aged 15-24 years (ranked third of all mental disorders) (GBD

2019 Mental Disorders Collaborators, 2022). Further, mortality studies indicate that bipolar disorder is associated with a loss of approximately 10 – 20 potential years of life. This is not solely because of suicide – indeed a significant proportion of this loss is because of the effects of the illness on the general medical health of individuals.

In addition, bipolar disorder carries the highest risk of suicide of any mental illness, (30-60 times that of the general population) and consequentially, approximately 30 – 50% of adult bipolar patients have a history of suicide attempts.

Management of Bipolar Disorder

Diagnosis and Formulation

The diagnosis of bipolar disorder is inherently complicated, partly because of the natural history of the illness, which typically begins with depression in adolescence (Ghaemi et al., 1999). Hence why several episodes of depression have usually occurred before mania emerges. However, as noted, the diagnosis of bipolar disorder can only be made once manic symptoms appear, and so there is often a delay in diagnosis which, in some cases, can be up to a decade. Another important diagnostic challenge is that some of the behaviours or ‘symptoms’ typical of the turmoil of adolescence, such as increased energy, not wanting to sleep, taking risks, and having heightened libidinal drive may be misconstrued as manic symptoms. The presentation can also be confounded by personality factors, especially when there is affective instability, and the symptoms of bipolar disorder can easily be obscured by those caused by substance misuse, including for example stimulants and alcohol. This is important because in addition to psychosocial adversity, substance misuse is associated with an earlier age of onset of bipolar disorder (Post et al., 2016).

A comprehensive assessment of an individual suspected of having bipolar disorder is essential. This should be repeated overtime as a longitudinal perspective is critical for accurate diagnosis and tailored management. It is important, for example, to establish if there is any specific pattern to the illness (such as seasonality), noting the duration of depression and mania, and periods of remission and recovery, so as to map the phases of illness (mania, mixed, depression), determine predominant polarity (that is, whether the person experiences mainly depression, mania or mixed states) and whether there is any sequence or order in which episodes typically occur. It is also important to note triggering events to individual episodes and how these have responded to any measures that may have been instituted. This includes identifying antidepressant-induced elevated states, which will necessitate withdrawing the medication promptly under close

supervision. It is again important to identify clearly the extent to which substances such as alcohol and other prescribed and illicit drugs precipitate episodes.

The context in which the illness arises is also important. Here, early trauma, family history, general medical health, developmental factors, life events, personality structure, current psychosocial issues and interpersonal context should be carefully assessed and reviewed. Pieced together, this information can be synthesized to provide a basis for formulating the nature of the illness, the problems that it is causing for the individual, and the factors that have likely contributed to it. Thus, understanding context is important for determining optimal management.

Treatment Principles

The management of bipolar disorder is more complex than that of major depression. First, bipolar disorder has additional acute episodes, namely mania and mixed presentations – that occur as well as episodes of depression. Second, the emphasis of treatment is not solely upon acute management, but also on long-term maintenance and prophylaxis, and so continuity of care and the development of a robust therapeutic relationship are essential.

Maintenance therapy is of particular importance in the management of bipolar disorder, because of the chronic and recurrent nature of the illness, and because it is only during this phase of comparative mood stability that, in addition to sustaining wellness, preventative strategies that will avert future episodes can be implemented.

As is the case for depression, the *Actions, Choices, Alternatives* framework should be used for the management of bipolar disorder. This framework provides a systematic and structured approach to the management of this complex illness.

Actions

The *Actions* for the management of bipolar disorder need to be considered in relation to the phase of the illness being treated (see Figure 17.4 below). Broadly speaking, *Actions* can be grouped into three types. First, those that can be instituted by the patient with appropriate support, such as lifestyle changes, where it is in the individual's best interest to maintain a healthy diet, engage in regular exercise, and ensure they maintain good sleep hygiene and stable circadian rhythms. For some patients, bipolar relapses are clearly linked to a disruption of core biological circadian rhythms such as the sleep-wake cycle. For example, missing sleep because of desire (e.g., socializing) or necessity (work commitments) or as a consequence of

travel (jet lag). Hence, the development of interpersonal social rhythm therapy (IPSRT) that is aimed at stabilizing circadian rhythms.

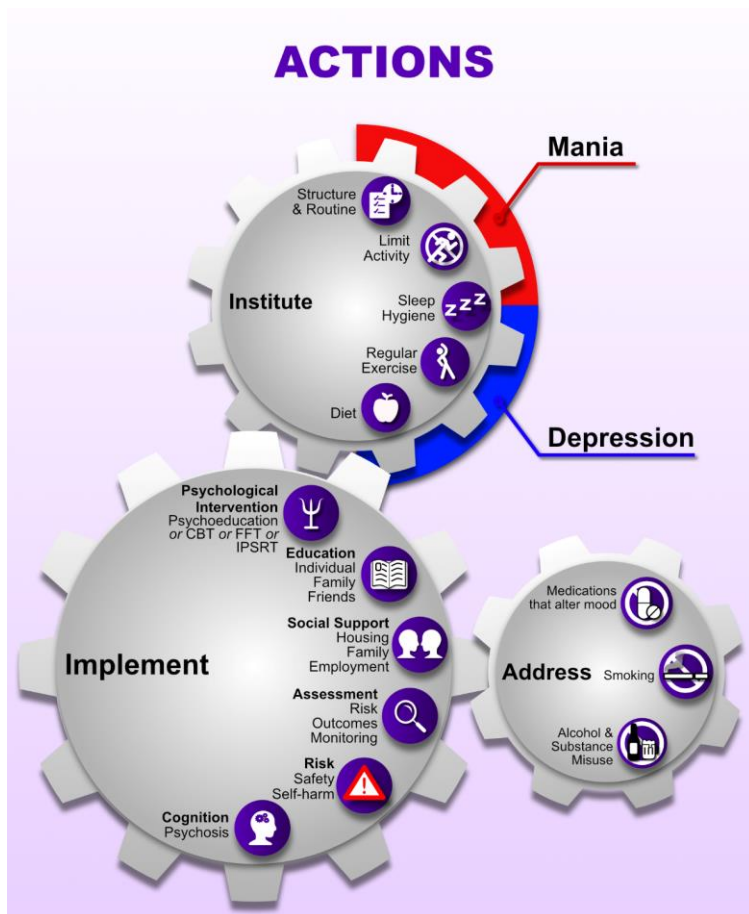


Figure 17.4. Actions for the management of bipolar disorder, adapted from Malhi et al. (2020a). The *Actions* for the management of bipolar disorder should be considered according to the phase of the illness being treated. For example, in the management of acute mania, limiting activity and instituting structure and routine are important as is restoration of normal sleep patterns. Restoring sleep is also important when treating bipolar depression, but in this case encouraging activity and exercise is necessary rather than restricting this. The key to long-term successful management of bipolar disorder is maintenance therapy and for this, psychoeducation and psychological interventions are essential, alongside restoration of normal biorhythms.

In addition, there are *Actions* that also must be implemented, such as the provision of psychoeducation to the individual, their family and friends, concerning both the illness and its prognosis as well as its treatments and broader management approaches. It is important to note that psychological interventions have been included as *Actions* because they should be offered to all patients, as early as possible, as they are particularly effective if implemented from the outset, and especially in conjunction with pharmacotherapy.

Finally, it is often necessary to address negative habits that the individual has developed (e.g., smoking, alcohol and substance misuse), and this usually requires the assistance of specialists.

Psychological Interventions

Adjunctive psychological intervention is central to the management of bipolar disorder (Murray, 2018; Chen et al., 2019; McIntyre et al., 2013). Four therapeutic evidence-based approaches include: Psychoeducation, Cognitive-Behavioural Therapy (CBT; see Chapter 33), Family-focused Therapy (FFT), and Interpersonal and Social Rhythm Therapy (IPSRT) (Malhi et al., 2021b). These four have a common treatment approach (active collaboration, skill-development focus), and share significant therapeutic content. Psychological interventions are foundational *Actions* in the management of bipolar disorder, but unlike the management of depression, they are not a substitute for pharmacotherapy.

Mania – Choices

The pharmacotherapy of mania encompasses the management of risk and the treatment of its three main components: acute manic symptoms, behavioural disturbance and cognitive symptoms and/or psychosis. It is important to note that the three components have different timescales of response to treatment. Hospitalisation is often required to contain risk, but in some cases, treatment can be administered as an out-patient. Most medications used to treat mania treat more than one syndromal component at a time, e.g., address agitation and psychosis as well as mania (see Figure 17.5).

The treatment of mania often requires combinations of medications, but ideally this should include a mood stabilising agent (MSA). The medications that are most commonly used are shown in Figure 17.5.

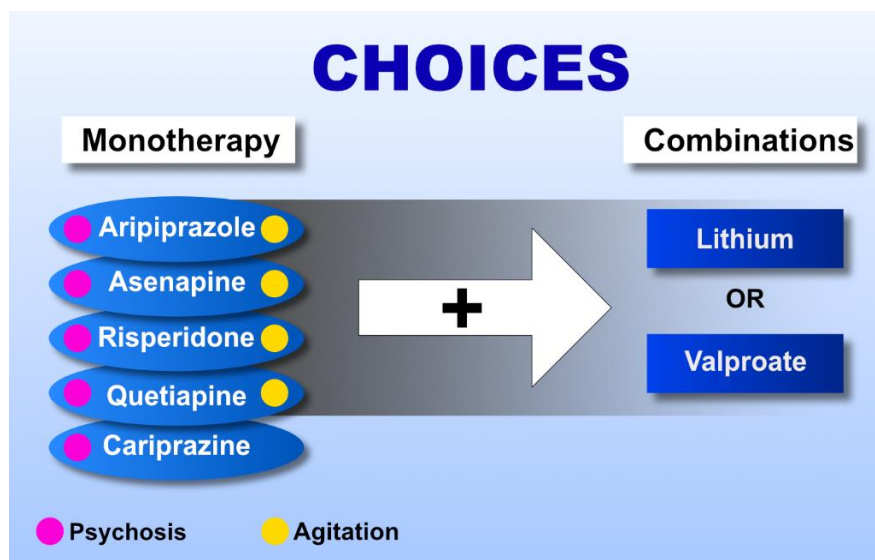


Figure 17.5. Choice treatments for the management of mania, adapted from Malhi et al. (2020a). Choice agents address all three components: mania, agitation, and psychosis. Where possible, monotherapy and oral treatment should be given preference. If monotherapy is insufficient then lithium or valproate can be added, and these can remain in place as the antipsychotic is gradually removed once the acute episode subsides. If combination therapy is insufficient, then *Alternatives* may need to be considered. Note that valproate is not recommended for women of childbearing age because of the high rates of teratogenicity and developmental delay among foetuses exposed to valproate

Mania – Alternatives

On occasion, *Choice* medications, even as combinations, may be insufficient, and *Alternatives* need to be considered. Here again, both monotherapy and combination strategies can be employed.

Another *Alternative*, if all else fails, is ECT, which may be indicated from the outset if, for example, pharmacotherapy cannot be tolerated or is undesirable (e.g. pregnancy). For further detail regarding ECT, see the later ‘ECT’ section in which the recommendations for administration are addressed collectively across all phases of bipolar disorder.

Bipolar Depression – Choices

Amongst the various phases of bipolar disorder, the treatment of bipolar depression is the most challenging. The medications used to treat bipolar depression belong to two broad groups: mood stabilising agents (MSAs) and second-generation antipsychotics (SGAs). Each group comprises three monotherapy options (see Figure 17.6). Where possible, mood stabilising agents should be given preference because they can be continued into maintenance therapy and are less likely to have significant long term side effects. Lithium for example is a good maintenance therapy and it is the only medication that protects against suicide.

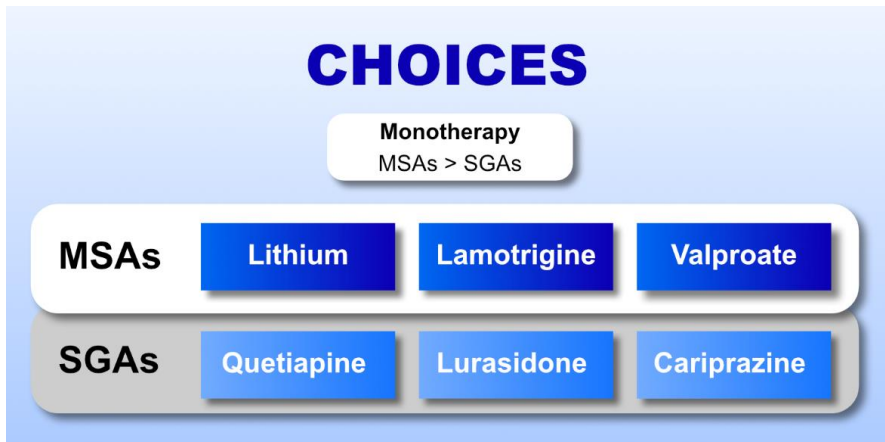


Figure 17.6. Choice treatments for the management of bipolar depression, adapted from Malhi et al. (2020a). Mood stabilising agents (MSAs) include lithium, lamotrigine and valproate (in sequence) and these are preferable to Second generation antipsychotics (SGAs) which include quetiapine, lurasidone and cariprazine (in sequence).

Bipolar Depression – Alternatives

When managing bipolar depression, it is not uncommon for the above monotherapy *Choices* to be insufficient, and so *Alternatives* may need to be considered as well as combinations of medications.

The combinations of medications in the treatment of bipolar depression can be easily remembered as combinations between either an MSA and an SGA, or an agent from either of these groups, with an antidepressant (for further detail, see the RANZCP Mood Disorders Clinical Practice Guidelines 2020). It is important to note that antidepressants, when prescribed on their own, may cause mood instability in bipolar disorder and therefore should ideally only be prescribed alongside an MSA

or SGA. ECT may be indicated, particularly if there are significant melancholic depressive features or psychotic symptoms present (see 'ECT' section below).

ECT

ECT (see Chapter 36) is an important *Alternative* across all acute presentations of bipolar disorder, it has proven efficacy in mania (Perugi et al., 2017), bipolar depression (Agarkar et al., 2018; Bahji et al., 2019a) and mixed states (Medda et al., 2015). The effectiveness and potential side effects of ECT is determined largely by placement of the electrodes and the pulse width of the current applied (see Figure 17.7).

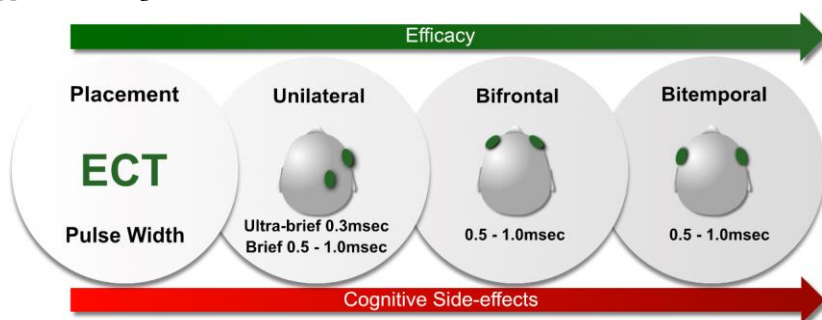


Figure 17.7. ECT in bipolar disorder, adapted from Malhi et al. (2020b).

Electroconvulsive therapy is effective in treating bipolar depression, mania and mixed states. Increased efficacy can be achieved by increasing the pulse-width and by shifting placement.

Lithium

In the sections above lithium has featured prominently and as such it warrants special consideration especially as it holds a unique position in the management of bipolar disorder, and it is an element about which there is often some confusion.

The properties of lithium were first definitively identified by John Cade who tested its effects on animals and himself before trialling it on his patients (Malhi, 2010; Shorter, 2009). It is the only true mood stabiliser as it not only has acute antimanic and antidepressant effects but also provides prophylaxis against both depression and mania. Further, it reduces the likelihood of suicide and is neuroprotective. Nevertheless, it is not as widely used as it should be because of its slow onset of action and the need for plasma monitoring (see Figure 17.8) (Rhee et

al., 2020). In addition, it can cause serious long term side effects involving renal and thyroid function and its acute side effects can also be troublesome.

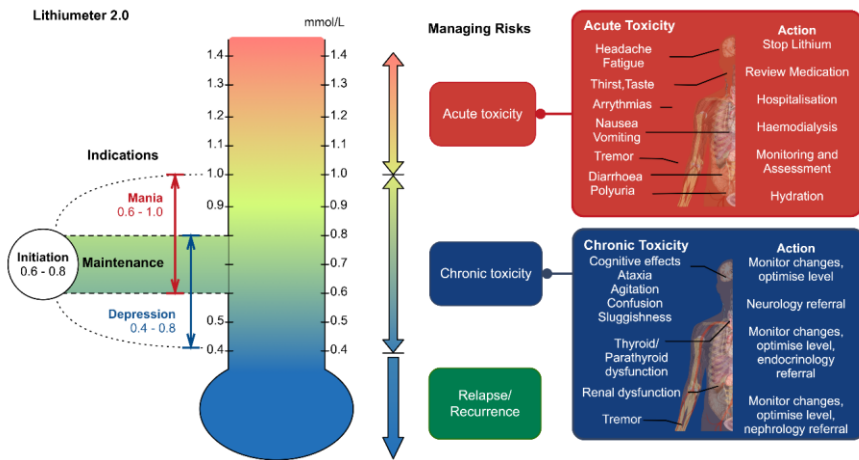


Figure 17.8. Lithiummeter 2.0, adapted from Malhi et al. (2016b). The Lithiummeter demonstrates the ideal serum concentration ranges of lithium for the various stages of treating bipolar disorder. The red and blue boxes illustrate the acute and chronic toxicity effects of lithium, and the actions needed to address these.

Conclusion

This chapter has outlined the framework for diagnosis and management of the manic and depressive phases of bipolar disorder. However, in real-world practice, bipolar disorder is a complex and heterogenous disorder, both in terms of diagnosis and treatment. In addition, its management is complicated by its chronicity, its cyclical nature, and the need for maintenance treatment. Thus, further reading regarding the management of this disorder is highly encouraged, and more detailed information regarding the nuances of treatment and the management of bipolar disorder in clinical practice can be found in the 'Further reading' list at the end of this textbook.

Further Reading

Please note, the clinical practice guidelines and the two summary documents are freely available online.

- McIntyre, R. S., Berk, M., Brietzke, E., Goldstein, B. I., López-Jaramillo, C., Kessing, L. V., ... & Mansur, R. B. (2020). Bipolar disorders. *The Lancet*, 396(10265), 1841-1856.
- Malhi, G. S., Bell, E., Bassett, D., Boyce, P., Bryant, R., Hazell, P., ... & Murray, G. (2021). The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian & New Zealand Journal of Psychiatry*, 55(1), 7-117.
- Malhi, G. S., Bell, E., Boyce, P., Bassett, D., Berk, M., Bryant, R., ... & Murray, G. (2020). The 2020 Royal Australian and New Zealand College of psychiatrists clinical practice guidelines for mood disorders: Bipolar disorder summary. *Bipolar Disorders*, 22(8), 805-821..
- Malhi, G. S., Irwin, L., Hamilton, A., Morris, G., Boyce, P., Mulder, R., & Porter, R. J. (2018). Modelling mood disorders: an ACE solution?. *Bipolar Disorders*, 20, 4-16.
- Malhi, G. S., Bell, E., Singh, A. B., Bassett, D., Berk, M., Boyce, P., ... & Murray, G. (2020). The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders: major depression summary. *Bipolar Disorders*, 22(8), 788-804.
- Malhi, G. S., Gershon, S., & Outhred, T. (2016). Lithiummeter: Version 2.0. *Bipolar Disorders*, 18(8), 631-641.

Chapter 18: Anxiety Disorders

Vladan Starcevic

Specialty of Psychiatry, The University of Sydney, Australia.

Lisa Lampe

School of Medicine and Public Health, The University of Newcastle, Australia.

and

Juliette Drobny

Specialty of Psychiatry, The University of Sydney, Australia.

Introduction

As a group, anxiety disorders are the most common mental disorders. The lifetime prevalence of anxiety disorders in various countries has been found to range between 9.2% and 28.7%, with the best-estimate lifetime prevalence rate for all anxiety disorders calculated at 16.6% (Somers et al., 2006).

The main feature of anxiety disorders is the presence of pathological anxiety, but emotions such as disgust, irritability and shame may also be prominent. Pathological anxiety differs from normal anxiety in a number of ways (Table 18.1). Individuals with anxiety disorders usually have a preserved insight that their anxiety is excessive and/or unreasonable.

Fear is closely related to anxiety. Both appear in response to a perceived threat and are often accompanied by similar bodily sensations, i.e., symptoms of autonomic nervous system hyperarousal (see Figure 18.1). However, there are also differences. While the threat in fear is easier to identify, external to the individual, more specific and immediate, the threat in anxiety tends to be elusive, internal, diffuse and lingering. The onset of fear is often abrupt and its course is acute or episodic, whereas the onset of anxiety is more often vague and its course is usually chronic. In contrast to fear where a typical response to threat is immediate “fight or flight”, the typical response in anxiety is vigilance. Most anxiety disorders have features of both fear and anxiety.

Table 18.1. Differences between normal and pathological anxiety.

	Normal anxiety	Pathological anxiety
Intensity	Minimal to mild	Moderate to severe
Duration	Short-lasting	Persistent
Quality of the experience	Understandable, rational	Puzzling, overwhelming, with little or no control over the anxiety
Sense of danger	Realistic	Exaggerated
Significant distress or impairment in functioning	No	Yes

Several conditions are classified in the group of anxiety disorders: panic disorder, agoraphobia, social anxiety disorder (social phobia), specific phobia and generalised anxiety disorder (GAD). The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) (American Psychiatric Association, 2022) also includes separation anxiety disorder and selective mutism, conditions that are usually encountered in childhood, although 40% of adults with separation anxiety disorder have been reported to experience an onset after the age of 18 years (Silove et al., 2015). If pathological anxiety is caused by a substance or a medical condition, such disorder is diagnosed as a substance-induced anxiety disorder or anxiety disorder due to another medical condition, respectively. Although obsessive-compulsive disorder and posttraumatic stress disorder are related to the anxiety disorders, in the DSM-5-TR each has been classified in its own category (American Psychiatric Association, 2022). Panic attacks are described below, followed by a brief outline of each anxiety disorder and treatment considerations.

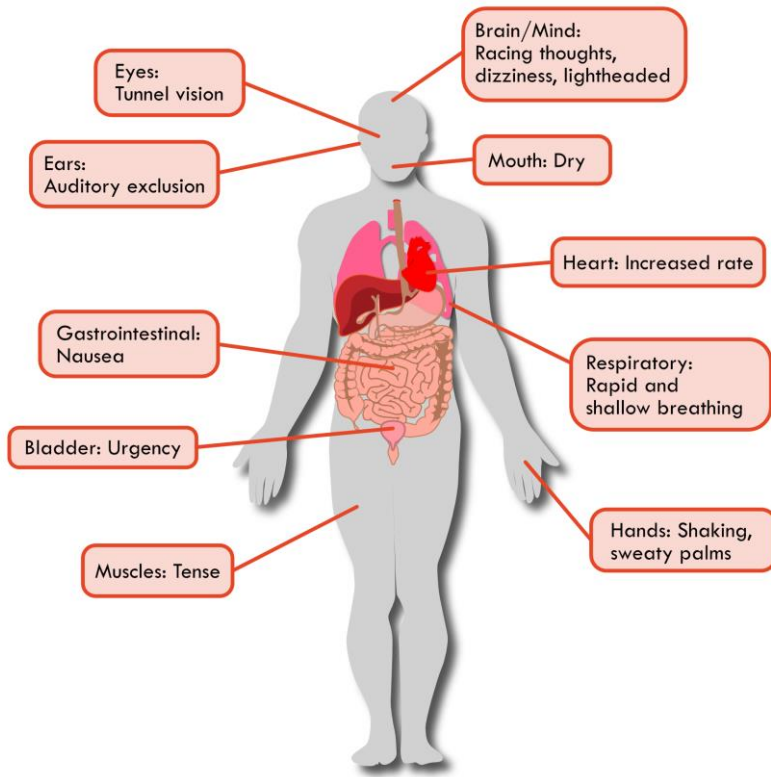


Figure 18.1. Physiological responses to anxiety.

Panic Attack

Panic attacks are characterised by an abrupt onset and rapid escalation of physical symptoms of anxiety, accompanied by intense fear. The numerous and distressing bodily symptoms are for the most part a consequence of autonomic nervous system hyperarousal. The peak of a panic attack is reached within minutes and attacks usually do not last very long – rarely more than 30 minutes.

The very first panic attack is often experienced as “out of the blue”; a similar experience may be reported for a few panic attacks occurring initially. Subsequent panic attacks are more likely to have cognitive (e.g., anxious thoughts), somatic (e.g., feeling hot, increased heart rate) or situational triggers, although these may be subtle and not obvious to the individual. Eventually, people usually learn to associate panic attacks with certain situations, and the attacks tend subsequently to be

anticipated and felt to be more likely in these situations. It is important to note that panic attacks may occur in any disorder: they are not pathognomonic of panic disorder.

Panic Disorder

Panic disorder comprises two components: recurrent panic attacks and anticipatory anxiety. Panic attacks in panic disorder are associated with intense fears of physical or mental catastrophe perceived as imminent. Such catastrophe includes dying, losing consciousness and passing out, losing control or “going crazy”.

Lifetime prevalence of panic attacks

- Up to 28.7%

Lifetime prevalence of panic disorder

- From 0.4% to 3.8%
- Best-estimate: 1.2% (Somers et al., 2006)

Male-to-female ratio

- 1:2-3

Usual onset of panic disorder

- Third decade of life

Help-seeking patterns

- Usually quick help-seeking due to intense and frightening physical symptoms and the accompanying fear
- Help is often sought in hospital emergency departments or primary care and specialised medical settings

Anticipatory anxiety is a key feature of panic disorder and represents a distressing and persistent fear of another panic attack. The essence of this fear is anticipation of the (unlikely) consequences of panic attacks, such as death, heart attack or other bodily catastrophe, loss of control, psychotic breakdown or humiliation. Anticipatory anxiety may lead to changes in behaviour, usually in the form of avoidance of the activities or situations (e.g., exercising or crowded shopping centres) that could trigger a panic attack or some of its symptoms. Epidemiological and help-seeking characteristics of panic disorder are presented above. In addition to the indirect costs of functional impairment and disability, panic disorder is associated with the direct costs of frequent visits to medical practitioners and numerous medical investigations.

Little is known about the aetiology of panic disorder. A genetic predisposition has been suggested, but it is unclear what constitutes a genetic vulnerability to panic disorder and how specific it is for this condition. A relatively specific risk factor for panic disorder is a high level of anxiety sensitivity (Schmidt et al., 1997), which denotes a fear of anxiety and its bodily symptoms based on beliefs about their dangerousness. The most commonly invoked psychological mechanism in panic attacks is catastrophic misinterpretation of bodily sensations and symptoms (Clark, 1986). In terms of neurobiology, an abnormally sensitive anxiety-regulating mechanism originating in the amygdala may be involved in all anxiety disorders. The frequent predominance of respiratory symptoms during panic attacks has been hypothesised to be due to hyperventilation, hypersensitivity to carbon dioxide of the brain-stem chemoreceptors or lower threshold for activating the suffocation alarm mechanism. Panic attacks may occur as a consequence of an increased central noradrenergic activity, with hypersensitivity of the presynaptic α -2 receptors and increased firing rate of the locus coeruleus. An additional factor may be a failure of the gamma-aminobutyric acid (GABA) neurotransmitter system to inhibit the locus coeruleus. Abnormalities in the serotonin neurotransmitter systems have also been implicated in the aetiology of panic disorder.

The diagnosis of panic disorder is based on clinical presentation and exclusion of the relevant medical conditions that may cause panic attacks and/or high levels of anxiety (Table 18.2). Panic attacks may occur in the course of other mental disorders and in such cases, an additional diagnosis of panic disorder may or may not be warranted. Substance misuse, other anxiety disorders and depression frequently accompany panic disorder.

The course of panic disorder varies from one person to another. Agoraphobia is a common complication. At least one third of patients recover, but as many as 50% report a chronic, fluctuating course, with remissions and exacerbations. The severity of panic disorder tends to decrease with ageing. Studies have established links between panic disorder and a higher risk of suicide attempts, suicide and cardiovascular disease, but these relationships are poorly understood.

Table 18.2. Medical conditions that may cause panic attacks and/or high levels of anxiety.

Frequent association with panic attacks	
Endocrine and metabolic disturbances	• Hyperthyroidism • Hypoglycaemia
Cardiac conditions	• Cardiac arrhythmias (e.g., paroxysmal atrial tachycardia) • Mitral valve prolapse
Neurological and vestibular conditions	• Complex partial (temporal, psychomotor) epilepsy • Vestibular dysfunction
Less common association with panic attacks	
Endocrine and metabolic disturbances	• Hypothyroidism • Hypoparathyroidism • Hyperparathyroidism • Cushing's syndrome • Menopause-associated endocrine disturbances • Electrolyte disturbances • Pheochromocytoma
Pulmonary conditions	• Pulmonary embolus
Gastrointestinal disorders	• Irritable bowel syndrome

Agoraphobia

Agoraphobia is a fear of multiple, interrelated situations from which it might be difficult or impossible to escape immediately (e.g., being in crowded or enclosed places such as shopping centres, cinemas, airplanes and tunnels or using public transport such as buses and trains), those in which the person is alone or outside of their safety zone so that immediate medical or other help might not be available (e.g., travelling far away from home, staying at home alone) and situations from which it might be awkward or embarrassing to escape immediately (e.g., standing in line, sitting in the middle of a row in a theatre). These situations are typically avoided. Avoidance may be of such proportions that the person becomes homebound and quite incapacitated. Individuals with agoraphobia often rely on a “phobic companion” (usually a partner or family member) to accompany them to feared situations.

Agoraphobic situations are frequently feared because of the expectation that panic attacks and symptoms would occur in these situations. Therefore, prevention of panic attacks and symptoms is the usual aim of avoidance in agoraphobia. This also explains a strong relationship between agoraphobia and panic disorder and their frequent co-occurrence. However, agoraphobia can also develop independently from panic, for example in the context of the fears of falling, being incontinent, getting lost, having an accident or being mugged.

Epidemiological and help-seeking characteristics of agoraphobia are shown below. Agoraphobia is associated with significant functional impairment, especially when the person is homebound.

Lifetime prevalence

- From 0.7% to 10.8%
- Best-estimate: 3.1% (Somers et al., 2006)

Male-to-female ratio

- 1:3-4

Usual onset

- Late adolescence or early adulthood

Help-seeking patterns

- Help-seeking depends on the extent of avoidance and the associated functional impairment
- Help is often sought from general practitioners or mental health services

The agoraphobic fear is usually explained as a consequence of learning, i.e., of associating unpleasant anxiety symptoms, panic attacks, with certain situations. These situations are subsequently avoided, with avoidance reinforcing the agoraphobic fear.

The diagnosis of agoraphobia is based on the fear and avoidance of a number of typical situations, as described above. A proper assessment includes ascertaining the specific fears linked to avoided situations and the extent of avoidance. It is important to distinguish agoraphobia from certain types of specific phobia (especially claustrophobia) and depressive disorders.

Agoraphobia is a chronic condition, with little or no spontaneous improvement. The extent of disability and prognosis may depend on the availability

of a phobic companion and other personal circumstances. If agoraphobia is aetiologically related to panic attacks, successful treatment of the latter may alleviate, but not necessarily eliminate, agoraphobic fear and avoidance. The most important long-term complications of agoraphobia are depression and alcohol misuse.

Social Anxiety Disorder (Social Phobia)

Social anxiety disorder is defined as a prominent and persistent fear of situations in which the person is or might be exposed to the scrutiny of others. These include both performance-type situations (giving a talk or doing something else in front of others) and various interpersonal interactions (ranging from simple conversations with unfamiliar people and asking strangers for directions to the more challenging situations involving communication with authority figures or expressing disagreement). Avoidance of social situations is the usual way of coping with social anxiety. If avoidance is not possible, there is endurance of social situations with much distress. In milder forms of social anxiety disorder, only performance-type situations tend to be feared and avoided.

The key phenomenon in social anxiety disorder is fear of negative evaluation, which is evident in concerns about coming across as “stupid”, abnormal or incompetent and the humiliation that the person fears will result. Fear of having noticeable anxiety-related bodily symptoms such as blushing, sweating or trembling, is also common. Social anxiety disorder is closely related to shyness, defined as the tendency to feel awkward, worried or tense during social encounters. However, the former denotes higher levels of anxiety, greater avoidance of social situations and decreased ability to adapt to social situations and respond to positive social cues. Unlike shy individuals, those with social anxiety disorder remain very anxious throughout their exposure to a social situation and despite any positive feedback or encouragement that they receive during such exposure.

Epidemiological and help-seeking characteristics of social anxiety disorder are shown below. Multiple areas of functioning are adversely affected. Severely affected individuals, about 30% of those with social anxiety disorder, may never form relationships, their educational achievement is not commensurate with their intellectual abilities and their participation in the workforce may only be marginal. A greater number of feared social situations is associated with greater severity and disability. Of all the anxiety disorders, long-term disability was reported to be most prominent in social anxiety disorder (Hendriks et al., 2016).

The aetiology of social anxiety disorder involves a number of biological and psychological factors. A genetic predisposition has been demonstrated, with most of

the vulnerability shared with other anxiety disorders and depression. A relatively specific risk factor is behavioural inhibition to the unfamiliar (Kagan et al., 1984), which can be observed very early in life as increased anxiety in response to strangers, increased startle response and difficulty settling after novel stimuli. A cognitive model (Rapee and Heimberg, 1997) postulates that social anxiety disorder results from the negative assumptions and beliefs about oneself, others and social situations (e.g., “I am inadequate, others are judgemental and social situations are a testing ground”). These assumptions and beliefs lead to appraisals of social situations as threatening and are maintained through avoidance of these situations, use of safety behaviours and multiple biases in information processing.

Lifetime prevalence

- From 0.5% to 16%
- Best-estimate: 3.6% (Somers et al., 2006)

Male-to-female ratio

- 1:1.5-2

Usual onset

- Early adolescence

Help-seeking patterns

- Delayed help-seeking, usually due to shame and embarrassment or lack of realisation that the condition can be treated
- When help is sought, this is often for complications such as depression or substance use disorder or because of an important life change (e.g., going to a different school or starting a new job)

The diagnosis of social anxiety disorder is based on the presence of severe anxiety in multiple social situations, which is usually accompanied by extensive avoidance. At the time of assessment, it is important to ascertain the presence of complications, particularly depression and substance use disorder. Due to its availability and anxiolytic and disinhibiting properties, alcohol is commonly misused by individuals with social anxiety disorder. While there is much overlap between social anxiety disorder and avoidant personality disorder, they seem to be distinct conditions (Lampe and Malhi, 2018).

The course of social anxiety disorder is chronic. Fluctuations in the intensity of social anxiety do occur, but without treatment, individuals with the condition typically remain impaired in many areas of functioning.

Specific Phobia

Various types of phobic disorders are subsumed under the diagnostic entity of specific phobia. They include phobia of animals, blood-injection-injury phobia (i.e., fear of the sight of blood, injured tissues or needle penetrating the skin), situational phobia (i.e., fears of driving and flying and claustrophobia), phobias of water, heights and storms, dental phobia and phobia of choking or vomiting. Individuals with specific phobia experience intense fear in the presence of their “phobic stimulus”, i.e., the specific object, situation, activity or natural phenomenon that they are afraid of. A fearful response can have the intensity and other characteristics of a panic attack. The fear is based on the perception of threat posed by the phobic stimulus. For example, a person with claustrophobia is typically afraid of being unable to breathe in a small, enclosed space. Some types of specific phobia, especially phobia of insects and blood-injection-injury phobia, are related to intense and distressing disgust responses. Blood-injection-injury phobia is unique among the phobias because it is as common in males as it is in females and is often characterised by a vasovagal reaction, with bradycardia, hypotension and fainting rather than the symptoms of autonomic hyperarousal generally seen in panic attacks.

Avoidance is the usual way of coping with fear in specific phobia. It is generally easier and less costly to avoid the circumscribed phobic stimuli in specific phobia than it is to avoid multiple agoraphobic situations in agoraphobia and social situations in social anxiety disorder. This explains the fact that individuals with specific phobia are usually less disabled by their fears and avoidance than those with agoraphobia and social anxiety disorder and that they are encountered less often in clinical settings. Epidemiological and help-seeking characteristics of specific phobia are shown below.

A genetic predisposition to specific phobia is likely to be nonspecific. Learning theories have provided a useful explanatory framework for specific phobia. Thus, phobia may be learned through a traumatic conditioning (direct aversive experience with the phobic stimulus), vicariously (observation of the fear in others) or via transmission of the information on the dangerousness of certain objects or situations. Some types of specific phobia (phobia of heights, water and spiders) have been conceptualised as “innate” fears, which may have a survival value and do not have to be learned (Menzies and Clarke, 1995).

The diagnosis of specific phobia requires the presence of the characteristic fear and avoidance and is warranted only if the fear is persistent and has caused significant distress or functional impairment. The fear is out of proportion to the actual threat posed by the phobic stimuli and to the sociocultural context. Insight that the fear is irrational or excessive is usually preserved, but may be absent in

children. In clinical practice, specific phobia may need to be distinguished from agoraphobia.

Specific phobia is typically a chronic disorder and fears rarely abate. The impairment is directly related to the degree of avoidance. Specific phobia may precede various other anxiety disorders. The prognosis is generally good, especially with treatment.

Lifetime prevalence

- From 0.6% to 12.5%
- Best-estimate: 5.3% (Somers et al., 2006)

Male-to-female ratio

- 1:2 (except for blood-injection-injury phobia)

Usual onset

- Childhood (except for situational phobia which tends to have an onset in adolescence or early adulthood)

Help-seeking patterns

- Delayed help-seeking, usually due to successful avoidance and relatively low functional impairment
- When help is sought, this is often due to changes in life circumstances that prevent ongoing avoidance (e.g., commencing a job that involves frequent air travel makes avoidance of flying no longer possible)

Generalised Anxiety Disorder (GAD)

The main characteristics of GAD are pathological worry and symptoms of tension. Pathological worry is a key symptom and GAD does not refer only to high levels of anxiety. Symptoms of autonomic hyperarousal may also be present, but they are usually less severe than in other anxiety disorders. Individuals with GAD worry about a range of issues, including health, relationships, family, work and finances. Pathological worry differs from normal worry in terms of the anxiety-amplifying and uncontrollable “what if” pattern of thinking. Such worry denotes an almost incessant and fruitless overthinking, whereby the threat is grossly exaggerated, focusing on the task to find a rational solution is diminished, further problems continue to be anticipated and there is no closure in sight. Pathological worry thus interferes with problem solving and decision making, which causes impairment in various domains of functioning.

Tension in GAD is usually experienced or observed as nervousness, feeling “keyed up” or “on edge”, irritability, exaggerated startle response, hypervigilance, restlessness or inability to relax. Muscle tension is one of the characteristic symptoms of GAD, with common complaints of tension headache and stiffness or pain in the neck, shoulder or back. Muscle spasms, tic-like movements, jerks, fine tremor and difficulty swallowing may also be present. Many individuals with GAD complain of sleep disturbance and fatigue, largely as a consequence of tension and constant and unproductive worrying.

Generalised anxiety disorder is arguably the most common anxiety disorder in the general population. Epidemiological and help-seeking characteristics of GAD are shown in the box below.

Various biological and psychological factors are likely to play a role in the aetiology of GAD. A genetic predisposition to GAD is so similar to the genetic predisposition to major depressive disorder that the two conditions have been regarded as genetically indistinguishable (Kendler et al., 1992). Various neurobiological mechanisms (e.g., hyperactivity of the noradrenaline system and a decreased function of the GABA-A receptors) have also been implicated, suggesting that GAD is a heterogeneous condition. Psychological models of GAD have mainly focused on the origins and functions of pathological worry. Thus, it has been hypothesised that pathological worry is largely driven by a difficulty in coming to terms with uncertainty (intolerance of uncertainty) (Ladouceur et al., 1997), with a notion that worrying could perhaps cease only with attainment of complete certainty. Another theory postulates that worrying allows avoidance of unpleasant bodily symptoms that accompany strong emotional states (Borkovec et al., 1998). Beliefs about the benefits of worrying (e.g., that worrying can prevent catastrophic outcomes) may reinforce pathological worry and thereby help maintain GAD (Freeston et al., 1994).

Making the diagnosis of GAD may not be easy. Patients rarely present with pathological worry as their main problem because many have reluctantly accepted it as a part of their personality that cannot be changed. Instead, a large proportion of patients present with symptoms such as insomnia or headaches, or vague complaints of “stress”, especially when seeking help from general practitioners. By the time they seek help, many individuals with GAD have become depressed, and GAD may be overshadowed by depressive symptoms. For these reasons, many cases of GAD are missed or misdiagnosed, GAD is rarely seen alone in clinical settings and is often diagnosed as a condition accompanying other mental disorders, usually major depressive disorder or another anxiety disorder.

The course of GAD is chronic. Besides depression, substance use disorders can also complicate its course. The impairment associated with GAD can be substantial.

Lifetime prevalence

- From 1.9% to 31.1%
- Best-estimate: 6.2% (Somers et al., 2006)

Prevalence across life-span

- High prevalence in all age groups
- Most common anxiety disorder among the elderly

Male-to-female ratio

- 1:2

Onset

- Usually in late adolescence or early adulthood
- Onset can occur at any age

Help-seeking patterns

- Delayed help-seeking, often due to perception of worrying and chronic anxiety as part of one's "normal" personality
- When help is sought, this is often due to complications such as depression

Treatment of Anxiety Disorders

The treatment approach to anxiety disorders is based on several components: 1) A thorough biopsychosocial and lifestyle assessment; 2) Exclusion of the relevant medical conditions and psychotropic substances as the causes of pathological anxiety; 3) Identification of the factors that maintain the disorder; 4) Collaboration between mental health professionals.

Treatment of the various anxiety disorders has many commonalities, and the same or similar treatment modalities are used across the anxiety disorders. The goals of treatment include a substantial decrease in anxiety and anxiety-related behaviours (e.g., avoidance), better coping with anxiety, decrease in vulnerability, prevention of recurrences and complications and improved functioning and quality of life. Treatments differ in terms of their potential to help patients reach these goals and how they go about reducing the impact of pathological anxiety.

Pharmacotherapy and cognitive behaviour therapy (CBT) have been most widely used and their efficacy has been demonstrated by numerous controlled trials (see Chapter 33). Modifications of CBT, such as mindfulness-based therapy and acceptance and commitment therapy, have also shown efficacy. In addition, several controlled studies have demonstrated the efficacy of psychodynamic psychotherapy. Pharmacotherapy and psychological treatment are often combined in clinical practice and there is some evidence to support this approach.

Pharmacotherapy

Pharmacological agents are used to alleviate distress and symptoms of anxiety and any co-occurring disorders, especially depression (see Chapter 35). Medications such as benzodiazepines are particularly effective in giving rapid relief of acute anxiety, which is often valued by patients because it improves their functioning and allows them to engage in other treatments, albeit it also carries the risk of dependence. Medications do not improve coping with anxiety directly and they are effective only for as long as they are taken. Therefore, relapses following the cessation of pharmacotherapy are common and prevention of relapse is enhanced by the addition of treatments that teach patients strategies for effective and long-term coping with anxiety, for example, CBT. The key task in pharmacological treatment is to find the right balance between the effectiveness of the medication and its adverse effects.

The aim of pharmacotherapy of anxiety disorders is remission, which is defined as the minimal presence of symptoms, little or no anxiety-related distress and return to normal functioning. Remission may decrease the risk of relapse after medication discontinuation and is most likely achieved by using the medication in an adequate dose and long enough (usually for at least 6-12 weeks). If an adequate trial with the initial (first-line) medication produces no improvement, the patient can be administered another (second-line) pharmacological agent. If there is some improvement, an augmenting agent (usually the second- or third-line medication) can be added. Approximately 75% of patients respond to the initial low dose of antidepressants, but patients with anxiety disorders typically take longer to respond than those with depression (Bandelow et al., 2012).

Maintaining remission requires an ongoing, daily use of the medication for at least 6 months. If the remission has lasted for at least 6-12 months and the patient is ready for discontinuation of pharmacotherapy, medication taper can be planned. The duration of this gradual reduction in dose until the medication is ceased depends on the type of medication, presence of any anxiety or withdrawal symptoms and various personal circumstances, such as ability to tolerate distress and cope. As a rule, medication should not be ceased abruptly after long-term pharmacotherapy.

Selective serotonin reuptake inhibitors and serotonin and noradrenaline reuptake inhibitors are considered by the evidence-based treatment guidelines (Katzman et al., 2014) to be the first-line pharmacotherapy for panic disorder, social anxiety disorder and GAD. Second- or third-line medications include benzodiazepines, tricyclic antidepressants for panic disorder and GAD, classical, irreversible monoamine oxidase inhibitors for social anxiety disorder and pregabalin, agomelatine and bupirone for GAD. Antidepressants are not always effective, they do not work quickly and are often associated with adverse effects. For these reasons, benzodiazepines can be used as second- or third-line pharmacotherapy or as augmenting agents. They are generally effective at relieving the symptoms of anxiety and well tolerated, but need to be administered carefully because of their own adverse effects and dependence associated with long-term use and because their use does not always result in functional improvement.

Pharmacotherapy is of little value for specific phobia. Medication treatment of agoraphobia is useful to the extent that it is effective in treating the associated panic attacks. Pharmacotherapy appears to have a limited role for agoraphobia unrelated to panic attacks.

Cognitive Behaviour Therapy (CBT)

The main advantages of CBT over pharmacotherapy are that it enables patients to cope with anxiety effectively and that treatment gains are maintained post-treatment and even enhanced with the passage of time. It achieves this by helping patients change unhelpful beliefs about the dangers of anxiety and by assisting them to tolerate anxiety symptoms better and reduce avoidance of feared situations. Such changes improve patients' functioning and decrease their vulnerability to future anxiety and the risk of relapse after treatment cessation. In addition, CBT fosters an active attitude towards treatment, which allows patients to develop a sense of ownership of their treatment gains. For these reasons, therapeutic effects of CBT are more likely to last longer than those of pharmacotherapy.

The techniques of cognitive and behaviour therapy for anxiety disorders are usually combined in clinical practice. They can be administered in individual and group formats, and in recent times CBT has also been successfully delivered via the Internet.

The first step in CBT is psychoeducation. This includes provision of information about anxiety, correction of any misconceptions and identification of the factors that maintain the disorder, which serves as the basis for treatment. Cognitive therapy techniques target maladaptive, anxiety-related assumptions,

beliefs, misinterpretations and appraisals, which play a role in the development and maintenance of anxiety disorders. In panic disorder, these techniques are used to challenge and correct catastrophic misinterpretations of bodily symptoms and to modify beliefs about body-based threat and the dangerous nature of anxiety. In social anxiety disorder, cognitive therapy addresses the negative assumptions and beliefs about oneself, others and social situations, as well as perception of the social environment as threatening and judgemental. Cognitive therapy techniques for GAD include imaginal exposure to the content of worries, modification of beliefs about the benefit of worrying and exercises that aim to improve coping with uncertainty.

The goal of behaviour therapy is to change and eliminate behaviours (e.g., avoidance) that help maintain pathological anxiety and impair function. The key behaviour therapy technique is exposure to the situations or stimuli that elicit anxiety. It is particularly useful for phobic disorders because they are characterised by prominent avoidance. Habituation – a decrease in anxiety upon repeated exposure to phobic stimuli – is believed to be the key mechanism that accounts for the effectiveness of exposure therapy. Exposure is best tolerated when it is performed gradually so that patients face in a step-by-step fashion the phobic stimuli that elicit increasing levels of anxiety. Exposure should ideally be self-directed and *in vivo*, but depending on the nature of the phobia and patient characteristics, therapist-assisted and imaginal exposure may need to be conducted first. This may be combined with role-play and modelling of therapist's behaviour (e.g., holding an insect) during therapeutic sessions. Patients with panic disorder are instructed to induce the specific symptoms of autonomic hyperarousal and thus conduct exposure to these feared bodily stimuli; this technique is referred to as interoceptive exposure.

Symptom control techniques reduce bodily symptoms of anxiety and may be used in conjunction with CBT. They include various muscle relaxation techniques that can be useful for all anxiety disorders, especially GAD. Another symptom control technique is breathing retraining, which may be helpful in the treatment of panic patients with hyperventilation and other breathing difficulties. It is important that such techniques do not become “safety behaviours”, used by patients for “prevention” of or distraction from anxiety. Research has shown that a focus on tolerating anxiety symptoms and being realistic about the consequences of anxiety is more effective than trying to prevent anxiety or panic. Social skills training, including assertiveness training, can be added to CBT in the treatment of social anxiety disorder when social skills may be lacking.

Further Reading

- Baldwin, D. S., & Leonard, B. E. (Eds.). (2013). *Anxiety disorders* (Vol. 29). Karger Medical and Scientific Publishers.
- Boyce, P., Harris, A., Drobny, J., Lampe, L., Starcevic, V., & Bryant, R. (Eds.). (2015). *The Sydney Handbook of Anxiety Disorders: A Guide to the Symptoms, Causes and Treatments of Anxiety Disorders*. The University of Sydney.
- Castle, D., Hood, S., & Starcevic, V. (Eds.). (2012). *Anxiety Disorders: Current Understandings Novel Treatments*. Australian Postgraduate Medicine.
- Simpson, H. B., Neria, Y., Lewis-Fernández, R., & Schneier, F. (2010). Introduction: The need for interdisciplinary approaches In Simpson. *HB, Neria, Y., Lewis-Fernández, R., & Schneier, F.(Eds.).(2010). Anxiety disorders: Theory, research and clinical perspectives*, 1-5.
- Stahl, S. M., & Moore, B. A. (Eds.). (2013). *Anxiety disorders: A guide for integrating psychopharmacology and psychotherapy*. Routledge.
- Starcevic, V. (2009). *Anxiety Disorders in Adults A Clinical Guide*. Oxford University Press.
- Stein, D. J., Hollander, E., & Rothbaum, B. O. (2010). *Textbook of anxiety disorders*. Second edition. Washington, DC/London: American Psychiatric Publishing.

Chapter 19: Obsessive-Compulsive and Related Disorders

Vlasios Brakoulis

Specialty of Psychiatry, The University of Sydney, Australia.

Introduction

The Obsessive-Compulsive and Related Disorders have arisen from the broader concept of Obsessive-Compulsive Spectrum Disorders first proposed by Hollander in 1993 (Hollander, 1993) which included related and co-occurring disorders. For the first time, DSM-5 (American Psychiatric Association, 2013) grouped a number of disorders into a new chapter called “Obsessive-Compulsive and Related Disorders” based on the presence of an obsessive preoccupation and/or repetitive behaviours. These disorders are listed in the box below. It is important to note that these disorders co-occur and that their treatment usually involves serotonergic antidepressants and/or psychological therapies based on similar principles.

Obsessive-compulsive and related disorders

- Obsessive-compulsive disorder
- Body dysmorphic disorder
- Hoarding disorder
- Trichotillomania
- Excoriation disorder (Skin picking disorder)
- Body-focused repetitive behaviour disorder (e.g., excessive nail biting)
- Obsessional jealousy
- Jikoshu-Kyofu (Olfactory reference syndrome)

Obsessive-Compulsive Disorder

Obsessive-compulsive disorder (OCD) is characterised by obsessions and/or compulsions. Obsessions are defined as recurrent, intrusive and distressing thoughts, images or impulses. Classically obsessions were defined as *ego-dystonic* in that they were thought to be in sharp contrast to the individual's wishes and desires. Compulsions are repetitive behaviours or mental acts. Obsessions and compulsions are usually recognized as irrational and most sufferers have full insight into their unreasonable and excessive nature. See the box below for some examples of common obsessions and compulsions. OCD can also present with indecisiveness, obsessional slowness, avoidance and excessive reassurance-seeking.

Obsessions	Compulsions
• I have left the stove on and the house will burn down. • If I touch something, I will be contaminated. • I might accidentally stab someone. • I feel an urge to touch a stranger's genitals. • A recurrent blasphemous thought or image. • I become distressed if I see a dining chair out of place. • If I am not clean, I might give my children food poisoning.	• Repeated checking of knobs, locks and windows. • Excessive handwashing or cleaning. • Excessive ordering and arranging. • Repeated praying. • Inability to stop counting. • A need to ask people things repeatedly. • A need to repeatedly touch things.

Epidemiology

OCD is thought to occur in 0.8 to 2% of the population (Karno et al., 1988). Subclinical obsessive-compulsive symptoms occur at a much higher rate, as do obsessive-compulsive or "perfectionistic" personality traits, which are commonly confused with OCD by the general public. Obsessive-compulsive disorder often has its onset in early adolescence, but also has a later peak in the mid-twenties. Early onset is associated with a male preponderance, tic disorders and higher incidence of OCD in other family members (Steven, 2011). Early onset OCD is also associated with greater severity of symptoms. Despite the common misperception that OCD is a trivial disorder, OCD has been ranked among the top ten most disabling of all

medical conditions (Murray and Lopez, 1996). Rates of OCD are similar in men and women and across different cultures, unlike anxiety disorders where women have higher rates of anxiety disorders than men (Weissman et al., 1994).

Aetiology

The aetiology of OCD is unknown but is likely to be multifactorial. Although no single gene has been linked to the development of OCD, twin studies indicate that both genetic and environmental factors play a role in its development (Taylor et al., 2010). Functional neuroimaging has shown abnormal and heightened connectivity of the ventrolimbic corticostriatal regions of people with OCD (Harrison et al., 2009). Cognitive models link OCD to an inflated sense of responsibility that has hypothesised to have arisen from over-protective parenting (Salkovskis, 1999), and psychoanalytic models propose a regression to the anal stage of psychosexual development (Freud, 1958). A cognitive model of understanding OCD symptoms is presented in Figure 19.1.

Presentation

It is important to appreciate that people with OCD are often too ashamed or embarrassed to present for help. They commonly present because their relatives can no longer tolerate their behaviour, or due to a co-occurring anxiety or mood disorder. When people with OCD self-present, they can no longer tolerate the distress associated with their OCD or their OCD symptoms are causing functional impairment. Compulsions can take many hours a day and can lead to delays in completing tasks and getting to appointments on time. The only common physical sign is the presence of erythematous and dry hands caused by excessive hand washing.

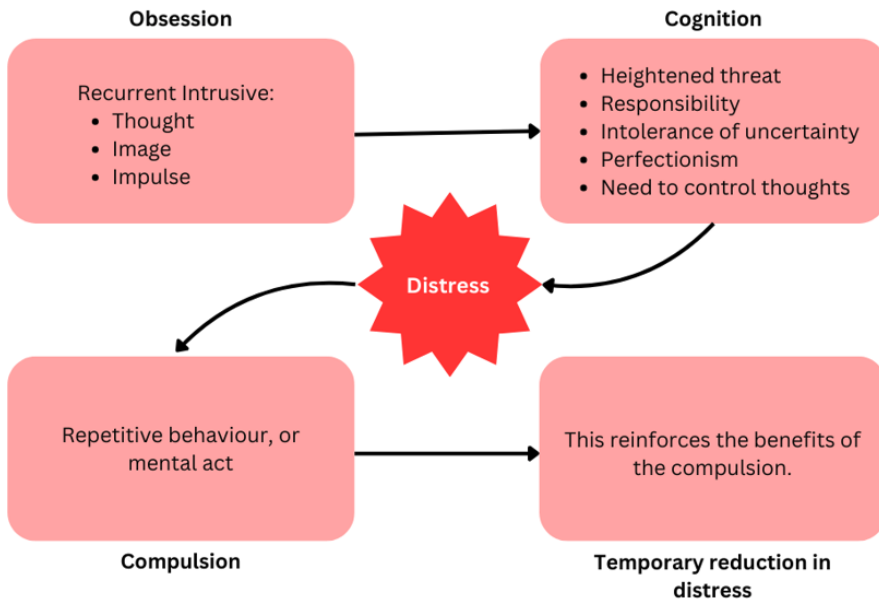


Figure 19.1. A cognitive model for OCD.

Assessment

Screening for OCD involves screening for common symptoms for OCD. Symptom severity is most commonly assessed using the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) (Goodman et al., 1989). OCD is diagnosed when the symptoms are distressing, time consuming (i.e. when they take more than one hour per day) or when they cause significant functional impairment.

Differential Diagnosis and Important Comorbidities

The most commonly co-occurring disorders are major depression and generalised anxiety disorder. It can be difficult to differentiate obsessions from depressive ruminations (reflections on the past) and excessive worries (worries about the future) associated with generalised anxiety disorder. The other diagnosis which one must be aware of is schizophrenia as obsessive-compulsive symptoms occur in approximately 15% of patients with schizophrenia, they can also occur in the prodrome of schizophrenia (see Chapter 15). Patients with severe OCD can present with poor insight where they are convinced that they need to do something compulsively, or where they believe that their obsession is reasonable.

Another important co-occurring disorder is tic disorder. Tics occur more commonly in patients who have early onset OCD and a family history of OCD. Tics are defined as spasmodic movements that often occur in facial musculature.

Tourette's syndrome is diagnosed when motor tics co-occur with vocal tics, for example, coughing or grunting noises.

Evidence-Based Treatments

OCD can be treated with either cognitive behavioural therapy (CBT; see Chapter 33) in the form of exposure and response prevention (ERP), or with high dose selective serotonin reuptake inhibitor (SSRIs). If either is ineffective, treatments are then combined.

Exposure and Response Prevention

The most effective psychological treatment for OCD is ERP for both adults and children (Abramowitz et al., 2006; Foa et al., 2007). This involves exposing the sufferer to the thing they fear and preventing their response or compulsion. For example, asking someone with contamination fears to touch a door handle and then to prevent their response of compulsively washing their hands. ERP typically involves 15 to 30 sessions which means that psychological treatment usually takes longer than for other anxiety disorders. ERP is thought to work by disconfirming any threat-related cognitions associated with OCD and improving one's confidence when faced with anxiety provoking situations. As family members often accommodate to the requests of sufferers associated with their compulsions or avoidance in order to reduce their loved one's distress, it can help to explain to family members how they can help reduce OCD behaviour and assist the patient with their ERP tasks.

Pharmacotherapy

Pharmacotherapy usually consists of a high dose serotonergic medication (SSRIs and clomipramine). SSRI medications are increased in dose over a number of weeks providing they are tolerated and that there is some degree of improvement. Providing there has been some improvement, the SSRI should be continued for at least 3 months to determine the maximum effect of the medication. Clomipramine, a serotonergic tricyclic antidepressant, is also effective in treating OCD. High doses are usually necessary and clinical response takes considerable time. In practice, it is not used first line because of a high rate of adverse effects. Common adverse effects include dry mouth, dizziness, low blood pressure, drowsiness, blurred vision, urinary retention, and sexual dysfunction. When high dose serotonergic antidepressants have only had a partial response, augmentation with antipsychotic medication can be useful.

Outcome

With appropriate therapy, most people with OCD can expect a reduction in the severity of their OCD symptoms. However, complete remission is

uncommon and many people with OCD remain chronically disabled by their symptoms. Hence, OCD is usually considered a difficult to treat relapsing and remitting disorder. Some patients have severe, intractable OCD which may require treatment with intensive in-patient ERP, deep brain stimulation or various forms of psychosurgery.

Body Dysmorphic Disorder

Body dysmorphic disorder is characterised by an excessive preoccupation with one's appearance or with a part of their body. People with body dysmorphic disorder believe that a part of their body, e.g. their nose, is misshapen, discoloured or deformed. This preoccupation can lead to repetitive checking in the mirror, seeking plastic surgery, and/or avoidance of social situations. The treatment of body dysmorphic disorder is similar to OCD. Despite a higher rate of more strongly held beliefs than OCD, SSRI treatment is more effective than antipsychotic medication.

Hoarding Disorder

Hoarding disorder refers to the excessive accumulation of household items accompanied by the inability to discard these items. In community surveys, hoarding disorder appears to affect approximately 1% of the population (Nordsletten et al., 2013). To meet criteria for a disorder, at least one room of the person's house is required to be cluttered to such an extent that it can no longer be used, for example the person with hoarding disorder is unable to sleep in his or her bedroom as it has too many items in it. Hoarding disorder is often confused with squalor which is characterised by much more disorganisation and filth with the aetiology of squalor often being dementia, schizophrenia or chronic alcohol abuse. Hoarding disorder is challenging to treat, but some studies have supported tailored cognitive behavioural strategies and SSRIs.

Trichotillomania

In trichotillomania, also known as "*hair pulling disorder*", sufferers excessively pull out hair from their head, eyebrows, eye lashes or pubic area to the extent that baldness develops. There is often a sense of pleasure associated with feeling the hair and a sense of relief when the hair is pulled out. The hair loss can be so severe that the sufferer needs to wear hats or wigs to hide their patches of hair loss. Children can swallow their hairs and this can lead to hair balls (bezoars) forming in the gastrointestinal tract that can lead to gastrointestinal obstruction. Although there is some evidence for treating trichotillomania with SSRIs, trichotillomania had traditionally been regarded as an impulse control disorder (i.e. a failure to resist

temptation, urges or impulses to hurt oneself or others) and behavioural techniques modified for trichotillomania such as habit reversal therapy are usually tried first. Habit reversal therapy is a psychological therapy that involves reversing bad habits or impulses by introducing other less deleterious actions, e.g. squeezing a stress ball rather than engaging in hair pulling. The amino acid supplement N-acetylcysteine may also be useful for treating trichotillomania when administered at doses between 600mg and 1200mg twice daily.

Excoriation Disorder (Skin Picking Disorder)

Excoriation disorder (also known as dermatillomania) is defined by recurrent picking of the skin to the extent that skin lesions develop. Similar behaviour can result from stimulant abuse. There is some evidence that habit reversal therapy, SSRIs and N-acetylcysteine can help reduce skin picking.

Body-Focused Repetitive Behaviour Disorder

Body-focused repetitive behaviour disorder refers to distressing and/or damaging repetitive behaviours that focus on the body and cannot be easily stopped by the sufferer. Examples of body-focused repetitive behaviours include excessive nail biting, lip biting, cheek chewing and chewing of the knuckles. As with trichotillomania, habit reversal therapy is often used as a specific psychological therapy aiming to reverse the body-focused behaviour by introducing a less harmful behaviour.

Obsessional Jealousy

In obsessional jealousy there is an excessive preoccupation with the idea that one's spouse or partner might be having an affair. This preoccupation is usually accompanied by excessive checking, e.g. of the spouse's underwear, telephone records, car seats for hairs, and car speedometer. There may also be associated stalking. If the preoccupation becomes delusional where the patient firmly believes that their spouse or partner has been unfaithful without any proof to substantiate their claim, then the disorder is regarded as delusional jealousy or Othello's Syndrome. As some patients with delusional jealousy have been known to murder their spouse, one must always assess for any risk that the preoccupied individual may pose towards their spouse or the person that they might suspect that their spouse is having an affair with. Treatment with SSRIs and psychotherapy has been used (Stein et al., 1994).

Olfactory Reference Syndrome (Jikoshu-Kyofu)

Olfactory reference syndrome is also known as Jikoshu-Kyofu as it has been well documented in the Japanese literature. It refers to a disorder where effected individuals are preoccupied with a belief that they smell or that they are emitting a foul odour when they do not obviously smell. This belief is associated with avoidance of social settings and the excessive use of deodorants or mouth fresheners. This disorder can be treated with SSRIs (Matsunaga et al., 2001).

Further Reading

- Abramowitz, J. S., Taylor, S., & McKay, D. (2009). Obsessive-compulsive disorder. *The Lancet*, 374(9688), 491-499.
- Stein, D. J., Costa, D. L., Lochner, C., Miguel, E. C., Reddy, Y. J., Shavitt, R. G., ... & Simpson, H. B. (2019). Obsessive-compulsive disorder. *Nature Reviews Disease Primers*, 5(1), 52.

Chapter 20: Posttraumatic Stress Disorder

Richard A. Bryant

School of Psychology, The University of New South Wales, Australia.

Introduction

The capacity for people to develop psychopathological stress reactions has been recognised for over 100 years, however it is only in the wake of the Vietnam war that the definition of posttraumatic stress disorder (PTSD) was formally introduced. The current definition by the American Psychiatric Association's DSM-5-TR (American Psychiatric Association, 2022) stipulates that for a person to be considered for a diagnosis of PTSD, they must experience or witness a marked threat to themselves or others; this form of threat needs to be severe, such as war, disaster, assault, or severe accident. Although harassment or bullying can be very stressful, these types of events do not involve threat to safety or life and so tend not to trigger PTSD. If a person has experienced a traumatic event, then they need to experience four different clusters of symptoms. First, a person needs to experience a group of symptoms that comprise re-experiencing of the trauma, including intrusive memories, flashbacks, nightmares, and distress to reminders of the trauma. Second, one is required to display active avoidance of internal reminders of the trauma (e.g. thoughts, memories) or external reminders (e.g. situations, conversations). Third, the person needs to have a range of symptoms involving mood and thoughts, such as exaggerated negative thoughts (e.g. "no one can be trusted", "everything is ruined"), excessive blame, pervasive negative emotions (e.g. horror, fear, guilt or shame), diminished interest, feeling detached or estranged from others, or psychogenic amnesia. The final cluster comprises hyperarousal symptoms, such as exaggerated startle response, reckless behaviour, insomnia, aggressive behaviour, and sleeping or concentration difficulties.

Diagnostic systems have always recognised that most people will have stress reactions in the acute phase after a traumatic event. Many of these responses should not be pathologized because evidence indicates that most of these reactions will remit in the following weeks (Bryant, 2003). Accordingly, DSM-5-TR (American Psychiatric Association, 2022) stipulates that PTSD can only be diagnosed one

month after trauma. There is a provision for diagnosing people in the acute phase in DSM-5-TR in the form of acute stress disorder, which describes posttraumatic stress reactions that occur in the first month after trauma exposure. This diagnosis was initially introduced in DSM-IV as a means of describing severely distressed people who could benefit from treatment, as in many healthcare systems in the USA it can be difficult to access treatment without a diagnosis. It was also initially constructed as a potential means to identify people in the acute phase who are high risk for developing subsequent PTSD. Longitudinal studies have shown that the acute stress disorder diagnosis is only a modest predictor of PTSD because at least half of people who develop PTSD do not initially meet the acute stress disorder criteria (Bryant, 2010). In DSM-5-TR the acute stress disorder diagnosis does not require specific symptoms clusters to be satisfied but rather requires that at least 9 of 14 potential acute stress reactions be present (Bryant et al., 2011). This diagnosis is not intended to predict subsequent PTSD but instead aims to identify people who could benefit from treatment in the initial month after trauma (Bryant et al., 2011).

Prevalence of PTSD

Epidemiological studies indicate that most people will experience a traumatic event at some point in their lives, however these studies also suggest most people are resilient and do not develop a psychiatric disorder (Bonanno et al., 2015). Lifetime prevalence rates of PTSD are between 13%-20.4% for women and between 6.2%-8.2% for men (Breslau et al., 1991; Kessler et al., 2013). There is variability depending on the type of traumatic event, with interpersonal violence resulting in higher rates of PTSD (Forbes et al., 2011; Forbes et al., 2013). Consistent with this conclusion, the World Mental Health Survey found that organized, physical, or sexual violence increased the risk for PTSD (Liu et al., 2017). It is important to note that there are high rates of comorbidity between PTSD and other psychiatric disorders, including depression, anxiety disorders, and substance use disorder (Breslau et al., 1997; Kessler et al., 2013; Rytwinski et al., 2013).

The Course of PTSD

Many longitudinal studies have tracked trauma over time, and this has allowed understanding of how traumatic stress develops and maintains over time. We now know that rather than being a static condition, it actually fluctuates over time (Bryant et al., 2013). Further, modelling shows that there are four major trajectories of traumatic stress response: (a) a resilient class with few PTSD symptoms, (b) a recovery class with initial distress then gradual remission, (c) a delayed reaction class with initial low symptom levels but increased symptoms over time, and (d) a chronic distress class with consistently high PTSD levels (Bonanno et al., 2008; Bryant et al., 2015; deRoos-Cassini et al., 2010; Galatzer-Levy et al.,

2018). This pattern has resulted in a major challenge to early intervention following trauma exposure because we do not have robust metrics to predict in the acute phase who will subsequently develop PTSD. Although people with more severe psychological distress in the weeks after trauma are more likely to develop PTSD (Blanchard et al., 1997; Frommberger et al., 1998; Jehel et al., 2003; Karstoft et al., 2015; Shalev and Freedman, 2005), this association does not allow us to definitively identify who will and will not develop PTSD.

Risk Factors

What predisposes some trauma survivors to developing PTSD? Many of the risk factors are similar to risk factors observed across many psychiatric disorders, including female gender, low socio-demographic background, prior psychological disorder, family history of psychological disorders, and traumatic childhoods (Brewin et al., 2000). Risk also increases in relation to factors associated with the traumatic events, including more prolonged trauma, grotesque or interpersonal trauma, as well as the number of traumatic events that a person has experienced (Ozer et al., 2003). There is also considerable evidence that how a person responds to the trauma is also predictive of later PTSD, including dissociative reactions that comprise altered sense of reality or oneself after the trauma (Murray et al., 2002; Shalev et al., 1998) and catastrophic appraisals (Dunmore et al., 2001; Kleim et al., 2007; Wikman et al., 2011). There is also much evidence that poor social support and ongoing stressors contribute to the risk of the development of PTSD (Brewin et al., 2000).

Theories of PTSD

Neurobiological Models

Arguably the most influential theory of PTSD involves the concept of fear conditioning. In this theory it is proposed that stress hormones released at the time of trauma in association with the extreme fear experienced by a person leads to a strong learning response that associates the cues present at the time of trauma with the fear responses (Shalev et al., 2017). This learning leads to people reacting to these, and similar, cues in their environment as though the trauma is occurring again. A hallmark symptom of PTSD is the sense that memories of the trauma are very real and a sense that the trauma is recurring. For example, a firefighter may develop PTSD after pulling a dead baby out of a burning building. In the process the stimuli present at the time, such as the smell of smoke and burnt skin, may be strongly associated with the fear response. Subsequent to the trauma, each time the firefighter smells burnt meat (e.g., at a barbeque), he may experience intrusive memories and extreme distress because the memories of the trauma are readily activated and he

feels it is recurring. These models also propose that most people recover from initial stress reactions through a process called extinction learning, in which one is repeatedly exposed to reminders of the trauma but because there are no harmful outcomes, they learn that these stimuli are not indicative of threat but rather that they signal safety (Rauch and Drevets, 2009).

There is considerable evidence for fear conditioning models explaining PTSD. People with PTSD have dysfunction in neural circuits that are involved in fear conditioning, including the amygdala, prefrontal cortex, and hippocampus (Ressler et al., 2022). There is also much evidence of reduced activity of the medial prefrontal cortex, which is key for extinction learning (Kitayama et al., 2006).

There is also much evidence of neuroendocrinological abnormalities in PTSD, including noradrenergic and glucocorticoid abnormalities (Hendrickson and Raskind, 2016). Noradrenergic release interacts with cortisol to facilitate traumatic memories (McGaugh, 2000), and these abnormalities are well-documented in PTSD (Southwick et al., 1999; Southwick et al., 1997), and may explain the intrusive re-experiencing of trauma memories. Further support is from evidence that administration of propranolol, a beta-blocker that reduces noradrenergic activity, in the hours after trauma exposure limits subsequent reactivity to reminders (Pitman et al., 2002). In the context of the noradrenergic system, it is also worth noting that there is a potentially protective role for morphine in the acute phase after trauma. The locus coeruleus, which produces norepinephrine, is inhibited by morphine, and animal work indicates that morphine injections into the amygdala impairs memory for fear conditioning in rats (McNally and Westbrook, 2003). Numerous studies of traumatized populations indicate that greater morphine dose in the initial days after trauma exposure is associated with reduced PTSD at follow-up. Importantly, these studies are not randomized controlled trials, and so they do not allow us to conclude that morphine can limit subsequent PTSD.

One of the major mechanisms in the stress response is the glucocorticoid system. Interestingly, although increased cortisol is typically associated with chronic stress, PTSD is often associated with lower cortisol levels (Yehuda et al., 1990). It has also been shown that lower cortisol levels shortly after trauma predict subsequent PTSD severity (Delahanty et al., 2000). This paradoxical finding is interpreted in terms of cortisol binding to the glucocorticoid receptors in a negative feedback loop that promotes homeostasis of the stress response (Yehuda and McFarlane, 1997). It is proposed that lower cortisol in PTSD may result in elevated ongoing HPA activity, resulting in exaggerated catecholamine response and consequent over-consolidation of trauma memories. This proposition has received some initial support from early intervention studies that administration of

hydrocortisone shortly after trauma limits subsequent PTSD symptoms (Zohar et al., 2011b).

There is also much evidence of strong psychophysiological reactivity to reminders of the trauma in people with PTSD, which supports fear conditioning models. When given reminders of the trauma, people with PTSD display greater heart rate, skin conductance response, or facial electromyogram measurements than those without PTSD (Orr et al., 1993). Further, elevated resting heart rate in the days after trauma is predictive of later developing PTSD (Bryant et al., 2000).

Genetic Factors

Genetic factors play an important role in susceptibility to PTSD, accounting for 30%-72% of risk for developing PTSD (Sartor et al., 2011; True et al., 1993). Studies suggest that genes associated with PTSD are also linked with other common psychiatric disorders, including major depression, generalized anxiety disorder, panic disorder, and substance use (Koenen et al., 2008). For example, studies have indicated a role of the functional polymorphism in the promoter region of the serotonin transporter gene (SLC6A4) across many disorders, and this pattern extends to PTSD. The short allele (5-HTTLPR S), which reduces serotonergic expression and uptake by nearly 50% (Lesch et al., 1996), has been linked with impaired extinction learning (Galatzer-Levy et al., 2017), and gene x environment association studies indicate that *FKBP5* alleles increase risk for PTSD (Binder et al., 2008). Currently over 50 gene variants have been linked with PTSD, including HPA axis functions, noradrenergic, dopaminergic and 5-HT systems, and neurotrophins (Sheerin et al., 2017). To sum up this work, there is no single gene that represents a significant risk distinctively for PTSD, it appears that polygenetic approaches are more appropriate. One attempt to address this problem is the Psychiatric Genomics Consortium – Posttraumatic Stress Disorder Group, which has reported a genome-wide analysis of 20,730 people. Although no single-nucleotide polymorphism was found to be significantly associated with PTSD, this study did find a polygenetic risk profile that overlapped with risk for schizophrenia (Duncan et al., 2018).

Cognitive Behavioural Models

Cognitive behavioural models typically acknowledge that fear conditioning is instrumental in the development of PTSD, however they put much more emphasis on the influence of cognitive appraisals about the traumatic event, the person's responses to it, and their perceptions of future likelihood of harm. By engaging in excessively negative appraisals about the sense of threat or pessimism of recovery, these cognitive styles tend to maintain PTSD (Foa et al., 1999). Evidence indicates that engaging in negative appraisals prior to trauma is a risk factor for developing

PTSD (Bryant and Guthrie, 2007), and that the presence of these appraisals shortly after trauma predicts development and maintenance of PTSD (Dunmore et al., 1999; Kleim et al., 2007), as well as their decline after successful therapy (Kleim et al., 2013). This exaggerated sense of threat in PTSD can lead to strong avoidance of potential threats, which in turn deprives the person of learning that normal daily events (including those that are reminders of the trauma) are not threatening, and this impedes extinction learning (Foa et al., 1989).

Prevention of PTSD

Prevention

Programs have been initiated to prevent people developing PTSD by teaching them strategies that are intended to ‘inoculate’ them against the aversive elements of a traumatic experience. There is evidence that some prevention programs can mitigate the degree of mental health problems that emerge after trauma (Bisson et al., 2021). One example of attempting to limit PTSD focused on correcting attentional problems that have been observed in PTSD. Although most anxiety disorders are characterised by a bias to attend towards threat, PTSD is distinguished by having a bias to attend both towards (Bryant and Harvey, 1997; Buckley et al., 2000) and away from threat (Bar-Haim et al., 2010; Wald et al., 2011), resulting in greater attentional variability in PTSD (Naim et al., 2015). A large trial built on this evidence to test a prevention program in soldiers involved training them to control their attentional biases by using a brief computer task administered prior to deployment to combat. This trial found that soldiers receiving this program had fewer subsequent PTSD symptoms than those in a control condition and this result was mediated by a reduction in attentional variability, a finding that was subsequently replicated in a cohort of US troops (Badura-Brack et al., 2015). Apart from these isolated studies, however, the overall evidence for prevention of PTSD is very limited.

Psychological Treatments

Most international treatment guidelines recommend that the frontline treatment for PTSD is what may be termed “trauma-focused cognitive behaviour therapy” (Health, 2005). There are a range of treatments that can be described under this category, including Prolonged Exposure, Eye Movement Desensitization and Reprocessing, Cognitive Therapy, Cognitive Processing Therapy, and Imagery Rescripting Therapy. Although each treatment is somewhat different, they converge on two critical elements that are regarded as the core components of effective treatment of PTSD. The treatments involve some form of emotional processing of the traumatic memory and changing maladaptive appraisals so people have a more

realistic perspective of the experience and their future. This treatment has been shown to be effective for people PTSD after traumatic injury and assault, sexual assault, combat, terrorist attacks, refugees, and child sexual abuse (Bryant et al., 2008; Duffy et al., 2007; Foa et al., 1991; McDonagh et al., 2005; Neuner et al., 2004; Schnurr et al., 2003). A central strategy used in most treatments is exposure therapy in which the patient is assisted to 'relive' their trauma memory for approximately 10-30 minutes. Much evidence indicates that doing this over a series of therapy sessions results in less distress and fewer intrusive memories of the trauma, and accordingly is conceptualized as a form of extinction learning because the person learns that this trauma reminder is no longer threatening.

This approach has also been used shortly after trauma with patients who are more at risk of developing PTSD because they have high levels of acute stress. These programs employed a brief form of trauma-focused CBT (usually 5-6 sessions), and typically found that they were more efficacious than control conditions (Bryant, 2003; Bryant et al., 1998; Bryant et al., 2008; Bryant et al., 2005; Shalev et al., 2012). One trial even commenced trauma-focused CBT in the emergency department and provided subsequent sessions in the weeks after discharge, and this study also found that this approach can be beneficial (Rothbaum et al., 2014). Note that this approach is different from the universal prevention strategies described above because this 'secondary prevention' focused on patients with elevated stress responses and so more at risk of PTSD. The utility of early provision of trauma-focused CBT is supported by meta-analytic studies that suggest this intervention can limit later PTSD (Kornør et al., 2008; Roberts D Clin Psy et al., 2009). Although trauma-focused CBT has been shown to be effective in reducing PTSD, it is important to note that only two-thirds of patients respond adequately to this intervention (Bradley et al., 2005).

Pharmacological Treatments

PTSD is one of the psychiatric disorders in which psychological treatments are preferred over psychopharmacological interventions, which have only modest success. International treatment guidelines converge on the conclusion that stronger benefits from psychotherapy, potential adverse side-effects from pharmacotherapy, and possible relapse after medication discontinuation results in trauma-focused CBT being the first line of treatment. Despite this conclusion, the major psychotropic medications recommended for PTSD are selective serotonin reuptake inhibitors (SSRIs), which are the only medication approved by the US Food and Drug Administration for treatment of PTSD (sertraline and paroxetine). It should be noted that their effect sizes in PTSD is small (Thomas and Stein, 2017), underscoring the need to prioritise trauma-focused CBT. One reason why SSRIs may be useful in

treating PTSD is that they are effective in treating major depressive disorder, which is highly comorbid with PTSD.

Other medications have been used with PTSD, or rather specific symptoms of PTSD. For example, nightmares have been treated with prazosin (an α 1-adrenergic antagonist), which has been found to be effective in reducing nightmares and hyperarousal (Singh et al., 2016). It is also worth noting that benzodiazepines have often been prescribed in the context of PTSD, and particularly in the acute phase after trauma, as a means of reducing arousal and improving sleep. They are generally contraindicated because of limited efficacy and high risk of addiction. Further, benzodiazepines can result in more subsequent PTSD (Gelpin et al., 1996; Mellman et al., 2002). Potentially it can interfere with the regulatory function of the HPA axis (Zohar et al., 2011a).

Conclusion

As one in ten people will develop PTSD at some point in their lives, it is likely that most medical professionals will encounter patients with this condition regularly. Fortunately, we have efficacious treatments that can benefit most PTSD patients. One of the challenges for treating PTSD is that most patients only receive evidence-based care years after the onset of PTSD, which risks the development of chronic patterns of illness and entrenched psychiatric comorbidities. Medical and health practitioners can play a key role in promoting early help-seeking to stop PTSD from becoming chronic and debilitating.

Further Reading

Shalev, A., Liberzon, I., & Marmar, C. (2017). Post-traumatic stress disorder. *New England Journal of Medicine*, 376(25), 2459-2469.

Chapter 21: Feeding and Eating Disorders

Cornelia Kaufmann

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

Introduction

Feeding and eating disorders are highly distinctive and serious mental disorders characterised by persistent disturbance of eating behaviours which is responsible for impairment of physical and mental health and psychosocial functioning. The following disorders are in this category: Pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa and, recently added in DSM-5, binge eating disorder. These disorders share some diagnostic criteria, whilst other criteria are mutually exclusive and only one diagnosis can be assigned. However, due to the fact that there are a number of common psychological underpinnings and shared behavioural features, patients afflicted by these disorders can “migrate” from one disorder to another. For instance, someone might initially develop anorexia nervosa in middle adolescence and transition to binge eating and/or bulimia nervosa when starvation becomes untenable and impossible to control.

Aetiology

Biological, psychological and socio-cultural factors can all contribute to the development of an eating disorder. Genetic factors influence risk, psychosocial and interpersonal factors can trigger onset and changes in the neural network can sustain the illness (Zipfel et al., 2015). There is evidence that exposure to media images that promote the thin-ideal, may contribute to the development of eating disorders by causing body dissatisfaction, negative moods, low self-esteem, and eating disorders symptoms among women (Hawkins et al., 2004). Strict dieting as a response to body dissatisfaction can in itself trigger the development of an eating disorder.

A study from the Anorexia Nervosa Genetics Initiative (ANGI) and the Eating Disorders Working Group of the Psychiatric Genomics Consortium (PGC-ED) conducted a genome-wide association study of 16,992 cases of anorexia nervosa and 55,525 controls, identifying eight significant loci. The study found the genes associated with anorexia nervosa overlapped with other psychiatric disorders such as obsessive-compulsive disorder, depression, anxiety, and schizophrenia (Watson et al., 2019).

Psychologically, a low self-esteem, perfectionism and an anxious temperament seem to predispose individuals to an eating disorder. Impulsivity and emotional dysregulation are more common in bulimia nervosa. Life events, trauma and disrupted attachment in early childhood increase vulnerability to the development of an eating disorder (Brewerton, 2007). Being in an environment which encourages a certain physique, for instance working as a fashion model, professional ballet dancing, gymnastics or athletics can contribute to the development of an eating disorder.

An eating disorder often is a maladaptive coping attempt to regain control over unwanted and distressing emotions or life-situations. The individual feels that achieving in the eating disorder shows increased self-efficacy, self-control and “success”.

Epidemiology

Anorexia nervosa typically begins in mid adolescence and early adulthood. Lifetime prevalence in the general population is 1.9% for females (Wade et al., 2006) and around 0.1% in men (The National Eating Disorders Collaboration, 2010a). Bagaric et al. (2020) in their study found that the lifetime prevalence of bulimia nervosa was 1.21% (95% CI [0.87, 1.67]) for males and 2.59% (95% CI [2.07, 3.22]) for females. The lifetime prevalence for binge eating disorder was 0.74% (95% CI [0.49, 1.11]) and 1.85% (95% CI [1.42, 2.40]) for males and females, respectively, which is higher than reported in previous research.

Anorexia Nervosa (AN)

History

Reports of anorexia nervosa date back to the 1689 when it was described by Richard Morton, a British physician as a “nervous consumption”. It was first described as a diagnostic entity named *anorexia nervosa* in the 19th century by Sir William Gull, who was Queen Victoria’s personal physician. Awareness of the

condition was limited to the medical profession until Hilde Bruch, a German-American psychoanalyst, created wider awareness in her seminal work “The Golden Cage” in 1978 (Bruch, 2001). Since then, research into this disorder has advanced our knowledge of the genetic and biological processes and some of the psychological underpinnings.

Diagnostic Criteria

The diagnostic criteria for anorexia nervosa are a striving for weight loss that cannot be explained by another medical illness and an associated overvaluation of one’s weight and shape. There is an intense fear about weight gain and often behaviours are present which interfere with treatment attempts to increase food intake and hence, weight. This can take the form of using excessive amounts of laxatives, diet pills or diuretics as well as excessive exercise or self-induced vomiting. Individuals often do not acknowledge how unwell they are and experience their illness as ego-syntonic. It is often the relatives or partners of the individual who encourage engagement in treatment. The individual needs to have significant low weight which is defined as weight that is less than minimally normal, or for children and adolescents less than the minimally expected BMI percentile (Mild: BMI $>17 \text{ kg/m}^2$; Mod: BMI $> 16\text{-}16.99 \text{ kg/m}^2$; Severe: BMI $15\text{-}15.99 \text{ kg/m}^2$; Extreme: BMI $< 15 \text{ kg/m}^2$).

Two forms of anorexia nervosa have been established:

- *Restrictive subtype*: weight loss is accomplished mainly through fasting, dieting and/or excessive exercise. No binge eating or purging occurs.
- *Binge-eating/purging subtype*: recurrent episodes of binge-eating and purging occur.

Severe and enduring anorexia nervosa (SEAN) is a group of individuals with a prolonged course of anorexia and several treatment failures. They generally present with significant physical and psychiatric sequelae as well as compromised social, interpersonal and occupational functioning.

Bulimia Nervosa (BN)

History

In contrast to anorexia nervosa - which appears to have been noted throughout history - bulimia nervosa appears to be a more modern development. It was first described in 1979 by British psychiatrist, Gerald Russell, who published his seminal paper, “*bulimia nervosa: an ominous variant of anorexia nervosa*”, a case series of

30 patients who reported self-induced vomiting as an attempt to mitigate the effects of episodes of overeating. It was determined that these represented a syndrome that was distinct from anorexia nervosa but shared the same fear of weight gain.

Diagnostic Criteria

DSM-5-TR diagnostic criteria are recurrent episodes of binge eating with inappropriate compensatory behaviours in order to mitigate the caloric effect of binge eating (American Psychiatric Association, 2022). These behaviours can take the form of self-induced vomiting, laxative abuse, using diuretics or diet pills and engaging in prolonged fasting or excessive exercise. Binge eating episodes are discrete episodes of eating a much larger amount of food than what most individuals would eat in the same time (about 2-3x normal meal sizes). These episodes have to occur at least once per week.

Binge Eating Disorder (BED)

History

Binge eating disorder was first described by psychiatrist Albert Stunkard, who coined the term “Night Eating Syndrome” (Stunkard et al., 1955). He later specified that binge eating could occur without the nocturnal component (Stunkard, 1959). In DSM-IV, Binge eating disorder was listed under the category “Eating Disorders Not Otherwise Specified” (EDNOS), but now in DSM-5-TR it has its own place as an eating disorder diagnosis (American Psychiatric Association, 2022).

Diagnostic Criteria

Individuals with this disorder will eat a larger than normal amount of food with a sense of loss of control. They will eat much more rapid than normal and until feeling uncomfortably full, despite not always feeling hungry during those episodes. These episodes are followed by feelings of shame, disgust and embarrassment. Importantly, there are no compensatory behaviours following the binge eating episodes in the form of food restriction, excessive exercise, self-induced vomiting, taking of laxatives, diet pills or diuretics.

Other Specified Feeding or Eating Disorders (OSFED)

In this category, any Feeding and Eating disorders are grouped when they do not meet full criteria for any of the above disorders, but still cause significant psychological, social, occupational distress.

- Atypical anorexia nervosa - criteria for AN are met, except that despite significant weight loss, the individual's weight is within or above the normal range.
- Bulimia nervosa (of low frequency and/or limited duration) - inappropriate compensatory behaviours occur less than once per week.
- Binge eating disorder (of low frequency and/or limited duration) - binge eating occurs less than once per week.
- Purging disorder - recurrent purging behaviours without binge eating.

Assessment and Diagnosis

A full psychiatric assessment needs to be undertaken, as individuals with eating disorders experience high rates of other mental disorders, with reports of up to 97% having a comorbid condition. The most common of these are depression, anxiety disorders, followed by substance abuse and personality disorders (58%) (The National Eating Disorders Collaboration, 2010b; The National Eating Disorders Collaboration, 2012). Of note is that anxiety, depression, OCD and cognitive impairment can all represent the reversible effects of starvation. Additional to a full psychiatric assessment, it is important to ask questions specific to eating disorders:

- Food intake (*"what do you eat on a typical day?"*)
- Compensatory mechanisms: exercise, self-induced vomiting, laxatives, diet pills or diuretics.
- Body image (*"what do you see when you look into the mirror"* and *"does your weight influence how you see yourself?"*) This can reveal significant disturbances.
- It is advisable to ask about the highest and lowest weight during the course of the illness and prior to its onset. *"How do you feel about gaining weight?"* can reveal an intense fear of weight gain.
- Medical complications specific to eating disorders: amenorrhoea, reduced bone density, bradycardia, dental issues, constipation, lack of energy and fatigue, feeling cold, dizziness and fainting.

Physical Examination

Additional to a comprehensive physical examination:

- Measure weight and height to calculate BMI
- Blood pressure (postural), HR, temperature

- Bloods: FBC/ EUC/ LFT's/ Ca/ Mg/ PO4/ Glucose/ TFT's/ Amylase/ Lipase/ Serum Osmolality/ lipids
- ECG- bradycardia, ST segment elevation, T wave flattening, more serious: arrhythmias, U waves, QTc -prolongation
- Consider a Bone Mineral Density scan every 12 months (adolescents) to 2 years (adults)

Look out for:

- Hypotension, postural hypotension, bradycardia, cardiac arrhythmias
- Cachexia
- Conjunctival pallor, skin pallor
- Brittle nails and hair
- Dry flaky skin, Carotoderma
- Bruising
- Lanugo (new development of fine hair on face and body)
- Hypothermia
- Dehydration
- Delayed capillary refill, acrocyanosis (peripheral cyanosis)
- Peripheral oedema
- Parotid hypertrophy, dental decay and Russell's sign (calluses on the dorsum of the fingers due to purging)
- Stress fractures

Medical Complications

All eating disorders have a high prevalence of concomitant medical complications. This applies particularly to anorexia nervosa (see Table 21.1) due to the often severely underweight status of these individuals (Mehler and Brown, 2015). All body systems are adversely affected by malnutrition. Most medical complications are reversible with weight restoration, or cessation of binge/purge behaviours but some are not, for instance osteoporosis is unlikely to be reversible after puberty.

Table 21.1 Medical complications of anorexia nervosa.

Organ System	Pathology	Possible Outcome
Cardiovascular	Bradycardia, Hypotension	Syncope
	Cardiac arrhythmias Mitral valve prolapse Takotsubo cardiomyopathy	Sudden death Chest pain Heart failure
Endocrine	Low levels of reproductive hormones	Amenorrhoea Osteoporosis and stress fractures, arrested growth Hypoglycaemia Hypernatraemia Hypothyroidism
	Loss of hepatic glycogen Central diabetes insipidus Hypercortisolaemia Low T3 and T4 (TSH often normal)	
Renal	↓Na, ↓K, ↓Cl, ↓Mg, ↓Ca, ↓PO ₄ , Loss of HCL from purging	Cardiac arrhythmias Refeeding syndrome Metabolic hypokalaemic alkalosis
	Volume depletion (laxatives/ diuretics/ restriction)	Secondary hyperaldosteronism (Pseudo Barter syndrome)
Gastrointestinal	Gastroparesis Parotid hypertrophy	Constipation, atonic colon Can be painful when purging is ceased
	Refeeding pancreatitis Liver transaminitis Increased HDL Loss of adipose tissue between spine and aorta	Epigastric pain, nausea, vomiting Hepatic steatosis Hypercholesterolaemia Superior mesenteric artery syndrome– upper quadrant abdominal pain
Neurological	Loss of adipose tissue	Local compression neuropathies (peroneal nerve palsy), Difficulties with set shifting, and evidence of cognitive rigidity, impaired emotional regulation, motivation and memory
	Loss of cerebral grey matter and white matter, reduced connectivity between limbic system and frontal lobe	
	High levels of cortisol lack of glucose, fatty acids and thiamine	affecting memory and learning
Haematological	Pancytopenia due to fatty atrophy of bone marrow	anaemia, leukopenia and thrombocytopenia
Ophthalmologic	Orbital fat atrophy	Lagophthalmus

Anorexia Nervosa Medical Complications

Refeeding Syndrome

Refeeding syndrome is an extremely rare but serious and potentially fatal complication of refeeding. The cause is a rapid uptake of electrolytes from blood plasma into cells in response to the release of insulin to metabolise carbohydrates, causing a significant fluid shift. The consequence can be hypophosphataemia, hypomagnesaemia and hypokalaemia in the blood plasma, especially as stores of electrolytes tend to be low already. This can cause muscle weakness, cardiac arrhythmias, cardiac failure, seizures and confusion. Osmotic shifts of fluids into cells can cause hypertension and oedema. Peripheral oedema can occur, and concerningly so can pulmonary oedema. Most at risk are individuals who are severely underweight. A wide range of incidence has been cited (0-45%) due to a heterogeneity in population and definition of refeeding syndrome (Cioffi et al., 2021). Refeeding should be done with care and in consultation with physician colleagues if needed. Most difficulties occur in patients with conditions other than anorexia nervosa and when carbohydrate rich Total Parenteral Nutrition (TPN) was used (Madden et al., 2015).

Bulimia Nervosa Medical Complications

Treatment tends to be stepped and should ideally include a range of inpatient and outpatient options. Ideally there is a smooth transition between inpatient and outpatient settings, according to the patients need and level of recovery (Hay and Claudino, 2012). Relevant factors to consider regarding the level of care are medical (in)stability, level of cognition and insight, ability to cooperate with treatment and the individual's level of community supports. Table 21.2 displays a summary of medical complications of bulimia nervosa.

Table 21.2. Medical complications of bulimia nervosa.

Organ system	Pathology	Potential outcome
Metabolic	↓K, ↓Cl from self-induced vomiting	Metabolic alkalosis Cardiac arrhythmias, sudden death
	Dehydration	Volume depletion, Pseudo-Barter syndrome
	Loss of bicarbonates due to laxatives	Metabolic acidosis
Dental	Tooth enamel erosion	Teeth sensitivity, increased tooth decay
Gastrointestinal	Self-induced vomiting	Gastroesophageal reflux, Barrett's oesophagus Russel's sign (callus on the dorsal aspect of the hand)
	Mallory Weiss tear Laxative use	Haematemesis Chronic constipation, atonic colon
Ophthalmologic	Increased intraocular pressure due to self-induced vomiting	Subconjunctival haemorrhage

Engagement

The first step in treatment is always engagement of the individual and their family, which can be difficult due to the ego-syntonic nature of the disorder – this means that, particularly for anorexia nervosa, the illness is perceived as congruent with the individuals' values and needs, and hence any change in the status quo is resisted. Ideally, self-determination and self-management are encouraged with the aim of conducting treatment in the least restrictive context. Psychoeducation and discussion about the medical implications of the disorder is an important part of this process.

Medical stabilisation is the next step to achieve a reversal of physical and cognitive effects of starvation. This means refeeding and cessation of compensatory behaviours. Inpatient treatment is preferred if the patient is medically unstable and/or BMI <14, an inpatient medical treatment is indicated.

Indicators of medical instability (in adults):

- HR < 40bpm or tachycardia on standing (>20bpm increase)
- blood pressure < 90/60 mmHg or with a postural drop of >20mmHg
- temperature <35.5°C.
- Admission is also indicated if there has been rapid weight loss, several days of no oral intake at all and uncontrolled purging, exercise or suicidality.

Involuntary treatment is a last resort, but when done sensitively does not have to be the end of a therapeutic relationship.

Outpatient treatment is an option if the patient is medically stable and should ideally consist of a multidisciplinary team with involvement of the patient's GP, a psychiatrist, dietician and psychologist (if possible, all should have clinical experience with eating disorders). Day programs, if available can be a step-down from the inpatient setting.

Refeeding:

- Options include oral intake of meals, use of nutrition supplements (Ensure, Fortisip etc.) and nasogastric feeding
- Establishment of regular and appropriate meals and snacks sufficient to achieve nutritional rehabilitation is the first step. Involvement of a dietitian is desirable.
- Some components of refeeding will be considered non-negotiable (e.g. steady weight gain, being weighed, consuming a specified amount of nutrition and calories, not engaging in compensatory behaviours). Excessive negotiation about these basic components is best avoided.
- A combination of nasogastric feeding overnight and oral intake of food during the day is an option.
- Determining the adequate energy requirement for refeeding can be difficult due to metabolic changes occurring during refeeding, diversion of food (e.g. into pockets and serviettes) and secret compensatory behaviours (purging, exercise etc.). Recommendations regarding actual kcal/kg/day can vary and is individual. It is not advisable to discuss calories with the patient.
- The patient and family should be provided with a meal plan in order to optimise consistency in approach and minimise debates about alterations to food portion sizes or food types.

- Establishing increased social eating with family members and friends will help to normalise eating.
- Minimise eating disordered behaviours at mealtimes (e.g. no playing with food, not taking very small bites or cutting food into tiny pieces, sitting at the table for a specified time)
- Post meal support can help with anxiety and agitation and can involve socialising, relaxation or distraction.
- In order to prevent purging behaviours, access to the bathroom might be restricted for one hour after meals.

Psychological therapies with evidence for eating disorders:

- Maudsley family-based treatment (for children and adolescents) is a family-based therapy with three definitive phases: Phase 1 consists of weight restoration with parents taking control of all meals. Phase 2 works on returning control overeating to the adolescent and the goal of phase 3 is establishing a healthy adolescent identity.
- Cognitive Behaviour Therapy for Eating Disorders (CBT-Enhanced) – challenges eating disorder related behaviours and patterns of thinking (Fairburn et al., 2015)
- The Maudsley model of anorexia nervosa treatment for adults (MANTRA) – a combination of motivational interviewing, cognitive remediation and flexible involvement of carers.
- Focused psychodynamic therapy for anorexia nervosa (Zipfel et al., 2014)
- Specialist supportive clinical management (SSCM) with a primary focus on resumption of normal eating and restoration of weight, but also addresses life issues impacting on the eating disorder.

Pharmacotherapy in eating disorders:

- Evidence for medication treatment in eating disorders is weak to moderate
- There is support for the use of high dose fluoxetine in bulimia nervosa and binge eating disorder (Hay, 2012)
- Lisdexamphetamine also has shown efficacy in binge eating disorder (Fornaro et al., 2016)
- Low dose antipsychotic medication may be useful in anorexia nervosa especially in the context of high anxiety and obsessive eating disorder related ruminations (Hay and Claudino, 2012).

Prognosis

In anorexia nervosa, there is an almost 18-fold increase in mortality including a high suicide rate. Among the surviving patients, less than one-half recovered on average, whereas one-third improved, and 20% remained chronically ill. The normalization of the core symptoms, involving weight, menstruation, and eating behaviours, was slightly better when each symptom was analysed in isolation. The presence of other psychiatric disorders at follow-up was very common. Longer duration of follow-up and, less strongly, younger age at onset of illness were associated with better outcome. There was no convincing evidence that the outcome of anorexia nervosa has improved in the second half of the last century (Hans-Christoph, 2002).

Most people with bulimia nervosa, binge eating disorder or other specified eating and feeding disorders (OSFED) have a relatively good prognosis with 50% or more free of symptoms after five years of treatment (Steinhausen and Weber, 2009).

Avoidant/Restrictive Food Intake Disorder (ARFID)

This disorder has some similar criteria to anorexia nervosa, however, here the predominant feature is a concern about the sensory qualities about food (texture, smell, colour, taste or temperature of food) or worry about a potential aversive consequence of eating certain foods (choking, vomiting, nausea, gastrointestinal discomfort, etc). This leads to a failure to meet adequate nutritional and/or energy needs and causes significant weight loss. However, in contrast to anorexia nervosa, there is no concern about weight or body shape. Some individuals become dependent on nutritional supplements or even nasogastric feeding. ARFID is more common in children than any other age group. Anxiety disorders, autism spectrum disorder, obsessive-compulsive disorder and attention-deficit/hyperactivity disorder may increase the risk for developing ARFID.

Pica

Pica is the persistent eating of non-nutritive and non-food substances (for instance soil) which is inappropriate for the individual's developmental level and is not part of a socially and cultural normative practice. Pica often occurs in the context of another mental disorder like autism spectrum disorder or intellectual developmental disorder, but can also occur in medical conditions, including pregnancy. Onset is most often in childhood, but can occur throughout the ages. Neglect and lack of supervision can increase the risk for this condition.

Rumination Disorder

Repeated regurgitation of food after eating, which then may be re-chewed and re-swallowed or spat out. This occurs in the absence of a medical disorder affecting the gastrointestinal system and the behaviour occurs without any apparent disgust, involuntary retching or voluntary purging behaviours. It cannot occur together with a diagnosis of another feeding and eating disorder. Rumination disorder is more common in individuals with intellectual disability and can occur at any age. Predisposing factors can be lack of stimulation, neglect, stressful life events and difficulties in the parent-child relationship.

Further Reading

- Mehler, P. S., Anderson, K., Bauschka, M., Cost, J., & Farooq, A. (2023). Emergency room presentations of people with anorexia nervosa. *Journal of Eating Disorders*, 11(1), 16.
- Rowe, E. (2017). Early detection of eating disorders in general practice. *Australian Family Physician*, 46(11), 833-838.

Chapter 22: Personality Disorders

Janine Stevenson

Specialty of Psychiatry, The University of Sydney, Australia.

and

Lisa Lampe

School of Medicine and Public Health, The University of Newcastle, Australia.

Introduction

Patients with personality disorders present for treatment in all medical settings, and every medical practitioner will encounter such patients frequently. People with personality disorders have problems with interpersonal relationships that can affect their interactions with health professionals. Having a comorbid personality disorder is a strong predictor of poor treatment outcome, a cause of premature mortality and a great cost to society.

What is a Personality Disorder?

Personality disorders are generally described as a pervasive and consistent pattern of maladaptive traits, cognition, inner experience and behaviours evident by adolescence or early adult life, leading to substantial personal distress or social dysfunction or both, and disruption to others. The individual thinks about himself/herself, others and their relationships, and acts in interpersonal situations in ways that cause distress and impairment.

To make a diagnosis of personality disorder the maladaptive patterns must be life-long or of many years duration and must be consistent across time and situations. The maladaptive thinking or behaviour must not be limited to times where the individual is unwell with some other illness, such as depression, or experiencing severe stress. We all can display difficult personality traits when

stressed. It only becomes a disorder if it is persistent and affects the individual in a negative way or affects others who encounter the individual.

Diagnostic Criteria for Personality Disorder

An enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture. This pattern is manifested in two (or more) of the following areas:

1. Cognition (i.e., ways of perceiving and interpreting self, other people, and events).
2. Affectivity (i.e., the range, intensity, lability, and appropriateness of emotional response)
3. Interpersonal functioning.
4. Impulse control.

B. The enduring pattern is inflexible and pervasive across a broad range of personal and social situations.

C. The enduring pattern leads to clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The pattern is stable and of long duration, and its onset can be traced back at least to adolescence or early adulthood.

Classification

There is disagreement among experts regarding whether personality disorders are best classified categorically or dimensionally. In categorical systems of classification (such as in DSM-5), people are described as having clusters of traits that form a recognizable pattern, for example, a dependent personality, a schizoid personality or a borderline personality. When the person's symptoms meet criterion level on a minimum number of criteria they are diagnosed as having that personality disorder. However, there is considerable overlap between these categories, so that severe personality disorder seems to be manifest as multiple pathologies. An alternative way of classifying personality disorders is a dimensional system utilising trait-based personality theory. The five-factor model (FFM) is arguably the best-supported dimensional system and classifies people on five traits each on a continuum: extraversion, openness to experience, conscientiousness, agreeableness, and neuroticism, each person being somewhere on the continuum for each of these traits.

The DSM-5 categorical classification identifies general features of a personality disorder (see box above) and then distinguishes 10 categories of

personality disorder divided into 3 clusters: Cluster A (paranoid, schizoid and schizotypal), Cluster B (antisocial, borderline, histrionic and narcissistic), and Cluster C (avoidant, dependent, and obsessive-compulsive). The evidence informing the categories and clusters is limited, and decisions are based more on consensus and opinion. The available evidence supports some aspects of these clusters but fails to support the validity of other aspects. DSM-5 also includes an ‘alternative model for personality disorders’ which sees personality disorders in terms of impairment in personality functioning – deficiencies in self-functioning (self-awareness and self-directedness); or interpersonal functioning (empathy, intimacy, and mutual understanding), or both; essential elements in the capacity of an individual to form good relationships. This model acknowledges the severity of the disorder (the best predictor of outcome) and the form or style of personality pathology, which are represented as traits that lie on dimensions from normality to frank disorder.

The ICD-11 classification abolishes all type-specific categories of personality disorder, identifying simply the presence or absence of personality disorder itself. Different degrees of severity are defined (this can fluctuate over time), and this can be qualified by a description of domain traits (negative affective features, dissocial features, disinhibition, anankastic features, and detachment). Four dimensions have been suggested in ICD-11 to cover the range of personality disorder: emotional dysregulation (vs stability), extraversion (vs introversion), antagonism (vs compliance), and constraint (vs impulsivity).

Epidemiology

Prevalence and Demographics

Community-based surveys in western countries report a prevalence rate of individuals with personality disorders of 12.16% (95% CI 8.01 – 17.02%) (Volkert et al., 2018). A quarter of patients in primary care have been reported to have personality disorder, 50% of psychiatric outpatients, and an even greater proportion of psychiatric inpatients (Stevenson et al., 2011b). There are some gender and cultural differences. Antisocial personality disorder (ASPD) is more prevalent in males while borderline personality disorder is more prevalent in females. There are higher rates in urban areas for all personality disorders than rural areas and two-thirds of prisoners have a personality disorder (predominantly antisocial and borderline). Narcissistic personality disorder is reportedly more prevalent in Western than Eastern countries.

Age at Onset

The manifestations of a personality disorder are usually evident by adolescence or early adulthood, and generally become manifest under stress, depending on current supports and pressures. Complexity and severity usually diminish in older adults, who display less neuroticism (negative affectivity) generally. Although personality disorder is considered to be a lifelong diagnosis, the severity and form of the disorder typically fluctuate over time, depending on many factors. Recognition of the fluctuating nature of the disorder should help destigmatisation of the diagnosis.

Implications of Personality Disorder/ Burden of Disease

Personality disorder is associated with both higher morbidity and mortality. Life expectancy is shorter by 13.3 years for women and 15.5 years for men (suicide and homicide, but also increased cardiovascular and respiratory diseases, poor relationships with health professionals, smoking, alcohol and drug abuse) (Chan et al., 2023).

The presence of a personality disorder is a risk factor for developing another psychiatric disorder such as major depression, anxiety and substance use disorders, and typically has a negative effect on treatment outcome. The presence of more than one personality disorder confers more burden. Some personality disorders show a genetic relationship to other psychiatric illnesses, for example, increased rates of schizophrenia have been reported in the first-degree relatives of persons with schizotypal and paranoid personality disorders (Kendler et al., 1993). Comorbid anxiety disorders are common in Cluster C personality disorders, and comorbid depression in avoidant personality disorder.

The overriding issues for people with a personality disorder are relationship problems. The person with the personality disorder is usually unable to recognise a problem in themselves and so projects the blame onto others, leading to problems with intimacy, difficulties relating to professionals, inability to function well at work and social isolation. Repeated self-harm is frequent in borderline personality disorder, and impulsive risk-taking in borderline and antisocial personality disorders, contributing to the burden of disease. This propensity for risk taking and substance use increases the likelihood that people with these personality disorders will come in contact with health professionals.

Be aware that personality disorder cannot be diagnosed in the absence of distress or significant impairment. For example, a person with a schizoid personality might be quite comfortable working as a computer programmer at home alone but

could become acutely distressed at having to work in an office among other people, in which case they would be described as having a personality disorder.

Coping Strategies

An Australian study of 240 psychiatric inpatients aged from 18-100 demonstrated that patients with personality disorder used more dysfunctional coping strategies (denial, drug and alcohol abuse, projection, avoidance etc) than patients without personality disorder. The latter used more active and effective coping strategies (facing the problem to do something about it) and seeking support from others (Stevenson et al., 2012).

Risk/Vulnerability Factors (Aetiology)

Biological

There is a large genetic component predisposing a person to develop a personality disorder (probably up to about 50%; (Kendler et al., 2008; Reichborn-Kjennerud, 2010; Torgersen, 2009) but environment, particularly early attachment relationships, plays an important but variable role, depending if it is nurturing and supportive or disruptive and traumatic.

There have been some neurobiological theories postulated. Individual differences in the regulation and organization of cognitive processes, affective reactivity, impulse / action patterns, and anxiety may, in the extreme, provide susceptibility to personality disorders such as borderline and schizotypal personality disorder. A low threshold for impulsive aggression, as observed in borderline and antisocial personality disorders, may be related to excessive amygdala reactivity, reduced prefrontal inhibition, and diminished serotonergic facilitation of prefrontal controls. Affective instability may be mediated by excessive limbic reactivity in GABAergic/glutamatergic/cholinergic circuits, resulting in an increased sensitivity or reactivity to environmental emotional stimuli as in borderline personality disorder and other Cluster B personality disorders.

Disturbances in cognitive organization and information processing may contribute to the detachment, desynchrony with the environment, and cognitive/perceptual distortions of Cluster A or schizophrenia spectrum personality disorders. Epigenetic remodelling of glucocorticoid receptor genes in response to stress has also been proposed as a mechanism for how the environment can interact with the genome contributing to long term changes in behaviour (Bagot and Meaney, 2010). Brain trauma, pre or postnatally can result in personality dysfunction.

Culture/Social Processes

Personality can also be shaped by culture, such that someone who fits well in their own culture might be considered aberrant in another (e.g. Zulu versus Amish societies; the withdrawn and restrained Asian versus the outgoing, exuberant Australian). Social processes, our cognitive/perceptual organisation, and heritable temperamental traits such as affective instability and proneness to impulsivity/aggression all influence personality.

Psychological

Childhood abuse or neglect increased the risk of developing a personality disorder by four times in one study (Johnson et al, 1999). People with borderline personality disorder frequently have a history of emotional, physical or sexual abuse or neglect. A major life crisis, such as suddenly becoming disabled can also result in personality change.

Attachment

Attachment represents another intensive area of study in personality disorder and forms the basis of some treatment approaches. A disorder of attachment to the carer is common. The infant/child looks to the carer for caring and protection, but a carer who is unreliable, erratic, devaluing or frankly abusive can disrupt attachment (see Chapter 13). Attachment style forms an important basis for future relationships. Children require security in their early caregiver relationships, and sufficient emotional attunement and nurturing to develop their capacity to self-soothe when distressed. Without security, consistency, validation and affection it is likely that individuals will struggle to develop a positive sense of self and adaptive strategies for coping with loss and distress (see Chapter 13).

Presentation

Individuals with personality disorders may present due to the distress experienced directly from their maladaptive thoughts or emotional disturbance (e.g., depressed mood or anxiety, suicidal ideation), because of associated behaviours (e.g. self-harming) or the responses of others (criticism or rejection), or because of complications arising from their attempts to manage their problems (e.g. substance misuse). They often present with mental disorders, especially with depressive or anxiety disorders, eating disorders, but they can also present with relationship issues, marital problems, difficulties at work or attempted suicide or deliberate self-harm.

Assessment

The assessment of a patient's personality functioning requires a detailed personal and developmental history. It is critical in establishing that the symptoms with which the person presents, although possibly more severe and acute than usual, are typical of their long-term functioning. But note that some personality disorders can manifest late if they have previously been compensated; for example, a dependent woman who copes until her husband dies and then her neediness manifests itself. Also be wary of diagnosing a personality disorder in the presence of another psychiatric disorder; for example, if the woman with dependent traits was experiencing a major depressive illness, we would need consider whether some of her apparent dependent behaviours are not in fact due to the loss of confidence, concentration and clarity of thinking associated with her depression. More than one assessment may be needed, as well as collaborative history from a relative or friend.

Semi-structured interviews typically take 1h-2h to complete and even self-rating instruments take a long time. Semi-structured instruments administered by a clinician are the most reliable, but all such instruments tend to over diagnose, and clinical assessment tends to under diagnose.

Obtaining a collateral history is important. Comorbidities must be explored as well as the burden of symptoms, for example, has there been a likely adverse impact on social and occupational functioning? There are no essential medical investigations for personality disorder but there may be for any comorbidity such as an eating disorder, or for any impacts of self-harm or risk-taking behaviour (e.g., drug use, unprotected sex).

Factors to elicit from history

- History of abuse (physical, sexual, psychological) or neglect
- Difficulties with interpersonal relationships
- Problems maintaining employment
- Dysfunctional coping strategies
- History of self-harm/ suicidality
- Lack of a sense of identity
- Emotional dysregulation
- Impulsivity
- Lack of empathy

Diagnosis

People with personality disorders do not usually present for treatment of their personality disorder but with symptoms such as depression, anxiety, or in the case of borderline personality disorder, often with recurrent self-harm. Therefore, it is important to have your mind open to the possibility of an underlying personality disorder. Ask yourself the questions: does this person have a treatment-resistant disorder? Consider especially the person's long-term functioning. Does this person have relationship problems? Difficulty working? Is this simply a cultural difference? Is this person displaying personality traits but no impairment in functioning? A narrative approach to history taking (get the person to tell their life story) is often helpful.

Threshold for diagnosis varies with the particular personality disorder, but it is the extent of interference with intrapersonal and interpersonal function that determines whether the patterns of thinking and behaviour represent traits or disorder. Keep in mind the difficulty of diagnosing personality disorder in the context of acute psychiatric illness, for example psychosis, major depression, substance dependence/intoxication/withdrawal. Be aware that behaviours could also be due to some other cause, for example cognitive impairment, intellectual disability, stage of development, cultural difference, or acute stress.

Clues to a diagnosis of personality disorder

- Multiple emergency presentations
- Conflicted opinions about the patient (splitting)
- Chaos in the patient's life
- Negative counter-transference reactions
- Negative self-image
- Inappropriate anger
- History of dysfunction in occupational settings
- History of unhappy or failed relationships

As stated above, DSM-5 describes 10 subtypes (see Table 22.1): antisocial, avoidant, borderline, dependent, histrionic, narcissistic, obsessive-compulsive, paranoid, schizoid, schizotypal; and a group of 'other personality disorders' which includes personality change due to a general medical condition, and 'other specified personality disorder' and 'unspecified personality disorder' (where symptoms characteristic of one or more personality disorders are present, together with

distress and impairment, but criteria are not met for any specific personality disorder).

Significant problems with the categorical approach to personality disorder diagnosis, including arbitrary diagnostic thresholds, extensive overlap among categories, lack of evidence for 10 distinct categories, and insufficient clinical utility have led the 11th edition of the International Classification of Diseases (ICD-11) to adopt a dimensional approach to the classification of personality disorders.

Table 22.1 Personality disorder subtypes in DSM-5

Personality Type	Description
Cluster A	
Paranoid personality	Distrusting, suspicious, malevolent perception of others
Schizoid personality	Social detachment, restricted emotional range
Schizotypal personality	Eccentric, odd beliefs, discomfort with intimacy
Cluster B	
Narcissistic personality	Grandiosity, exhibitionism, lacking empathy
Antisocial personality	Disregard for and violation of others' rights, lack of guilt, narcissism
Borderline personality	Unstable interpersonal relationships, self-image and affect, emotional dysregulation, impulsivity
Histrionic personality	Attention-seeking, excessive emotionality
Cluster C	
Avoidant personality	Excessive sensitivity to negative evaluation, feelings of inadequacy, social inhibition and avoidance
Dependent personality	Submissive, clingy, needy, avoids decisions
Obsessional personality	Preoccupied with rules, likes to be in control, perfectionism

The lack of clear boundaries between types of personality disorder, state vs trait factors (temporary versus lifelong) and poor agreement among clinicians is leading towards a general diagnosis of “personality disorder” based on the problems and disabilities characteristic of personality disorders, with some description of traits. They refer to a diathesis or vulnerability model (rather than a disorder model) conferring a tendency to develop other psychiatric disorders or abnormal behaviours. Exacerbation occurring in response to a stressful event is consistent with

the stress diathesis model. Social dysfunction persists and persistent maladaptation is seen as the core of the condition.

The ICD-11 classification of personality disorders focuses on core personality dysfunction, while allowing the practitioner to classify three levels of severity (Mild Personality Disorder, Moderate Personality Disorder, and Severe Personality Disorder) and the option of specifying one or more prominent trait domain qualifiers (Negative Affectivity, Detachment, Dissociality, Disinhibition, and Anankastia). Additionally, the practitioner is also allowed to specify a Borderline Pattern qualifier.

Differential Diagnosis

At times, a person with a psychiatric disorder can behave in a manner which may suggest personality disorder because of dysfunctional interpersonal interactions, but which fully resolves when the acute disorder is treated. This is referred to as “state dependent personality dysfunction”. It does not mean the person has an enduring disturbance of personality, but that they have decompensated under stress or the effects of the primary mental disorder. There may be diagnostic uncertainty around schizotypal personality and schizophrenia. The presence of delusions rather than overvalued ideas, and hallucinations should point to a diagnosis of schizophrenia, and longitudinally there would usually be evidence of functional decline, and deficit symptoms such as apathy and amotivation.

Social phobia and avoidant personality look similar and may in fact represent variations of the same spectrum. Anxiety symptoms are generally more prominent in social phobia, and the sense of inferiority more pervasive in avoidant personality disorder. DSM-5 allows both diagnoses to be given. A mood disorder with low mood, self-harm, and social withdrawal can appear similar to borderline personality disorder. Mood symptoms are common in a borderline personality disorder, but the depressive states tend to be more transient, reactive and angry.

PTSD with depressed mood, anger, relationship problems, substance abuse and inability to work can look like borderline personality disorder. The history should usually identify a traumatic stress. Diagnostic difficulties can arise with ‘complex PTSD’, where an individual experienced sustained abuse over a period of time. Some patients will have symptoms of both disorders.

Personality change can be linked to brain pathology caused by medical conditions such as systemic lupus erythematosus (SLE), encephalitis, and acquired immunodeficiency syndrome (AIDS). Substance abuse can affect personality, both

acutely and with chronic use. Substance abuse is also common in personality disorders characterised by impulsivity.

State Dependent Personality Dysfunction

People who show evidence of personality pathology only when suffering a symptom disorder, which disappears when the symptom disorder is treated are considered to have a state dependent personality disorder only, for example someone who displays self-harming behaviour, impulsive acts, angry mood and relationship problems while suffering from major depression and not when well. They could also be considered to lie along the spectrum from “normal” to “severe personality disorder” or be said to have a personality diathesis (Tyrer and Tyrer, 2015).

Borderline Personality Disorder

This personality disorder is dealt with in more detail as it is likely to be the most commonly encountered and most problematic personality problem in medical practice.

Symptoms include:

- **impulsivity** (self-harming, drug abuse, reckless driving, eating disorders).
- **mood instability (emotional dysregulation)** with frequent fluctuations from depression, anger, anxiety, and temporary euphoria. This can at times look like bipolar disorder and is often misdiagnosed as such (though both can occur together).
- **black and white** thinking (someone is all good (idealized) or all bad (devalued) which can lead to
- **splitting**, especially in the inpatient situation where staff develop very different opinions and behaviour towards a patient which can cause clashes between staff if not recognized.
- frequent changes regarding **relationships** with one person being a friend this week and rejected the next because of some perceived injustice.
- **identity** issues are common, for example feeling empty, unsure of sexual orientation, future plans, often a noxious sense of self,
- **intolerance of being alone/ severe separation anxiety**; frequent **altercations** –arguments, fights, recurrent accidents.
- chronic **anger** and angry outbursts out of proportion to the trigger.

Patients with borderline personality disorder have frequent presentations to emergency departments with self-harm or serious suicide attempts. They present for surgery after ingesting sharp objects; they present to their general practitioner with multiple somatic complaints. Because of difficulty with trust, they can anticipate rejection and easily interpret responses as critical or rejecting which can lead them to be aggressive and abusive to medical staff, often complaining and uncooperative with care. Counter-transference reactions (emotional reactions to the patient and/or their behaviour) of staff are often negative and can be neglectful. It is important to react with empathy, realising that these patients are not deliberately difficult, as their traumatic childhood experiences, lacking nurturing and support, have communicated a hostile world where there is no-one to trust. Listening to their concerns, understanding their pain is the better approach. There is also the danger of a positive countertransference to an idealizing (or “seductive”) patient who then becomes “special”. Appropriate, consistent boundaries should be set, never wavering from the patient/doctor relationship. Apparent kindness can be misinterpreted as a special friendship, and there are cases where patients have intruded into health care workers’ lives, a situation, which ends badly for both.

The intrinsic symptoms of borderline personality disorder – chronic angry dysphoria, self-harm, relationship difficulties – often prompt help-seeking. Whatever area of medicine you enter you will encounter these patients. Failure to recognize the problem can result in poor rapport, non-compliance, either idealization or vexatious complaints and a difficult relationship. As a result, the individual will receive less than optimal medical care and be at risk of reduced quality of life and life expectancy.

Evidence Based Treatments

Outcome Studies

Psychotherapy has a long tradition dating from Freud and Jung. The publication of DSM-III in 1980 provided a stimulus for the development of psychotherapies to treat personality disorders, particularly borderline personality disorder, given its prominence in acute psychiatric presentations. Subsequently, most studies examining the effectiveness of psychotherapy in personality disorder have focused on the treatment of borderline personality disorder

Marsha Linehan in the USA introduced dialectical behaviour therapy (DBT). Bateman and Fonagy in the UK introduced mentalization-based therapy. Interpersonal psychotherapy was developed by Klerman and Weissman, and in Australia the Conversational model of psychodynamic psychotherapy was developed by Russell Meares (Meares, 2004a; Stevenson and Meares, 1992). Schema-focussed

therapy was developed by Jeffery Young. Acceptance and Commitment Therapy by Hayes, Strosahl and Wilson. All these forms of therapy have been shown to have efficacy in treating borderline personality disorder in clinical trials.

Dialectical Behaviour Therapy

Marsha Linehan developed a treatment program for young, suicidal, self-harming women (Linehan et al., 1991). An offshoot of cognitive behaviour therapy, the approach is a highly structured, directive, individual and group therapy format that seeks to teach patients general problem-solving skills, emotional regulation strategies, interpersonal skills, and distress tolerance. The strategies and techniques are outlined in manual form.

The group treatment is organised didactically, divided into 16 weekly modules with an emphasis on experiential learning through formal instruction, modelling, behavioural and cognitive rehearsal, cognitive restructuring, interpersonal feedback, reinforcement, and homework assignments. The therapy is aimed at teaching the individual new coping and problem-solving skills and helping them tolerate and find meaning as it currently exists in their life. It has the best evidence base for its effectiveness (Stoffers-Winterling et al., 2022).

Schema-Focused Therapy

Schema-focused therapy can be done individually or in a group (Clarkin et al., 2006). It can help to identify unmet needs that have led to negative life patterns, which at one time may have been helpful for survival, but as an adult are hurtful in many areas of their life. Therapy focuses on helping the person get their needs met in a healthy manner to promote positive life patterns. It integrates elements of cognitive therapy, behaviour therapy, object relations, and gestalt therapy into one unified systematic approach that focuses on maladaptive schemas. Schema therapy is easily applicable to Cluster C personality disorders as well as borderline personality disorder and is noted to be effective across a number of personality disorders (Zhang et al., 2023).

Mentalization-Based Psychotherapy

Mentalization-Based Psychotherapy (MBT) aims to increase mentalization capacity, leading to better affect regulation and interpersonal relationships (Bateman and Fonagy, 1999). Mentalization refers to the capacity to represent feelings in the mind, and to connect behaviours, events, feelings and thoughts. Often patients with personality damage lack the capacity to imagine what is in the mind of another person, what they might be feeling. Mentalization is a process by which patients

learn to think about others and themselves before acting. The aims of MBT are to improve behavioural control and affect regulation as well as to foster better relationships. It serves to stabilize the patient's sense of self and to enhance stability in emotions and relationships.

Transference-Focused Psychotherapy

There are many transference-focused psychotherapies that are based upon a psychodynamic approach to treatment. It is presumed that there has been an arrest in development at an early stage of life due to emotionally traumatic events during childhood (Clarkin et al., 2006). The approach is maturational, resulting in the development of a more mature, realistic, and flexible sense of self and others, and more coherent sense of self-identity. The therapist helps the patient build a positive sense of self, and well-being involving validation of their experience. This is then followed by dealing with the trauma and integrating it into the person's narrative. Although these treatments are known by a confusing range of terms and acronyms and have seldom been subjected to a randomised controlled trial there is an evidence base that supports their use as another possible treatment approach to borderline personality disorder (Setkowski et al., 2023).

General Psychiatric Management

Developed by Gunderson who views personality disorders in terms of disturbed attachment, symptoms are taken to indicate maladaptive ways of coping with emotional distress. Gunderson developed General Psychiatric Management after noting that less intensive and easier-to-learn therapies could be nearly as effective as more intensive psychotherapies and more accessible to more patients. General Psychiatric Management includes case management, encouragement of emotional processing, and relational management (Smith et al., 2001). Gunderson describes this therapy as a once-weekly, generalist model that medicalizes borderline personality disorder, emphasizes psychoeducation, and focuses on social adaptation (Gunderson et al., 2018).

Systems Training for Emotional Predictability and Problem-Solving (STEPPS)

STEPPS was developed with an assumption that personality disorder reflects dysfunctional ways of dealing with emotions. It is a group intervention that helps patients develop better ways of dealing with emotions (Black et al., 2013; Stoffers-Winterling et al., 2022).

Common Features

All these treatments include a clear, detailed and mutually agreed upon treatment framework in terms of appointment times, fees, cancellation policy, and termination policy. Secondly, all treatments pay close attention to the emotional experiences of patients. All therapies focus on the quality of the treatment relationship and assign an active role to the therapist who is encouraged to be mentally and emotionally engaged with the patient. They encourage exploratory interventions and change-oriented interventions. Use of interpretations in a strict psychoanalytic sense is not encouraged, nor is advice giving. All encourage support for the therapist (e.g., supervision with an experienced peer).

Partial Hospitalisation and Step-Down Treatments

This allows more containment during crises without the patient developing emotional dependence on the hospital environment. The patient attends during the day and goes home at night. The program involves psychoeducation, learning and practising better coping strategies, individual therapy and support. The next step down is to less frequent outpatient care, group programs and social support, followed by weekly individual therapy, which may last one year or more.

Medication

Medication is not indicated as a first line treatment for personality disorder, but may be needed for comorbid symptom disorders (Bohus et al., 2021). Antidepressants may be useful for comorbid major depression and sometimes for the dysthymia commonly comorbid with personality disorders, perhaps especially borderline personality disorder and avoidant personality disorder.

Medications are also commonly used to alleviate specific symptoms experienced in personality disorders, although there is no evidence that they improve overall outcome. SSRIs may be useful to treat anxiety. Anticonvulsants, such as sodium valproate and carbamazepine may help control the anger felt by people with borderline and antisocial personality disorder. Antipsychotics help manage aggression and psychotic symptoms.

Benzodiazepines are not recommended because of the risk of disinhibition (especially in antisocial personality disorder), misuse and dependence. In practice it is common to see patients with personality disorder who are already taking an antidepressant, an antipsychotic, a mood stabilizer, and one or more benzodiazepines – the polypharmacy attesting to the fact that medications have not helped, yet trials of medication are repeated relentlessly.

Crisis Management

Patients present in crisis when their usual defences fail, resulting in suicide attempts, self-harm, failure to cope. Deliberate self-harming (cutting, burning, overdosing) may occur without suicidal intent, driven by painful emotions and memories. Physical pain may relieve emotional pain albeit temporarily. Individuals may also attempt (and often succeed) suicide when feeling hopeless and overwhelmed by emotional pain, failed relationships and other social stressors. Patients with borderline personality disorder are the most likely to present to an emergency department after an overdose, cutting, burning or mutilating themselves in some other way. They act impulsively and are often also involved in car accidents or other reckless behaviour. Drug abuse and misuse and dependence is also common.

It is vitally important to be empathic, not condemning or critical of their behaviour, for that compounds their already high levels of distress and intensely negative self-concept. Unfortunately, critical and rejecting responses are common from emergency staff, who may see the same person come in again and again with self-harming. It is likely that staff feel frustrated at the apparent inability of the person to learn better coping strategies, and perhaps at their own seeming impotence to make a difference.

Be mindful that this is most likely a person who experienced significant adversity in childhood and on into adult life. This behaviour may be the only strategy they know to manage their symptoms, the physical pain masking the emotional pain for a short time, sometimes to prove to themselves that they are alive, that they actually exist (feelings of emptiness are common in borderline personality disorder), and sometimes to punish themselves, consistent with a view of worthlessness or feelings of guilt. It is important to listen, and hear their story so that they feel heard, and to validate their feelings. Treat them with respect, give them time, help them deescalate, treat their physical needs gently and carefully and seek psychiatric review. The person should also receive a follow-up appointment with a member of the mental health team within a couple of days. This is ideally followed by an arrangement for more long-term therapy to treat the underlying personality disorder. Treatment of comorbidities such as substance abuse and dependence, anorexia, mood disorders, and anxiety must also be considered.

Outcome/Prognosis

Stability of Diagnosis and Impairment

Longitudinal work suggests that people diagnosed with antisocial, avoidant, borderline, histrionic, and schizotypal personality disorders are likely to continue to meet these diagnoses for many years. However, research has also underscored the

potential for ongoing personality change and maturation (Nestadt et al., 2010). Maturity and life experience, good relationships, and therapy enables some people to develop more mature coping strategies and discard the maladaptive ones. Arguably this may be most prominent for borderline personality disorder and antisocial personality disorder where survivors may show significantly better function over time. However, symptoms can recur in old age when supports are lost and coping strategies are fewer.

Comorbidity

Any comorbid symptom disorder such as schizophrenia, bipolar disorder, anxiety disorder worsens prognosis, and vice versa. Comorbid drug abuse also hampers progress in therapy, because either they are impaired during therapy sessions, because sessions are not remembered or because the individual continues to avoid directly dealing with problems or learning to tolerate emotional distress, instead escaping into intoxication. Persons who abuse drugs also have an increased mortality.

Age at Treatment/Diagnosis

Treatment beginning in adolescence or early adulthood takes less time and is more likely to be successful than therapy started, say in middle age, when maladaptive traits are well-developed and entrenched. On the other hand, young people may be less willing to engage in therapy, and less convinced that interpersonal problems originate from their own thinking and behaviour, or that their problems may be enduring and unlikely to resolve spontaneously.

Interpersonal Relationships

A stable long-term relationship such as an intimate partnership (or therapy) confers a better prognosis, either because the disorder is less severe, allowing enough trust to develop a relationship or because the relationship itself is healing.

Social Supports

Developing other social supports, such as a stable job, which provides a sense of achievement, and good friendships also seem to help.

Severity of Disorder

More severe disorders usually have features of more than one personality disorder indicating more pathology. This usually means a worse prognosis for recovery.

The Older Patient with a Personality Disorder

It was thought until recently that personality disorders “burn out” in old age. We now know differently. The trend is for personality disorders to ameliorate in middle age, but with the stresses encountered as a person ages (loss of spouse, loss of health, eyesight, hearing, occupation etc) coping strategies may no longer be available, and the personality disorder can again become a problem. Nevertheless, personality disorders in older adults are less severe and of lower prevalence than in young people (Abrams and Horowitz, 1999).

Prevalence

Overall prevalence in older adults (over 65s) is approximately 10%. In the community it ranges from 2.8%-13%, from 5%-33% among psychiatric outpatients and from 7%- 61.5% among inpatients. In an Australian inpatient survey, the prevalence was 59% (Engels et al., 2003; Stevenson et al., 2011b). The most common personality disorders were obsessive-compulsive, dependent and paranoid. Older patients with a personality disorder are more likely to be single, separated or divorced. Men were more likely to have antisocial or schizoid personality disorder than women. Never married people were more likely to have obsessive-compulsive or paranoid personality disorder. Obsessive-compulsive personality disorder was also associated with a higher level of income and education.

Although each personality disorder is known to be comorbid with other personality disorders and with psychiatric syndromes over the lifetime, in older people single personality disorders are more common, meaning overall they have less personality pathology. The hypothesis is that personality disorders are not qualitatively distinct syndromes but rather maladaptive variants of personality functioning. Vaillant and Milofsky described a process of maturation and a reduction in the detrimental effects of dysfunctional personality from age 18 to 65. A decline in neuroticism (negative affectivity) up to the age of 70 has been described (Vaillant and Vaillant, 1990).

Personality disorders that caused impairment in the young adult, but which ameliorated in mid-life, can again become a problem in older adults as coping strategies that were available earlier are lost. Increased social isolation, loss of supports and less effective coping, as well as inflexibility of reactions all combine leading to exacerbation of personality disorder symptoms.

Aetiology

Personality disorders generally develop early in life – adolescence or early adulthood – due to a combination of genetic predisposition and environmental trauma. Change in personality can also be caused by brain disease such as dementia, tumour, infection, chronic illness and major life trauma.

Presentation

Personality disorder features may present differently across the life span because of maturational processes and context changes. The outward expression of personality can change although the internal structure of the underlying personality remains stable. For example, impulsivity and reckless behaviour occurs less in older adults with borderline personality disorder, but underlying dysphoria and identity conflicts still exist. The changes of aging further complicate assessment – increased introspection, slowing of thought and motor function, physical disability, hearing and visual impairments.

When faced with a treatment-resistant psychiatric disorder such as chronic depression, be mindful of the possibility of an underlying personality disorder that needs to be addressed as well.

Assessment

Information from those who know the patient well is necessary in addition to that from the patient. Look for patterns, interpersonal difficulties, blaming others, trouble keeping jobs, relationships. If you see any of this, ask specific questions.

Lifetime Psychiatric Comorbidities

All personality disorders have been shown to be significantly correlated with each other except for antisocial personality disorder. The strongest association was between paranoid and schizoid (Zimmerman et al., 2012). The presence of a personality disorder also confers susceptibility to mental disorders, such as major depression, schizophrenia, anxiety disorders as well as physical disorders such as cardiovascular disease (Grilo et al., 2010).

As at other ages, people with personality disorders tend to use dysfunctional coping strategies such as denial, use of alcohol or drugs. They also have fewer or no social supports and less satisfaction with social supports to rely on (Stevenson et al., 2012). Late life personality disorders are associated with impaired

general, social and psychological functioning, interpersonal relationships and quality of life.

Treatment

When mental disorders and personality disorders coexist, treatment efforts are generally directed first at the mental disorder. However, in those with coexisting personality disorders symptoms of the mental disorder are usually more severe, take longer to treat, result in more functional impairment and often cannot be completely resolved.

Treatment of the mental disorder is often pharmacological, but the personality disorder is better treated with psychological approaches, such as dialectical behaviour therapy, interpersonal therapy, cognitive behavioural therapy or problem-solving therapy. Simply teaching more adaptive coping strategies and providing extra social supports can help (De Leo et al., 1999).

Prognosis

The presence of a personality disorder makes treatment of the mental disorder more complicated and may see it take longer and be less successful. The older adult with a personality disorder often has a poorer prognosis because they lack the social supports and the coping skills to aid recovery. They tend to be isolated; family is often estranged and when entering a residential facility, they tend not to “fit in”. Medical morbidity is higher, lifespan often decreased, and suicide is more common (Stevenson et al., 2011a).

Strategies to Sustain Care

We cannot cure the personality disorder quickly, but we can manage the deficits caused by the disorder.

Firstly, treat as any mental health disorder:

1. Establish an empathic understanding of the patient’s situation
2. Do not blame
3. Provide social supports, physical supports
4. Teach coping strategies

Further Reading

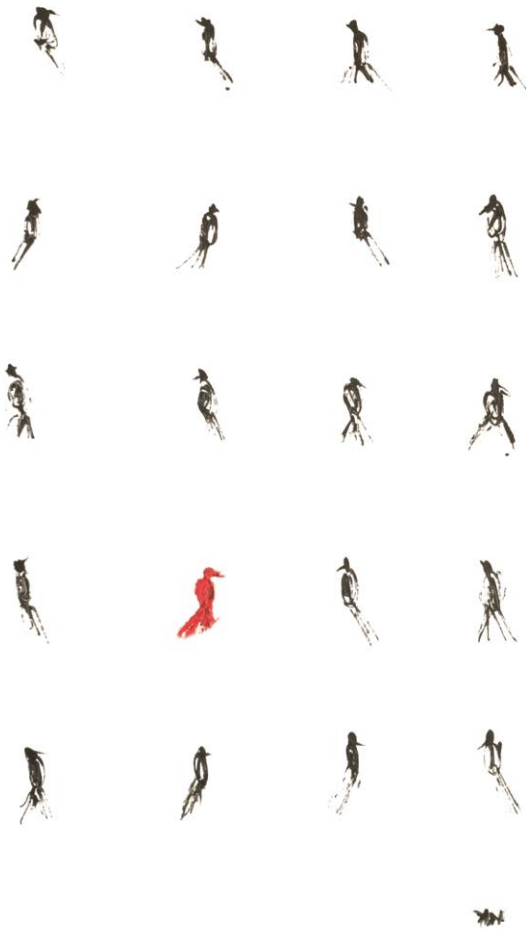
The Harvard Study of Adult Development:

www.adultdevelopmentstudy.org/

Tyrer, P., Mulder, R., Kim, Y. R., & Crawford, M. J. (2019). The development of the ICD-11 classification of personality disorders: An amalgam of science, pragmatism, and politics. *Annual Review of Clinical Psychology*, 15(1), 481-502.

Vaillant, G. E. (1998). *Adaptation to Life*. Harvard University Press.

SPECIFIC CLINICAL GROUPS



Artist: Anne Naylor
Title: *There's Always One*
Medium: Acrylic Paint on Canvas
Dimensions: 500 x 400 mm

Chapter 23: Australian Aboriginal and Torres Strait Islander Psychiatry

John Towney

School of Rural Health, The University of Sydney, Australia.

Robyn Shields

Justice Health and Forensic Mental Health Network, New South Wales Health, Australia.

Emma Walke

School of Public Health, The University of Sydney, Australia, and School of Medical Sciences, The University of Sydney, Australia.

Daniel Talbot

Westmead Hospital, Western Sydney Local Health District, Australia, and Specialty of Psychiatry, The University of Sydney, Australia.

and

Nicholas Burns

Greater Western New South Wales Local Health District, Australia.

Social and Emotional Well-Being

The closest word for ‘health’ in Aboriginal and Torres Strait Islander languages translates best as “wellbeing”, though the equivalent word often has a much broader meaning. For example, the word ‘punyu’, from the language of the Ngarinman from the Northern Territory, is sometimes translated as wellbeing, but the word also encompasses ‘person’ and ‘country’ in its meaning. It is also associated with being strong, happy, knowledgeable, socially responsible, beautiful, clean and safe.

Social and emotional wellbeing is the basis for understanding concepts of physical and mental health for Aboriginal and Torres Strait Islander Australians. It is an holistic approach to health that encompasses the importance of connection to land, culture, spirituality and ancestry, and how these affect the wellbeing of the individual and the community as a whole (Gee et al., 2014). Western medicine tends to understand the disease process by emphasizing the natural history of the disease within the individual. However, for Aboriginal and Torres Strait Islander people, mental health is approached from the broader perspective of social and emotional wellbeing.

The concept recognises that a person's wellbeing is influenced by key social determinants of health, such as stable housing, employment opportunities and access to other societal benefits and privileges. For Aboriginal and Torres Strait Islander people these social determinants have been shaped by major historical and contemporary social forces such as government policies, institutional racism, and the effects of colonisation.

Jiman/Bundjalung woman Professor Judy Atkinson explains the centrality of land and country to Aboriginal and Torres Strait Islander ideas of identity and wellbeing. "Land is a story place. Land holds the stories of human survival across many generations. Land shapes people, just as people shape their countries". Far from being an uninhabited wilderness, as conceived of by Europeans and named 'Terra Nullius', the land called Australia "is a moral sphere, the seat of life and emotions, and a place of the heart". "The land grew the people and people grew the country" (Atkinson, 2002).

Social and emotional wellbeing is not the same as mental health and mental illness, but they are related concepts. They can interact and influence each other. You can have good social and emotional well-being and still have a mental illness. (Department of the Prime Minister and Cabinet, 2017).

Domains of Social and Emotional Wellbeing

In the diagrammatic representation below (Figure 23.1), the self is pictured as being surrounded by 7 overlapping domains that are sources of wellbeing and connection. The domains support a strong and positive Aboriginal and Torres Strait Islander Australian identity. The model also acknowledges the historical, political and societal forces that have shaped, and continue to shape, the social and emotional wellbeing of Aboriginal and Torres Strait Islander Australians (Gee et al., 2014).

The 7 key domains are:

1. **Connection to body** which concerns physical health; feeling strong and healthy and being able to physically participate as fully as possible in life.
2. **Connection to mind and emotions** which concerns mental health; the ability to manage thoughts and feelings. Maintaining positive mental, cognitive, emotional and psychological wellbeing is fundamental to an individual's overall health.
3. **Connection to family and kinship** These connections are central to the functioning of Aboriginal and Torres Strait Islander Australian societies. Strong family and kinship systems can provide a sense of belonging, identity, security, and stability for Aboriginal and Torres Strait Islander people.
4. **Connection to community** which provides opportunities for individuals and families to connect with each other, support each other and work together.
5. **Connection to culture** helps maintain a secure sense of cultural identity by participating in practices associated with cultural rights and responsibilities.
6. **Connection to Country** helps to underpin a person's identity and a sense of belonging. Country refers to an area on which Aboriginal and Torres Strait Islander people have a traditional or spiritual association. Country is viewed as a living entity that provides nourishment for the body, mind and spirit.
7. **Connection to spirituality and ancestors** provides a sense of purpose and meaning. The mental health and emotional wellbeing of Aboriginal and Torres Strait Islander Australians can be influenced by their relationship with traditional beliefs and metaphysical worldviews.

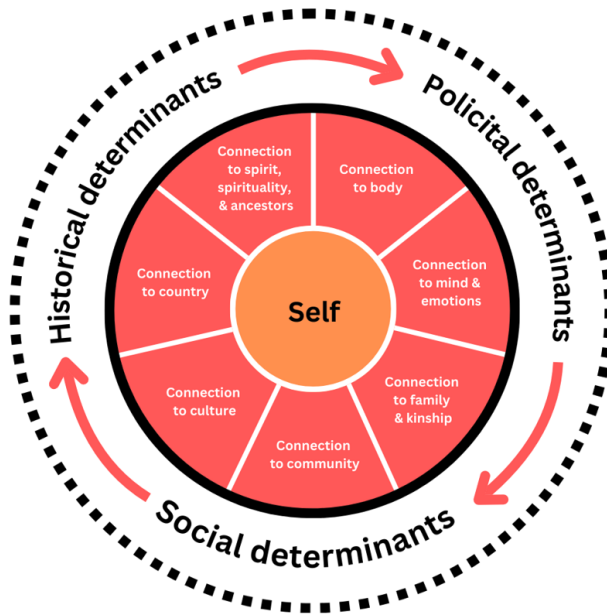


Figure 23.1. Overlapping domains that are sources of wellbeing and connection. Adapted from Gee et al. (2014).

Epidemiology of Psychological Distress and Mental Disorders in Aboriginal and Torres Strait Islander Australians

A Definition of Good Mental Health

At an individual level, good mental health can be defined as a positive state of wellbeing in which a person can manage their thoughts and feelings to cope with the normal stressors of life and reach their potential. For many Aboriginal and Torres Strait Islander Australians, good mental health goes beyond this. It indicates feeling a sense of belonging, having strong cultural identity, maintaining positive interpersonal relationships, and feeling that life has purpose and value (Dudgeon et al., 2014).

Life Expectancy

Life expectancy and deaths are widely used as indicators of population health. Given current mortality patterns, Aboriginal and Torres Strait Islander males

born in 2015–2017 can expect to live 71.6 years, and Aboriginal and Torres Strait Islander Australian females 75.6 years, which is 10 years less than for the general population (Australian Bureau of Statistics, 2019). In general, life expectancy is lower in remote areas, with Aboriginal and Torres Strait Islander males and females living in major cities expected to live around 6 years longer than those living in remote and very remote areas.

Causes of Death

When considering causes of death, in 2020, intentional self-harm was the fifth leading cause of death among Aboriginal and Torres Strait Islander Australians (behind coronary heart disease, diabetes, chronic respiratory diseases, and cancers of the airways), suggesting a high burden of psychological distress in the Aboriginal and Torres Strait Islander community (Australian Bureau of Statistics, 2022).

Burden of Disease

The burden of disease describes the number of years of healthy life lost, either through chronic ill health or premature death. Almost half of the burden of disease in Aboriginal and Torres Strait Islander Australians is due to modifiable risk factors (Australian Institute of Health and Welfare, 2022b). Mental illness and substance use as a disease group is by far the single biggest causal group of burden of disease for Aboriginal and Torres Strait Islander Australians (Australian Institute of Health and Welfare, 2022b). This disease group accounts for 20% of the total burden of disease, overtaking cardiovascular disease in recent years. This is highest in the age group late childhood to age 44. When considering individual diseases, 4 of the 5 diseases with the highest burden among Aboriginal and Torres Strait Islander Australians in 2018 were mental health related; anxiety disorders (accounting for 5.3% of the burden of disease), suicide and self-inflicted injuries (4.6%), alcohol use disorders (4.4%) and depressive disorders (4.3%).

Suicide

Data from 2016–2020 show that the rate for suicide of Aboriginal and Torres Strait Islander Australians is almost twice the rate of non-Indigenous Australians, with the differences being greater for people aged under 45 (Australian Bureau of Statistics, 2022). Suicide was the fifth leading cause of death among Aboriginal and Torres Strait Islander Australians in 2020, accounting for 5.5% of all deaths. It was also the leading cause of death for Aboriginal and Torres Strait Islander children aged 5–17 (Australian Bureau of Statistics, 2022). By comparison, suicide was the 14th leading cause of death for the general Australian population (1.9% of all deaths).

Rates of Mental Distress and Mental Illness

There are two main sources of information for estimating rates of psychological distress and mental illness. The first source is the National Aboriginal and Torres Strait Islander Health survey, which is conducted by the Australian Bureau of Statistics periodically (Australian Bureau of Statistics, 2019). The second is the Mayi Kuwayu study, which has been created by and for Aboriginal and Torres Strait Islander peoples and in response to the general community's perceived lack of understanding of the importance of Aboriginal and Torres Strait Islander culture and its link to wellbeing (Gee et al., 2014). Interestingly, the results of the two studies are similar.

Rates of Psychological Distress

In the 2018-2019 National Aboriginal and Torres Strait Islander Health Survey (Australian Bureau of Statistics, 2019), over two-thirds (66%) of Aboriginal and Torres Strait Islander Australian adults reported some degree of psychological distress. The rate was higher among males (70%) than females (63%). An estimated 31% reported high or very high levels of psychological distress in the last 4 weeks. The number reporting high or very high levels of psychological distress has increased compared to previous surveys. Those aged 45 to 54 years were most likely to report 'high or very high' levels of psychological distress. The Mayi Kuwayu study suggests that up to half of the psychological distress experienced is caused by discrimination.

Mental Disorders

Mental disorders are diagnosed according to standardized criteria based on internationally standardized diagnostic tools such as the DSM-5-TR (American Psychiatric Association, 2022). It is important to note that the main diagnostic tools used in population surveys in Australia have not been validated for Aboriginal and Torres Strait Islander Australian populations. However, this notwithstanding, in the 2018–19 survey, an estimated 24% of Aboriginal and Torres Strait Islander Australians reported having a diagnosed mental health or behavioural condition with a higher rate among females (25%) than males (23%). More than one in 10 individuals reported having an anxiety disorder (16.5%) or depression (13.3%) (Australian Bureau of Statistics, 2019).

Within the group of mental and substance use disorders (that is, considering all mental disorders as accounting for 100%) the leading disorders were anxiety disorders (23%), alcohol use disorders (19%), depressive disorders (19%), drug use disorders (excluding alcohol) (9%) and schizophrenia (7%) (Australian Institute of Health and Welfare, 2022b).

Drugs and Alcohol Use

Hazardous alcohol and substance use is a major modifiable risk factor that contributes to the burden of disease. Hazardous substance use occurs at approximately twice the rate for Aboriginal and Torres Strait Islander Australians when compared to the general population (Australian Institute of Health and Welfare, 2011). In a 2008 survey by AIHW, 23% had used an illicit drug recently and 4 out of 10 had used an illicit drug in the last 12 months. Cannabis and amphetamines were the most commonly used drugs.

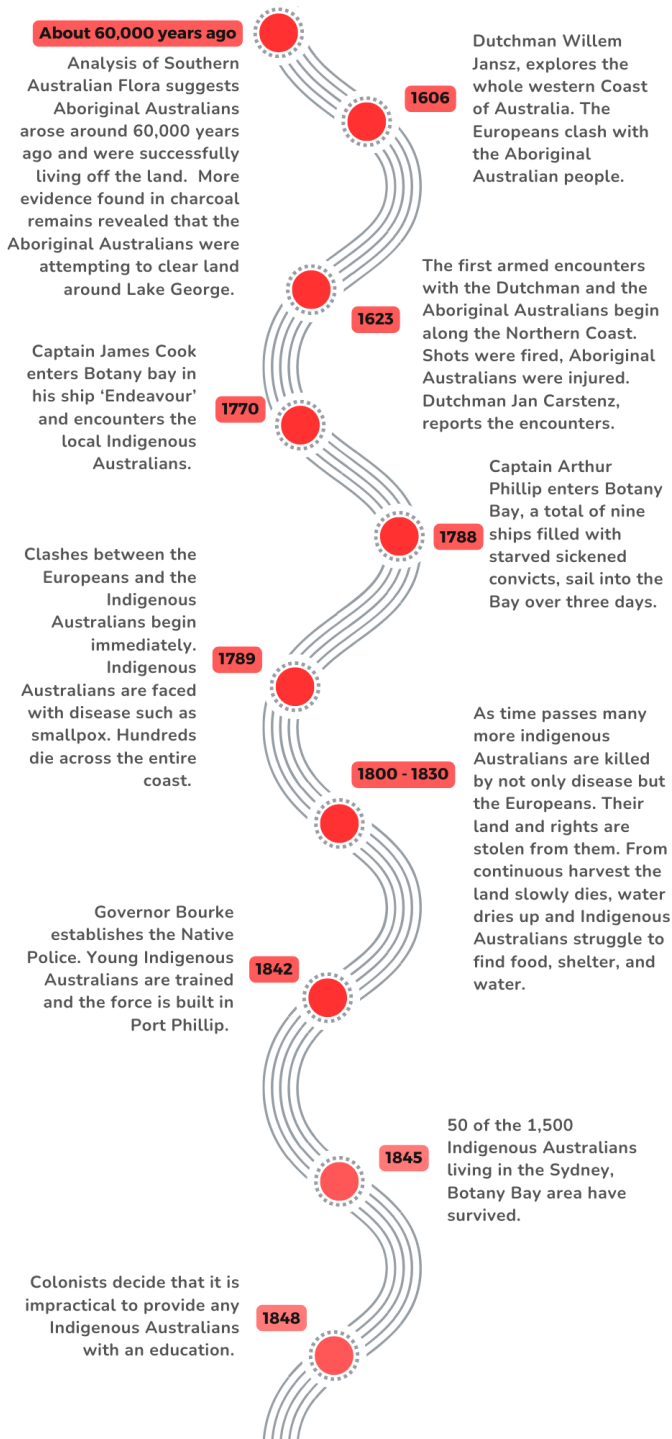
Cigarette smoking occurs in over 40% of Aboriginal and Torres Strait Islander Australians which is twice the general population rate (Australian Institute of Health and Welfare, 2011). With regard to alcohol use, whilst the overall rate for Aboriginal and Torres Strait Islander Australians is lower than the general population rate, patterns of hazardous use (including binge pattern use and chronic use) are greater, especially in the age 16-24 (Australian Institute of Health and Welfare, 2011).

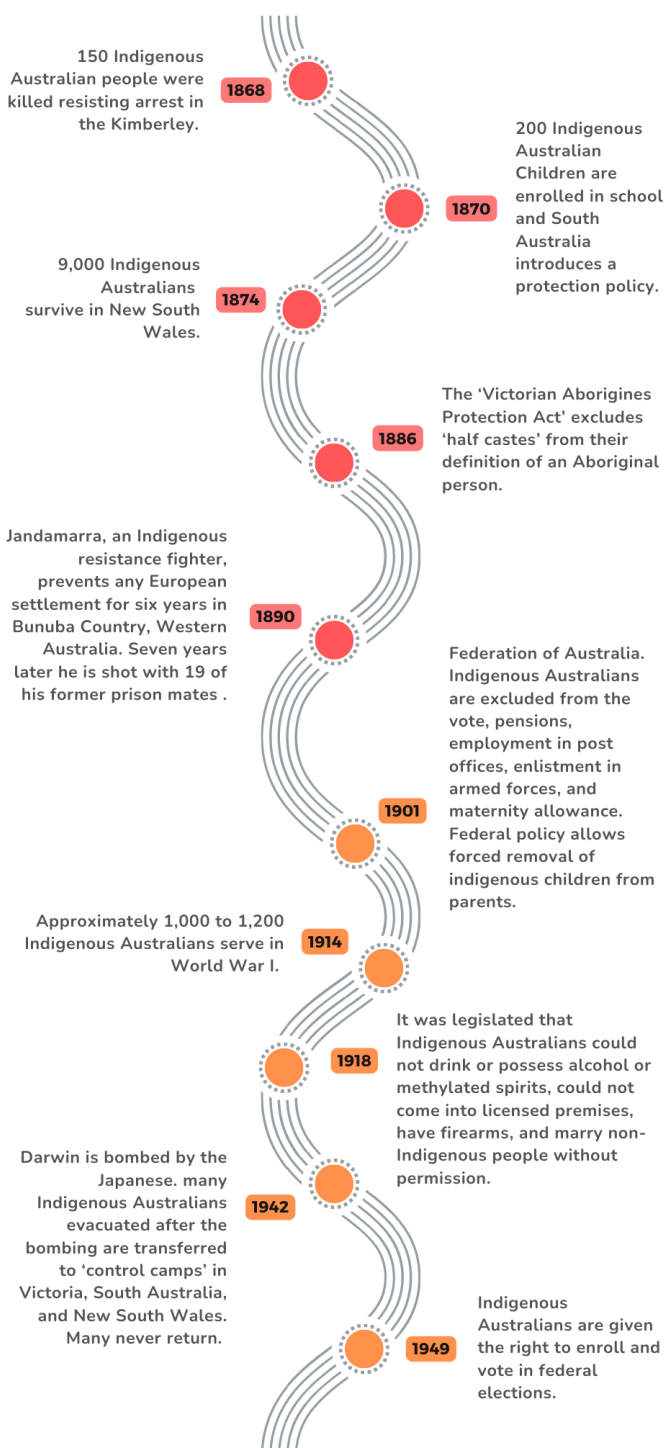
Hospital Use

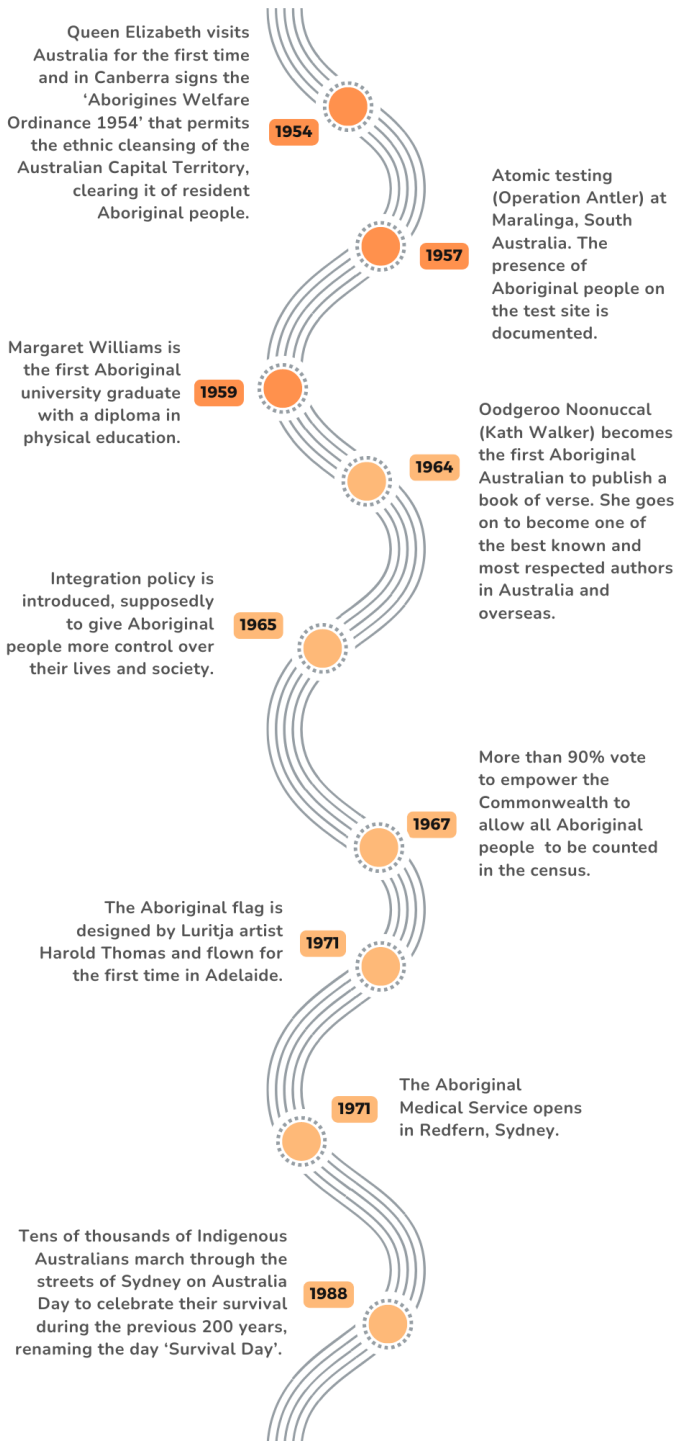
Hospitalization rates can be used as a proxy measure for psychiatric morbidity in the community. In the period 2020-2021, hospitalizations for mental and behavioural disorders in Aboriginal and Torres Strait Islander Australians accounted for 8.4% of all hospitalizations for Aboriginal and Torres Strait Islander Australians, of which 0.5% were for self-inflicted injury (Australian Institute of Health and Welfare, 2022a). The rates of admission were approximately twice the rate for psychotic illnesses compared to the general population and more than 1.2 times the rate for depressive disorders, suggesting greater severity of illness.

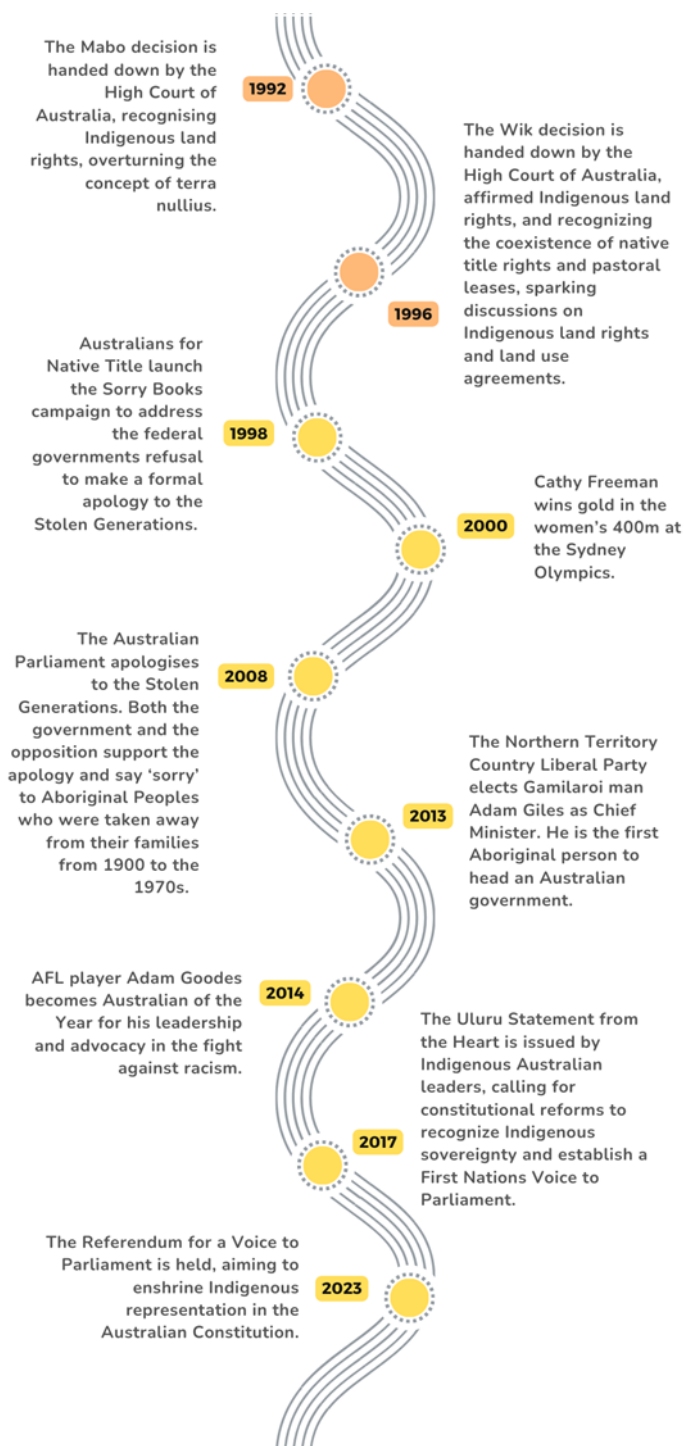
History Timeline

The timeline on the following pages provides a snapshot of some key social and political events that have shaped the welfare of Aboriginal and Torres Strait Islander Australians in the post-colonial era. It highlights the traumatic nature of the historical interactions between Aboriginal and Torres Strait Islander Australians and their European colonizers.









Inter-Generational Trauma and Determinants of Health

The psychological impacts of trauma are explored in detail in a separate chapter of this book and is essential pre-reading for this chapter. Going beyond this, given the seismic shift in the world of Aboriginal and Torres Strait Islander Australians that occurred with the dawn of the colonial era, it is important to understand the impacts of these historical forces on Aboriginal and Torres Strait Islander Australians as an entire race.

The encounter between Australia's Aboriginal and Torres Strait Islander peoples and European colonizers has been described as an asymmetrical collision of cultures with the burden of trauma almost exclusively confined to Aboriginal and Torres Strait Islander Australian individuals, families and communities (Hunter, 1998). The traumatic events can be broadly divided into two phases; the phase of physical violence, beginning in the early colonial period, and marked by mass extinctions through killings, the introduction of foreign diseases, and starvation due to the forced removal of lands and food sources. The second phase is one of structural violence, in which state and federal government policies were enacted with the underlying philosophy of crude social Darwinism, that is the belief that Aboriginal and Torres Strait Islander peoples were an inferior race of humans that would inevitably die out. These policies had two aims: assimilation, and the elimination of Aboriginal and Torres Strait Islander culture, including its history and language. As examples, such policies allowed for segregation of Aboriginal and Torres Strait Islander people into settlements, the forced removal of children from their families (the 'stolen generation') and sanctioned slavery.

Trauma that is experienced by an entire group or race of people as a whole is not unique to Aboriginal and Torres Strait Islander Australians. It is an experience common to many First nations peoples across the world. There are also many studies of war-related trauma showing the intergenerational transmission of trauma through the effects on family structure and functioning (Harkness, 1993).

There are numerous terms which essentially describe the same process. The terms 'collective trauma', 'transgenerational trauma' and 'intergenerational trauma' are synonyms that describe the phenomenon of trauma that impacts universally throughout a group of people, and which then reverberates from one generation to the next within that group, so that the effects are felt for many generations to come. Although these experiences may be more difficult to describe at the individual level, the trauma results from experiences that impact a whole community, and is felt by the community. For Aboriginal and Torres Strait Islander people these terms describe the impact of colonisation, dislocation, and

discrimination affecting Aboriginal and Torres Strait Islander Australian individuals, families, and communities (Atkinson et al., 2014).

According to Erikson (1994), the impact of collective trauma is a ‘blow to the basic tissues of social life that damages the bonds attaching people together and impairs the prevailing sense of community’ (p. 233), which effectively renders the group powerless and deprived of the communal scaffolding, structures, and systems to navigate traumatic situations (Menzies, 2019).

Professor Helen Milroy (Australian Institute of Health and Welfare, 2016) offers a comprehensive description about the multi-layered effects of intergenerational trauma on Aboriginal and Torres Strait Islander Australians, displayed in the box below.

Professor Helen Milroy on intergenerational trauma

“The transgenerational effects of trauma occur via a variety of mechanisms including the impact of attachment relationships with care givers; the impact on parenting and family functioning; the association with parental physical and mental illness; disconnection and alienation from the extended family, culture, and society. These effects are exacerbated by exposure to continuing high levels of stress and trauma including multiple bereavements and other losses, the process of vicarious traumatising where children witness the on-going effects of the original trauma which a parent or care giver has experienced. Even where children are protected from the traumatic stories of their ancestors, the effects of past traumas still impact on children in the form of ill health, family dysfunction, community violence, psychological morbidity, and early mortality.”

A central pathogenic mechanism in transgenerational trauma is the disruption to the building blocks of stable attachment formation, those being a stable and secure home environment. Menzies argues that due to practices of removal from family and relocation to unfamiliar environments there is loss of basis for developing healthy and sustainable family structures (Menzies, 2019).

“Forced separation and assimilation have meant that significant numbers of Aboriginal people experienced the destruction of the family unit, the dislocation from home and community to unfamiliar places with unfamiliar people, because as children they were denied what is now considered to be a fundamental and universal human right: the right to grow up in a family environment, in an atmosphere of happiness, love and understanding”.

When considering the impact of trauma within Aboriginal and Torres Strait Islander communities, it is important to engage with communities and to learn about and acknowledge some of the experiences that have left their legacy. This journey of ‘truth telling’ is part of the process of healing. The story in the text box below provides an illustration of collective trauma. This story is from the Brewarrina community (Ngemba Country). The story is from an Aboriginal and Torres Strait Islander elder from that area who knew some of the old tribal men when he was young, and he didn’t know why they had a pronounced limp. When he asked about it this is what he was told.

When settlers first came to the Brewarrina region, they invaded and occupied existing traditional lands for farming purposes. Due to the clash of cultures between the traditional custodians and the settlers, there was much disharmony between these groups. Over time the settlers stole Aboriginal women and girls as they needed servants to look after the house duties until their settlements were established, and they could then encourage colonial women to relocate and start families and support the settlers.

Once the Aboriginal women and girls were stolen, the Aboriginal men would come to the properties at night and leave with the Aboriginal women and girls. The Aboriginal groups would then travel away, and the settlers had no one to do the house duties. This was an ongoing problem for the settlers. The traditional owners could travel across land very fast and were able to avoid being found by the settlers.

To reduce the problem of losing Aboriginal women and girls, the settlers then captured some of the Aboriginal men looking to free their family members, and they would cut one of their Achilles tendons so that the men could not travel very far on foot, and they could not leave the area with their families. Once this happened the Aboriginal men would be easily caught if they tried to leave, and the Aboriginal women and girls were able to be kept as servants.

Atkinson et al. (2014) argue that western diagnostic classification systems are not adequate for describing the impact of trauma in First nations Australians, and therefore may not be helpful in guiding management. Mental illness diagnoses such as PTSD do not conceptually capture the levels of chronic, ongoing stress that Aboriginal and Torres Strait Islander peoples experience in their everyday lives. The impacts of trauma are cumulative and so pervasive that they are too numerous to mention. It is virtually impossible to identify and overcome a single source of stress. Individuals, families and communities continue to feel so disempowered and let down by Australia’s laws, institutions and societal attitudes that they have entered a state of learnt helplessness, in which they have learnt not to hope for change or improvement.

Key Determinants of Health

Health outcomes for Aboriginal and Torres Strait Islander peoples are influenced by a complex interaction of environmental (non-medical) factors, behaviours and biological factors. These factors are called the ‘determinants of health’, and they can be barriers or enablers to health and wellbeing (Australian Indigenous HealthInfoNet, 2024).

An important aspect of this interaction is the role played by the key social determinants of income, education, and employment. According to the World Health Organisation, the social conditions in which people are born, live and work is the single most important determinant of good health or ill health (Australian Institute of Health and Welfare, 2016). As factors that affect health, social determinants can be seen as ‘causes of the causes’—that is, as the foundational determinants which influence other health determinants.

These and other related indicators of material wealth combine to determine one’s socioeconomic position within the wider community. The social determinants of health related to socioeconomic position help to explain both the gaps in the average health status of Indigenous and non-Indigenous Australians, and the wide variation observed in the health outcomes within the Aboriginal and Torres Strait Islander Australian population.

Other key determinants include:

1. **Environmental factors** including access to safe water supply, good waste management, food security, caring for country, personal hygiene, and containment of communicable diseases.
2. **Cultural factors** such as exposure to institutional racism (for example in the judicial system), discrimination, and the ongoing effects of colonisation.
3. **Community capacity** factors that promote strong communities. These include family safety, child protection, interactions with criminal justice, governance, access to transport, and digital services. On some of these indices, Aboriginal and Torres Strait Islander Australians are grossly disadvantaged. For example, Aboriginal children are removed from their families at 7 times the rate of non- Aboriginal and Torres Strait Islander children. Aboriginal and Torres Strait Islander people are imprisoned at a rate of 13 times the national rate (Australian Bureau of Statistics, 2023).
4. **Health behaviours:** healthy eating (which is dependent on access to healthy fresh food), physical activity, smoking, substance use and immunisation.

Assessment and Diagnosis

While assessment and diagnosis are important tools for mental health professionals, their misuse can perpetuate racial and cultural stereotypes, and be used as a method of cultural control. Much of the suspicion that Aboriginal and Torres Strait Islander people have regarding these processes derives from its political nature. In the context of the traumatic history of European impact on Aboriginal and Torres Strait Islander Australians, it is easy to understand the caution that most Aboriginal and Torres Strait Islander people have about psychiatric assessment and diagnosis. Psychiatric assessment has been deeply rooted in the power differential between the coloniser and colonised. It is important that mental health practitioners be careful not to further pathologize Aboriginal and Torres Strait Islander people in their assessment and diagnostic process (Adams et al., 2014).

The RANZCP (Royal Australian and New Zealand College of Psychiatrists) highlights the important role psychiatrists can play in breaking down myths and stereotypes about Aboriginal and Torres Strait Islander people, and in changing societal attitudes (Royal Australian New Zealand College of Psychiatrists, 2014). The college has articulated some key principles to guide mental health practice:

- Adapt Western-style individualistic models of diagnosis and assessment to accommodate the broader conceptual frame of Aboriginal and Torres Strait Islander well-being, incorporating the importance of land, spirituality and culture.
- Recognize the role of trauma, racism, marginalization, mainstream ignorance and hostility, and its impact on Aboriginal and Torres Strait Islander mental health and burden of distress.
- The workforce should adopt an Aboriginal and Torres Strait Islander frame of reference and be skilled in cultural competence.
- Caution must be taken against perpetuating ongoing trauma in our practices of assessment and care.
- Practice in partnership with Aboriginal and Torres Strait Islander people and work with respect and dignity.
- Recognize and seek out culturally relevant models of care as alternatives to Western biomedical models of care.

With these principles in mind, the challenge is to conduct diagnostic assessments in a culturally sensitive manner, ideally using tools that have been devised and validated for culturally diverse Aboriginal and Torres Strait Islander populations. Western diagnostic assessment is generally individualistic and symptom-focused, aiming at syndromal identification. Furthermore, it has been criticized for its focus on deficits,

rather than strengths, and a tendency to pathologize the ordinary. This increases the risk of discrimination and stigmatization.

The most internationally recognized and commonly-used diagnostic tools DSM-5 and ICD-11, have attempted to improve the cultural sensitivity of their diagnostic instruments. The DSM-5-TR now includes guidelines for cultural assessment of people from diverse cultural backgrounds, which separates out the cultural elements from the context of psychiatric diagnosis. It identifies some key dimensions to be explored in any assessment process. These include cultural identity, cultural explanations of the illness, cultural factors associated with psychosocial functioning, and cultural elements of the relationship between the client and the practitioner. The DSM-5-TR now offers the 'Cultural Formulation Interview', which can be used to assist clinical understanding and decision making, not merely diagnostic process (American Psychiatric Association, 2022).

The Practitioner-Client Relationship

It is important to consider the power differential inherent in the relationship between the assessor and the person being assessed and how this may impact on presentation and interpretation. The national practice standards for the mental health workforce 2013 (Department of Health and Aged Care, 2013), dictate that all practitioners must undergo training in cultural competency to enhance culturally safe assessments. The Australian Psychological Society guidelines encourage clinicians to consider the high burden of trauma, discrimination, the high burden of psychological distress and the need to look for strengths.

Preparation and Context for Assessment

The assessor should be patient and respectful. It is important to introduce themselves and offer some details about who they are. They should take time to get to know the person before deciding on which assessment tools are useful. They should also get to know the community and their cultural context. Consideration should be taken when deciding on the location for the assessment, aiming to conduct it in the natural ecological setting for the person. It may be appropriate to use a cultural broker. Finally, the assessor should be aware of their own beliefs and attitudes and consider how this may impact on the assessment process.

Language and Communication

Language barriers and problems with inter-racial miscommunication may impact on the quality of the assessment. Be aware that English may not be the preferred language for the service user. Good communication skills are critical. It is

important to be sensitive to the language skills of the person and their educational level. Try to adopt a yarning style of interview, rather than the traditional western interrogative approach to medical interviewing. Be sensitive of non-verbal cues such as posture, gesture, eye contact. It is also important to be aware of sensitive topics that may be retraumatizing.

Considerations in Mental State Examination

Caution should be taken when interpreting mental state findings. In particular, practitioners should avoid over-interpretation and culturally-bound inferences. For example, in terms of behavioural presentation, posture, gesture, touch and eye contact, all have cultural interpretations that may differ from one setting to the next. Avoidance of eye gaze, for example, may be a sign of respect rather than avoidance or low mood. Affective responses too, may be a function of cultural clash. Anger and resentment in Aboriginal and Torres Strait Islander people may be more historical than circumstantial. Non-Aboriginal and Torres Strait Islander clinicians conducting assessment are representatives of the dominant culture and may invoke feelings of anger and resentment that are substantially exacerbated by the context, particularly if the review is involuntary. Table 23.1, adapted from Adams et al. (2014), outlines some factors to be aware of in assessing mental state.

Table 23.1. Factors to consider when examining mental state of Aboriginal and Torres Strait Islander Australian peoples.

Appearance	Understand the person’s ‘usual’ standard of self-care and appearance. Identify any changes that may indicate a mental health issue. Consider cultural influences and manifestations such as grief.
Behaviour	Have a good understanding of Aboriginal and Torres Strait Islander culture as it relates to a person’s behaviour. Behaviours can be culture-specific.
Affect	Affect can take on cultural forms as not all human emotions are universal. Anxiety and depression for example, may be difficult to diagnose as the manifestation of these conditions could be vastly different across cultures
Mood	An Aboriginal and Torres Strait Islander person’s mood may not be expressed in the same way as a non-Aboriginal person. The language used to describe mood may not have meaning for Aboriginal and Torres Strait Islander people and may need to be translated into meaningful terms.
Speech and thought form	Thought disorders may be difficult to detect if the client does not have good English. The clinician would then need to rely on the services of an Aboriginal and Torres Strait Islander health worker or community member.
Thought Content	Aboriginal and Torres Strait Islander spirituality may display as delusional or otherwise cultural. The clinician needs to ascertain whether the primary symptoms pre-date the culturally based retrospective attributions.
Perception	These may be pathologically or culturally based. It is advisable to seek advice from the Aboriginal and Torres Strait Islander mental health worker. Auditory hallucinations are less commonly sited and may be indicative of a mental disorder.
Cognition	Assessing cognition is difficult due to the lack of culturally appropriate assessment tools— assessments of function and activities of daily living are not appropriate in remote communities where living is collective in nature. It is not uncommon for families to seek help as a last resort.

Culture-Bound Syndromes

The DSM-5-TR describes culture-bound syndromes as recurrent, locality-specific patterns of aberrant behaviour and troubling experience that can be

understood within a cultural context (American Psychiatric Association, 2022). Furthermore, they can only be addressed in that cultural context. They may or may not be linked to a particular DSM diagnostic category. Many of these patterns are indigenously considered to be “illnesses,” or afflictions, and most have local names. At heart is the need to recognize the different ways of communicating distress. To make sense of these, there is a need to reference culture for understanding experiences.

Tracey Westerman (Westerman, 2021) has validated several culture-bound syndromes in which an understanding of culture is crucial. She highlights the need to understand Aboriginal and Torres Strait Islander grieving practices, and practices of retribution for wrong-doing (‘being sung’), which may lead to misinterpretation of presenting phenomena. For example, spiritual visitations may be misinterpreted as hallucinations, or sorry cuts may be interpreted as self-harming related to depression or complex trauma rather than a response to a bereavement. Symptoms such as a weakened spirit and community disconnection (longing for country) may require cultural resolution and healing with culturally appropriate counselling services.

Assessment Models and Tools

As discussed above, the commonly used Western tools DSM and ICD have made cultural adaptations to assist in assessment and diagnosis. In addition, there are numerous validated culturally sensitive tools now available when working with Aboriginal and Torres Strait Islander people. One such example is the *Mental Health Stay Strong Care Plan* (Menzies School of Health Research, 2008). The comprehensive package of support materials provides a series of culturally appropriate stages of assessment and care planning options for supporting mental health in Aboriginal and Torres Strait Islander communities. The package incorporates an understanding of the holistic nature of Aboriginal and Torres Strait Islander health and mental health. Their ‘grow strong mental health tree’ combines the visual with the written word to explain mental health issues and provide a framework for exploring them in a cultural context. In this area, more work needs to be done to develop a comprehensive suite of culturally validated tools. Table 23.2, adapted from Adams et al. (2014), lists other culturally validated instruments.

Table 23.2. A List of Culturally Validated Assessment Tools.

Assessment Tool	Description
The Westerman Aboriginal Symptom Checklist Youth (WASC-Y), The Westerman Aboriginal Symptom Checklist-Adult (WASC-A)	Youth 13-17 years, Adults aged 18 years and over. Identifies risk of anxiety, depression and suicidal behaviour whilst factoring in cultural resilience.
Kimberley Indigenous Cognitive Assessment tool (KICA)	Cognitive screening tool that assesses dementia in older Aboriginal and Torres Strait Islander Australians living in rural and remote areas.
Strong Souls	Assesses social and emotional wellbeing of Aboriginal and Torres Strait Islander youth. Measure for depression, anxiety, suicide risk, and resilience.
Negative Life Event Scale	Assessment of psychological wellbeing in Aboriginal and Torres Strait Islander Australians. Measures exposure to stress.
Kearins' Visual Spatial Memory test	Assessment based around research with Aboriginal and Torres Strait Islander children aged 6-17 years that focused on task behaviour.
Australian Aboriginal Version of the Harvard Trauma Questionnaire (AAVHTQ)	Culturally competent measure of specific traumatic stressors and trauma symptoms criteria for PTSD. This questionnaire includes specific cultural idioms of distress reactions that are relevant to Aboriginal and Torres Strait Islander people.
The K-5 measure of psychological distress	A subset of five questions adapted from the Kessler Psychological Distress Scale-10 (K-10) developed in 1992 by Kessler and Mroczek (ABS 2003).
The K-10	A non-specific psychological distress scale of 10 questions to measure levels of negative emotional states experienced in the 4 weeks prior to interview.
Kimberley Mum's Mood Scale	Currently in development as an adapted version of the Edinburgh Postnatal Depression Scale and is currently being validated across the Kimberley in Western Australia. This tool screens for depression and anxiety.
Q Test	Cultural-fair, language-free assessment measure of cognitive functioning screening and trainability.

In summary, in participating in the acts of mental health assessment and diagnosis, the practitioner needs to be aware of the need to decolonize these processes. We should aim to shift the discourse from one of distress to a discourse of hope. This can be facilitated by the adhering to the key principles of

acknowledging white privilege and understanding Aboriginal and Torres Strait Islander terms of reference.

Considerations in Management

In theory, all the treatment modalities and technologies described in this book can and should be considered when treating Aboriginal and Torres Strait Islander people. In this section we describe some overarching principles to take into consideration when working with Aboriginal and Torres Strait Islander service users.

To work effectively with Aboriginal and Torres Strait Islander people, practitioners must recognize that the Western biomedical model is often inadequate, and if used in isolation is potentially harmful. The vision articulated in the NSW Aboriginal Mental Health and Well-being Strategy 2020-2025 (New South Wales Ministry of Health, 2020) articulates the key cornerstones of care: that Aboriginal and Torres Strait Islander people will have access to holistic and person centred/family centred care. Care should be delivered in a culturally safe and trauma-informed manner. And finally, there should be a focus on healing practices.

Practitioners must always keep in mind the risk of engaging in paternalistic and coercive practices that mimic the process of colonisation. It is important to be aware that Aboriginal and Torres Strait Islander people may be mistrusting and suspicious of mainstream services. They may be wary of presenting to services voluntarily and may do so only when their symptoms are advanced. Practitioners must be aware of the need to promote a sense of trust and safety. In this regard, the same principles discussed in the previous section about assessment and diagnosis also pertain to management. In addition to the principles listed in the section above, some further considerations include:

1. The location for care - Hospitals and emergency departments can be noisy and frightening places. If possible, try to conduct assessments or therapy in quiet locations with access to the outdoors, or in designated culturally safe spaces.
2. Informed consent - When a treatment plan is explained, care must be taken to consider the educational level and proficiency in the language being used. A cultural broker can help in these circumstances or a family member.
3. Keep family and community informed of any assessment outcomes and treatment decisions as soon as possible, if they are not present.
4. Respect the need for privacy and confidentiality at all times.
5. Avoid the use of coercive treatment wherever possible, including involuntary treatment orders and restrictive practices under mental health legislation or other legislation. If this is necessary, keep involuntary

treatment as brief as possible. This pertains in particular to the use of seclusion and restraint, as well as involuntary hospitalisation.

6. Be aware of social hierarchies that may exist within communities and the need to respect elder status and community leader status. The practitioner's gender and age may impede the development of a strong working relationship.

The practitioner should keep in mind the importance of connection to country and kinship ties. If there is an opportunity for a person to undergo healing on country and in their own community, this should be facilitated. If possible, try to create opportunities for the person to reconnect to their country and the cultural and spiritual practices of their elders. Connection to community is an important part of healing.

Enhancing or restoring cultural identity is a healing practice. Formulating a 'cultural wellness plan' can be helpful. This process identifies key cultural signifiers for the person, including the person's country or nation of origin, relevant language group, significant family or community members, identifying their totem, relevant community dreaming stories and other relevant cultural and spiritual practices.

Self-determination is also an important factor in promoting good health. At the level of the individual, this means allowing the person to work alongside practitioners to make informed choices about their own treatment. Management plans should be co-designed, ideally with the support of family and community members. This will help to identify which are the important healing practices for that particular person.

Practitioners should be aware that navigating health services may be difficult for Aboriginal and Torres Strait Islander people. Help may be needed in identifying local health resources, both general and Aboriginal and Torres Strait Islander Australian-specific. Again, the Aboriginal Mental Health workforce is helpful in navigating health systems.

There are some particular considerations in regard to biomedical aspects of care. Some of the pharmacological agents used in psychiatric practice can have a strong impact on weight, cardiac and metabolic function, including in particular the second-generation antipsychotics, clozapine and mood stabilizers. Given the relatively high incidence of obesity, type 2 diabetes, heart disease and hypertension, caution should be taken to use the lowest possible doses, or to choose a cardiometabolic-sparing agent where possible. Similarly, caution is recommended when using lithium and other agents that are entirely renally excreted, given high community rates of renal disease.

Compliance with prescribed medication poses challenges in circumstances where coercive treatment practices may retraumatize, especially in involuntary administration of depot medication. Where possible, enlist the assistance of family or community members to increase the likelihood of oral medication compliance, in preference to depot medication.

Finally, practitioners should be aware of the presence of traditional healing practices and the possibility that these can co-exist with Western medical practices, or even replace the need for them. There are multiple examples of innovative traditional healing practices, resources, and registered Ngangkari traditional healers.

Broader Considerations

Thinking more broadly, health service providers can assist in ensuring that their services are ready to provide care that is culturally safe, and trauma informed. This means ensuring that all practitioners are trained in cultural competence and that the physical environment is a welcoming space with readily recognizable symbols of Aboriginal and Torres Strait Islander cultural and language.

There are considerations at a national level. In keeping with the principle of empowerment and self-determination, Aboriginal and Torres Strait Islander people should be given the chance to design and operate health services that are tailored to their particular needs. This has been the vision of Aboriginal Medical Services which have been operating across the country since 1970s.

Furthermore, the burden of mental health problems in the Aboriginal and Torres Strait Islander community cannot be comprehensively addressed until the existing social inequities are corrected. The Closing the Gap scheme aims to rectify the social disadvantage Aboriginal and Torres Strait Islander Australian experience on key social determinants of health yet has failed deliver on almost all measures so far, especially in relation to Aboriginal and Torres Strait Islander suicides.

Finally, healing cannot take place without truth telling and reconciliation. Aboriginal and Torres Strait Islander people need to have their perspectives on historical events heard and recognized as valid. Judy Atkinson (Atkinson, 2017) talks of the value of deep listening. Until we start listening and learning about the depth and complexity of historical and contemporary factors affecting the health and wellbeing outcomes for Aboriginal and Torres Strait Islander Australians, we cannot hope to support individuals and communities in the work that is currently being undertaken to resolve some of these outcomes. By valuing the experiences, and

better understanding the circumstances of many Indigenous populations, we can improve our own capacity to support those who are most vulnerable and in need.

Now brood no more
On the years behind you,
The hope assigned you
Shall the past replace,
When a juster justice
Grown wise and stronger
Points the bone no longer
At a darker race.

From Song of Hope (Oodergoo of the tribe Noonuccal, formally Kath Walker).

Further Reading

Australian Indigenous Health infonet: healthinfonet.ecu.edu.au

The Mayi Kuwayu Study: mkstudy.com.au

Chapter 24: Perinatal Psychiatry

Ankur Srivastava

*Westmead Mother and Baby Unit, Westmead Hospital, Western Sydney Local Health District, Australia, and
Statewide Outreach Perinatal Services, New South Wales, Australia.*

and

Philip Boyce

*Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia, Westmead Institute of
Medical Research, Australia, and Faculty of Health, Charles Darwin University, Australia.*

Introduction

The birth of a child is a significant milestone and life transition for any woman and her family. It is usually a joyous event, as is the pleasure in observing the extraordinary development from the highly dependent newborn into an exploring and chattering toddler. While this is generally a positive life transition, it is also a time of risk for women for developing, or having a recurrence, of a mental disorder, either during pregnancy or over the first year postpartum (what is referred to as the perinatal period). The age of first-time mothers, in the mid to late 20s, coincides with the peak incidence of the common mental disorders as well as for the psychotic disorders. It is, therefore, not surprising to find high rates of the common mental disorders over the perinatal period. The more severe mental illnesses, schizophrenia and bipolar disorder can also present during the perinatal period, presenting unique problems for the clinician.

Maternal well-being is critical for the optimal development and maturation of the human infant. Mental illness during pregnancy, and its treatments, can have adverse effects on the developing foetus and, by the same token, maternal mental illness can cause disruption to the mother-infant relationship, and attachment, that may have long-term repercussions, with the offspring at risk of developing mental disorders later in life.

From a perinatal psychiatry point of view, there is a risk of exposing the foetus and the infant to possibly harmful medications. But interestingly, it is also the time when there is high contact with health professionals, and it is a time when we can identify women who are at risk of the common mental health problems or other

disorders and provide intervention for them. It's an ideal opportunity for prevention and/or early intervention in this period.

Importance of the Perinatal Period

The foundations of healthy development are laid down during the perinatal period; the origins of some disorders, such as schizophrenia, can be found in disruptions to healthy foetal development. Disruptions to the mother-infant relationship can also increase the risk of psychiatric disorders in later life (Box 1). This has contributed to an increased focus on the wellbeing of women during pregnancy and over the first year postnatally to prevent the intergenerational transmission of mental disorders.

Factors before birth that can influence childhood outcomes

Genes

- Schizophrenia
- Bipolar Disorder
- Common mental health disorders

Influences on the intrauterine environment

- Vitamin D deficiency
- Famine
- Influenza
 - Schizophrenia
 - Bipolar

Maternal illness

Medications

Lifestyle

- Substance misuse
- Smoking
- Diet

Schizophrenia and, to an extent, bipolar disorder, are, in part, neurodevelopmental in origin and we now know that disturbances to foetal development can increase the risk of the offspring developing these psychiatric disorders in later life (see (Boyce et al., 2018)). Epidemiological studies have

demonstrated that there is an increased risk of developing schizophrenia in the offspring of women who:

- Experienced famine (severe nutritional deficiency, with lack of micronutrients) during pregnancy.
- Delivered their infants in winter or spring. It is suggested that the women may have experienced vitamin D deficiency during pregnancy.
- Had influenza during the second trimester of pregnancy; this also increases the risk of the offspring developing bipolar disorder.

We now recognise that maternal mental illness during pregnancy can also have an effect on foetal development (Stein et al., 2014) that puts their offspring at risk of:

- Internalising and externalising disorders in childhood
- ADHD
- Anxiety and/or depression in adulthood

Medications used to treat maternal psychiatric disorders that pass through the placenta, can also have adverse effects on the foetus:

- There is a *small* increased risk of congenital abnormalities with the antidepressants (they are considered to be safe to use after a detailed risk benefit assessment) (Grigoriadis et al., 2013)
- Sodium Valproate carries a high risk of congenital abnormalities (it also affects neurodevelopment and can lead to children having significant cognitive impairment) (Meador et al., 2012).

Lifestyle choices such as substance misuse and poor diet, of the pregnant woman can also affect the outcome. There is increasing evidence now about maternal diet and the risk of the child developing depression and anxiety in later life (Jacka et al., 2013).

The Stress of Becoming a Mother

Becoming a mother is generally a positive experience but it is also stressful (see Table 24.1) with a mix of biological, psychological and social stresses. Biological stresses include the physical changes, and symptoms, related with pregnancy (nausea, vomiting, constipation, dizziness, breast and other body changes, etc.) along with hormonal changes (primarily B-HCG, progesterone and estrogen) that significantly affect a pregnant woman's daily routine. The growing uterus, later in

pregnancy, brings about pressure effects in the abdomen and impairs venous return to the heart in the supine position disrupting the sleep.

Table 24.1. The stresses of becoming a mother.

Biological stress	Physical changes Hormonal changes
Psychosocial stress	Role change to ‘mother’ Loss of working role Relationship changes 2 to 3 person relationship
Socioenvironmental stress	Financial pressure Change in social networks
Intrapsychic stress	Coping with dependence Own parenting experiences

Psychological changes include the role change from being a ‘woman’ to becoming a ‘mother’; a challenging and demanding role, both physically and emotionally. She might be taking the full-time mother’s role from being a working and probably, a financially independent, woman. The relationship with her partner changes from them being a dyad (or couple) to a triad (family) with the infant coming into it; changing the entire focus of her relationships. This can impact on her relationship with partner and other family members.

In addition, there are socio-environmental stresses, especially the financial pressure following maternity leave (if it is available) or job loss. There is obvious additional financial pressure associated with raising a child. Motherhood brings about changes in social networks, for example the one associated generally around work, and close friendships to new social networks comprising of other mothers and childcare.

Intra-psychic stresses for the new mother relate to her having to cope with her newborn’s dependence on her and the associated loss of freedom (this can be a particular issue among adolescent new mothers). This is strongly influenced by their childhood experiences with their own parents. How their mother related to them in childhood can significantly affect their parenting skills and ability to cope with the newborn’s emotional needs.

Perinatal Mood Disorders

There are four different mood disorders (see Chapter 16 and 17) specifically related with perinatal period as shown in Table 24.2. Each disorder is expanded upon below.

The ‘Blues’

The Blues affect at least 70% of women and it is most likely a stress response related to the demands of labour and the sudden changes in the hormonal profile. The blues generally emerge on the 3rd to 5th day postpartum. The common symptoms are lability of mood, with a mix of anxiety and depressive symptoms, tearfulness, irritability and emotionality. It is a benign and transient condition and does not require any treatment as it spontaneously resolves after a day or two. It is important to be aware of this condition to avoid over-diagnosing postnatal depression which is a much more serious condition. In some women, the blues may precede a postpartum psychosis or postnatal depression.

Postnatal Depression

Postnatal depression is defined as an episode of major depression that develops within the first six months after childbirth with a similar clinical picture of depression at other times in the life cycle. It is not an official diagnosis in the DSM or ICD; however, DSM-5-TR allows for a ‘peripartum’ specifier for disorders that have their onset during pregnancy or in the first 4 weeks postpartum (American Psychiatric Association, 2022).

Many women suffering from an episode of major depression in the first year after childbirth have their onset outside of this 4 week window, hence the term postnatal depression is used for such episodes of depression. Up to 10-15% of women will experience postnatal depression.

Postnatal depression was originally thought to be a unique form of depression because of its close association with childbirth when it was considered to be caused by the changes in hormonal levels following parturition; however, we now recognise that there is nothing unique about this form of depression as the symptom pattern is no different from major depressive disorder and any link to hormonal changes only applies to a few women with postnatal depression (O'Hara and McCabe, 2013). The best way to conceptualise postnatal depression is to use a stress-diathesis model, in which the stress of becoming a new mother triggers depression among women with an underlying vulnerability (risk factors).

Table 24.2. Perinatal mood disorders.

	The blues	Puerperal psychosis	Antenatal depression	Postnatal depression
Prevalence	~70%	0.1%	~10%	10-15%
Time of onset Following childbirth	3-5 days	Within 3 weeks	Any time during pregnancy	Gradual onset over first 3-6 months
Symptom profile	Anxiety Depression Tearfulness Lability of mood Irritability emotionality	Psychotic symptoms Manic presentation Melancholic presentation Indecisiveness confusion	Symptoms of major depressive disorder Low mood Fatigue worry	Symptoms of major depressive disorder + anxiety
Course	Benign/ transient	Severe May require hospital admission Good remission with treatment	Variable	Gradual onset Possible chronicity
Causal factors	Hormonal Stress response	Biological/genetic oestrogen sensitivity	Relapse of pre-existing depression Stress Sleep disturbance Interpersonal difficulties	Psychosocial Poor relationship Poor support Dysfunctional personality style

Risk Factors for Postnatal Depression

The risk factors for postnatal depression are predominantly psychosocial (Howard et al., 2014; Boyce and Hickey, 2005; O'Hara and McCabe, 2013) (see Table 24.3), however genetic factors play a part and, for some women, biological factors, in particular oestrogen sensitivity, may be the major contributing factor to the onset of depression. While there is a genetic component to postnatal depression, no specific gene or SNP has been identified for the disorder as yet, but there is a familial component to postnatal depression and postnatal depression runs in families.

Increased rates of adverse early life experiences have been found to increase the risk of women developing postnatal depression. Such experiences include trauma, neglect, or physical, sexual or emotional abuse. Women who have experienced poor parenting, characterised by overcontrolling or critical and uncaring parents, also have an increased risk of developing postnatal depression.

Girls under the age of 16 have a heightened risk of developing postnatal depression, although this risk may be associated with the factors that lead to them becoming pregnant at a young age. Increased risk of postnatal depression is found among women who have previously experienced episodes of depression and/or anxiety and among women with a vulnerable personality style characterised by being overly sensitive, anxious, perfectionistic or a worrier. While postnatal depression can affect women of all strata of society, higher rates are found among socially disadvantaged women and women in developing nations.

Table 24.3 Risk factors for postnatal depression.

Genetic endowment	
Early life experiences	Trauma
	Neglect and abuse
	Poor parenting
Sociodemographic	Young age
	Social disadvantage
Social environment	Dysfunctional intimate relationship
	Poor instrumental or emotional support
	Poor social support network
	Life event stress
‘Internal environment’	Oestrogen sensitivity
	Personality style
	Anxious, timid, unstable
	Previous history of depression and/or anxiety

There is a well-known saying “it takes a village to raise a child”; a woman’s social network, and the amount of support she receives, is critically important in the perinatal period and women who have poor social network or lack social support are at a heightened risk of developing postnatal depression. While the broader social network is important, a dysfunctional intimate relationship is one of the strongest risk factors for postnatal depression (Boyce and Hickey, 2005; Boyce et al., 1991). Women with partners who provide poor instrumental (helping in practical ways around the house and with attending to the baby), or emotional support (listening

to and being empathic) in the first year after the baby is born, are at high risk of developing postnatal depression. Additional life events stress, such as bereavement during pregnancy, financial stress, or the stress of moving home (a relatively common experience among new mothers) can also contribute to postnatal depression.

Impact of Postnatal Depression

The critical importance of postnatal depression is the impact it has on the developing infant (Stein et al., 2014). Women with postnatal depression:

- May have a poor interaction with their infants, unresponsive or intrusive
- May be insensitive and unresponsive to infant cues
- May not provide a rich environment for the infant to explore to enhance cognitive development

As a consequence, these infants may have attachment problems (attachment insecurity) which poses a risk for subsequent mental health problems later in life.

The impoverished environment with an unresponsive mother may lead to the infant having cognitive difficulties (identifiable by 1 year of age) and behavioural difficulties identifiable by the time they are four years old. These attachment, cognitive and behavioural problems seen in the children of mothers with postnatal depression are known to be the precursors of mental health problems and disorders in later life. This highlights the importance of treating women with postnatal depression to prevent such difficulties arising in the next generation. A critical issue here is to identify women with postnatal depression because they often are unaware that they are depressed and hence, do not seek treatment. To overcome this, screening tools have been developed to identify possible perinatal depression (see below).

Depression in Pregnancy

Postnatal depression is the most well recognised mood disorder associated with childbirth and little attention was paid to women during pregnancy until the last 20 or so years. Depression during pregnancy is as common as postnatal depression (whether it is a *de novo* episode of depression or a pre-existing major depressive episode) (Bennett et al., 2004). The symptoms of depression in pregnancy are the same as major depressive disorder, but in assessing depression in pregnancy, care needs to be taken in eliciting symptoms. Some depressive symptoms may in fact be due to the women being pregnant rather than due to depression (such

as fatigue, loss of energy, poor sleep, appetite change and loss of libido). Women who are depressed in pregnancy are at higher risk of having a:

- preterm delivery,
- low birth weight baby,
- small for gestational age baby and
- increased risk of gestational hypertension.

The importance in recognising and treating depression during pregnancy is that it can have an adverse impact on the developing foetus. The offspring of mothers with depression during pregnancy have a higher risk of:

- Emotional and behavioural problems as children
- Developing depression in later life

The mechanism for this is not fully understood. However, there are speculations that depression in pregnancy can lead to over-arousal of the foetus's HPA axis (Chaudron, 2013), either directly by increasing the amount of glucocorticoids produced in the placenta, or indirectly by activation of the HPA axis leading to placental hypersecretion of corticotrophin releasing factor.

Screening for Perinatal Depression

Many women with perinatal depression (depression during pregnancy or postnatal depression) do not seek treatment, or even consider themselves to be unwell. As a consequence, many women are not identified as being depressed (or having anxiety) and so are not treated. This is a matter of significant concern because of the potential adverse consequences of perinatal depression on the developing foetus and infant. Screening programs have now been implemented to identify women at risk of having depression (Buist et al., 2002; Austin and Marce Society Position Statement Advisory, 2014). The major screening tool is the Edinburgh Postnatal Depression Scale (EPDS) (Cox et al., 1987). This is a 10 item self-report scale scored from 0-3 for each item. A score above 12 on this scale indicates a high probability of the woman meeting DSM-5-TR criteria for major depressive disorder (American Psychiatric Association, 2022). A detailed clinical assessment must follow to confirm the diagnosis.

There is a lot of debate about using screening tools, particularly regarding their psychometric properties (validity, sensitivity and specificity). There are two important issues related to perinatal screening. First, does the screening process lead to better outcomes for women identified as being depressed? Screening, without

having the appropriate referral pathways (for full assessment and treatment) in place, achieves nothing. A second concern relates to the instrument's sensitivity and specificity, especially identifying 'false' positives. In other words, women who score high may not meet criteria for major depression (leading to costly interventions) and women with major depression who score low (false negatives) will miss out on necessary treatment.

Treatment of Perinatal Depression

The treatment of perinatal depression is essentially no different from the treatment of depression at any other time in the life cycle, except that the developing foetus and the nursing infant always need to be kept in mind. Management strategies include lifestyle interventions (sleep hygiene, healthy diet, alcohol and tobacco cessation and abstaining from illicit drugs), psychoeducation, supportive therapy, evidence-based psychotherapies and antidepressant medications.

The first step in the management is to identify that the disorder is present. At least half of the women with perinatal depression do not have it recognised by health professionals and won't recognise that they are depressed (the reason these screening questionnaires were introduced).

Once the depression is recognized, a comprehensive biopsychosocial and lifestyle assessment should take place. The specific risk factors to the woman's depression should be identified and formulated so that treatment can be planned to target specific risk factors.

Psychoeducation about postnatal depression should be provided, explaining what perinatal depression is and how it can affect women. The key messages, in addition to explaining about depression, are that many women experience perinatal depression ("you are not alone") and many will feel ashamed about this, but there is nothing shameful about depression, it is an illness that can be treated. She will need to be told that becoming a mother is stressful and 'mothering' is a demanding role and not something that women naturally do (it has to be learned). The risk factors that have led to her depression should be explained to her along with the strategies that can be put in place to deal with them. Linking women with support groups such as the Perinatal Anxiety and Depression Association (PANDA) or Beyond Blue, either on-line or in person, will help them find new social support networks.

She should be given permission to talk about her stressors. For example, she should be allowed to talk about how motherhood is not what she expected, or that her baby won't settle at night, and she is exhausted, or that her partner is too preoccupied with his work and is not helping her with the baby. Careful, non-

judgemental active listening to the woman and allowing her to develop strategies may be a sufficient intervention for many women.

Treatment of Postnatal Depression

1. Recognition
2. Clarification of risk factors
3. Permission to talk
4. Psychoeducation
 - Prioritizing activities
5. Non-directive counselling
6. Specific treatments
 - CBT
 - IPT
 - Antidepressant medications
 - *Safety with breastfeeding*

Prioritizing her daily activities so that she can focus on what she needs to do (look after her infant) rather than spending time on things that may not be a priority (making sure the home is spotless) and encouraging her to take time for self-care will reduce her sense of being overwhelmed (Parker et al., 2014).

Table 24.4. Risks and Benefits on taking medication over the perinatal period.

	Not taking medication		Taking medication	
	Benefits	Risks	Benefits	Risks
Mother	No adverse effects of medication	Persistent symptoms or relapse	Maternal wellbeing	Adverse effects of medication
Foetus	No risk of harm from medication exposure	Impact of maternal illness on foetal development	Reduced risk of impact of maternal relapse	Risk of foetal exposure to medication

Some women may require more specific psychological treatments such as Cognitive Behaviour Therapy (CBT; see Chapter 33) or Interpersonal Psychotherapy (IPT) (Dennis and Hodnett, 2009). Some women who do not respond to non-pharmacological interventions or have a moderate to severe perinatal depression, will require antidepressant medications (in conjunction with psychological therapy) (Chisolm and Payne, 2016). There is an absence of evidence concerning the efficacy of antidepressants among women over the perinatal period (pregnant women and women not on contraception are typically excluded from participating in clinical trials), but there is no reason why they should not be efficacious for perinatal women. Electroconvulsive therapy (ECT; see Chapter 36) is a safe option for pregnant women with severe postpartum depression and psychotic depression, but careful monitoring of the foetus is required.

Table 24.5. Psychotropic medications in pregnancy and breastfeeding.

	Pregnancy	Breastfeeding
SSRI	Probably safe Moderate to severe depression Poor Neonatal Adaptation Syndrome (PNAS)	Low passage through breastmilk
SNRI	Probably safe Moderate to severe depression PNAS	Low passage through breastmilk
SGAs	Weight gain and metabolic syndrome Avoid starting clozapine	Sedation
Lithium	Caution in pregnancy Careful monitoring	Not to breastfeed
Anticonvulsants	AVOID Valproate Lamotrigine – possibly safe	Valproate (safe) Contraception

A decision about using antidepressants in the perinatal period requires a risk benefit assessment as medications will cross the placenta exposing the foetus to the drug. There is a potential, but small teratogenic risks of the medications

(approximately 4% risk of congenital cardiac defect), recognising that the rate of congenital abnormalities in the population is around 3%. The newborn may go through a withdrawal syndrome called postnatal adaptation syndrome (PNAS) if the mother has been on an antidepressant during pregnancy (especially an SSRI or SNRI). The symptoms in the newborn are distress, fussiness, being jittery, abnormal crying, hypoactivity and difficulty feeding in the first few days of life. Antidepressants will pass through breastmilk exposing the breast feeding baby to the medication. The risks and benefits of women taking medication during pregnancy and while breastfeeding is shown in Table 24.4. These risks and benefits apply for the treatment of all psychiatric disorders over the perinatal period.

Perinatal Anxiety Disorders

While considerable attention has focused on perinatal depression, anxiety disorders are equally as prevalent over the perinatal period (Dennis et al., 2017). The origins of the anxiety disorders (panic disorder, social anxiety disorder and generalised anxiety disorder) are, like those for perinatal depression, predominately psychosocial. Anxiety over the course of pregnancy, like depression, can have an adverse impact on the developing foetus, with increased risks of behavioural and emotional disorders in the offspring (Stein et al., 2014). While there are concerns about the effect of anxiety on the developing foetus, little attention has been paid to screening for anxiety during pregnancy, but the Edinburgh Postnatal Depression Scale does identify anxiety symptoms, and so if a high score is returned, asking about anxiety symptoms as well as depressive symptoms is essential. The same applies postpartum; anxiety symptoms are common but women often do not disclose them and they have to be specifically asked. This is particularly important where women have panic attacks, and these can often be related to caring for her newborn baby. In addition to the common anxiety disorders, many women will have pregnancy-specific anxiety, related to the wellbeing of her foetus, foetal development and how she will be able to manage when she becomes a mother.

The management of perinatal anxiety disorders follows the same approach as for the management of anxiety disorders at other times in the life-cycle (Wilkins et al., 2019). Focused psychological treatments, in particular CBT, should be considered as the first line of treatment as they are safe and effective. However, some women may not respond to, or have the time to engage in, psychological treatments. In such cases, treatment with an SSRI will be required, as it will for women with severe and disabling anxiety symptoms. It is important to note the same caveats about prescribing medication over the perinatal period apply for women with anxiety disorders as it does for perinatal depression.

Postpartum Psychosis

There is a close association between childbirth and the onset of a new psychotic illness among women that have shown no sign of mental illness beforehand. One to two women out of 1000 deliveries will suffer from this (Kendell et al., 1976). Postpartum psychosis (previously called puerperal psychosis) is one of the most interesting disorders in psychiatry. Its incidence has been consistent over time, with papers published in the early 1800s reporting the same rate as we see now with the same unique clinical picture (Boyce and Barriball, 2010; Bergink et al., 2015a). Bipolar disorder has a high risk of relapse following childbirth as do women that have had a prior episode of postpartum psychosis (Wesseloo et al., 2016).

Postpartum psychosis is not officially recognised as a unique disorder (it had been until the 1960s). However, it does seem to be related to bipolar disorder as many women who had a postpartum psychosis, are subsequently being diagnosed with bipolar disorder (Platz and Kendell, 1988; Munk-Olsen et al., 2012). Using the current DSM-5-TR criteria, women with a postpartum psychosis will be diagnosed with bipolar disorder with a peripartum onset (American Psychiatric Association, 2022). It generally has its onset within three weeks following childbirth and will present with a mix of mood and psychotic symptoms along with perplexity, confusion and catatonic symptoms.

Mood symptoms:

- Depression: often with a melancholic presentation with agitation, retardation or psychosis
- Manic symptoms: with grandiosity, increased energy, reduced need for sleep, pressured speech, impulsivity and disorganisation.
- Mixed features: Women can present with a mix of depressive and manic symptoms.

Psychotic symptoms:

- Delusions: These delusions are often mood congruent and related to the baby. For example, the baby is the reincarnation of God/Satan. However, paranoid delusions, and bizarre delusions can also be present.
- Hallucinations: These can also be mood congruent. For example, God is speaking to her or hearing derogatory voices telling her she is a bad mother.
- Disorder of thought form

Other symptoms:

- Perplexity and/or confusion: Here, she may appear perplexed or confused as if she had a delirium, but the perplexity is a feature of her psychosis (she is internally preoccupied) and she is oriented to time, place and person.
- Catatonic symptoms: She may present with catatonic symptoms, such as posturing or staring off into space.

Postpartum psychosis is a psychiatric emergency because these women are at high risk of harming their infant and/or themselves. Women that kill their newborn baby (neonaticide) will often be suffering from postpartum psychosis. Women are also at risk of suicide, especially if they have a psychotic depression when they believe the baby would “be better off without me”.

Management of postpartum psychosis

1. Psychiatric emergency
 - Risk of harm to infant
 - Infanticide / Neonaticide
2. Admission to a mother-baby unit
 - Support for partner
 - Support for mother-infant relationship
3. Treat underlying pathology
 - Antipsychotics – first generation
 - Antidepressants
 - Mood stabilizers
 - Breastfeeding contraindicated for lithium
 - Benzodiazepines for catatonic features
4. ECT

They require an admission, ideally into a mother-and-baby unit (if one is available), so that she won't be separated from her baby. If she is admitted to a general psychiatric bed, the baby has to be nursed elsewhere. This condition needs an aggressive management with the primary goal of reducing the symptoms and achieving remission. We also want to ensure that there will be minimal separation between the mother and the baby while taking care for the infant's safety. A long separation between the mother and her infant, may lead to a poor mother-infant relationship and less secure attachment.

Generally, women with postpartum psychosis will need an involuntary admission under the Mental Health Act, as they lack insight into their illness and are likely to be non-compliant with treatment. The primary goal of treatment is to bring the acute symptoms under control, with antipsychotic medication for psychotic symptoms (delusions and hallucinations) and manic excitement (Bergink et al., 2015b). Depressive symptoms require antidepressant medication, and if there are psychotic symptoms or marked agitation, antipsychotic medication as well. As this disorder is closely aligned with the bipolar disorder, lithium is an important treatment as a mood stabilizer. However, if lithium is used, the woman is not able to breastfeed because lithium passes freely through the breast milk. Benzodiazepines can be used as first-line treatment especially in the presence of catatonic features (Bergink et al., 2015b).

ECT is a highly effective treatment for postpartum psychosis, especially for those women with a delusional/agitated depression. ECT can bring about resolution of symptoms promptly reducing the duration of separation of the mother and her infant. The partners should be supported as well, as they may be struggling with their own difficulties related to new parenthood and their partner's sudden and acute mental illness.

The aetiology of postpartum psychosis is not yet clear (Jones et al., 2014). Underpinning the disorder is a genetic predisposition, although no specific gene(s) have yet been identified (Jones and Craddock, 2001). There are three possible triggers for the disorder, over and above the genetic predisposition:

- A neuroendocrine hypothesis that suggests that some women have oestrogen sensitivity and when oestrogen levels change rapidly following childbirth, this has an effect on dopamine receptors triggering the psychosis.
- A circadian rhythm hypothesis. Disruption to circadian rhythms can trigger relapse in individuals with bipolar disorder. Childbirth often leads to loss of sleep and this is a common trigger for the onset of a postpartum psychosis.
- Another possible trigger for the onset of psychosis is an inflammatory process. There is a build-up of pro-inflammatory cytokines during pregnancy and parturition can lead to oxidative stress triggering episodes of bipolar disorder/psychosis.

Postpartum Relapse and Bipolar Disorder

Women who have had an episode of postpartum psychosis are at risk of having a relapse with subsequent pregnancies, as are women with a bipolar disorder (Wesseloo et al., 2016).

- Risk of relapse with bipolar disorder: 38%
- Risk of relapse for postpartum psychosis: 31%

The risk of relapse is higher among women that do not take any medication (66%), but if they take prophylactic medication (predominately lithium), the risk of relapse is reduced to 23%. The use of prophylactic medications in preventing relapse in bipolar disorder can be crucial. However, there should be careful discussion with the woman and her partner about the risks and benefits of using and avoiding these medications both for the mother and the baby.

Mood Stabilizers in Pregnancy

Lithium

Lithium is one of the most commonly used mood stabilizers and is a gold standard for treating bipolar disorder. However, infants exposed to lithium in utero early in pregnancy have an increased risk of developing cardiac abnormalities, specifically Ebstein's anomaly. If a woman is taking lithium during pregnancy, appropriate and regular scans must be done to rule out a cardiac abnormality in utero (Galbally et al., 2010). Lithium has a narrow therapeutic index and the serum concentration can go to toxic levels relatively easily. There are considerable hemodynamic changes happening especially towards the latter half of pregnancy warranting frequent blood monitoring and lithium dose adjustments. A safe strategy is to reduce the dose of lithium closer to birth to avoid dehydration-related toxicity in the mother and toxicity in the newborn. The dose can be increased again as soon as the baby is born. It is important to note that Lithium is secreted in breast milk in considerable amounts and it is not safe to breastfeed while being on lithium.

Sodium Valproate

Sodium valproate is a widely-used anticonvulsant mood stabilizer. However, its use is NOT recommended for women of childbearing age as it is a potent teratogen with at least 17% of exposed foetuses having a congenital abnormality, specifically spinal tube defects. It also has an effect on the developing

brain, with infants exposed to sodium valproate in utero having an IQ at least one standard deviation below other infants.

Preconception Counselling

The use of mood stabilizers and antipsychotics during pregnancy and breastfeeding requires careful planning, and women with bipolar disorder require preconception counselling to plan out how to manage their pregnancy and the postpartum period. Three important points need to be made about this:

1. Women should be informed about this at the time of diagnosis of bipolar disorder, so that they do not have an unplanned pregnancy. There should be a discussion about contraception at the time of diagnosis.
2. Women should be advised not to stop medication abruptly should they conceive.
3. There needs to be a detailed discussion about the risks and benefits of taking a mood stabilizer over the course of the pregnancy, and in the postpartum period.

Schizophrenia

Little attention had been paid to women with schizophrenia (see Chapter 15) and their reproductive life until recently. There are a number of reasons for this. Women with schizophrenia had interpersonal difficulties and tended not to form intimate relationships that could contribute to them not having children. Their treatment with the first-generation antipsychotics that raise prolactin levels may also have contributed to them failing to conceive. Furthermore, women with schizophrenia were thought to have low fecundity. We now know that women with schizophrenia do not have low fecundity and, with the second-generation antipsychotics, infertility is no longer a major side effect of the medication.

A major issue concerning women with schizophrenia is their capacity to be able to effectively care for, and look after, a newborn baby. Two important factors contribute to this:

- the presence of positive symptoms (such as, being distracted by hallucinations, preoccupied by delusional thinking) and disorganisation can interfere with their ability to look after the baby.
- Cognitive impairment can make it difficult for them to be able to structure their life to look after the baby.

It is important, in the management of women with schizophrenia, to have a discussion with them about contraception (this is a population at high risk of exploitation by others and at risk of having an unplanned or unwanted pregnancy), and the potential risks of medication on the developing foetus as part of their psychoeducation at the time of diagnosis.

Should they do plan to have a baby, they should undergo effective preconception counselling to determine the capacity and capability of looking after a baby. Such preconception counselling will need to review her medications to ensure that they are safe during pregnancy and if they wish to breastfeed. There will also need to be extensive planning about how she will be able to manage her baby and ensure that she has adequate support. An unfortunate aspect about women with schizophrenia is that up to 40 to 50% of them will have their infants removed by social services because they are unable to adequately care for the baby.

Further Reading

- Austin, M.P., Highet, N., & the Expert Working Group (2017) *Mental Health Care in the Perinatal Period: Australian Clinical Practice Guideline*. Melbourne: Centre of Perinatal Excellence.
- Bergink, V., Rasgon, N., & Wisner, K. L. (2016). Postpartum psychosis: madness, mania, and melancholia in motherhood. *American Journal of Psychiatry*, 173(12), 1179-1188.
- Boyce, P., & Barriball, E. (2010). Puerperal psychosis. *Archives of Women's Mental Health*, 13, 45-47.
- Boyce, P., & Buist, A. (2016). Management of bipolar disorder over the perinatal period. *Australian Family Physician*, 45(12), 890-893.
- Dennis, C. L. (2014). Psychosocial interventions for the treatment of perinatal depression. *Best Practice & Research Clinical Obstetrics & Gynaecology*, 28(1), 97-111.
- Galbally, M., Snellen, M., Walker, S., & Permezel, M. (2010). Management of antipsychotic and mood stabilizer medication in pregnancy: recommendations for antenatal care. *Australian & New Zealand Journal of Psychiatry*, 44(2), 99-108.
- Howard, L. M., Molyneaux, E., Dennis, C. L., Rochat, T., Stein, A., & Milgrom, J. (2014). Non-psychotic mental disorders in the perinatal period. *The Lancet*, 384(9956), 1775-1788.
- Stein, A., Pearson, R. M., Goodman, S. H., Rapa, E., Rahman, A., McCallum, M., ... & Pariante, C. M. (2014). Effects of perinatal mental disorders on the fetus and child. *The Lancet*, 384(9956), 1800-1819.

Chapter 25: Child and Adolescent Psychiatry

Philip Hazell

Specialty of Psychiatry, The University of Sydney, Australia, and School of Medicine, Charles Darwin University, Australia.

Iain E. Perkes

School of Clinical Medicine, The University of New South Wales, Australia.

Jean Starling

Specialty of Psychiatry, The University of Sydney, Australia.

Angela Dixon

The Children's Hospital at Westmead, Australia

and

Zoe Quick

Formerly with The Children's Hospital at Westmead, Australia

Introduction

Child and adolescent psychiatry is concerned with mental health problems occurring in individuals from birth to between 16 and 25 years of age. The field may be further subdivided into perinatal and infant, child, and adolescent or youth psychiatry. Child and adolescent psychiatrists are trained to deal with patients across the age spectrum but may choose to focus on one or other of these subspecialty areas.

How Does Child and Adolescent Psychiatry Differ from General Adult Psychiatry?

Child and adolescent psychiatry is not miniaturised adult psychiatry. Child and adolescent psychiatry differs from general adult psychiatry in several important

ways. Child and adolescent psychiatry is especially dynamic because disorders are often expressed differently at different ages, and disorders evolve over time. Child and adolescent psychiatry is related to adult psychiatry in the same way embryology is related to gross anatomy. There is an opportunity to intervene early and positively influence the trajectory of patients who are maturing.

Classification of Mental Disorders Affecting Children and Adolescents

Psychiatric diagnoses are limited in young people by the lack of criterion validity (biological markers of illness), understanding of aetiology and pathophysiology. A lack of diagnostic stability is exaggerated in child and adolescent psychiatry. The same child (with the same brain!) may transition through enuresis, separation anxiety disorder, oppositional defiant disorder, substance use disorder and major depressive disorder.

Child and adolescent psychiatric disorders are diagnosed according to ICD-11 and DSM-5-TR. To use these classifications, note the following:

1. Problems are seen in the context of normal development, i.e. what is common for 1-year-olds can be maladaptive for 7-year-olds (e.g. nocturnal enuresis, separation anxiety).
2. Disorders are often about a failure to achieve developmental milestones. The difference therefore between normality and disorder is less clear-cut than in adult psychiatry.
3. Symptoms may be diagnosable because of their intensity or persistence; thus temper tantrums are common in pre-schoolers, but deemed problematic only if they occur frequently, are of long duration and lead to functional impairment.

Table 25.1. Mental disorders that can affect children and adolescents.

Conditions restricted to childhood	Elimination Disorders (enuresis, encopresis)
	Separation Anxiety Disorder
	Selective Mutism
Conditions that have their onset in childhood but may affect adult adjustment	Intellectual Disabilities
	Communication Disorders
	Autism Spectrum Disorder
	Attention-Deficit/Hyperactivity Disorder
	Specific learning Disorder
	Motor Disorders (coordination, stereotyped movements)
	Disruptive Mood Dysregulation Disorder
	Tic Disorders
	Gender Dysphoria
	Oppositional Defiant Disorder
	Conduct Disorder
	Intermittent Explosive Disorder
Conditions that can recur across the lifespan	Depressive Disorders
	Anxiety Disorders (other than separation, mutism)
	Obsessive-Compulsive and Related Disorders
	Trauma-and Stressor Related Disorders
	Dissociative Disorders
	Somatic Symptom and Related disorders
	Anorexia Nervosa
	Sleep-Wake Disorders
	Pyromania
	Delirium
Conditions usually associated with adulthood but which may have their onset in childhood or adolescence	Schizophrenia Spectrum and Other Psychotic Disorders
	Bipolar and Related Disorders
	Bulimia and Binge-Eating Disorders
	Sexual Dysfunctions
	Substance-Related and Addictive Disorders
	Dementia
	Personality Disorder
	Paraphilic Disorders

Many of the mental disorders described in this chapter will be familiar to you from previous chapters in this book. Other conditions are either only seen early in life, or are described differently in children than they are in adults. An example

of the latter is conduct disorder, which if persistent in adulthood is called antisocial personality disorder. One way of grouping mental health problems of childhood is to think about: 1) conditions restricted to childhood such as selective mutism, 2) conditions that have their onset in childhood but may affect adult adjustment, such as attention deficit hyperactivity disorder, 3) conditions that can recur across the lifespan such as anxiety and 4) conditions usually associated with adulthood but which may have their onset in childhood or adolescence, such as bipolar disorder. Table 25.1 lists the mental disorders that can affect children and adolescents according to this grouping. Cross-sectionally a very simple way to organise mental disorders affecting children and adolescents is into Internalising and Externalising problems.

The approach has scientific validity as factor analysis of common symptom items has shown that problems do cluster roughly into these two categories. Internalising problems refer to emotional symptoms such as worry or sadness while Externalising problems refer to behavioural symptoms such as aggression or hyperactivity. Later in the chapter, when we discuss a few disorders in more detail, we have grouped them according to the Internalising/Externalising dichotomy. Note, however, that it is common for children with Externalising problems to also experience Internalising symptoms. In addition, this simple classification does not work so well for rare symptoms such as odd and eccentric beliefs or dissociation.

Epidemiology of Mental Disorders Affecting Children and Adolescents

The prevalence rates of common psychiatric conditions in major epidemiological studies are summarised in Table 25.2. Rates vary from study to study owing to the diagnostic criteria and research instruments used, as well as characteristics of the population surveyed. Internalising problems are more common in girls, while Externalising problems are more common in boys. Some conditions such as attention-deficit/hyperactivity disorder (ADHD) occur less frequently with increasing age, in others such as depression, prevalence goes up with age. The recent Australian survey made a distinction between mild, moderate and severe problems based on impairment and suicidality. About 5.5% of those surveyed fell into the moderate or severe range of impairment, and represent the group most likely to need specialist child and adolescent mental health care. Children in out of home care, juvenile detention and immigration detention have higher rates of mental health problems than the general population, as do Aboriginal and Torres Strait Islander Australian children, and children with chronic medical illnesses or intellectual disability. Offspring of parents with a mental disorder also have higher rates of problems. Rates of depression and conduct problems are rising in the population, while rates of ADHD remain stable.

Impact of Mental Disorders Affecting Children and Adolescents

Over the lifespan serious mental illnesses cause a high burden of disease with reduced life expectancy of more than ten years and severely impaired social and economic functioning (Lothgren, 2004). In 2010 mental illnesses were responsible for 7.4% of DALYs (disability-adjusted life years) and 22.9% of YLDs (years lived with disability) worldwide. This particularly affects the young, with the highest total proportion of DALYs in those aged between 10 and 29 years (Whiteford et al., 2013). The top five ranked causes of YLDs worldwide in 10-14 year olds include unipolar depressive disorders and anxiety disorders. In 15-19 year olds alcohol use disorders are added to the list, becoming the number two cause of YLDs in 15-19 year old males. In total mental illnesses are responsible for nearly 50% of YLDs in adolescents 10-19 years. When adolescents themselves are surveyed they rate mental health problems as their highest concern (World Health Organisation, 2012).

Mental illnesses greatly increase the risk of long-term disability not only because of severity but also because these disorders tend to present early in life - of those with mental health problems at 26, half met criteria for diagnosis at 15 and 75% by their late teens (Kim-Cohen et al., 2003). Both emotional and behavioural disorders lead to poor self-esteem, peer difficulties and reduced academic performance, as well as reducing family wellbeing, with increasing severity and number of diagnoses increasing the risk of difficulty (Sawyer et al., 2000). Young people with mental illness are at risk of dropping out of school, becoming involved with delinquent peers, and family breakdown. In addition, in later life severe mental illness reduces life expectancy because of the higher risk of chronic physical ill health (Fagiolini et al., 2008; Heald, 2010).

Mental illnesses are also a major cause of mortality in the younger age groups, as the suicide rate in mental illness is at least nine times higher than the general population (Inskip et al., 1998). Worldwide, suicide was one of the five leading causes of death in adolescents in 2012 (World Health Organisation, 2012). In developed countries including Australia and the United States of America suicide is one of the three leading causes of death in adolescents and young adults (United States Census Bureau, 2016).

Table 25.2. Prevalence of common child psychiatric conditions in major epidemiological studies.

Study	Sample	Measures	Prevalence
Second Australian Child and Adolescent Survey of Mental Health and Wellbeing (Australia, 2015)	5500 participants aged 4 to 17	Strength and Difficulties Questionnaire; Diagnostic Interview Schedule for Children, version IV (DISC-IV; some modules)	Any mental health problem (last 12 months): 13.9% Severe mental health problem (last 12 months): 2.1% DSM-IV diagnoses: anxiety disorders 6.9%; depressive disorders 2.8%; CD 2.1%; ADHD 7.4%
Great Smoky Mountain Study of Youth (US, 1993–2000)	1420 children from North Carolina aged 9 to 13 assessed annually to age 16	Child and Adolescent Psychiatric Assessment (CAPA)	DSM-IV diagnoses (3-month prevalence): any diagnosis 13.3%; anxiety disorders 2.4%; depressive disorders 2.2%; CD 2.7%; ODD 2.7%; ADHD 0.9%
British Child and Adolescent Mental Health Survey (UK, 1999)	10438 participants aged 5 to 15	Development and Well-Being Assessment (DAWBA)	DSM-IV diagnoses (current): any diagnosis 9.5%; anxiety disorders 3.8%; depressive disorders 0.9%; CD 1.5%; ODD 2.3%; ADHD 2.2%
Puerto Rico (US, 2000)	1886 children aged 4 to 17	Diagnostic Interview Schedule for Children, version IV (DISC-IV)	DSM-IV diagnoses (last year): any diagnosis 16.4%; anxiety disorders 6.9%; depressive disorders 3.4%; CD 2.0%; ODD 5.5%; ADHD 8.0%
South-eastern Brazil (2000–01)	1251 children aged 7 to 14 attending school	Development and Well-Being Assessment (DAWBA)	DSM-IV diagnoses (current): any diagnosis 12.7%; anxiety disorders 5.2%; depressive disorders 1.0%; CD 2.2%; ODD 3.2%; ADHD 1.8%

Services to Treat Mental Disorders Affecting Children and Adolescents

Children and adolescents make up almost a quarter of the population (Australian Bureau of Statistics, 2015; United States Census Bureau, 2016), yet they access only 6% of mental health services and only one percent of mental health

inpatient beds (Morris et al., 2011). It would be nice to think that the low utilisation of specialist mental health resources by young people was a result of lower rates of morbidity compared with adults, but the data tell us otherwise. Some young people are receiving mental health care outside of the mental health system, but others are not receiving treatment at all. Mental health services for children and young people are organised through a network of agencies and facilities based on the Tier nomenclature developed by the National Health Service in the UK.

Tiers of mental health service

- **Tier 1** – Practitioners who are not mental health specialists e.g., GPs, teachers, social workers.
- **Tier 2** - Child and Adolescent Mental Health Services (CAMHS) specialists working in the community in a uni-disciplinary way e.g., psychologists funded through the Better Access scheme.
- **Tier 3** - Specialist multi-disciplinary teams working in the community or outpatient clinics to address severe, complex and persistent disorders.
- **Tier 4** – Services for young people with the most serious problems such as inpatient units, day units and highly specialised community teams.

Schools are in a good position to observe warning signs such as behavioural deterioration or academic withdrawal and refer to school counsellors who can triage to other appropriate services or assess in more depth, as needed. School services also serve as the most common point of referral to Child and Adolescent Mental Health Services (CAMHS). General practitioners are often the first port of call for families and may refer to more widely available services such as paediatricians, particularly for neurodevelopmental disorders such as autism spectrum disorders or attention deficit hyperactivity disorder. Other private practitioners such as clinical psychologists provide evidence-based therapies including cognitive behavioural therapy or family interventions for many mental disorders. Many non-government agencies also provide counselling or family support. Welfare services and juvenile justice provide care for young people whose mental health problems have become so severe that they live in out of home care or are involved in the forensic system.

In the absence of access, or knowledge about access to community-based mental health services children and adolescents with mental health issues may present to hospital emergency departments. This is unavoidable when there is a medical complication such as poisoning, a laceration or delirium, or when there is

imminent danger of suicide. The emergency department is not a good environment, however, to conduct a comprehensive biopsychosocial assessment of other issues. It is better, if possible, to divert the patient to an urgent (within 24 hours) review by a community Child and Adolescent Mental Health Service (CAMHS). Some health services have specialized mobile teams who will visit young people in their homes or other non-hospital settings to conduct an assessment and initiate or coordinate treatment.

Interviewing a Family

Interviewing family members is essential to the practice of child and adolescent psychiatry. It is rare to see children without their parents or carers, and the assessment of an adolescent is not complete until parents or carers have been involved in the consultation. Family involvement has several practical aspects associated with it. Parents possess the historical information about the child's disorder and development the clinician will need to accurately assess the problem. For a child under 16 years of age the parents have the legal power to initiate assessment and consent to treatment. In addition, parents typically provide financial and practical resources (such as transport) to enable the patient to access care. An advantage for young people who attend the clinic with other family is that they are not the sole focus of attention. Of course there are exceptions. We sometimes agree to see young people 16 years and older without parental knowledge if this is the only way to engage the young person in assessment. Typically, such referrals are brokered by a school counsellor.

Family interviewing attends to both content (the history provided) and process (observation and assessment of the interaction between family members). The emphasis can shift according to the nature of the presenting problem. When a problem seems to be reactive to circumstances within the family such as conflict, the interviewer will be interested in the role of the family in the genesis and maintenance of the problem. When a problem seems to be “hardwired”, such as autism spectrum disorder or intellectual disability, the focus shifts to psychoeducation and the capacity for the family to adapt to the situation. A task of the family assessment is to identify strengths or competencies in the family. This can be probed by asking how the family have responded to other challenges. Disorders or problems of other family members can be identified. This is relevant, because if a young person has a mental health problem there is a reasonable likelihood a parent or sibling may also be affected by mental illness. Sometimes it becomes clear the referred person, or “identified patient”, is not the most unwell member of the family system. Family interviewing affords the clinician the opportunity to observe parenting style in real time. Aspects include how well the parents have prepared the child for the appointment (including bring age-appropriate activities for younger children), and

how they respond to the child's affect and behaviour. Finally, the interview is an opportunity to engage family (particularly parents) in the treatment process.

The clinician starts by assuming the family cares about the child and has areas of competency in promoting child wellbeing. Even very disadvantaged and seemingly chaotic families may possess good fundamental child rearing skills. However, sometimes the assessment reveals significant deficits in caregiving (neglect) or harmful practices (abuse). Procedures and approaches for interviewing a family are not as well standardized as the psychiatric interview of the individual, nevertheless there are some common elements. The techniques have been derived from the family therapy movement (see treatment modalities below) that comprises many different schools. Each school has its own identifying characteristic or signature, but most schools of family therapy have more in common than they have differences. Opinions vary over whether all family members should be invited to the first appointment. Our default option is to invite all family members living in the same household, and any non-custodial parent who lives elsewhere. Parents may not wish to bring preschool aged children out of concern that they will be disruptive. They are reassured that the service likes and will accommodate young children at appointments. A specific circumstance where involvement of all family members is contraindicated is when there is documented or suspected abuse of one individual by another. Participation of the perpetrator may even be legally precluded through a Domestic or Apprehended Violence Order. Sometimes estranged parents are so conflicted it is better to see them separately.

The content of the family interview attends to, as a minimum, five key areas; family demographics, clinical symptomatology of the child, individual parent history, parent relationship history, and the history of family as a unit (Josephson, 2007) . The story of the family will typically link to the story of extended family who are not present at the interview. One technique for gathering and displaying complex family information is to draw a genogram (usually on a whiteboard) to which can be added salient information such as health and psychiatric status, personal characteristics, key dates, previous relationships, and the closeness or distance of existing relationships (Figure 25.1).

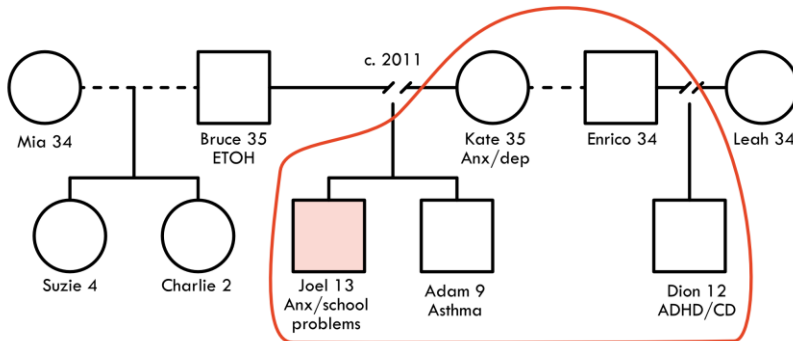


Figure 25.1. Example of a genogram. The genogram encompasses two generations. Joel, the referred patient, lives with his mother Kate, her new de facto partner Enrico, a younger brother, and Enrico's son from a previous relationship. Kate divorced Joel's father in 2011. Bruce has repartnered in a defacto relationship with Mia and they have two young daughters. Enrico is separated but not divorced from his first wife Leah. Various health problems are annotated.

Concurrently the clinician is attending to family processes, which can be grouped into the following four domains; structure, communication, family beliefs and family regulation of child development (Josephson, 2007). Specific examples are provided in Table 25.3. Below is a transcript of the first few minutes of a family interview. Adam (11 years of age) has been referred to a child and adolescent mental health clinic because of anxieties that have escalated since a change in family circumstances. There is a younger sibling with an intellectual disability. In this case, two clinicians have been assigned to the first assessment. Examine the transcript with reference to the process domains described in the box on the following page.

Clinician 1: Hello and welcome to the service. I am Philip, a child and adolescent psychiatrist.

Clinician 2: And I am Michael, a social worker.

Clinician 1: Would you like to introduce yourselves, maybe say a little about yourselves?

Mother: I am Anthea. I'm....34. Uh, I work part time, 3 days a week. As a research assistant.

Father: I'm Stewart..... I'm 32. I have a nursing background and I work as a pharmaceutical rep.

Child 1: I'm Adam

Mother: And this is our second son Bryce, he is 4 years old.

Clinician 1: Adam, what sort of things are you in to?

Child 1: I play soccer. Dad is....was....our coach. I like [insert a current popular computer game].

Clinician 1: And Adam, what is Bryce in to?

Child 2: Need a...need a....need a wee.

(Pause to see if anyone responds. No-one does)

Child 1: He, he likes it when we build a cubby house under the dining table

Clinician 1: And where does Bryce go when Mum is working?

Child 1: He....

Mother: Bryce goes to a preschool. A special preschool.

Father: Anthea needs time out. Bryce can be....

Mother: Hard going. Not that you....

Father: Say it

Mother: Well since you took the promotion...on the road a lot...both the boys....I don't know

Clinician 2: It sounds like there may have been some important changes for the family recently

Child 2: Need a...need a....need a wee.

(Pause to see if anyone responds. No-one does)

Clinician 2: So I wonder what you hoped would come from meeting with us today?

Mother: Adam has...a lot of worries. At night he thinks people might break into the house. Has to check, or actually get us to check, that doors and windows are locked. He gets bad dreams, can't sleep. Comes into our bed...

Father: Because you let him....

Mother: When Stewart is away. If Stewart is home he takes Adam back to his own room

Clinician 1: You have different um, attitudes, rules about kids co-sleeping....

Mother: And Adam gets very upset if Stewart doesn't phone in on the nights he is away from home...

Father: That was once...

Adam: I thought...I thought...

Mother: That Daddy had been in an accident

Clinician 1: Do Adam's worries spill over to other places....like school?

Mother: Adam likes school

Father: No, there he is just a normal kid...

Clinician 2: So I am curious. Stewart you took on a new work role recently...

Father: Two months ago

Clinician 2: Which has led to some changes in how things run in the family

Father: I guess

Clinician 2: I wonder what sort of conversation you both had [gestures to parents] about how things would work when Stewart took the new job

Mother: [looks away]

Father: ...we didn't

Child 2: Need a...need a....need a wee.

Table 25.3. Family processes (adapted from AACAP, 2007).

Domain	Example
Structure	
Adaptability	The family's behaviour as a group in response to the unfamiliar experience of the clinic visit. Do they for example bring toys or activities with them to engage younger family members?
Cohesion (connectedness v separateness)	On a continuum from close to the point of intrusiveness through to distanced to the point of disengagement. An intrusive family member may persistently answer questions on behalf of others, while a disengaged family member may be ignorant of important information relevant to other family members
Boundaries	A loose intergenerational boundary may occur when a parent shares intimate information about the relationship between the parents with a child
Communication	
Clarity	People may express clearly what they think and feel or alternatively the content may be disguised or suppressed
Emotional expression	On a continuum from exaggerated expression of emotion through to constricted or suppressed emotion.
Problem solving	How the family go about arrangements for future appointments if another child in the family has a conflicting commitment
Family beliefs	Shared and non-shared beliefs about matters such as the respective roles of mothers and fathers, and at what age children may be expected to leave home or to pay rent
Family regulation of child development	
Responsiveness	How attentive older family members are to the needs of the younger family members
Consistent	Children cope naturally with different rules and expectations according to the environment and context. Difficulties arise when parents are internally inconsistent (for example ignoring behaviour sometimes and punishing it harshly at others) or when parents undermine each other's strategies.
Degree of regulation	How the parents respond if a child becomes, for example ,giggly and overexcited

Interviewing an Adolescent

Adolescence is characterised by rapid change in several developmental domains. Sometimes described as an “in-between” age, interviewing an adolescent requires accommodating their evolving autonomy while still appreciating the limitations in their decision-making abilities. Adolescents vary as to what they will discuss with a professional and working with them effectively necessitates flexibility of style and particular attention to interpersonal communication skills.

In the mental health setting, the adolescent is always evaluated in the context of the family, school, community, and culture (King, 1997). While collateral information from other sources is expected for a comprehensive assessment, meeting with the adolescent on their own is an important part of the evaluation. There is no fixed order or manner of conducting the adolescent interview, though some general considerations can guide the process.

The interview comprises history taking and mental status examination (see Chapter 5). History is obtained about significant areas of the adolescent’s life and functioning, past and present, including factors leading to the referral, presenting problem, relevant life circumstances and psychological factors. The mental status examination involves observing and recording the adolescent’s appearance, behaviour, communication, emotions and thought processes during the interview. The clinician will attend concurrently to the content of the history and to the mental state features (King, 1997). A few features distinguish the mental state examination of an adolescent with that of an adult. The clinician will be on the lookout for dysmorphic features that could signal a neurodevelopmental disorder. Pubertal stage relative to age is important, as precocious puberty is a risk factor for psychosocial problems in girls, and delayed puberty is a risk factor for boys. Observation of behaviour and attitude should include the interaction between the adolescent and parents. Interpretation of phenomenology is also important. A surprisingly high proportion of otherwise normally developing adolescents report hallucinations and ideas of reference. A degree of mood instability is also to be expected. More information about cognitive function will usually be gained from examining school reports than from conducting a Mini Mental State Examination.

For many adolescents, meeting someone new and talking about potentially difficult issues is anxiety provoking and threatening. Taking time to establish rapport and put the young person at ease from the outset of the interview is a critical initial step. Orienting the adolescent by introducing yourself, explaining your role and the purpose of the interview, is a good starting point. Rather than beginning with discussion of the presenting problem, establishing the young person in context first (e.g., where they live, go to school, who they live with etc.), as well as their interests

and hobbies, can foster rapport and trust and encourage disclosure. During the introduction, the clinician should also explain the concept and limitations of confidentiality.

Employing an approach that progresses from less threatening subjects to more difficult topics is recommended. Inquiring about the adolescent's understanding of why they have come to see you can segue into discussion of the presenting problem, and can also provide an opportunity to address misapprehensions or to relay to the adolescent your understanding of the reasons for referral. Listening to what the adolescent is telling you, showing interest and trying to understand their perspective can further consolidate rapport. The clinician should be discreet about note taking. When discussing sensitive topics, it is better to give your full attention to the adolescent and leave documentation for later.

Topics such as sex, family, peer group and drug use are common components to history taking with adolescents. A widely used approach to assess the major areas of adolescent functioning is the HEEADSSS interview, developed at the Children's Hospital of Los Angeles. This focuses on assessment of the **H**ome, **E**ducation and employment, **E**ating, peer-related **A**ctivities, **D**rugs, **S**exuality (activity, orientation, and sexual abuse), **S**uicide/depression, and **S**afety from injury and violence. These areas are explored using a developmentally oriented approach. A 12-year-old pre-pubertal female, for example, would not be asked the same questions in the same manner as would an 18-year old fully mature female.

Ask open-ended questions where possible and avoid making assumptions. When inquiring about the adolescent's home environment, for example, starting with "Tell me about your mum and dad," immediately assumes that the patient lives with two parents and that the parents are of different genders. Compare this to "*Where do you live?*" or "*Tell me about your living situation,*" followed by "*Who lives with you?*" This allows the adolescent to describe what is most important in his or her home setting. Further questions may then explore areas such as what relationships are like at home and whether there have been any recent changes, e.g., moving, running away, divorce etc. (Klein et al., 2014).

Similar exploration of school, peers, interest, eating and exercise habits can follow. Is the young person attending school? What subjects do they enjoy? What goals do they have for when he or she finishes school? What does the adolescent do to have fun and with whom? Do they play any sports? Does their weight or body shape cause them any stress? Remember to search for the adolescent's strengths as focusing on risk factors alone can induce shame and deter engagement.

The HEEADSSS interview

1. **Home Environment:** This section explores the adolescent's living situation, family dynamics, relationships with family members, and any potential sources of stress or conflict at home.
2. **Education and Employment:** Here, the interviewer assesses the adolescent's school or work situation, including performance, satisfaction, and any challenges or concerns related to education or employment.
3. **Eating:** This part focuses on the adolescent's eating habits, attitudes toward food and body image, as well as any potential issues related to eating disorders or nutritional concerns.
4. **Activities:** The interviewer explores the adolescent's extracurricular activities, hobbies, interests, and social life, including friendships and peer relationships.
5. **Drugs:** This section addresses the adolescent's history of substance use, including alcohol, tobacco, and other drugs. It assesses frequency, quantity, patterns of use, and any associated problems or consequences.
6. **Sexuality:** Here, the interviewer explores the adolescent's sexual behaviours, sexual orientation, reproductive health, contraceptive use, and any concerns or questions related to sexuality.
7. **Suicide and Depression:** This part focuses on the adolescent's emotional well-being, including symptoms of depression, anxiety, stress, and thoughts or behaviours related to self-harm or suicide.
8. **Safety:** The last section addresses safety issues, including risk-taking behaviours, exposure to violence or abuse, use of protective equipment (e.g., seat belts), and any concerns related to personal safety.

Some areas, like drugs and sexuality, can be sensitive. These topics can be approached obliquely: *“We talked about what you and your friends do to have fun. Do any of your friends get drunk or high?”* Young adolescents who may be somewhat reticent about discussing their own drug or alcohol use are often more willing to talk about such behaviour by their friends. This might be followed by: *“Tell me about a time that you felt pressured by friends to use drugs or alcohol, if any.”* This may enable a more specific discussion of the circumstances and types of substances tried (Klein et al., 2014). Older adolescents can often be asked about drugs more directly, such as *“What types of drugs or alcohol have you tried?”*

Before proceeding with questions about the adolescent's sexual history, seeking the young person's permission can help maintain rapport, such as: *“Do you mind if I ask you a few more personal questions in order to work out how I can best help?”* Inquiring into current relationships is a good starting point, including whether the

adolescent is sexually active. For example: “*Are you going out with anyone right now?*” followed by: “*Can you tell me about that relationship?*” can lead into: “*As you know, many young people your age are sexually active. By that I mean that they have had sexual intercourse. There are also many young people who have chosen not to have sex. How have you handled this part of your relationship with Josh or with other people you have gone out with?*” (Klein et al., 2014). It is important to ask such questions sensitively, but also matter-of-factly. Oblique questioning about sex may convey to the adolescent a level of discomfort in asking the questions, which can further add to theirs.

Other areas to cover include suicidality and abuse. Has the adolescent had any prior suicide attempts? Does the adolescent have any current suicidal ideation? It is critical to ask about sexual or physical abuse in adolescents who are reporting significant problems in the areas such as family dysfunction, decline in school grades, lack of friends, substance abuse, early-onset of sexual activity, history of suicide attempts or runaway behaviour.

In addition to major areas of psychosocial functioning, inquiry should be made about the presence of specific psychiatric symptoms. Often information about psychopathological symptoms emerges during the course of the interview, whereby specific enquiry to determine the presence of a particular disorder is required (e.g., depression, anxiety, psychosis, conduct disorder, substance abuse disorder, etc.).

At the completion of the interview, the clinician should give the adolescent a brief summary of their understanding of the issues as well as a future plan and/or treatment options, where appropriate. Negotiate with the adolescent what information will be discussed with the parent(s) before inviting them into the assessment. The adolescent should also have time to ask final questions.

Internalising Disorders

Anxiety Disorders

These are similar to those in adults with the exception of separation anxiety (see Chapter 18), and are the most common conditions in primary school aged children. Clinical presentation takes many forms (see Table 25.4). Associated somatic complaints (e.g., headaches and abdominal pain, sleep disturbance and nightmares) are reasons for frequent visits to the GP. Anxiety, sometimes masquerading as a physical complaint, is a common reason for school refusal in primary school aged children. Anxiety may be complicated by the presence of other comorbid states like depression and poor impulse control (e.g., ADHD and oppositional behaviour). School performance, recreation and social life are often

impaired due to poor concentration and distractibility (which may be diagnosed incorrectly as ADHD).

Table 25.4. Types of anxiety-based disorders seen in children.

Disorder	Features
Generalised anxiety	Pervasive worries and fears
Social phobia	Timid and socially avoidant
Specific phobia	Unrealistic fears focused on a specific object (such as a dog) or situation (travelling in a lift)
Post traumatic stress	Recurrent thoughts about or reliving in play a stressful experience such as sexual abuse, a bush fire or exposure to violence in war-torn areas
Obsessive compulsive	Obsessive thoughts (such as contamination) and repetitive compulsive behaviours (such as handwashing)
Separation anxiety	Distress, agitation, restlessness and irritability upon threat of separation from a caregiver.

Anxiety is more likely to occur at times of transition (e.g. moving from pre- to primary school). Children who refuse to attend school may fear leaving home out of concern for a depressed mother but then become secondarily anxious because of missed education and social embarrassment. Over-anxious or distant parents and traumatic events (e.g., accident, death of a relative) can reinforce age-appropriate fears. Some children are temperamentally predisposed to react to stress by becoming anxious.

Effective *treatment* comprises CBT (see Chapter 33) combined with exposure to the feared situations (e.g., separation from parents), relaxation training and reinforcement of confident behaviour (Bernstein and Shaw, 1997). The cognitive aspect entails formulating positive thoughts to counter overly critical self-appraisal. A useful treatment for concurrent anxiety and depression in either parent is counselling aimed at reducing over-protection and inadvertent reinforcement of maladaptive behaviour. Return of the child to school is a priority and is eased by consulting with the class teacher and providing a rewarding, non-threatening and structured educational program. RCT evidence supports the use of specific serotonin reuptake inhibitor antidepressant medication, SSRIs, particularly when

combined with CBT. Prognosis is good with treatment, particularly if anxiety is detected early.

Mood Disorders

While depression (see Chapter 16) does occur in younger children, there is a marked upswing in incidence in the teenage years, especially among females. The onset of depression is typically gradual, making it difficult to pinpoint when the problem began. Instead of looking sad or unhappy, depressed young people often appear cranky, grouchy and irritable. Atypical presentations (e.g. sleeping or eating more than usual) are common, contrary to the more typical picture (sadness, insomnia and loss of weight) observed in adults. Comorbidity is frequent, particularly with disruptive disorders, drug and alcohol problems and anxiety.

Prior to seeking professional help for their depression some young people access self-help guidance via the internet. There is evidence that internet delivered psychological treatments can prevent the progression of depressive disorder in its prodromal or very early stages. About 20% of young people will try dietary supplements or herbal remedies, but there is a lack of quality clinical trial evidence to support the practice (Hazell, 2015). Mild symptoms of brief duration (a few weeks) may remit spontaneously. Therefore, it is reasonable to simply wait, or recommend self-help material that the young person will be able to access via the internet. Persistent mild or moderate symptoms (a few months) respond to psychological interventions such as CBT and interpersonal psychotherapy (IPT) (Hazell, 2011). Patients who do not respond to psychological treatment, or have severe symptoms, require pharmacotherapy. Among the antidepressant medications, only fluoxetine seems to be effective in inducing remission (Hazell, 2011). Time to response can be lengthy. Time on treatment seems to be more important than dose for achieving that response. While new generation antidepressants are well tolerated, they may induce suicidal behaviour (in about 4% of young people, compared with 2% taking a placebo). The mechanism underlying this phenomenon is unclear. Symptoms of 'activation' may be an indicator of increased suicide risk and needs to be monitored closely. To prevent relapse or recurrence antidepressant treatment must continue for at least 6 months after remission has been achieved. A patient who has not responded to an adequate course of fluoxetine may be switched to a different class of antidepressant such as venlafaxine (usually in older teenagers). If this is also unsuccessful augmentation with a mood stabilizer or a second generation antipsychotic may be required.

Key points about depression in young people

- About one in ten adolescents will endure a major depressive episode (i.e., meet conventional diagnostic criteria).
- Only a small proportion will be diagnosed or receive treatment.
- Depressed young people have increased rates of suicide, academic or vocational impairment, interpersonal problems, and substance use.
- Episodes of depression eventually remit, but about 40% of affected children experience a relapse. Relapses are more likely among those who in previous episodes, have been depressed for a long time, had incomplete recovery despite treatment, continue to face environmental stressors (i.e., family conflict), or suffer comorbid psychiatric or other medical conditions.
- One third of affected children will attempt suicide,
- Three to four percent of affected children will complete suicide.
- Depression leads to more functional impairment than any other commonly occurring mental illness.

Typical features of mania seen in adults—grandiosity, pressured speech and excessive spending—are not easily identified in young people. Flight of ideas, poor judgement, hypersexuality (which may be falsely attributed to sexual abuse), aggressiveness and auditory hallucinations may occur. Further complicating the diagnosis of bipolar disorder is the ambiguous onset and offset of episodes. Diagnosis is not difficult when symptoms are classical. The usual age of onset is late adolescence or young adulthood, but many patients describe childhood difficulties. Reliability of diagnosis of bipolar disorder in preadolescents, when there may be an overlap with ADHD, is questionable.

Second generation antipsychotic medications are the treatment of choice for the management of acute mania (Hazell and Jairam, 2012). Bipolar depression is best treated with a combination of antidepressant medication and a second-generation antipsychotic (to prevent a manic switch). Prevention of relapse demands the application of the full range of specialist clinical skills including the maintenance of a therapeutic alliance, individual psychological support, motivational interventions, attention to physical wellbeing and lifestyle, education and support of family or carers, liaison with education providers and other key people in the young person's life, and the capacity to monitor and fine tune pharmacotherapy. On the basis of existing evidence there is slightly more support for second generation antipsychotic than there is for mood stabilizers for preventing relapse of bipolar disorder in children and adolescents, but there is a major knowledge gap in this area.

Externalising Disorders

Attention Deficit Hyperactivity Disorder

Danny (see case example below) manifests many of the features typical of ADHD including motor overactivity, impulsivity, distractibility, and inattention. We can suspect inattention because of the poor handwriting and the avoidance of tasks that require mental effort. Danny's problems are apparent in more than one setting, which is essential for fulfilling the ADHD diagnosis. Were Danny younger (say 3 years old) his behaviours might be acceptable, but he is not regulating himself in the manner expected of a 9 year old. Danny was less than 12 years old when symptoms first became apparent, another requirement to fulfil diagnostic criteria. If it hasn't happened already, Danny will soon fall behind at school. His parents will show signs of parenting fatigue and to an outsider their parenting may seem ineffective or maladaptive. Danny may develop secondary problems such as oppositionality, anxiety or even depression. Danny has combined type ADHD, but it is possible for children to have just problems with concentration (inattentive subtype) or just problems with overactivity and impulsivity (hyperactive-impulsive subtype) Referrals for ADHD peak between 8 and 11 years of age. Boys are affected three times more than girls. Clinical features can manifest at preschool but a diagnosis made in children less than 4 years is unreliable.

Case example

Danny's parents dreaded taking him to the supermarket because his behaviour was not that which would be expected from a 9-year-old boy. He would excitedly run from aisle to aisle, shouting the names of the products on the shelves. If not watched carefully Danny would help himself to sweets and ice cream, even though he knew he was only allowed one treat. He would engage other shoppers in random conversation. Mostly they were amused, but sometimes he could be offensive. Danny's parents also dreaded parent teacher meetings. They were weary of hearing about his exuberant but dysregulated classroom behaviour, his tendency to talk when he should be listening, his fidgetiness, and the manner in which he seemed to disrupt the other children. His handwriting was immature and scrawling, and his spelling barely phonetic. At home Danny could be loving and fun, but his parents had learned that it was best to keep him busy with physical activity. A deal had been struck with school that Danny was exempt from homework, on the grounds that getting him to do it had become too traumatizing for the family.

Functional neuroimaging shows underactivity in the frontal lobes (associated with the organization of behaviour) and caudate (associated with regulating movement) and overactivity in the amygdala (associated with regulating emotion). Serial MRI studies show a delay relative to typically developing children in thickening across a range of cortical structures.

Assessment requires a careful history from parents and other sources such as teachers. Behaviour rating scales for parents and teachers help to quantify symptoms and allow comparisons with similar-aged children. Hearing, visual and neurological problems are excluded. IQ and reading assessment are indicated if intellectual impairment or learning disorders are suspected.

Management is multimodal (Health, 2009). Behavioural counselling helps the parents to increase the young person's behavioural compliance but ADHD symptoms respond less well. Techniques include praise and reward for appropriate behaviour, clear instructions (e.g. one specific request at a time) and consistency (e.g., both parents adhere to a simple set of rules). Classroom strategies include: presenting work in modules under 10 minutes to avoid overtaxing capacity to attend; interspersing classroom learning with brief periods of exercise; permitting movement provided it does not disrupt peers; using cues to alert students they have strayed from the task; seating them close to the teacher or students who concentrate well; and articulating expectations of behaviour.

CNS stimulants such as methylphenidate and dexamphetamine have a pivotal role in reducing symptoms where these interfere with learning, social interaction and family life. Stimulant medications increase the activity of dopamine and noradrenaline in neurons. The putative mechanism of action is better filtering of extraneous information (noise) coupled with a boost in focus on salient information (signal). Efficacy and safety are well established in the short term and, provided there is treatment adherence, is maintained long term. Common side-effects are loss of appetite, weight loss and sleeplessness.

In about a quarter to a third of cases ADHD persists into adulthood. By this time hyperactivity has usually resolved but inattention and impulsivity remain a problem. Remission may be attributable to compensatory neural mechanisms as much as it is to normalisation. Antisocial behaviour and substance misuse are the most consistently reported adult outcomes of childhood ADHD, but individuals are also at increased risk of mood and anxiety disorders. Secondary impairments impact on functioning in domains such as: education, occupation, relationships, parenting, and complex skills such as driving.

Oppositional Defiant Disorder

Oppositional Defiant Disorder (ODD) applies to children who lose their temper persistently, argue with adults, annoy people, blame others, are touchy, and have frequent or intense temper tantrums. This disrupts relationships with family and peers, and makes them 'difficult to manage' at school. ODD can be a response to an overly authoritarian or overly permissive family.

Defiance, often the presenting feature, can manifest in other conditions. Depressed adolescents may be angry and oppositional; anxious or obsessive children may be unco-operative when forced to confront feared situations or prevented from performing their rituals; psychotic young people can appear defiant. Disruptive Mood Dysregulation Disorder (DMDD) shares features in common with ODD, but is characterized by severe temper tantrums and chronic irritability. DMDD was only established as a diagnosis in 2013. Children currently meeting DMDD criteria may formerly have been diagnosed with ODD, or childhood onset bipolar disorder. First line treatment for ODD is parent behaviour management training.

Conduct Disorder

Young people with Conduct Disorder (CD) display behaviours such as bullying, initiating fights, truancy, cruelty or violence to people or animals, deception, and theft. CD is not diagnosed when behaviours are better explained by another condition such as schizophrenia, bipolar disorder or autism spectrum disorder. Depression, ADHD, substance abuse and learning problems are common co-morbidities. An isolated antisocial act does not warrant the diagnosis. An earlier onset has a worse prognosis. Most young people with CD will have shown symptoms of ODD earlier in development. ODD behaviours are always overt and easily observed, while CD behaviours can be covert and difficult to detect. This is relevant to treatment, because observable behaviours are more readily responsive to behaviour management strategies. Most aggression seen in association with CD is an excessive reaction to a situation, is associated with heightened arousal, and is sometimes referred to as 'hot' aggression. In contrast, some people with CD manifest 'cold', calculated aggression that is associated with normal or suppressed arousal. They show a striking lack of empathy, referred to as callous unemotional traits. The basis for the lack of empathy seems to be a deficit in emotion recognition. Individuals with callous unemotional traits do not direct their gaze to the eyes of picture faces expressing emotion, thus failing to receive salient information (Dadds et al., 2008). Aetiological factors for CD include the parent child interaction difficulties associated with ODD, but extend to biological and social factors. CD is heritable, affecting neurotransmitter systems such as the monoamine oxidase transporter gene. Perinatal hazards such as hypoxia and maternal smoking, alcohol and heroin use increase the risk of CD, as does exposure to environmental toxins

such as lead. Finally, CD tends to aggregate in communities where there is disadvantage.

Parent training in behaviour management is the treatment of choice for primary school aged children with CD, but the intervention is less effective with teenagers. Cognitive and social problem solving groups are helpful for children 10-14 years with CD. Adolescents with CD typically require multimodal interventions including individual therapy, family work, community advocacy, diversionary activity (such as cultural groups, Police Boy's Club) and medication. Medication is prescribed for comorbid conditions such as ADHD. A prominent guideline recommends the second generation antipsychotic risperidone for severe aggression (National Institute for Health and Clinical Excellence, 2013). We do not endorse this practice, because of the problems of metabolic side effects, and our observation that benefits attenuate with time (the guidance is based on evidence from short term clinical trials). Clonidine has more enduring effects. Note that callous unemotional traits do not generally respond to pharmacotherapy.

Treatment Modalities

Treatment flows from formulation and can be categorised into biological, psychological and social components. Goals are collaboratively established to target problems or behaviours, and monitoring mechanisms are used. Informed consent is obtained from parents and adolescents over 15 years. It is desirable that younger children also assent to treatment. Because co-morbidity is common, a multi-modal approach is usually indicated.

Psychoeducation

Education for the parents and child about the nature of the condition, its longitudinal course and treatment is essential. Parents may be provided with written information prepared by professional organisations. An example would be parent's guide to depression and anxiety in young people developed by Beyond Blue. Mobile devices provide opportunities to deliver mental health information, treatment, and monitor function and symptoms. An example would be the Love, Talk, Read, Sing, Play mobile App developed to assist parents to engage in and enjoy the development of their preschool aged children. Psychoeducation sometimes extends to other adults engaged with the child. A school visit to meet relevant teaching and student support staff can help address uncertainties about the nature of a child's problem, the significance of symptoms, and the best way to address behaviours that occur in the school environment. It is always best that parents also participate in such school meetings.

Advocacy

Young people often lack shared decision-making in terms of their social environment and the expectations of them in terms of output, that is, the family they live with and the school they attend are often not of their own choosing. Some young people may view school refusal and running away from home as their only available option to modify the situation. Child and adolescent psychiatrists have roles in communicating, advocating for systems change and managing young people in out of home care. Advocacy extends to providing reports to enable children to gain access to additional educational funding and support, and parents to gain access to carer's allowance or other financial support. This is relevant because costing studies demonstrate that childhood mental illness places a considerable financial burden on families.

Parent Behaviour Management Training

Behaviour management refers to the actions or inactions taken to aid an individual (or group) to learn to act appropriately and pro-socially relative to the given context and their developmental stage. In essence, the overall goal of behavioural management for parents and carers is to help young people develop adaptive self-regulation skills. Self-regulation is the ability to recognise, direct and modulate one's own thoughts, feelings and behaviour. This is done by teaching parents and carers skills to help scaffold their young person's ability in an age-appropriate manner.

Behaviour management is essential in ensuring young people grow up to become functional and pro-social members of society. This means following the rules and expectations of society and managing their own emotions and behaviours adaptively. Throughout childhood and adolescence behavioural non-compliance is a normal aspect of development. For example, it is very normative for a two year old to throw a temper tantrum. However, if non-compliance and externalising behaviours continue and increase with age, this can lead to significant difficulties and poorer later life outcomes. Indeed, a temper tantrum in an adolescent is not only less acceptable; it is also likely to be much harder to manage and may result in significant risk to the teenager and others. Where non-compliance and acting out becomes severe, persistent and unmanageable across multiple settings, the young person is at increased risk for a number of negative life trajectories. These may include peer relationship problems, academic difficulties, increased mental health problems (including increased alcohol and drug use) and a criminal lifestyle.

Successful behaviour management approaches make use of regular routines, structure and expectations. They teach young people what to do (making the adaptive, desired behaviour explicit), as opposed to what they are not allowed

to do. For example, “Please keep your hands and feet to yourself” rather than “Do not punch or kick others”. This allows the parent or carer to positively reinforce desirable behaviour rather than having to set down consequences for poor behaviour. Similarly, feedback on the form of praise towards the young person should be specific rather than general. For example, “I appreciate that you made your bed this morning”, “I like the way you took your plate to the sink” rather than generic “Good girl” or “Good boy”. This helps the young person to know what it is they have done well and repeat the desired behaviour.

Reinforcement can be both interpersonal (such as attention, praise, and carer time) and/or tangible (such as toys, screen time). Reinforcement should be meaningful for the young person and where possible be associated with the desired behaviour. For example, “You may play on the computer after you finish your homework”. If possible, it is generally advisable to avoid using food or money as the primary reinforcer.

Types of reinforcement

Positive reinforcement. Adding something to increase the frequency of a behaviour. A teenage son will be more likely to empty the dishwasher if he is praised for doing it.

Negative reinforcement. Removing something to increase the frequency of a behaviour. Parents have nagged their son about emptying the dishwasher for weeks. One day he voluntarily empties the dishwasher and to his surprise the nagging stops. He is more likely to repeat the behaviour.

Punishment. Adding something aversive to decrease the frequency of a behaviour. A teenage son breaks curfew by more than an hour and is grounded from outings for a week. In this case the addition is the temporary restriction in freedom.

Extinction. Removing something to decrease the frequency of a behaviour. A four-year-old child makes frequent ‘curtain calls’ to the living room after bedtime. Her parents have typically made a fuss, engaged her in conversation and even made her hot milk drinks to ‘help her sleep’. Fun! Parents change their strategy, are cool, offer no drinks and walk the child to her bedroom without comment. The ‘curtain calls’ cease.

Reinforcers also need to be age and capacity appropriate. For example, factors such as trauma or intellectual disability are likely to impact a young person's ability to manage their emotions. If this is the case, behaviour management may need to initially start with co-regulation techniques. This involves adult modelling of how to deal with difficult emotions alongside the young person, using attachment bonds to help the young person regulate their emotions. The adult may have to help the young person to first identify and then manage the difficult emotion. Similarly, parents or carers may need to scaffold the young person through successive approximations in order to get to the appropriate pro-social response. Typically, as the young person gets older, they should take a larger role in helping identify expectations and reinforcers. Some families may find it helpful to incorporate written or visual guides and achievement charts ("star charts") especially around routine expectations. These can be helpful guides for the young person and provide direct, often rewarding, feedback.

All behaviour management approaches take consistency and persistence. If the young person has a history of non-compliance or comes from a background where there has been previous inconsistency in behaviour management then it is highly likely there will be an initial increase in non-compliant behaviours when strategies are first implemented. Parents and carers should be warned about this when implementing behaviour management. Where reinforcement of adaptive behaviour alone is not successful, short amounts of time in or time out are often used as a consequence to manage maladaptive behaviours. Time out refers to a low intensity punishment in which the child is briefly isolated from others. As the child learns to manage themselves, they may choose to remove themselves to a safe and calming place as a way of coping while learning more advanced skills. General guidelines for time out are typically one minute per year of age (i.e., six minutes of sitting calmly for a six-year-old child). On occasions where the young person is placing either themselves or another at risk, restraint may be implemented. Ideally, seclusion or restraint should be used only as a last resort. This makes sense when one considers that the young person is being taught that they cannot manage themselves when these strategies are implemented.

There are a number of well researched and empirically supported programmes available to teach parents and carers behaviour management in the community. These include Triple P Parenting and Magic 1-2-3. There are also play-based parenting guides such as Parent Child Interactional Therapy (PCIT), which focus on the attachment relationship as a means of improving difficult behaviours. The aetiology of disruptive behaviour is multifaceted and should be considered in a full assessment. Areas of consideration include individual psychological and biological factors, family factors and environmental factors.

Individual factors as mentioned earlier include aspects such as temperament, trauma and developmental level. Even within families some young people require more limit setting while others are acutely aware of social roles and expectations. Trauma is well known to disrupt regulation capacity and therefore the young person may be more sensitive and require more scaffolding in relearning adaptive strategies. Likewise, certain developmental disabilities and syndromes are frequently associated with regulation difficulties which may amplify acting out behaviours (such as Foetal Alcohol Syndrome, Pervasive Developmental Disorders, Noonan Syndrome and Tuberous Sclerosis).

Relevant family factors include parental and carer health issues, both mental and physical. Issues such as parental history of abuse and neglect and of antisocial behaviour (parents who themselves have a history of non-compliance) should be considered as these may impact a parent or carer's own ability to self-regulate. Other relevant family factors include marital conflict (including domestic violence); stress (including work and financial pressures); the level of support the family has, such as extended family and community involvement; and environmental disruptions (such as moving house regularly).

At a broader level, factors such as cultural expectations, gender biases and the role of generational timing should be considered. For example, the influence and implication of social media on youth behaviour is a relatively new but increasingly pertinent factor to consider in regards to behaviour management.

When considering a young person's disruptive behaviour, it is important to try to understand the meaning of the behaviour. Developmentally, young people around certain ages are expected to push the boundaries (toddlers and teenagers particularly). Indeed, this is part of the natural individuation process. Most young people are not deliberately trying to do the wrong thing. It is unhelpful to label a young person as "bad". Instead, it is helpful to consider why the young person might be acting in the way they are and in what way the environment is reinforcing the current behaviours (either explicitly or inadvertently). Examining the aetiology above, acting out behaviours may be a bid to engage a parent or carer, a distraction from something else in the family system or the result of an underlying difficulty or recent trauma. Consider all members of the family and their role in perpetuating the acting out behaviour. For example, a sibling with a recent health diagnosis may be impacting the previous balance of allocated time and attention.

The following factors have been identified as being present in young people who have come from difficult backgrounds, or have faced a number of risk factors, but have managed to maintain adaptive self-regulation skills. Individual factors include high intelligence, easy temperament and alternate areas of competence (such

as a young person struggling at school but doing well at sport or music). Protective family factors include supportive relationships, strong extended family networks and a secure attachment to at least one adult within the family network. Social and environmental factors that are likely to aid a young person in developing adaptive self-regulation capacities include a supportive school environment that fosters responsibility taking, pro-social peers and a group with which the young person identifies. For example, this may include a religious group, an ethnic community group or a sports group. Finally, good role models able to provide examples of adaptive self-regulation across any setting (home or external) are also likely to be of benefit.

As with the aetiology factors that elucidate risk, different protective factors are stronger at different developmental stages. In children, family factors are paramount, whereas in adolescence individual, school and peer influences are more important. The task for professionals is to promote these protective factors in order to lessen the effects of risk factors, as well as to intervene where possible to reduce risk factors. As young people grow older, they tend to accumulate more risk factors and so stronger protective measures are needed. This highlights the importance of intervening early to prevent effects on development, and to address the problem behaviours before they are entrenched. Hence it is important to consider the degree of impairment of life functions in assessment, not just diagnostic symptoms.

Family Therapy

Family therapy is a form of psychological treatment that focuses on the interaction of family members as a means of understanding a symptom and effecting change. It is a problem oriented rather than diagnosis-oriented treatment. Family therapy has grown from the observation that emotional or behavioural problems in an individual often impinge on others with whom they live. Most family therapists do not view the family as necessarily dysfunctional, but rather see the family as a forum in which to solve a problem. The family therapist may work alone, but it is common for two people to work together as co-therapists. Sometimes the therapist may work with the assistance of a 'team'. The team consists of one or more co-therapists who do not participate actively in the session but who observe the interactions of family members with each other and with the therapist and may provide advice or feedback to the family through the therapist. Commonly the team will observe the session through a one way screen or on a video monitor.

Family therapy is informed by systems theory, which in turn is derived from the basic biological and physical sciences and is concerned with describing and predicting the behaviour of complex systems on the basis of the interaction of the parts. The important principles underpinning systems theory are:

1. The notion of homeostasis, or a dynamic steady state, which is sustained by monitoring shifts of parts of the system away from the balance, and the correction of these shifts (negative feedback).
2. The notion that the steady state of a system or organism may shift abruptly from time to time through a process of 'deviation amplification' or positive feedback. This shift in steady state is conceptualised as essential for the adaptation of the system or organism.

While there are many different schools of family therapy, they share some common techniques:

1. Reframing is restating a problem or behaviour in a different (usually more positive) way. An example would be reframing a child's disruptive behaviour in the context of marital discord as a caring and concerning activity designed to distract the parents from their conflict with one another.
2. Circular questioning is a technique used to elicit differences in relationships the family have experienced before and after a problem began, or has resolved. An example would be asking what a younger sibling has noticed about his parents' interaction with each other since a teenage sister re-emerged after months of isolating herself in her bedroom.
3. Metaphor is employed by many therapists as a means of illustrating a problem, or assisting the family to distance themselves from a problem so they can look at it more objectively. An example would be to liken the arguments between a mother and daughter over the issue of responsibility versus freedom to a strange tennis match in which the object seems to be to hit the ball as far out of court as possible.
4. Paradox is a technique that stimulates the family to resolve a problem through the mechanism of deviation amplification. Instead of offering strategies that might reduce the intensity of the problem, the therapist suggests a counter strategy that intensifies difficulties in the short term. Rather than ban internet access, parents of a teenager may encourage increased usage, while at the same time demonstrating intense interest in the content that has been accessed.

A specific application of family therapy is in the treatment of anorexia nervosa in younger people. Family Based Treatment is recommended as first-line treatment for people with anorexia who are aged less than 19 years, have had a duration of treatment of less than three years, and are not in need of emergency refeeding (Hay et al., 2014). Family Based treatment encourages parents to engage

in restoring the weight of their child to a healthy level, while at the same time re-engaging with the child emotionally.

Therapy with the Child

Psychological treatments developed for the treatment of adults have been adapted for application in older children and youth. Modalities include cognitive-behaviour therapy (CBT), interpersonal psychotherapy (IPT) dialectical behaviour therapy (DBT) and mindfulness based therapy (MBT). Play therapy directed to younger children may be psychodynamically informed, or structured as in CBT; for example, a doll may be used to provide exposure play therapy for an upcoming insertion of an intravenous canula. Art therapy may be used in a psychodynamically-informed fashion for the young person to express intra-psychic conflict, and is particularly useful in cases where that would not be possible verbally. Group approaches are effective in treating anxiety and depression in teenagers and have been successfully delivered in schools. Group therapy has also been successfully delivered in schools directed to young children with evolving conduct problems. Socially awkward children can be helped by group-based social skills programs such as the Secret Agent Society.

Medication

Few medicines are approved by regulatory bodies for the treatment of psychiatric illness in children and adolescents (see Table 25.5), therefore most prescribing is “off-label”. The most common classes of medication prescribed for children are the antidepressants, and medications for the treatment of ADHD. Less commonly prescribed are antipsychotic medications and anxiolytics.

Children are not just undersized adults. They metabolise and eliminate drugs more quickly than adults leading to shorter drug half-lives. As a consequence they typically require higher weight-adjusted doses and more frequent dosing than adults. Children with neurodevelopmental disorders are vulnerable to the side effects of psychotropic medication, and may have idiosyncratic reactions. Noradrenergic pathways develop more slowly than serotonergic pathways, rendering noradrenergic specific drugs less effective. This is thought to account for the lack of effectiveness of tricyclic drugs for depression in young people. About one in five medicated children are receiving more than one psychotropic drug. Some drug combinations are supported by clinical trial evidence. An example is the combination of a stimulant medication and clonidine for children who are both hyperactive and aggressive (Hazell and Stuart, 2003). Much polypharmacy however is a reflection of desperation and a lack of planning and should be avoided since it increases the chances of drug interactions and reduces adherence. Prescribing is done

cautiously and by well-informed clinicians, for example SSRI prescription in adolescents is accompanied by information about the increased risk of suicidal ideations. Not only should assessment information come from multiple sources, but so should information to help evaluate treatment effectiveness and tolerability. Symptom or behaviour checklists can be of help in this regard. Growth parameters, pulse rate and blood pressure should be measured regularly in all children receiving psychotropic drugs, and a screen for common side effects should be undertaken at each review. Specific precautions against metabolic side effects must be taken when prescribing second generation antipsychotic drugs.

Table 25.5. Psychotropic medications approved by the Australian Therapeutic Goods Administration for use in children.

Medication	Indication
Methyphenidate (short and long acting)	Attention-Deficit/Hyperactivity Disorder
Dexamphetamine (short and long acting)	Attention-Deficit/Hyperactivity Disorder
Atomoxetine	Attention-Deficit/Hyperactivity Disorder
Guanfacine extended release	Attention-Deficit/Hyperactivity Disorder
Risperidone	Conduct disorder, other disruptive disorders in patients with subaverage intellect, intellectual disability, and prominent destructive behaviour Behavioural disorders associated with autism
Quetiapine	10-17 years: monotherapy in acute mania associated with bipolar I disorder; 13-17 years: schizophrenia treatment
Fluvoxamine	Obsessive Compulsive Disorder

Further Reading

Rey, J. M., & Martin, A. (2006). JM Rey's IACAPAP e-textbook of child and adolescent mental health. *Lancet*. <https://iacapap.org/resources/e-textbook.html>

Chapter 26: Old Age Psychiatry

Carmelle Peisah

Specialty of Psychiatry, The University of Sydney, Australia, and School of Clinical Medicine, The University of New South Wales, Australia.

John Snowdon

Specialty of Psychiatry, The University of Sydney, Australia, and School of Clinical Medicine, The University of New South Wales, Australia.

Janine Stevenson

Specialty of Psychiatry, The University of Sydney, Australia.

Shalini Narchal

Western Sydney Local Health District Mental Health Services, Australia.

Anne Wand

Specialty of Psychiatry, The University of Sydney, Australia, and School of Clinical Medicine, The University of New South Wales, Australia.

Introduction

Management of mental illness in older people is both the same as, and different from, younger people. Although older people develop the same mental illnesses as younger people, the biopsychosocial aetiologies and perpetuating factors are different, as are the treatment strategies which, by necessity, must be shaped by both the biological and social contexts of ageing. Specifically, older people are burdened with more physical illness, neurodegenerative disease and disability than younger people, and age-related changes in organ systems render them more vulnerable to the adverse effects of psychotropics. The psychosocial factors contributing to mental illness in older people are more commonly associated with loss, both personal and interpersonal. Treatment settings for older people are heavily influenced by carer availability and support and may include residential aged care facilities. The management of mental disorders in older adults requires an

understanding of these unique biopsychosocial factors that both contribute to mental illness in older age and influence treatment.

Mood Disorders in Older Adults

Depression

There is a perception that old age is miserable. For most this is not true, despite the challenges of aging. In fact, cross-sectional community surveys have frequently revealed that happiness with life and life satisfaction across the lifespan is represented by a U-shaped curve, with the nadir in mid-life and higher levels of wellbeing in younger and older adults (Blanchflower and Oswald, 2008; Steptoe et al., 2015). This is not the case in nursing homes where it has been estimated that at least 23% of nursing home residents are depressed (Snowdon et al., 2010).

Is Depression Different in Older People?

There is ongoing debate about whether older people have more melancholic and psychotic depression. Brodaty et al. (1997), found in a mixed inpatient and outpatient sample that older people did have more melancholic depression and more psychotic features. Other purely community-based studies have not shown these differences and in general it is inpatient studies (which reflect patients with more severe depression) that show the most difference between depression in older and younger people (Blazer, 2003).

Subsyndromal anxiety symptoms such as unrealistic fears, irritability, behavioural agitation, and excessive and uncontrollable worries, may be more common in older people with depression (Diefenbach and Goethe, 2006), but are often under-recognised and unreported (Wetherell et al., 2009). At the same time, anxiety disorders are also commonly comorbid with major depression, present in 47- 61% in primary care (Beekman et al., 2000). Older depressed patients tend to be more somatically focused (Drayer et al., 2005). Atypical depression characterised by overeating and hypersomnia is probably less common in older people (Husain et al., 2005).

Genetic factors may be more relevant in people with early onset depression (i.e. family history is more common), while brain pathology may be more important in late onset depression. Alexopoulos et al. (1997), coined the term “vascular depression” to describe major depressive disorder (MDD) associated with deep white matter hyperintensities on MRI. It has been hypothesised that the white matter changes cause depression by interrupting frontal-striatal circuits known to be associated with mood. Patients with this type of depression typically display

executive impairment that does not respond to treatment. The depression itself is also more treatment resistant than non-vascular types (Aizenstein et al., 2016).

Overlap of Depression with Other Medical Illness

There is a complex interaction between medical illness and depression (see Chapter 30), which is particularly relevant in late life. Medical illness, especially when painful, disabling or chronic, increases rates of depression (Krishnan et al., 2002) and medical co-morbidities are common among older people with depression. Neurological diseases such as Parkinson's Disease are associated with higher rates of depression beyond similarly disabling conditions, implying a direct neural effect. Estimates vary, but around 40% of those with Parkinson's Disease have MDD (Cummings, 1992). There are also high estimated rates of depression comorbid with other neurological diseases. Around one third of stroke survivors develop depression (Paolucci et al., 2006) and 40% of those with Alzheimer's Disease (Wragg and Jeste, 1989). Conversely, depression can worsen not only the symptoms of other illnesses, but also their course. For example, depression influences rehabilitation outcome, morbidity and mortality after myocardial infarct and stroke (Krishnan et al., 2002). Most importantly, depression has a bi-directional relationship with pain. Pain syndromes exacerbate depression and vice versa. Chronic pain in older people is a risk factor for suicide (Van Orden et al., 2015).

Assessment

A number of well-validated screening tools for depression in older populations have been developed. For example, the Geriatric Depression Scale (GDS) is a 30-item self-administered questionnaire (Scogin et al., 2000). The Cornell Scale for Depression in Dementia (Alexopoulos et al., 1988) relies on semi-structured interviews with the patient and an informant and has been administered in Australian nursing homes as part of routine screening (Snowdon et al., 2011b). Although these all have good sensitivity and specificity, they do not replace proper clinical assessment. Often test scores are noted but not followed up with treatment, particularly in nursing homes. Morning is a good time to assess for depression: if there is marked diurnal variation, there can be striking signs and symptoms in the morning and an almost normal mental state by afternoon- a marker of melancholic depression. Assessing people at home is helpful to give information about how well they are functioning, particularly with regards to self-care. In addition, family members or care staff may be able to give information about sleeping, eating and behavioural changes.

Important differential diagnoses of depression in older people

1. **Medical illness** – symptoms such as low mood, lack of energy, sleep disturbance, poor concentration, and poor oral intake are seen in both depression and medical illness. Look for cognitive symptoms such as thoughts of guilt, worthlessness, pervasive pessimism, feelings of being a burden or suicidal ideas, to distinguish a significant co-morbid depression;
2. **Delirium** – hypoactive delirium is most often confused with depression in medical settings. Mood symptoms will generally resolve when the delirium resolves. Starting treatment for depression during delirium can worsen delirium by adding to distressing physical symptoms, anticholinergic load and polypharmacy;
3. **Dementia** – apathy, agitation and emotional lability (e.g., tearfulness) are common in dementia. Additionally, people with dementia may not be able to articulate emotions, especially if language is affected, in which case look for behavioural changes as signs of depression – for example anorexia, screaming, agitation or increased social withdrawal;
4. **Grief** – for example after losing a partner, grief should not be pathologized, nor should a major depression occurring in this context be undertreated. Insomnia, low mood, weight loss and tearfulness are all normal in the months following such a loss, and may occur in waves rather than be pervasively present. However, when symptoms are severe, pervasive, do not improve with time, or there is excessive guilt or suicidal ideas, consider depression.

The person themselves may report that they are continuing to participate in activities but a spouse may reveal this is not the case. It is important always to check for suicidality. Do not be reassured by a non-lethal sounding plan; it is the intent that is crucial. Be aware also of psychotic features, cognitive impairment (with associated impulsivity), substance use disorders and access to means of self-harm, all of which may increase the risk of a suicide attempt.

Treatment

Psychosocial interventions and psychological therapies are recommended as first line for mild to moderate depression (Malhi et al., 2018c). Older people benefit from cognitive behavioural therapy (CBT; see Chapter 33) (Serfaty et al., 2009), especially when modified for age and targeting both depression and anxiety

symptoms (Wuthrich et al, 2016). Mild to moderate depression responds as well to CBT as to selective serotonin reuptake inhibitors (SSRI's) (Laidlaw et al., 2008). Family therapy is also an important, but often neglected, modality of treatment for all mental health disorders of older adults (Peisah, 2006).

Side effects and antidepressant use in older people

Principle 1: Secondary amine tricyclic antidepressants (e.g. nortriptyline) are safer than tertiary amines and are recommended in older people. Avoid tertiary amine (e.g. imipramine, doxepin, amitriptyline, and dothiepin) due to greater anticholinergic and noradrenergic side-effects:

- delirium
- urinary retention
- postural hypotension
- falls
- worsening of glaucoma

Principle 2: Monitor for Syndrome of Inappropriate Antidiuretic Hormone (SIADH), and consequent low sodium, which is particularly associated with SSRIs but can occur with any antidepressant;

Principle 3: Always **START LOW AND GO SLOW** with dosing when using psychotropic drugs in older people.

Principle 4: Consider drug interactions, which can be complex and may depend on the subtype of P450 enzyme used for metabolism.

In general (as when treating younger people), the evidence supporting pharmacotherapy is strongest for more severe depression, which also applies to depression in dementia. The range and use of available antidepressants is discussed elsewhere in the text and includes secondary amine tricyclics, SSRIs, venlafaxine, mirtazapine and other newer products. Deciding which antidepressant to use is partly determined by consumer preference, medical comorbidity, the side-effect profile of the medication and potential drug interactions.

For psychotic depression and depressive episodes in bipolar affective disorder, treatment protocols are similar to treatment for younger patients. However, regarding antipsychotics, older people are more prone to manifest extrapyramidal and other side-effects, including sedation, gait disturbance and falls, swallowing problems, QT prolongation and metabolic problems.

Despite good evidence that lithium can be effective as an adjuvant (or sometimes alone) in treating depression in older populations, lithium has increasingly been replaced by sodium valproate without any evidence of superiority in terms of safety profile or efficacy (Sajatovic et al., 2005; Shulman et al., 2003).

The metabolism of lithium is likely to be different due to reduced renal clearance, decreased volume of distribution and possible changes in the blood-brain barrier, plus the effect of any concomitant medications. For all these reasons, lithium may be used, but with caution to enhance efficacy and minimize adverse effects, namely:

- start at lower doses and increase slowly while checking 12-hour trough lithium levels;
- for the prophylaxis of bipolar depression, and management of acute unipolar depression for patients over 50 years (and those with renal or thyroid impairment, on diuretics, ACE inhibitors or NSAIDs/COX-2 inhibitors), aim for the lower end of the target serum lithium concentration range (i.e., 0.5-0.6 mmol/L) (Wijeratne and Draper, 2011).
- Lamotrigine can also be used in older populations as augmentation therapy for treating treatment resistant depression.

ECT is recommended for major depression with psychosis, catatonia and life-threatening depression (e.g., associated with inadequate oral intake and suicidality), as well as depression that has not responded to other agents. Research suggests ECT works as well, and possibly better, in older people (Dombrowski and Mulsant, 2007). The concern that ECT causes more cognitive side-effects in older populations can be addressed by careful choice of the mode (e.g. ultra-brief, brief etc.), type of electrode placement (e.g., right unilateral, bifrontal) and frequency of ECT administration, although there is often a trade-off between efficacy, speed of response, and side effects (Tor et al., 2015).

Mania

Mania in older people can be thought of as either early onset or late onset. Early onset means the person has had bipolar affective disorder since they were young and continues to experience episodes. Late-onset mania can be either secondary mania (most commonly) or late-onset bipolar affective disorder (Brooks III and Hoblyn, 2005). Causes of secondary mania, where there is a clear organic cause, include: medication (e.g., antidepressants, steroids, some antibiotics) and neurological illness such as stroke and cerebrovascular disease. Although treatment

is similar as for other causes of mania, the underlying medical cause needs to be treated in parallel with the mania.

Late-onset bipolar affective disorder - usually defined as occurring after 50 - is diagnosed where mania is not secondary to medication or illness and there is no known history. Similar to early onset bipolar affective disorder, the first episode is usually depressive in the late-onset disorder (Montes et al., 2013).

Presentation

Some authors have suggested older people with mania present with more irritability, dysphoria and more persecutory delusions but less overall psychotic symptoms (Kessing, 2006) than younger adults; while other studies suggest a similar clinical picture regardless of age. There is less comorbid substance abuse than in younger people with bipolar affective disorder; however, this may be a cohort effect. The severity of depressive and manic symptoms are reduced in older age compared to younger adults (Beunders et al., 2023). Cognitive impairment is common and present during periods of euthymia in 40-50% of older adults with bipolar disorder (Beunders et al., 2023). Dementia is more prevalent in people with bipolar disorder than their non-affected peers.

Treatment

Lithium is effective in treating mania in older people, although caution is advised with dosages and target serum concentration range, as discussed above. The higher end of the range (0.7-0.8 mmol/L) is recommended in the management of acute mania and prophylaxis of mania (Wijeratne and Draper, 2011).

The evidence regarding sodium valproate and other anticonvulsants is sparse. They are subject to hepatic metabolism and consequently have interactions with some medications, as well as reduced clearance in people with hepatic impairment. Beyond this, tolerability and efficacy have not been adequately studied in older age groups.

Lastly, there is some research into atypical antipsychotics suggesting agents such as quetiapine, olanzapine and lurasidone are effective and reasonably well tolerated in older age, although postural hypotension and sedation can be a problem (Sajatovic et al., 2008).

Late Life Suicide

Epidemiology

Suicide in later life remains a relatively unaddressed public health problem in Australia, receiving far less attention than suicide in younger age groups (Wand and Peisah, 2020). Notable is the peak suicide rate among men and women aged 85 years or more; although for men, the suicide rate peaks around 45-49 years then drops, while for women the suicide rate is fairly constant, both peaking again in very old age (Figure 26.1). The suicide rates of Australian men aged between 60 and 84 markedly decreased between the early 1980s and 2016-2017 (Snowdon et al., 2017). Suicide rates for older women (aged 60 or more) have remained comparatively low over the same period (Snowdon et al., 2017). Possible reasons for the decreases include not only improved recognition, understanding and treatment by clinicians of late life depression (and anxiety and psychosis), but also advances in and availability of treatments for physical problems. Certainly, Australia has improved its recognition of and provision for the needs of older people and there has been widespread interest in the notion of wellbeing and ageing (NSW Ministry of Health, 2014). However, although there has been some reduction in the suicide rate of women aged over 85 years, the rate among men aged over 85 years is the same as it was in the early 1980s. Suicides of men in this age group are commonly related to loneliness, distress about physical decline and not wanting to be a burden on loved ones (see below). Why the male rate at over 85 years is sixfold higher than that of females in the same age group remains to be elucidated.

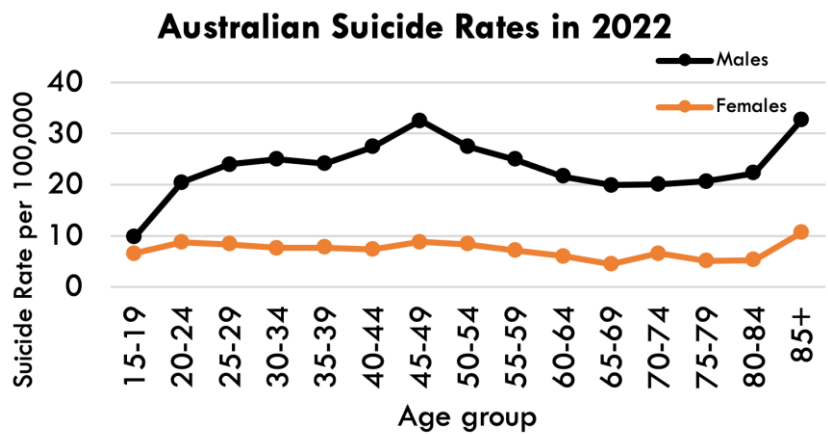


Figure 26.1. Suicide rates in Australia in 2022 (rate per 100,000).

Aetiology

Suicide is commonly attributable to a complex interplay of multiple factors, mental disorder being but one. Studies of older people who have completed suicide have provided insight into the biopsychosocial context of suicide in older people. In late life, self-harm and suicide are closely linked; they share common risk factors, and there is greater intent to die and higher lethality of self-harm. It is therefore important that any self-harm in an older person be fully assessed and the underlying contributing factors holistically managed (Wand et al., 2019).

Half or more of those who have completed suicide with depressive illnesses will have had late onset (age 65+) depression and may have had vascular brain changes. Depression is an important and potentially treatable factor. The older person is less likely than younger persons to have been referred to a mental health specialist or team, although 77% (in Australia) will have visited a GP in the three months prior to death (De Leo et al., 2013). The responses of families and healthcare professionals to older adults who have self-harmed for a variety of various physical, psychological and social difficulties, may exacerbate or attenuate thoughts of self-harm (Wand et al., 2019), providing an opportunity to prevent late life suicide. Responses characterised by hopelessness, helplessness and invalidation may be particularly associated with self-harm and suicide in older people.

Suicidal responses to stressful or depressing circumstances may occur at any age and may be accompanied by painful emotions such as shame, guilt and sadness, either part of, or separate to a depressive syndrome. Personality factors affect how a person responds to distressing or threatening circumstances, such as onset of physical or cognitive impairments. A psychological experience useful for understanding suicide is the concept of ‘psychache’ (Shneidman, 1999), an intensely felt psychological pain deemed to be unbearable. Functional impairment due to disability, pain or chronic illness (Fässberg et al., 2016) and the feeling of being a burden are strongly associated with old age suicide (Van Orden et al., 2015). Loneliness and loss (of health, spouse, role, home, self-esteem) are commonly contributing factors to late life suicidal thoughts. Commonly, the older person who dies by suicide lives alone, their partner having died or been admitted to residential care. They may have lost their friends, are no longer involved in work and social groups, and they may have had to change accommodation, including entering a nursing home (Murphy et al., 2012).

Prevention and Management

Although the ratio of attempted to completed suicide in old age is much lower than among younger people, attempted suicide and self-harm (regardless of intent) provide important opportunities for intervention. Any self-harm should be

taken very seriously. It is essential to sensitively ask the older person why they harmed themselves, and to develop an individualised management plan which directly addresses the potentially reversible contributing factors. This must include attention to underlying diagnoses (e.g., psychiatric disorders, cognitive impairment) and factors which might otherwise be dismissed as small or of ‘lesser’ importance (e.g., loneliness, impaired vision, chronic nausea, fear of placement) (Wand et al., 2018). Our aim as clinicians should be to acknowledge and recognise the individual factors that drive suicidal intent in older people, and not dismiss them as trivial or ‘expected with aging’. Identification of emotional distress and/or depression requires ongoing monitoring and care, though assessing the extent of the risk is often problematic. Collaborative care models, using depression care managers or community health services can improve outcomes.

It is important to include the carers of older people who self-harm due to their knowledge of the older person’s risk factors for self-harm and as allies in implementing care plans. Most importantly, providing support to carers may alleviate the significant carer burden affecting their own mental health and that of their older relative who self-harmed (Wand et al., 2019). Decreasing isolation is important and understanding the person and their circumstances is the key to being able to help. Being aware of the services available to older people is vital when establishing a management strategy. Psychotropic medication may also be appropriate but needs to be at the lowest effective dose with a plan for monitoring response and adverse effects. Studies have shown that the use of lithium for depression and clozapine for schizophrenia can help prevent suicide. Antidepressants may be considered where depression and/or anxiety disorders are present, but because of the many factors that may contribute to suicidal thinking, the doctor’s role is far wider than merely deciding which antidepressant to prescribe.

Psychosis in Older Adults

Introduction

Psychotic symptoms in older adults occur in the context of a number of syndromes including schizophrenia (see Chapter 15), mood disorder (depression and mania; see Chapters 16 and 17), delusional disorder, dementia and delirium. In older people psychotic symptoms can be of new onset or may have existed since younger age.

Prevalence

The lifetime prevalence of schizophrenia in patients over 65 years of age is 0.3% and lifetime prevalence of delusional disorder 0.18% (Colijn et al., 2015).

There is a wide reported range in the prevalence of psychotic symptoms in dementia depending on the population sampled and the disease stage, with the prevalence of delusions approximately 40% and hallucinations 25% (Reinhardt and Cohen, 2015).

Assessment

Assessment of psychotic symptoms in the elderly is similar to and follows the same format as the general psychiatric assessment, including a strong focus on social history, activities of daily living and cognitive functioning. Obtaining collateral history from family carers or professional staff in residential care is essential to obtain an accurate assessment of functioning. Assessment must include review of medications and medical problems that can both contribute to psychotic symptoms and make the person more vulnerable to the side effects of antipsychotics and functional impairment. A comprehensive mental state examination including cognitive testing and consideration of risks (such as harm to others, self-neglect and suicide) should be part of each assessment (see box below).

Differential diagnosis of psychosis in older people: disorders and key features

- **Schizophrenia** - can be broadly defined by onset, i.e. Early onset < 40, Late onset between 40-60; Very Late onset > 60. All three subtypes share many clinical features of positive and negative psychotic symptoms with functional deficits, although later onset is associated with female gender, hearing impairment, and less severe positive symptoms (Maglione et al., 2014).
- **Delusional disorder** - onset often in middle age. Symptoms are usually characterized by focal, systematized, non-bizarre delusions and are often paranoid (jealous, erotomanic or hypochondriacal) in nature and can be associated with mild auditory hallucinations. Individuals with delusional disorder usually function well and do not deteriorate with time (Nagendra and Snowden, 2020).
- **Mood Disorders** - psychotic symptoms may be seen as part of Major depression or manic episodes.
- **Dementia** – psychotic symptoms can occur as **Behavioural and Psychological Symptoms of Dementia** (see below) (Burns et al., 2012). Lewy Body Dementia is associated with visual hallucinations.
- **Delirium** - Psychotic symptoms are of acute abrupt onset and are associated with fluctuating inattention (by definition), disorientation, and disorganized thinking both in form and content. Delusions are usually paranoid in nature but differ from those observed in schizophrenia in being vague, changeable in theme, and poorly systematized. Visual hallucinations are more commonly seen but auditory hallucinations can also be present.
- **Primary medical disorder** - psychotic symptoms seen with brain tumours (mainly frontal lobe), seizure disorders (temporal lobe seizures), autoimmune encephalopathies, Parkinson's disease (often due to anti-Parkinsonian medications), thyroid and parathyroid disorders, adrenal dysfunction (Cushing's disease), systemic lupus erythematosus, vitamin-B deficiencies and strokes.
- **Medications** and substance intoxication/withdrawal – for example benzodiazepines, anticholinergics, antihistamines, anti-Parkinsonian drugs, opiates, anti-Inflammatory drugs, anticonvulsants and steroids.

Management

Management of psychotic symptoms in older people starts with systematic assessment of symptoms including assessment of safety and excluding possible organic causes such as delirium, medical illnesses and/or medications. Therapeutic alliance and rapport have an important role in assessment and further management as they facilitate symptom disclosure as well as adherence to treatment. Baseline investigations should be carried out including physical examination, EUC, FBC, liver function, renal function, calcium, magnesium and phosphate, and a CT brain for new presentations. An EEG may be considered if there are focal neurological signs or symptoms suggestive of an underlying seizure disorder (e.g., episodic symptoms, olfactory hallucinations).

If the psychotic symptoms are part of a primary psychiatric disorder then treating the disorder should be the aim. With secondary psychosis (i.e. that associated with medical illness, delirium etc.) the focus of treatment should be the underlying cause.

Use of antipsychotics in older adults, especially those with dementia, is associated with a dose-related risk of stroke and sudden death (Maust et al., 2015). Notwithstanding this, consensus guidelines (Alexopoulos et al., 2004) recommend the use of antipsychotics in older people for schizophrenia, delusional disorder, psychotic depression, and mania with or without psychotic symptoms albeit at significantly lower doses compared with those used to treat younger people with schizophrenia. See below for discussion of management of psychosis in dementia.

An ECG (assessing the baseline QTc interval) should be performed prior to initiation of antipsychotics. The principle for use of these medications in older people remains “start low and go slow”, given the increased sensitivity and risk of side effects. Antipsychotics should be initiated at the lowest possible dose and titrated very slowly to the therapeutic dose. Close monitoring for side effects is needed, including sedation, falls, swallowing difficulties, extrapyramidal side effects, hyperglycaemia, postural hypotension, anticholinergic effects and QTc prolongation.

Non-pharmacological strategies including family education and support are essential adjuncts to drug treatment. There is randomised control evidence for cognitive behavioural social skills training (CBSST) in improving functioning in older people with schizophrenia, and both CBSST and other supportive goal-focused interventions can reduce symptom distress, and improve self-esteem, motivation and life satisfaction (Granholt et al., 2013).

Personality Disorders in Older Adults

Introduction

It was thought until recently that personality disorders “burn out” in old age. We now know differently. Although personality disorders (see Chapter 22) attenuate in middle age, the stresses and losses associated with ageing may cause symptoms to re-emerge in later life. Nevertheless, personality disorders in older adults are generally less severe and of lower prevalence than in young people (Stevenson et al., 2011b).

Prevalence

The overall prevalence of personality disorders in community dwelling older adults is approximately 10-15% (Penders et al, 2020), although higher rates are seen amongst psychiatric outpatients (up to 33%) and inpatients (up to 60%) (Stevenson et al., 2011b). The most common personality disorders in older people are obsessive-compulsive, dependent and paranoid subtypes. Older people with a personality disorder are more likely to be single, separated or divorced. Markedly higher rates of personality disorders have been reported in people with cognitive impairment and nursing home residents, raising concerns about the validity of diagnostic criteria in these populations (Penders et al., 2020). For adults across the lifespan, personality disorders often occur comorbidly with other personality disorders and with other psychiatric syndromes, although in older adults single personality disorders tend to occur more frequently. The hypothesis is that personality disorders are not qualitatively distinct syndromes but rather maladaptive variants of personality functioning. Vaillant and Milofsky (1980), described a process in men of maturation and a reduction in the detrimental effects of dysfunctional personality from age 18 to 65, related to successful marriage, work achievements, close friendships and overall mental health. There is also a decline in neuroticism (negative affectivity) up to the age of 70.

Aetiology

Personality disorders generally develop early in life – adolescence or early adulthood – due to a combination of genetic predisposition and environmental trauma. Personality disorders which caused impairment in the young adult, but which ameliorated in mid-life, can again become a problem in older adults as coping strategies that were available earlier are lost. Increased social isolation, loss of supports and less effective coping, as well as cognitive inflexibility, all combine to produce an exacerbation of symptoms of personality dysfunction. Movement into

residential aged care facilities can also pose new challenges for the older person with personality disorder. Change in personality can also be caused by brain disease such as dementia, tumour, infection, chronic illness and major life trauma.

Presentation

Personality disorders in older people are often missed by clinicians. Thus, it is important to consider this diagnosis amongst older patients, particularly given the high prevalence, associated distress, reduced functioning, and impact on treatment (Stevenson et al., 2011b). Features of personality disorder may present differently across the lifespan because of maturational processes and context changes. The outward expression of personality can change although the internal structure of the underlying personality remains stable. For example, impulsivity and reckless behaviour occur less in older adults with borderline personality disorder, but underlying dysphoria and identity conflicts may persist. The changes of aging further complicate assessment – increased introspection, slowing of thought and motor function, physical disability, hearing and visual impairments. When faced with a treatment-resistant psychiatric disorder such as chronic depression clinicians should consider the possibility of an unrecognised underlying personality disorder that needs to be concurrently addressed.

Assessment

Biographical information from informants is necessary in addition to taking a history from patients themselves. Collateral information can help identify patterns of psychosocial dysfunction and interpersonal difficulties, such as blaming others, trouble keeping employment and estrangement from family, suggestive of enduring personality pathology over the lifespan. A thorough medical assessment is important too, as clinical presentations of personality vulnerabilities in late life may be complicated by the contributions of cognitive impairment, medical comorbidities, polypharmacy and psychopathology - all more common with ageing.

Treatment

The treatment of personality disorders in older adults is complex. Personality disorder in older adults is associated with strain in the clinician-patient interpersonal relationship, treatment rejection and non-adherence. The prognosis of medical comorbidity, which is higher among older adults with personality disorders, is worse than those who do not have personality vulnerabilities. This is often attributed to lack of social supports and coping skills to aid recovery, and conflict with clinicians resulting in inadequate or suboptimal physical health care. As a result, compounded by the increased rate of suicide, lifespan is often decreased. When

major psychiatric disorders and personality disorders coexist, treatment should be directed first at the psychiatric disorder. However, in those with a comorbid personality disorder, symptoms of the psychiatric disorder are usually more severe, take longer to treat, result in more functional impairment and may not completely resolve. Older adults with personality disorder are more likely to be socially isolated due to family estrangement and when entering a residential aged care facility they tend not to “fit in” and may engender conflict.

These complexities explain the frequent therapeutic nihilism associated with personality disorder. Treatment of personality disorders in older adults has been underexplored and may stem from this therapeutic nihilism (Penders et al., 2020). However, one recent study examining schema therapy for Cluster C personality traits/disorders, (Videler et al., 2018), and some small case series, demonstrated that treatment of personality disorders in late life may be both feasible and effective (Penders et al., 2020). When embarking on treatment, in addition to evaluating the individual needs, degree of motivation and engagement, cultural perspectives, type and severity of the personality vulnerability in the older adult, clinicians need to consider sensory, functional and cognitive impairment. The approach to treatment therefore needs to be tailored to the individual.

While treatment of the major psychiatric disorder is usually pharmacological, personality disorder responds better to psychological approaches, such as dialectical behaviour therapy (DBT), interpersonal therapy, cognitive behavioural therapy (CBT), schema therapy or problem-solving therapy. The spectrum of treatment may range from supportive structured approaches (e.g., psychoeducation and behavioural advice), to coping or adaptation (e.g., social skills training) or interventions to change personality traits (e.g., schema therapy, DBT). Teaching more adaptive coping strategies and providing extra social supports can also be of benefit.

Delirium in Older Adults

Delirium is the most common serious mental disorder in old age and is frequently unrecognized, particularly hypoactive delirium, the most prevalent subtype. Around 20% of older people are delirious at hospital admission, while up to 40% develop delirium during their hospital stay. For people with dementia, rates of delirium during hospitalisation may be as high as 80% (De, et al 2017). Up to 60% of patients aged 75 and older who live in nursing homes have delirium (Inouye, 2006).

Aetiology and Risk Factors

Predisposing risk factors for delirium in hospitalised patients are (Fong et al., 2009; Inouye, 2006):

- advanced age (over 70)
- pre-existing cognitive impairment (such as dementia)
- depression
- dehydration
- visual impairment
- male gender
- other cerebral injury or underlying chronic organic mental syndrome
- addiction to alcohol or drugs
- polypharmacy
- terminal illness
- frailty and functional impairment
- hearing impairment

As advanced age per se confers vulnerability, almost any physical illness can precipitate delirium in older adults. Precipitating factors include physical restraint, catheterisation, malnutrition, having more than three new medications added and any iatrogenic event such as a procedure, fall, infection or complication (Inouye and Charpentier, 1996; Kalish et al., 2014). Pain, constipation, urinary tract infections and urinary retention are common precipitants for delirium. Medications – commonly anticholinergics and benzodiazepines – are the sole precipitant for delirium in up to 40% of cases.

Diagnosis and Treatment

Prompt assessment, differential diagnosis and treatment are essential. First, identify the syndrome, and second, determine and treat the organic aetiology (Wu and Hilmer, 2015). The hallmarks of delirium are sudden onset, inattention, and fluctuation in cognition and level of consciousness. Thought disorder, delusions and hallucinations may also occur as part of the syndrome but are present in less than 40% of cases of delirium. There are numerous validated screening tools for delirium including the 4AT, Confusion Assessment Method (CAM), and the Single Question in Delirium (Sqid) (De and Wand, 2015). Delirium must be distinguished from other organic syndromes such as dementia and other psychiatric disorders such as depression, mania, schizophrenia, and atypical psychoses.

Multicomponent non-pharmacological interventions are effective for both the prevention and treatment of delirium (Wilson et al., 2020). Up to a third of incident delirium can be prevented using such interventions. These strategies include reorientation and improving communication (hearing and vision aids, dentures), early mobilisation, pain relief, normalisation of sleep patterns, optimising oxygen saturation, and addressing hydration, nutrition and constipation. It is important to educate the older person's family about delirium, as they may be distressed by the symptoms in their relative, but also willing to assist in management. Any form of physical restraint should be avoided, including catheters, high bed rails, and intravenous lines where possible, as they are associated with prolonged delirium and adverse outcomes. These non-pharmacological measures are an important part of the secondary prevention of complications of delirium, including falls and associated fractures, dehydration, pressure areas, acute renal failure and malnutrition. Educational interventions for healthcare professionals about delirium have been shown to improve professional practice of clinicians and outcomes for patients (Lee et al., 2020).

There is no single effective medication treatment for delirium. There is little evidence to support the routine use of psychotropics in delirium, but significant risk of adverse effects (Wilson et al., 2020). Therefore, pharmacotherapy is considered second line for the management of delirium and should be reserved for people at risk to themselves (for example by misadventure, severe agitation, self-neglect or behaviours preventing treatment of the underlying medical condition) or others (for example, through assaults on family/clinicians). It is important to obtain informed consent for use of antipsychotics in this context.

Recent systematic reviews and meta-analysis of antipsychotics specifically, for the prevention and treatment of delirium concluded that there was no evidence of benefit (delirium incidence, duration, severity, hospital or ICU length of stay, mortality) (Burry et al., 2018; Neufeld et al., 2016). Further, antipsychotics may be associated with greater delirium symptomatology and reduced survival when compared to placebo in certain populations such as palliative care (Agar et al., 2016). Studies do not suggest significant differences in efficacy in treating delirium with haloperidol versus atypical antipsychotic agents, such as olanzapine, risperidone and quetiapine, but report higher rates of extrapyramidal side effects with haloperidol (Meagher et al., 2013), especially at doses 2.5mg/day, and particularly in people with Dementia with Lewy Bodies (which is characterised by neuroleptic sensitivity, which is often severe). The oral route is preferred to the intramuscular route (Wu and Hilmer, 2015) and intravenous administration should be avoided.

Benzodiazepines are associated with greater incidence of delirium when given prophylactically. In terms of delirium treatment, two partially controlled

studies, one examining alprazolam and the other lorazepam, showed no advantage in treating agitation associated with delirium, and less benefit and more adverse effects compared to antipsychotics, respectively (Lonergan et al., 2009). There are no adequately controlled trials to support the use of benzodiazepines in the treatment of delirium among hospitalized older adults, but there is evidence of increased sedation and worsening the health status of delirious patients (Lonergan et al., 2009). The exception to this is delirium secondary to substance withdrawal, where short acting benzodiazepines are the drugs of choice (e.g., alcohol or benzodiazepine withdrawal delirium).

Prognosis

Delirium is associated with adverse outcomes such as increased length of hospital stay, greater morbidity and mortality, increased risk of, earlier onset and progression to dementia, development of post-traumatic stress disorder, functional decline, loss of ability to live independently in the community and increased rates of admission to nursing homes. Mortality rates among hospitalised patients with delirium range from 22% to 76% (Inouye, 2006). However, if recognized early and the underlying cause(s) are treated aggressively, full recovery is possible.

Dementia (Major Neurocognitive Disorder) and Changed Behaviours (Formerly Behavioural and Psychological Symptoms of Dementia, or “BPSD”)

Approximately 9% of Australians over 65, and 30% over 85, have a diagnosis of dementia (Laver et al., 2016). Dementia is a clinical syndrome characterised by a cluster of symptoms and signs manifested by difficulties in memory, disturbances in language, psychological and psychiatric changes, and impairments in activities of daily living (Burns and Iliffe, 2009). According to DSM-5-TR (American Psychiatric Association, 2022) dementia is defined by:

1. Evidence of substantial cognitive decline from a previous level of performance in ≥ 1 domains based on the concerns of the individual; a knowledgeable informant, or the clinician.
2. Decline in neurocognitive performance, typically involving test performance in the range of ≥ 2 standard deviations below appropriate norms (i.e. $< 3^{\text{rd}}$ percentile) on formal testing or equivalent clinical evaluation.
3. The cognitive deficits are sufficient to interfere with independence (i.e., requiring minimal assistance with instrumental activities of daily living).

The kind of cognitive or intellectual functions affected by dementia include language (expressive and receptive), orientation, attention, memory, judgment, planning and praxis (e.g., motor, visual tasks).

There are more than 100 causes of dementia. Alzheimer's disease is a specific disease entity and is the commonest cause of dementia followed by Vascular Dementia, although mixed dementia commonly occurs. Other causes of dementia include Lewy Body Dementia, Fronto-Temporal Dementia and Alcohol Related Brain Damage. For an approach to diagnosis, see the box below.

A major initial aim when assessing people with neuro-cognitive disorder is to identify any reversible causes of dementia. In particular consider whether the person developing memory loss or word-finding difficulty could have, for example, a meningioma, other space-occupying lesion (e.g., subdural haematoma, cerebral abscess), vitamin or calcium deficiency, or thyroid disorder. This is particularly relevant in people with young-onset dementia.

In managing dementia, Australian Clinical Practice (Guideline Adaptation Committee, 2016) recommend an emphasis on promoting and maintaining independence with activities of daily living, continuing exercise and the pursuit of meaningful activities, as well as maintaining adequate nutrition and oral health and the treatment of medical and psychiatric co-morbidities (Kurrle et al., 2012; Laver et al., 2016).

A systematic approach to diagnosing dementia

- Patient and informant history taking,
- Cognitive assessment using a screening tool with established reliability and validity:
 - The Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005); or
 - Kimberley Indigenous Cognitive Assessment tool for Aboriginal and Torres Strait Islander populations, (KICA; LoGiudice et al., 2006); or
 - Rowland Universal Dementia Assessment Scale (RUDAS; Story et al., 2004) for non-English speaking populations]].
- Medication review,
- Blood screening tests (full blood count and profile, thyroid function, B12 and folate)
- Brain CT or MRI to rule out reversible causes of dementia (Laver et al., 2016).

Acetylcholinesterase inhibitors and memantine may be prescribed for people with Alzheimer's dementia and mixed (Alzheimer's + vascular) dementia. These medications have modest evidence for slowing functional decline (Laver et al., 2016) and reducing changed behaviours. Anti-amyloid monoclonal antibodies such as lecanemab and aducanumab have recently been approved for the treatment of Alzheimer's disease in countries including Australia and the USA (Cummings, 2023). These medications are the first disease-modifying therapies available for Alzheimer's disease. However, clinically significant benefits are modest and potential adverse effects may be serious.

Changed behaviours, (formerly Behavioural and Psychological Symptoms of Dementia; BPSD) are symptoms of disturbed perception, thought content, mood and behaviour occurring in people with dementia (Burns et al., 2012). They occur so commonly as to be virtually ubiquitous in most forms of dementia, with up to 97% of people experiencing changed behaviours of variable severity during the course of their illness (Brodaty et al., 2001). They include:

- agitation
- aggression
- calling out/ screaming/swearing
- disinhibition (sexual)
- wandering
- nighttime disturbance
- shadowing
- depression
- irritability
- elation/euphoria
- anxiety
- apathy
- delusions
- hallucinations

Reliance on medications to “manage” these symptoms occurs commonly and is increasing in Australia (Snowdon et al., 2011a). While admittedly there has been some reduction in antipsychotic use in response to national de-prescribing campaigns (Capacity Australia), benzodiazepine prevalence is higher, and sedating antidepressant and ‘PRN’ psychotropic prescribing is widespread (Westbury et al., 2018). This is despite a growing body of evidence for non-pharmacological interventions (Chenoweth et al., 2009; O'Connor et al., 2009; Testad et al., 2014). Instead, consensus guidelines recommend the use of multidisciplinary,

individualised (person-centred) psycho-social and environmental approaches as a first line approach to changed behaviours (NSW Heath, 2022).

Such person-centred care relies on comprehensive assessment involving observation, measurement and monitoring of changed behaviours, to assess the antecedents, triggers and consequences of behaviours (the ABC approach) – often a communication of unmet needs or a response to too much or not enough stimulation in the environment. One of the most common unmet needs in dementia is pain, the detection of which can be problematic in people who have language deficits, and therefore pain assessment in dementia should be based on observation of pain behaviours rather than self-report. If pain is suspected in the presence of BPSD, use of analgesia should be the first line pharmacological treatment (Husebo et al., 2011).

Psychotropic medications may be indicated when there is severe and complex risk of harm, when symptoms are psychotic in nature, and when psychosocial interventions have been exhausted or when there are comorbid pre-existing mental health conditions (Peisah and Skladzien, 2014). These medications have a range of serious side effects and are associated with increased mortality for people with dementia. The evidence supporting the effectiveness of psychotropics in treating BPSD is modest at best, with most support for atypical antipsychotics. International data suggest that up to 20% of people with dementia who receive antipsychotic medications derive some benefit from the treatment. The only psychotropic which is listed by the PBS for treating BPSD is risperidone and this is the medication which has the strongest evidence for its effectiveness (recommended dose range from 0.25mg- 2mg daily) (Peisah and Skladzien, 2014). Severe sensitivity to both typical and atypical antipsychotics is seen in approximately 50% of patients with Lewy Body Dementia and antipsychotics are best avoided in this group without consultation (McKeith et al., 2005).

Family and professional caregiver education and support is also essential to management of changed behaviours, to minimise restraint use and to improve the quality of life for the person with dementia (Laver et al., 2016).

Capacity, Ethical Issues and the Older Adult

Human rights frameworks such as the United Nations Convention on the Rights of Persons with Disabilities (CRPD) (2006), of which Australia is a signatory, inform best practice care for older people with mental disorders and dementia. The CRPD also underpins our approach to capacity and decision making (O'Neill and Peisah, 2021).

Capacity (or competence) is the ability to make decisions. Capacity is task - or domain - specific. This means that capacity to consent to medical treatment is different from capacity to make a decision to execute a power of attorney, write a will, enter a contract or deed, appoint an enduring guardian or to manage financial affairs. Global capacity, where a person is deemed as either capable or incapable of making all decisions, has been rejected both in law and in human rights discourse. Therefore, it is inappropriate to state that a person “lacks capacity” without further reference to the type of capacity task. A person’s capacity can vary in different circumstances, at different times, and even within domains for different types of decisions. For example, within the domain of capacity for medical treatment consent, capacity to give consent to a blood test or antibiotics (a much simpler decision) is different from capacity to give consent to amputation (O’Neill and Peisah, 2021).

Medical practitioners have human rights, ethical, legal and professional responsibilities to obtain consent before treating any patient. This equally applies to patients who lack capacity to give consent for their own treatment. For a person’s consent to be valid, the person must be:

- competent (or have capacity) to make the treatment decision;
- acting voluntarily without pressure or duress; and
- provided with enough relevant information about the treatment options and alternatives to enable them to make the decision.

Capacity is broadly defined as the ability to:

- (i) To understand the specific situation, relevant facts or basic information about choices
- (ii) To use reasoned processes to weigh the risks and benefits of the choices
- (iii) To communicate relatively consistent or stable choices.

As a starting point, in the common law (judge-made law across the Commonwealth), there is a presumption of capacity in relation to anyone 18 years or more. Following from this, it is often said that incapacity is not “status” or diagnosis bound, which means that just because someone has a diagnosis of dementia or schizophrenia, a doctor cannot assume that they lack capacity (Peisah et al., 2009). A valid “trigger” such as an acute deterioration in mental state, or apparent inability to understand what has been explained must exist to rebut the presumption of capacity. When assessing capacity, after responding to such a trigger, it is important that the doctor provides the patient with all the relevant information in a form that the patient can understand. This includes information about the nature of their condition, and the nature and effects of any treatment options, including

benefits and side effects, and the consequences of refusing treatment. The patient should then be asked to repeat their understanding of this in their own words. An answer to the closed question: “Do you understand?” is never proof of capacity.

In New South Wales, if a person lacks capacity to give consent for their own treatment, consent must be obtained from their “person responsible”, (Guardianship Act 1987(NSW)) which is, in order of priority:

- (i) an appointed guardian (including an enduring guardian) who has been given the right to consent to medical and dental treatments or, if there is no guardian,
- (ii) the most recent spouse or de facto spouse (including same-sex partner) when the spouse or de facto has a close and continuing relationship with the person or, if there is no spouse or de facto spouse,
- (iii) the unpaid carer or the carer at the time the person entered residential care (note: recipients of a government carer benefit are not considered to be paid) or, if there is no carer,
- (iv) a relative or friend who has a close personal relationship with the person.

Concluding Statement

The treatment of mental disorders in old age is complex and involves consideration of a myriad of bio-psycho-socio-ethical-legal and human rights considerations. Specifically, older people have a human right to equitable access to high quality mental health care on a par with that provided to younger people. Moreover, in Australia, there is no reason to expect less than a high quality of life in later life, regardless of physical or mental co-morbidities. To undertreat and dismiss such as “part of ageing,” is ageist.

Further Reading

Capacity Australia. Empowered Project: Empoweredproject.org.au

Guideline Adaptation Committee. (2016). Clinical Practice Guidelines and Principles of Care for People with Dementia. Sydney. Guideline Adaptation Committee:

sydney.edu.au/medicine/cdpc/documents/resources/dementia-guidelines-full-document-website.pdf.

NSW Ministry of Health (2014). Wellbeing later life your guide to programs and activities.

health.nsw.gov.au/mentalhealth/resources/Publications/wellbeing-in-later-life.pdf

O'Neill N., Peisah C. (2021) *Capacity and the Law* 4th Edition, Australasian Legal Information Institute (AustLII) Communities. Online:

austlii.community/wiki/Books/CapacityAndTheLaw/

The program resource page for Behaviour Management – A Guide to Good

Practice: *Managing Behavioural and Psychological Symptoms of Dementia (BPSD)*
dementiaresearch.org.au/bpsdguide.html

Chapter 27: Emergency Psychiatry

Ralf Ilchef

Specialty of Psychiatry, Sydney Medical School, The University of Sydney, Australia, and Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

and

Nick O'Connor

Specialty of Psychiatry, Westmead Clinical School, The University of Sydney, Australia.

Introduction

Psychiatric emergencies are acute disturbances in thought, behaviour, mood, or social relationship that require immediate intervention to save the patient and/or others from imminent danger (Baker, 2022). Emergency psychiatry is the clinical assessment and management of these events.

Psychiatric emergencies can arise in any setting, including the person's home or a public place. Increasingly, and for a number of reasons, people experiencing psychiatric emergencies are brought to the Emergency Departments of our hospitals. This chapter describes these psychiatric emergencies and their management.

A doctor's approach and style of thinking and acting in a psychiatric emergency is quite different to a psychiatrist's behaviour in a quiet consulting room with a cooperative patient who is a good historian. Frequently, there is an imperative to make a rapid assessment on imperfect information. A sound knowledge of psychiatric conditions and treatments is essential, but not sufficient. In the emergency situation, the psychiatrist needs to be assessing the patient and the situation on a number of levels. What is the nature of the patient's distress? What are the likely diagnostic and aetiological hypotheses? What information can family/ambulance officers/police/Emergency Department staff provide? What are the immediate risks (aggression, self-harm, absconding, misadventure, medical emergency)? What is going to be the safest setting to manage the patient? Does the

patient require involuntary detention under mental health legislation? Who needs to be involved in the care of the patient? What advice and direction do they require? All the while, psychiatrists dealing with a psychiatric emergency must contain their own anxieties in order to bring calm and authority to the situation and to provide explanation, direction and reassurance to the patient, the family and colleagues. The urgent clinical situations seen in psychiatry can be divided into behavioural and medical emergencies.

Behavioural Emergencies

Acute Severe Behavioural Disturbance

Acute Severe Behavioural Disturbance (ASBD) can come in many forms, from agitation, pacing, shouting, nonsensical speech and bizarre posturing to withdrawal, mutism and torpor. Assessment follows the principles of general medicine and includes (i) obtaining as full a history as possible; (ii) corroborating or clarifying that history by speaking to the patient's family and friends and to witnesses and health care staff; (iii) performing as complete an examination as possible, with specific emphasis on the neurological and endocrine systems; and (iv) ordering appropriate investigations. An early priority is assessing your own safety as well as that of the patient, other patients, visitors and staff. A life-threatening medical emergency has usually, but not always, been identified at triage.

Once this has been done, the diagnostic schema usually known as Fould's hierarchy (see Figure 27.1) can be helpful. This framework used in emergency assessment grossly oversimplifies Fould's and Bedford's complex and interesting work on relationships between classes of mental illness (Foulds and Bedford, 1975) but is nonetheless clinically valuable. The hierarchical pyramid reinforces three helpful concepts. Firstly, each item on the pyramid is a more urgent diagnostic consideration than those below; secondly, each can mimic the symptoms of those below; and thirdly, items lower down the pyramid are encountered more frequently than those above.

The first diagnostic consideration, the tip of the pyramid, is 'organic', in other words, a physiological cause of acute brain disturbance. It is critical to consider this as these are the disorders that can kill patients rapidly. No great harm is done when a psychiatric disorder such as mania or schizophrenia is misdiagnosed as an organic condition, but the reverse error can be life-threatening.

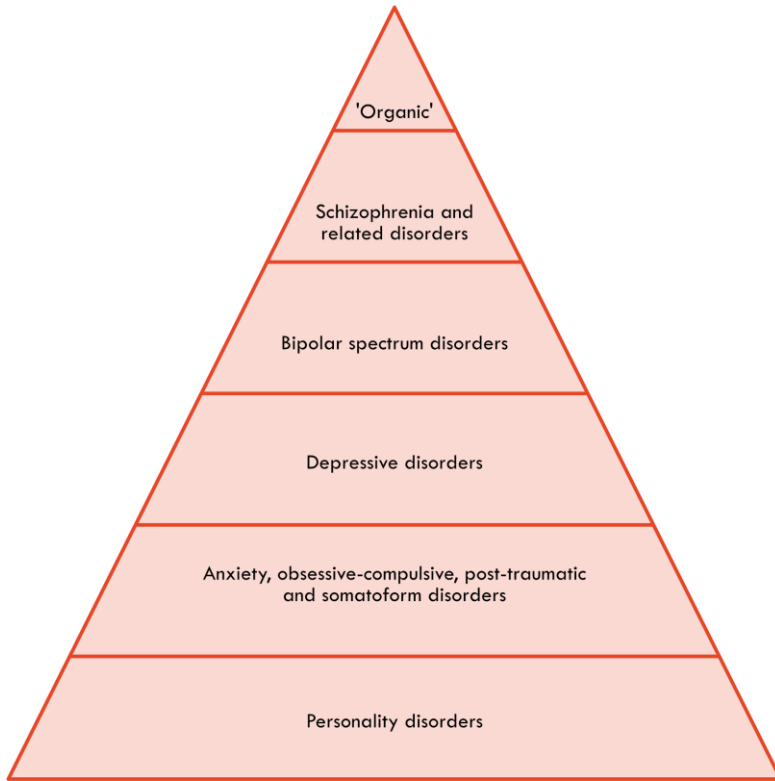


Figure 27.1. A Primer of clinical psychiatry, adapted from ClinicalGate (clinicalgate.com)

When considering an organic diagnosis, think of three broad categories: (i) exogenous (in other words intoxication or withdrawal states involving recreational drugs or prescribed medications); (ii) intracranial (such as bleeding, space-occupying lesions, cerebrovascular events or viral or autoimmune encephalitis); and (iii) systemic (any end organ failure or marked metabolic disturbance can cause a confusional state, most commonly hypoxia, sepsis, hypo- or hyper-glycaemia, acute renal failure, acute hepatic failure, profound hypo- or hyper-thyroidism and silent myocardial ischaemia). Context is important, and you are likely to have different diagnostic priorities for a young man seen in ED at 2am on a Sunday morning than for an elderly woman assessed twelve hours post-operatively on a surgical ward.

If you are satisfied that the disturbance is unlikely to be organic, actively consider psychosis. It is quite easy to spend an hour with a patient and not uncover psychotic symptoms unless they are specifically pursued. Ask about auditory

hallucinations, hallucinations in other modalities, unusual (persecutory, grandiose, bizarre) thoughts, ideas of reference, thought interference and passivity phenomena.

Next specifically enquire about symptoms of mania and major depressive disorder. Then consider anxiety disorders, post-traumatic states and OCD. Finally think about an acute stressor in a person with personality vulnerabilities.

If an 'organic' diagnosis is made, definitive medical management is indicated, with liaison psychiatry involvement to assist in psychological, environmental and (where indicated) pharmacological management of the condition. For others, reassurance, a low stimulus environment, presence of a staff member if indicated, active involvement of family or friends, judicious pharmacological management of agitation (usually with benzodiazepines) and specific treatment of the underlying psychiatric disorder are priorities.

Aggression

Approaching the patient with Acute Severe Behavioural Disturbance (ASBD) who is loud, pacing, agitated, or threatening requires as much clear thought as possible, in a very tense situation ("First take your own pulse..."). If you are asked to see a person who is agitated, aggressive or escalating in behaviour, try to ensure that it is done in a clearly visible area, where you can be seen by other staff, and can readily leave. Ensure that the patient can't get between you and the door. Find out as much as you can about the patient ahead of time by reading the notes, looking at the ambulance or police report and talking to anyone who might have accompanied the patient.

Approach the patient slowly, in a calm and low-key manner, and with an open stance (arms by your sides, palms forward). Pay close attention to your own subliminal cues; your body is trying to tell you something. If the patient's manner is very threatening, ensure that a sufficient number of staff members are very close by (at least five, including security). Carry out the history and examination as fully as possible in the circumstances. Do whatever eases a charged atmosphere, for example offer the patient a drink of water. Don't react, don't be aggressive and don't make promises you can't keep. If the person is highly agitated, offer an anxiolytic medication (usually a benzodiazepine, or a sedating antipsychotic medication). The most likely diagnoses to consider include:

1. Alcohol or stimulant (methamphetamine, cocaine) intoxication
2. Organic brain syndrome (developmental disorder, acquired brain injury, dementia)
3. Acute psychosis and/or mania (see Chapters 15 and 17)

4. Cluster B (antisocial, borderline, narcissistic) personality organization in crisis (see Chapter 22)

The initial approach to the irritable, agitated, or aggressive patient is one of verbal de-escalation. The aim is to restore a workable clinician-patient dialogue. The Mental Health clinician is often the leader and 'culture bearer' in these challenging situations. The most important issue is attitude. Fundamental to successful interventions with an agitated patient are: positive regard for the patient, the capacity for empathy and patience. De-escalation frequently takes the form of a verbal loop in which the clinician listens to the patient, finds a way to respond that agrees with or validates the patient's position, and then states what he wants the patient to do, for example, accept medication, sit down, etc. (Accinni et al., 2021).

In rare situations, physical restraint and parenteral sedation may need to be considered. This should not be used unless there is no other possible way of ensuring the safety of your patient, other patients, visitors and staff (and of course yourself) from harm. The principles in this situation are to ensure sufficient preparation (medications drawn up, space cleared of potential implements), sufficient numbers of staff (five-point restraint: one person for each limb, and the head)), prompt action and a paramount consideration for the safety, welfare and dignity of the patient. Recall that many patients presenting with severe behavioural disorders will have a history of trauma and may find restraint especially distressing. Before working in this area, ensure you are familiar with a detailed and up-to-date protocol for the use of restraint of the agitated patient.

Acute Psychosis

The patient with an acute psychosis may not be aware that they are unwell. The hospital, and especially the Emergency Department, can be a strange and frightening environment. While many feel reassured that they are in a medical facility, the hospital setting exacerbates the condition of some patients. The person with acute psychosis may find the world a threatening place and may not trust people, or even believe they are not who they purport to be. It is important to understand that the patient's agitation may arise from persecutory or referential ideas. The patient requires careful assessment, explanation and reassurance. If the person is presenting with their first episode of psychosis, they and their family are entering a strange and intimidating world of mental health services and often the psychiatric inpatient unit. How the clinician interacts with the person and their family and manages the situation can leave a lasting impression. Again, as with the aggressive or agitated patient, the mental health clinician has an important role as leader and culture bearer. Positive regard, empathy and patience are required.

Investigations are done on patients presenting with a first episode of psychosis to exclude organic causes and contributory factors, and may include: FBC, EUC, LFTs, TFTs, Brain MRI or CT, EEG, B12, Folate, antineuronal antibodies to exclude autoimmune encephalitis, urine drug screen and ECG (as many medications have potential cardiac side effects). Further investigations may be required depending on the history obtained.

If the patient is known to have schizophrenia or a similar diagnosis, an antipsychotic may be initiated in emergency. However, if a drug-induced psychosis is a possible diagnosis, it may be worth holding off on commencing an antipsychotic with a view that a drug-induced psychosis will usually resolve without antipsychotics over a few days to a week. In either circumstance the patient's distress may be assisted by the short-term use of benzodiazepines.

Acute Mania

The manic patient presents as elevated, loud, expansive and overactive or irritable, argumentative and hostile. Usually they are distractible and this can often be used to advantage to avoid confrontation. Often the manic patient will not have slept for a number of days and may not have attended to their hydration and self-care. One severe case was a man in his 40s who was brought by police having been found half-naked, bare foot and hollering in a national park. He had been out there for 4 days in the burning sun by day and low temperatures at night, eating and drinking only what he could find in the bush. He was hoarse from shouting "God's Word", severely sunburned and his feet lacerated from walking on sharp stones. Here the disruptive behaviour is an interaction between his mania, dehydration, sunburn and his injuries. All require management.

The principles that apply to the care of the acutely psychotic patient apply with the manic patient. The investigations required are the same as for other psychotic illnesses such as schizophrenia. One important point of emphasis is that the manic patient requires containment and sleep. Sedation in the first 48 hours is frequently required. Antidepressant medication should be discontinued. If the patient is known to suffer bipolar disorder an antimanic agent (lithium, valproate) or a second generation antipsychotic (or a combination of the two) may be commenced in the emergency department. However, as is the case with acute psychosis, if a drug-induced psychosis is a possible diagnosis, it may be worth providing benzodiazepines alone initially.

Catatonia

Catatonia is a rare presentation of acute psychosis, most often seen in organic disorders, schizophrenia-spectrum disorders, acute mania and amphetamine-associated psychosis. The clinical hallmarks are marked psychomotor slowing often to the point of immobility, mutism, resistance to passive movement (“gegenhalten”) and so-called “waxy flexibility” (limbs remain as positioned by the examiner) although incomplete forms are more commonly seen. In fact some studies suggest that in an attenuated form catatonia can be seen in up to 10% of psychiatric inpatients (Heckers and Walther, 2023). The initial clinical priority is prompt exclusion of neurological, metabolic and other causes of sudden onset rigidity and unresponsiveness. Immaculate medical and nursing care of the immobile patient is essential, including adequate hydration (intravenous fluids if needed), regular turns, monitoring of pressure areas and where indicated DVT prophylaxis. Up to three quarters of patients will respond to parenteral lorazepam 1-2mg 6th hourly as needed, up to 6mg/day. For patients who are unresponsive or deteriorating clinically, electroconvulsive therapy is the treatment of choice. Antipsychotic medication is generally held to be ineffective and should in any case be withheld until neuroleptic malignant syndrome is excluded, but may be needed during recovery for residual psychotic symptoms.

Suicidality and Self-Harm

Assessing a patient for suicidality might occur after a suicide attempt or act of self-harm, after a patient has presented with suicidal thoughts or after concerns have been raised during an unrelated presentation. Previously guidelines to suicide risk assessment have tended to focus on risk factors and encouraged stratification into ‘high’, ‘moderate’ and ‘low’ risk groups. However, the evidence now makes plain that a risk-stratification approach cannot guide management and should not be undertaken (Heckers and Walther, 2023). A careful, unhurried history, augmented by any corroborative sources you can find, is of paramount importance, for at least five reasons.

Firstly, it helps to allow a diagnosis to be made, and use of Fould’s hierarchy can let us determine whether organic factors, psychosis or severe mood disturbance are present, each of which require a specific response. Although diagnosis is not a strong prognostic factor for suicide overall, some symptoms seen in particular mental disorders (persecutory delusions in schizophrenia, severe major depression with agitation and/or psychotic features) indicate a need for a particularly careful assessment. Secondly, elements in the history should influence your formulation and management plan: these include past suicide attempts, a family history of completed suicide, impulsiveness, poor physical health, chronic pain and substance abuse. Thirdly, it allows an assessment of capacity, which is vital if consideration is to be

given to involuntary treatment. Fourthly, it allows for engagement with the patient; there is nothing like taking the time to really hear someone's story to lessen resistance and build trust.

Fifthly, with empathy and understanding it can lead to a formulation, to try to answer the question "Why is this person presenting with this problem at this time"? If uncertain, come back and see the patient a second time. This will give you important information about the level of engagement and the variability of their clinical presentation.

A decision to discharge a patient who has expressed or elicited suicidal concerns should not be made without a thorough discussion with a senior mental health clinician. Factors that will influence this decision include:

- severity of the attempt or intensity of the ideation,
- the quality of engagement,
- the patient's current expressed attitudes,
- the availability of a responsible adult who is aware of the situation to be with the patient and
- the availability of after-hours mental health services.

The Patient with Borderline Personality Disorder in Crisis

People living with borderline personality disorder (see Chapter 22) have usually had the unlucky combination of an exquisitely sensitive and over-emotional temperament and an invalidating or abusive developmental background. They are prone to discontinuities of affect, cohesion and sense of self. The most common emergency presentations are with overdose, self-cutting and suicide attempts. Many self-harm presentations are attempts to cope with unbearable and overwhelming emotions or complete lack of feeling in a numbed and often depersonalized state. With patients who present repeatedly there can be a temptation to discount the importance of an individual act of self-harm, but it is important to remember that members of this group have a 10% lifetime risk of suicide.

The key to assessing and managing the patients with borderline personality disorder who present in crisis is the ability to connect and engage (Meares, 2012). In addition to the positive regard, empathy and patience required in most psychiatric emergencies, how the clinician handles transference and countertransference is vital. There are some particular points additional to those of the standard emergency psychiatric assessment principles that apply to the patient.

Connecting with the patient with borderline personality disorder

1. Listen (put your agenda on hold)
2. Choose what is most alive (amplification)
3. Find and affirm that which is positive (validation)
4. Use the language of the patient
5. Attunement to the patient's feeling state (empathy)
6. Stay aware of your own feelings, thoughts,
7. Avoid using the personal pronoun 'you' (speak about things in a more general sense: "it seems.....")
8. Acknowledge disruptions and your own disjunctions

The Patient with an Eating Disorder in Crisis

Patients with severe eating disorders (see Chapter 21) have the highest mortality rate of any psychiatric disorder. Most often the acute crisis involves a patient with anorexia nervosa presenting after a period of food restriction and purging. Many of these patients also have depression or borderline personality disorder and so may present with an episode of self-harm when the more life-threatening aspect of their situation is their physiological state of starvation.

The key parameters that must be assessed to determine whether the anorexic patient requires a medical admission of re-feeding and restoration of their physical health are:

1. BP < 90/60mmHg or BP postural drop (> 20 mmHg).
2. BMI < 14kg/m² in women or 16kg/m² in men plus acute medical issue (e.g. infection).
3. Significant electrolyte disturbance (i.e. serum potassium < 2.5mmol/L, hypophosphatemia).
4. Haematological abnormalities such as significant anaemia (Hb < 70) or severe neutropaenia < 1.0 x 10⁹/L.
5. LFT dysfunction (3 upper limit of normal).
6. Hypothermia (temperature < 35.5°C).
7. Renal failure
8. Symptomatic hypoglycemia (< 2.7mmol/L).
9. Heart rate < 40 bpm or > 110 bpm or postural tachycardia of increase of > 10 bpm.

Refeeding syndrome is a potentially life-threatening complication of medical treatment for profoundly starved patients, in which an array of electrolyte and haematological imbalances including hypophosphataemia, hypokalaemia, low thiamine and white cell dysfunction can lead to multi-organ damage, neurological impairment and death. Recent studies suggest the risk may be less than previously considered, and that in most cases rapid re-feeding under close medical and nutritional supervision can safely take place (Staab et al., 2022).

Medical Emergencies

General Considerations

The following conditions are life-threatening medical conditions seen in patients with psychiatric disorders. There remains a stigmatising tendency to view unexpected clinical events in medically hospitalised psychiatric patients as “behavioural” until proven otherwise, sometimes with catastrophic results. Any acute deterioration in your patient needs prompt review, with active consideration of these life-threatening medical complications.

Delirium

The syndrome of delirium results from disruption of normal brain function due to biochemical, electrical, or mechanical disturbances. Delirium can be thought of as a ‘brain fever’. As with fever in a respiratory infection, it is a reliable indicator that something serious is going on, and that investigation and intervention are required. Possible outcomes include death and dementia and as with any other acute organ failure, delirium should be considered a medical emergency. The patient requires hospitalisation and investigation unless the cause is known and imminently correctable.

While each patient’s clinical presentation will vary, sometimes considerably, because of personality factors or the nature of the organic insult, certain features make the diagnosis of delirium relatively simple. The hallmark of delirium is acute onset of a state of *clouded consciousness*: a reduction in the clarity of awareness of the environment. This means the patient has difficulty shifting, focusing and sustaining attention, and is easily distracted by irrelevant stimuli. Impairment of consciousness ranges on a continuum from excited hypervigilance and overactivity - to barely discernible dulling - to coma. Characteristically this fluctuates, often worsening at night with fatigue and decreased environmental stimulation.

Often the patient is unable to accurately describe: the hour of the day, the day of the week, the month, the year. He is unable to find his way in the ward. He

does not know which ward he is in, or which hospital, nor indeed which city and sometimes which state or even country. He may indicate he does not know who the doctor is or not recognize family members. A corroborative history from family confirms these capacities were recently intact. Memory impairment occurs because of failure to register new information, disturbance of short term processing and distorted thinking.

Perceptual disturbances are characteristically a hypersensitivity to stimuli, illusions, misinterpretations and hallucinations. Illusions are distorted perceptions: the folds of bedclothes become a geography of mountain ranges. Misinterpretations are correct perceptions given a wrong meaning: a door slamming is thought to be a pistol shot. Hallucinations are false perceptions in the absence of sensory stimuli. Visual hallucinations commit the physician to *excluding* the diagnosis of delirium.

Thinking is disturbed, usually slowed, and characteristically the patient's utterances are no longer goal-directed. Reasoning is defective, complex ideas cannot be formulated, and there is difficulty grasping and understanding. The thought disorder of delirium is most commonly manifest in fragmented and rambling speech. Delusions may be reported, often secondary to perceptual anomalies but these are disorganised rather than systematised.

Psychomotor activity is also disturbed. The patient with hyperactive delirium is restless and hyperactive and this activity is purposeless. He gropes and picks at his bedding or attempts to stand in or get out of bed. Alternatively, and more commonly, the patient with hypoactive delirium may be slowed, sluggish and apathetic. The sleep-wakefulness cycle is usually disturbed, the patient somnolent and drowsy during the day, aroused and overactive at night.

Delirium is often missed in medical settings, especially hypoactive rather than hyperactive forms. As delirium indicates a very significant risk of morbidity and mortality, and a risk of progression to dementia, screening of vulnerable populations (especially elderly medical inpatients, older patients post-surgery, those with multiple medical co-morbidities and those on multiple medications) with well-validated brief instruments such as the Confusion Assessment Method (CAM) or Delirium Rating Scale (DRS) is vital (Helfand et al., 2021).

The patient with delirium requires medical investigation and management. The patient should be nursed in an easily observable bed, with a quiet environment. Attendance of a family or a one-to-one nursing special can provide orientation, reassurance and mitigate the risk of falls, wandering or absconding. The patient may require sedation if they are agitated, paranoid, or hyperactive. Care should be taken to not prescribe benzodiazepines where an intracranial cause is suspected.

There is no evidence that antipsychotics speed the resolution of delirium, and they should generally be avoided. If unavoidable, antipsychotics must be used very judiciously and in low doses with older patients as these medications are associated with increased cardiac and cerebrovascular morbidity.

Delirium in summary:

- Acute onset (hours–days)
- Clouding of consciousness
- Disorientation
- Memory impairment
- Perceptual disturbance
- Disrupted cognition
- Decreased or increased motor activity
- Disturbed sleep-wake cycle
- Fluctuating course

Acute Alcohol Withdrawal (Delirium Tremens) or Benzodiazepine Withdrawal

Delirium Tremens (the DTs) occurs in acute severe alcohol withdrawal. The clinical picture is of a patient with a history of substantial, prolonged alcohol intake who becomes agitated, tremulous and confused on day 2 or 3 of abstinence from alcohol. A very similar picture can occur with acute benzodiazepine withdrawal. Typically, the patient has a raised temperature, pulse and blood pressure, is shaking, sweating, confused, agitated, paranoid and reports visual hallucinations and illusions.

The patient is first given at least 300 mg parenteral thiamine – and substantially more if Wernicke encephalopathy (WE) is suspected. An oral multi-vitamin is commenced daily. At the same time, the severity of the withdrawal is assessed with an alcohol withdrawal scale (AWS). Sedation with 10-20 mg diazepam oral or IV every one to two hours is continued while the AWS score is greater than 4 and until the patient is lightly sedated. Small doses of haloperidol are sometimes required in the early stages (Schuckit, 2014). Signs of Wernicke encephalopathy include altered mental state, oculomotor abnormalities such as nystagmus, gait ataxia and evidence of nutritional deficiencies (Cantu-Weinstein et al., 2024).

Neuroleptic Malignant Syndrome

Four acute extrapyramidal syndromes can occur after starting or increasing an antipsychotic medication: neuroleptic malignant syndrome, acute dystonia, pseudoparkinsonism (slowing, shuffling gait, reduced arm swing) and akathisia (subjective motor restlessness). While all are distressing, the first two are also potentially dangerous and require immediate treatment.

Neuroleptic malignant syndrome has four cardinal features in a patient who has recently been treated with antipsychotic medication (Schönfeldt-Lecuona et al., 2020):

1. increased muscle tone
2. fever
3. elevated creatinine kinase (CK)
4. altered sensorium

Prevalence estimates vary widely, from one in six thousand to one in thirty neuroleptic-treated patients, although the most comprehensive review points to an occurrence of less than one in a thousand (Gurrera et al., 2007). Once the possibility of NMS has been raised, it should be rigorously evaluated, as a discharge comment of “? NMS” can greatly complicate future treatment. The diagnosis is unlikely without at least three of the features above, and the CK will usually be significantly elevated, typically > 1000 mU/mL. Remember that some degree of CK elevation is likely in a psychiatrically unwell patient who has walked long distances, lain for long periods of time, been involved in physical altercations or received intramuscular medication. Treatment involves supportive medical care, withdrawal of the causative agent and in severe cases consideration of specific pharmacological therapies (dantrolene, bromocriptine, and biperiden), lorazepam or ECT.

Laryngeal Dystonia

Acute dystonias occurring with antipsychotic medication typically affect the head and neck and can cause such distressing experiences as oculogyric crisis (forced involuntary upward gaze) and lockjaw. They are more commonly seen with potent D2 receptor blocking agents, such as haloperidol or risperidone. Laryngeal dystonia is a rare but unforgettable presentation, with hoarse voice, stridor and difficulty breathing. It can easily be mistaken for an anaphylactic reaction but should be distinguishable on history and lack of skin or mucosal changes (Ilchef, 1997). Treatment is with an intravenous anticholinergic agent such as benztropine.

Serotonin Syndrome

Serotonin syndrome arises from the use of substances that increase serotonin levels in the central nervous system, especially when these are taken at high doses or in combination (Scotton et al., 2019). The features of serotonin toxicity are neuromuscular excitation (hyperreflexia, clonus), autonomic changes (tachycardia, hypothermia) and altered mental state (agitation, confusion). Table 27.1 shows some agents associated with serotonin syndrome.

The principles of treatment are discontinuation of all serotonergic agents, cooling and fluid resuscitation where appropriate, benzodiazepines to reduce muscular rigidity and the risk of lactic acidosis and rhabdomyolysis, and consideration of a serotonin antagonist such as cyproheptadine, although this last measure has not been conclusively demonstrated to be of benefit (Nordstrom et al., 2016).

Conclusion

A variety of emergency presentations are seen in psychiatry, both behavioural and medical. Compared to the more predictable course of end-organ failure seen in other clinical disciplines, crises in psychiatry can be dramatic, dynamic and unpredictable, and require not only close attention to the person and their clinical state but also a rapid assessment of context, environment and safety, including of course your own.

Attending closely, anticipating and responding quickly to escalating distress, agitation or behavioural disturbance in a calm, non-threatening but authoritative manner can rapidly transform a fraught environment and improve outcomes. Intervene early in a deteriorating situation, prioritize the safety of everyone present, exclude an organic cause to the extent possible, be kind and always be mindful of your patient's dignity.

Table 27.1. Medications contributing to the Serotonin Syndrome. Adapted from Spadaro et al. (2022).

Class of Medications	Medications
Psychotropic	SSRIs: citalopram, fluoxetine, sertraline, escitalopram, paroxetine, fluvoxamine SNRIs: venlafaxine, desvenlafaxine, duloxetine TCAs: dosulepin, clomipramine, imipramine, amitriptyline MAOIs: phenelzine, tranylcypromine RIMA: moclobemide NaSSA: Mirtazapine
Anticonvulsants	Lamotrigine, carbamazepine, valproate
Antiemetics	Ondansetron, granisetron, metoclopramide
Anti-migraine	Triptans, ergotamine, methylergonovine
Opioids	Fentanyl, methadone, dextromethorphan, tramadol
Illicit Drugs	Methamphetamine, amphetamine, ecstasy (MDMA), psilocybin, LSD
Miscellaneous	Methylene blue, St. John's Wort, chlorpheniramine

Further Reading

Mental Health and Drug and Alcohol Office. Mental Health for Emergency Departments – A reference Guide. NSW Ministry of Health March 2015.
www.health.nsw.gov.au/mentalhealth/resources/Publications/mental-health-ed-guide.pdf

Chapter 28: Consultation-Liaison Psychiatry

Sara Ghaly

Specialty of Psychiatry, The University of Sydney, Australia, and New South Wales Director of Advanced Training and Chair of Binational Subcommittee of Advanced Training Consultation-Liaison Psychiatry RANZCP.

and

Christopher James Ryan

Specialty of Psychiatry, The University of Sydney, Australia, The University of Sydney, Australia, School of Clinical Medicine, The University of New South Wales, Australia, and School of Medicine, The University of Notre Dame Sydney, Australia.

What is Consultation-Liaison Psychiatry?

Like the other subspecialties in this section, consultation-liaison (CL) psychiatry is defined by the population it serves. In this case that is patients with a medical or surgical illness, who are inpatients of the general wards of a general medical hospital or outpatients of its clinics. Not surprisingly many of the disorders reviewed in this section also present frequently to general practitioners. The speciality's rather cryptic name derives from the two ways that psychiatrists get to see these patients in the general hospital.

'Consultation' refers to the fact that most of the time CL psychiatrists are asked to see general medical patients because the doctors looking after them think that they have a psychiatric problem of some sort and want assistance with this. The 'liaison' bit refers to the other way that patients can be seen. In some branches of medicine, in some hospitals, the physicians and surgeons feel that psychological issues occur so frequently that they need a psychiatrist embedded in their team. Psychiatrists in these liaison attachments attend team meetings, do ward rounds and either see patients themselves or advise on patients discussed with them. The nature of these liaison attachments vary from institution to institution. They are common in oncology, pain medicine and transplant medicine, but can arise in any speciality, subspecialty or service that offers a multidisciplinary approach to a particular disease – Parkinson's, HIV, multiple sclerosis and epilepsy are typical examples.

CL psychiatry is the discipline that covers this interface between the medical and the psychological - the realm between body and mind. This is a vast intellectual landscape and in this introductory chapter we can only hope to give you an overview, pointing out the major landmarks as we go. We'll start with what it's like to be a patient in hospital and the forces that shape normal and abnormal patient (and doctor) behaviour. Then we'll look at what we know about how the mind and body interact, before turning to a range of disorders at this interface. CL psychiatry also assists patients with medical conditions that present with, or that cause psychiatric symptoms. Those are beyond the scope of this chapter but appear in the earlier chapter on organic disorders.

Normal Patient Behaviour

What Is It Like to Be a Patient?

Not to put too fine a point on it, being an inpatient can be an unpleasant experience. First, of course, you only go to hospital because you are unwell and illness usually comes with a range of unpleasant symptoms, like lethargy, pain, or nausea. Second, because patients are unwell, they are also worried, because there is always a chance their illness could turn out badly. If that were not enough, they are in this unsettling and unfamiliar environment. Remember your first days of clinical placements, and how baffling the hospital seemed, that is what it's like for most of our patients, except it's worse because they're not going home in the evening.

Hospitals are huge, impersonal, institutions with hundreds of unwritten and often inflexible rules. Patients are often confined to their beds and dressed in pyjamas or a hospital gown. They need to deal with a range of professionals (and students) who appear and disappear often with little or no explanation. The usual rules of modesty are apparently thrown out the window and patients are poked and prodded and pricked at all hours of the day and night.

CL psychiatry is often called in when patients' reactions to hospitalisation are out of the ordinary – usually when they get upset or angry or frustrated. When you think about it though, it's amazing how infrequently this happens. The reason that so few people get very upset, angry or frustrated, is tied up with the answer to another question.

Why Do Patients Wear Pyjamas?

When patients come into hospital they almost always get changed into pyjamas. Of course, that makes sense if they are literally confined to bed, but most

patients stay in their pyjamas even when they're quite able to pop down to the hospital coffee shop. Why?

The answer lies in the fact that when patients enter hospital, they are also expected to enter a particular social role - the sick role (Parsons, 1975). The sick role is a sociological concept originally outlined in the 1950s and while there are problems with applying the model too tightly, it provides a useful heuristic to explain various aspects of patient behaviour.

The sick role has it that when people become unwell, they willingly take on a role that, on the one hand allows them some privileges but, on the other, demands some duties. The first privilege is that while sick, patients are exempt from their usual roles and responsibilities. Sick people need not go to work for example, or need not sit exams, for the period of their illness. The second privilege is that they are exempt from responsibility for their condition – it is not their fault that they can't work or do exams.

Privileges like these place a significant burden on others and some *quid pro quo* is required. That takes the form of one major duty. Whenever people assume the sick role, they must do all they can to leave it as soon as possible. In our society that means that patients must seek out medical care, regardless of any inconvenience associated with it, and then they must conform to any treatment recommendations offered.

Viewed through this lens, hospitals are large institutions that utilise the obligations of the sick role to get people back to their normal societal roles as efficiently as possible. Viewed through this lens, pyjamas play the same role in hospitals as baggy green prisoner uniforms play in gaols – they underline who's in charge. They reinforce the obligations of the sick role, and that patients must follow instructions and get well as quickly as possible.

So powerful is the sick role and, frankly, so robust are most people, that despite the worry, the pain, the nausea, the sleeplessness, the prodding and the pricking, most patients enter and leave hospital without major psychological incident. However, things can go wrong.

Abnormal Illness Behaviour

When people become unwell they exhibit a variety of illness behaviours (Mechanic, 1961). That is, they perceive, evaluate and act upon their symptoms in different ways. In 1969 a psychiatrist working at the University of Sydney, Issy Pilowsky, proposed the term “abnormal illness behaviour” to cover the situation

where “the doctor does not believe that the patient’s objective pathology entitles him to be placed in the type of sick role he expects, for the reasons which he claims it” (Pilowsky, 1969: 349). Defined this way, “abnormal illness behaviour” seems to give the doctor a privileged view of the patient’s experience, which sits a little uncomfortably half a century later. Despite this though, the concept still provides a useful scaffold for thinking about issues that arise in this domain.

We all learn about, and are socialised into accepting, the sick role from our earliest years. Think about your experiences staying home from primary school; no school, but also no fun. Once you were well enough to get up and play, you’d be going back to school the following day.

Abnormal illness behaviour, perhaps more loosely defined than Pilowsky had it, arises when, for various reasons, patients cannot assume or positively reject the sick role and do not adhere to its strong obligations. While the overwhelming majority of patients will adhere, the reasons for a minority not doing so are myriad, and we’ll only review a few here.

If a patient is going to follow, with little question or dispute, all of the directions that their doctors or nurses or physiotherapists provide them, it’s going to be important that they trust that those people have their best interests at heart. Fortunately, we have also been strongly socialised to believe that doctors and other healthcare workers are extremely trustworthy, so for most people that is not an issue. However, for some people this trust breaks down for various reasons and these patients might seriously question or downright refuse to believe that what is being suggested is for their own good.

The most straightforward manifestation of his problem occurs in patients with persecutory, or other types of, delusions. If a patient feels that his doctors are all involved in an international conspiracy aimed to bring him down, or if he feels that they have been replaced by androids, it’s unlikely he’ll simply follow their directions. However, people don’t need to be psychotic to lack trust in their doctors. A small percentage of the population have a personality style marked by an inclination to view everyone, especially those in authority with suspicion. If you’ve never been inclined to trust anyone, even the strong social pull of patient role may be insufficient to reassure you about your healthcare workers.

It’s not only psychotic people or people with paranoid personalities that lack trust in doctors. Some of our patients have come to this country fleeing persecution. In their countries of origin, doctors may have been complicit in government sanctioned torture, so any socialised trust of doctors may have disappeared long before they reached our shores. Such people are likely to have a

well-founded fear of governments as well, and their experiences in detention centres during their attempts gain asylum may have generated an arguably justified suspicion of the Australian government as well.

Even previously healthy people born and bred in Australia may lose their trust in some circumstances. One such group of patients are those for whom things have gone badly wrong. Despite our best efforts some patients suffer terrible side-effects or receive and follow advice that was clearly ill-advised in hindsight. If the outcome was bad enough, or if bad outcomes happened more than once, these patients can lose their trust in the advice of healthcare professionals and, understandably, question everything they are told.

Other people, by nature of their illness, spend months, sometimes years in hospital. In these people the sick role simply wears off with time. They become “professional patients” and the sick role is swamped by other more normal social roles that arise because they have spent so long in the ward environment. Often these people behave more like the friends or workmates of the healthcare workers they have been around for weeks and months. This is not necessarily a bad thing of course, but it does dissolve the usual patient-doctor dynamic. Just as workmates will not automatically follow the directions of other workmates, so long-stay patients will often balk at directions that other patients would simply adopt demurely.

There is another sort of ‘professional’ patient that often finds it difficult to fit into the patient role – patients who are themselves health professionals. Doctors, particularly, find adoption of the patient role challenging. After all, their usual role is to be the person giving the barely questioned directions, not the person following them. Generally, they are the one’s asking the nurses to do things, not vice versa and of course, they know that everything they are being told to do may not be strictly necessary. If you are likely to have an operation at 11 am you are likely to be told to fast from midnight, but a doctor will know that that is not strictly necessary. Strict hospital protocols, designed for easy universal application, are not well suited to people who have an intimate and detailed knowledge of the system. Almost all doctors who find themselves as patients (or patient’s relatives) have to apply themselves to stay within the bounds of the usual patient (or relative) role. Not every doctor manages this successfully.

Whenever the sick role is eroded, the patient-doctor dyad will be impacted, and illness behaviour will become abnormal at least in the sense that it will fall outside the normal range. In most cases this can be addressed by first recognising what is going on, and then making allowances for it. Usually, when a patient’s trust is gone, this will mean taking much more time than usual explaining exactly why different requests are being made and reassuring the patient of

everyone's motives. On occasion, it will also require a more flexible than usual approach to normal hospital processes.

It's not only a lack of trust that may disrupt the patient role. Other examples that we lack space to examine in detail at this point include: patients who are used to prison environments, where a whole different social milieu applies that has little overlap with the patient role; patients who cope with their illness by utilising denial, so that they may show no recognition that they are ill at all; patients with borderline personality styles who might idealise some healthcare workers while demonising others, often causing a great deal of tension within the treating team.

Conditions Typically Encountered in Consultation-Liaison Psychiatry

There are a range of psychiatric conditions that are most frequently encountered in the hospital wards or clinics and therefore are sensibly dealt with in this chapter. As noted above, people with these conditions may also present to general practitioners. Many of them could also be regarded as displaying abnormal illness behaviour in Pilowsky's terms, but it is better perhaps to consider the conditions individually.

Medically Unexplained Symptoms

As the term suggests "medically unexplained symptoms" are symptoms, like those arising in the disorders discussed in this section, for which medicine has no secure explanation. It is important to understand that the term does not imply that psychiatry (as opposed to the rest of medicine) has somehow unearthed that explanation. In truth psychiatry is as much in the dark as the rest of medicine. However, psychiatry is the speciality that is happy to take conversion disorder, somatisation disorder and the rest as its own as there *might* be some psychopathological element and it is clear that the patient needs assistance.

The basic problem with all these disorders is that at this point we have no clear way of describing how the workings of the brain come to be manifest as consciousness as we all experience it. With no answer to the mind-body problem as it applies in normal circumstances, it is hardly surprising that our understanding is so poor when the system goes awry.

We will not be forever in the dark. There is no reason to think the problem is insoluble and a great deal of research is aimed at matching neurophysiological findings to clinical findings (Feinstein, 2011), but for the time being our understanding of these syndromes is so poor that "medically unexplained symptoms"

probably provides the most accurate and informative overarching heading. Note that DSM-5-TR groups these disorders together as “Somatic Symptom and Related Disorders” (American Psychiatric Association, 2022).

Conversion Disorder

In DSM-5-TR the main features of Conversion Disorder are the presence of “one or more neurological symptoms with clinical findings that provide evidence of incompatibility between the symptom and recognised neurological or medical conditions” (American Psychiatric Association, 2022).

The sorts of neurological symptoms encountered in Conversion Disorder range across the spectrum. They include: weakness or paralysis; and tremor, dystonia or gait abnormalities. There may be dysphagia, dysphonia or dysarthria or the sensation of a lump in the throat (globus). Possibly the most common are apparent seizures, without EEG change, best termed non-epileptic seizures. There may also be all manner of sensory symptoms such as anaesthesia, visual loss or diplopia.

The diagnosis should usually be made cautiously and DSM-5-TR rightly stresses that “it should not be made simply because results from investigations are normal or because the symptom is ‘bizarre’” (American Psychiatric Association, 2022). It is worth remembering that all investigations have false negatives and that there are a range of explanations for a lack of positive investigations in these cases that logically include: that the right investigation has not been conducted; or that the relevant investigation has yet to be invented. In the days before the invention of the CT scanner, a diagnosis of Conversion Disorder was often proven wrong on follow-up (Slater, 1965).

Conversion Disorder provides an excellent example of how psychiatric diagnostic classification (in this case the DSM system) can shift. DSM-IV had a diagnostic criterion that disappeared with the fifth edition, namely, the “[p]sychological factors are judged to be associated with the symptom or deficit because the initiation or exacerbation of the symptom or deficit is preceded by conflicts or other stressors” (American Psychiatric Association, 1994). The inclusion of that criterion was consistent with the view, previously central to the diagnosis, that the underlying cause of Conversion Disorder was some sort of internal psychological conflict. The name of the syndrome derives from the notion that emotional arousal is somehow “converted” into a physical symptom, a process known in psychoanalytic literature as “primary gain”. However, the authors of DSM-5 decided a change was needed. On the one hand, plenty of people with apparent Conversion Disorder did not appear to have an internal conflict and, on the other,

the vicissitudes of everyday life probably made it easy for examining psychiatrists to “discover” internal conflicts where perhaps they did not exist.

As a consequence of these perceived weaknesses with the previous understanding the creators of DSM-5 demanded that the diagnosis require “clear evidence of incompatibility with neurological disease” instead. Chief among the types of evidence suggested is internal inconsistency at examination and DSM-5-TR references a range of findings on neurological examination. However, whilst some of these tests (e.g. Hoover's sign) have at least some evidence to support their usefulness in making the diagnosis (McWhirter et al., 2011), many conversion symptoms have no empirically based test to support the diagnosis and it is not clear to what extent the evolution of the DSM's characterisation of Conversion Disorder has swapped one form of uncertainty with another.

All of this simply underlines the acknowledged weaknesses of the DSM or any classification system in psychiatry (and to some extent in medicine as a whole). The first paragraph of the preface of the manual notes that “DSM is intended to serve as a practical, functional, and flexible guide for organizing information that can aid in the accurate diagnosis and treatment of mental disorders. It is a *tool* for clinicians, an essential educational resource for students and practitioners, and a reference for researchers in the field” (American Psychiatric Association, 2013: xli) (emphasis added). That is to say it is not, and does not try to be, the definitive arbiter of psychiatric diagnoses.

Though patients are classically said to feature *la belle indifférence* – an odd sanguine acceptance of their deficit – there is no empirical data to support the sign's usefulness in making the diagnosis, and it is difficult to distinguish *la belle* from simple stoicism. Possibly more useful is the presence of a model for the symptom – for example a close relationship with an auntie say who had similar weakness after a stroke. The people most likely to experience non-epileptic seizures are people with established epilepsy and here, it is thought, they act as their own model.

Conversion Disorder is two or three times more common in females. Prevalence in the community is unknown, but in specialist neurology clinics, where it is most often encountered in practice, the diagnosis is made in around 5% of patients. It usually has its onset before the age of forty and it is brave indeed to use the diagnosis in unexplained neurological symptoms occurring for the first time over that age.

While the majority of conversion symptoms are only short-lived and tend not to recur, in a percentage of patients, with an estimated 2-5/100,000 per year, the symptoms persist and may be associated with significant disability. As one might

expect the most important differential diagnoses are those of actual neurological disease presenting somewhat atypically. Very often the clinician cannot but avoid ongoing uncertainty, but has no choice but to press on explaining the limits of our current knowledge to the patient.

Even in cases where conversion symptoms can be diagnosed with reasonable confidence, such as patients who exhibit a typical seizure which was observed whilst the patient's EEG showed no ictal phenomena, all that the clinician can say with confidence is that that particular seizure was a non-epileptic seizure. Carefully handled though, even such cautious advice can be of significant aid to the patient.

Management begins with an unavoidably lengthy explanation of an alternative underlying cause for at least some of the patient's symptoms. Oftentimes this is best expressed as "stress", since most people are happy to accept that "stress" can manifest itself physically. It's important to spontaneously declare that in hypothesizing that symptoms may not have a standard medical explanation, no one is suggesting that the deficit is all in the patient's head or that he or she is only pretending to be ill. The patient is ill. The deficits are real. It is just that it is now thought that the cause of symptoms is more likely to be related to (admittedly poorly defined) psychological factors than standard medical ones.

In a syndrome so poorly defined that is comparatively rare and which typically resolves spontaneously, there is, unsurprisingly, little or no evidence base to guide treatment (Feinstein, 2011). Beyond reassurance and education, management is usually focused on helping the patient deal with any identifiable ongoing stressors and encouraging ongoing psychological management to enable an avenue to talk about their worries. The hope is that this will provide an alternative avenue to cope with the stressors of everyday life and free patients from the disabling somatisation that has brought them to clinical attention. There is no role for medication except to treat clear comorbidities.

Somatic Symptom Disorder and Somatisation Disorder

The DSM-5-TR criteria for Somatic Symptom Disorder are outlined in the box below (American Psychiatric Association, 2022). In previous iterations of DSM and still in the ICD this disorder was known as Somatisation Disorder or Somatisation Syndrome and, in contrast to criterion A (for DSM-5-TR) below, to diagnose it the patient had to suffer a range of unexplained physical symptoms from a range of systems. In DSM-IV for example the patient needed a history of at least four pain symptoms, two gastrointestinal symptoms, one sexual symptom and one

neurological symptom. This is very different from the one symptom being sufficient in DSM-5-TR.

Because Somatic Symptom Disorder is relatively new, most of the information available in the literature concerns the more elaborate (and obviously much less common) Somatisation Disorder and the rest of this section will deal with this.

Epidemiologic studies suggested a lifetime prevalence of Somatisation Disorder of somewhere between 0.2% and 2.0% in women though the figures depended enormously on how exactly it was defined (Mai, 2004). How so ever it is defined though, it is far rarer in men. Traditionally it is regarded as a disorder with a young age of onset and DSM-IV demanded that symptoms began before age 30.

Somatic Symptom Disorder

A. One or more somatic symptoms that are distressing or result in significant disruption of daily life.

B. Excessive thoughts, feelings, or behaviours related to the somatic symptoms or associated health concerns as manifested by at least one of the following:

1. Disproportionate and persistent thoughts about the seriousness of one's symptoms.
2. Persistently high level of anxiety about health or symptoms.
3. Excessive time and energy devoted to these symptoms or health concerns.

C. Although any one somatic symptom may not be continuously present, the state of being symptomatic is persistent (typically more than 6 months).

The aetiology of Somatisation Disorder remains unknown. Theories, none of which are entirely satisfactory, often revolve around learnt behaviour perhaps first observed by parents during childhood, or some other mechanism by which the normal acquisition of the sick role is disrupted. Other theorists cite the importance of alexithymia – a difficulty expressing one's emotions in words - and suggest that the somatic symptoms become an alternative avenue for expressing one's feelings (De Gucht and Heiser, 2003). It is quite likely that in some cases normal bodily symptoms are amplified and misinterpreted. Though not a formal part of any of the

diagnostic formulations, anxiety and depression are very common in patients with these syndromes (Kirmayer et al., 1993).

Assessment of these patients begins as always with a careful history and examination. This is probably more important than usual in patients who have attracted these diagnoses as it is all too easy to assume that a diagnosis of a somatoform disorder (no matter which one) previously made is correct and then to miss an underlying medical condition. It is also important to realise that even a lifetime of suffering undisputed somatic symptoms offers no protection from developing a physical disease. Even if the patient is well known to you, take care at least to carefully enquire about new symptoms.

Utilisation of investigations represents something of a balancing act. New symptoms should be appropriately investigated but avoid repeating investigations, especially those with an associated morbidity, if they do no more than re-visit already well-trod paths.

Obviously, there is the risk that a diagnosis of Somatisation Disorder might obscure a medical diagnosis that presents with a range of physical symptoms. Diseases such as multiple sclerosis, systemic lupus erythematosus, and certain endocrine conditions should all be borne in mind when making the diagnosis. There is also considerable overlap between Somatisation Disorder (or indeed Somatic Symptom Disorder) and functional medical syndromes such as fibromyalgia, chronic fatigue, and irritable bowel syndrome, though the specifics of this are beyond the scope of this chapter. Many physicians exhibit a degree of therapeutic nihilism around patients with Somatisation Disorder, but this can only reflect an ignorance of the management of the condition (Mai, 2004).

As was the case with Conversion Disorder the first step in management is detailed feedback to the patient about what appears to be occurring, avoiding obviously, suggestions that there is nothing wrong with the patient. This done the next step is to recognise the reality of the symptoms and to bring into focus the existence of stressful life events that might also be relevant to the patient's symptomatology. After that various supportive, interpersonal or cognitive behavioural therapeutic approaches have been utilised that continue to link the two and shift the focus to the psychosocial realm. These interventions occur in sessions that are scheduled regularly, avoiding the need for the patient to have a somatic symptom as a ticket to gain support (Guthrie et al., 1999). As time goes on the regularity of the appointments can be spaced at longer and longer intervals. There is probably no role for pharmacotherapy beyond its judicious use in clear comorbid depression.

Illness Anxiety Disorder and Hypochondriasis

The big revamp of DSM-5's somatic disorders also saw Illness Anxiety Disorder replace Hypochondriasis, though, as was the case with Somatic Symptom Disorder and Somatisation Disorder, the two syndromes only somewhat overlap.

Hypochondriasis is “preoccupation with the fear of having, or the idea that one has, a serious disease based on the person's misinterpretation of bodily symptoms or bodily functions” (American Psychiatric Association, 1994: 445). Whereas those with Somatisation Disorder (traditionally defined) would make vague complaints of multiple symptoms in multiple domains patients suffering hypochondriasis would present with a limited range of symptoms, often described in precise detail and attributed to the possibility of one or more named diseases that they feared the symptoms revealed.

In the new Illness Anxiety Disorder by contrast, while the person remains preoccupied with the possibility of having (or acquiring) a serious disease, the somatic symptoms, if present at all, are mild and the person is more generally concerned about their personal health status. As the name reflects, Illness Anxiety Disorder appears to be a variety of anxiety disorder where anxiety is generated by the possibility of ill-health. Hypochondriasis while similar is probably better conceived of as featuring a conviction (falling short of delusional intensity) that the person has already developed, or is about to develop, some sort of nasty illness, and the anxiety arises from that belief. The management of Hypochondriasis largely overlaps in its approach with the management of Somatisation Disorder (Starcevic, 2015). Once again there is little role for pharmacotherapy.

Disorders of Body Image or Perception

The commonest disorder of body image or perception encountered in psychiatry is anorexia nervosa which is covered in the eating disorders chapter. However, a number of such conditions are best placed within the Consultation-Liaison chapter because that setting is the place they are most likely to be met.

Body Dysmorphic Disorder

Body Dysmorphic Disorder (BDD), sometimes called dysmorphophobia, is often described as a disorder of “imagined ugliness” (Morselli and Jerome, 2001). Sufferers are “preoccupied with one or more perceived defects or flaws in their physical appearance, which they believe look ugly, unattractive, abnormal, or deformed” (American Psychiatric Association, 2013: 243). Most commonly the perceived defects are around the person's face, especially the nose or hairline, or involve the person's skin or genitals, but any body area may be affected. As a result

of these concerns the patient may spend hours a day gazing at the affected body part in the mirror or picking at their skin. They are usually convinced that others are also appalled by their defects and may try to cover them up or avoid others entirely. Oftentimes the first clinical contact with individuals with BDD is made by plastic surgeons as they seek remedies for their problems, though often people devise their own treatments that bring them to medical attention such as efforts to bleach their skin.

DSM-5-TR has created a subclass of BDD called “muscle dysmorphia” where a sufferer, usually a male, is “preoccupied with the idea that his or her body build is too small or insufficiently muscular” (American Psychiatric Association, 2022). Whether or not this is best seen as a subcategory of BDD or its own condition is yet to be determined.

BDD, as a class, occurs in around 2% of the population with no clear sex difference (Buhlmann et al., 2010). Patients with BDD may account for up to 8-10% of patients in dermatology, cosmetic surgery, or maxillofacial clinics. BDD starts in two thirds of individuals by age 18. The disease causes significant disability as sufferers, usually still in adolescence, withdraw from friends, avoid intimate relationships and drop out of school. Depression is a frequent comorbidity and in one study over 40% of adolescents with BDD reported having attempted suicide (Phillips et al., 2006).

The causes of BDD remain obscure. It is more common in the first degree relatives of people with obsessive compulsive disorder (OCD) and like OCD, neuroimaging abnormalities in a range of areas, including the limbic system, visual processing areas, and the orbitofrontal cortex have been reported (Rossell et al., 2015). One conception of BDD has it as some middle ground between the normal concerns about appearance often expressed by individuals who may be so concerned as to seek plastic surgery, on the one hand, and psychotic concerns as experienced by those with delusional disorders on the other. Although people with BDD are not classically convinced of their concerns to a delusional degree, a number of studies have suggested that this apparent differentiation may be unusual in practice, so that some authors have suggested either including a subtype of delusional BDD as DSM-5-TR has done (American Psychiatric Association, 2022), or to simply label all similarly affected patients as suffering BDD regardless of the apparent intensity of their beliefs (Fang et al., 2014). Eating disorders, OCD and social anxiety disorder may also present challenges in terms of differential diagnosis.

The evidence base for treatment is marred by a poverty of randomised controlled trials. The management strategy most frequently subjected to empirical analysis has been the prescription of selective serotonin reuptake inhibitors or

clomipramine, where both open-label trials and some RCTs have suggested response rates from 53% to 73% (Fang et al., 2014). The same effects are not seen with other antidepressants and the evidence suggests that the doses of SSRIs need to be higher than are typically used in depression and trials need to be longer (perhaps 6-9 weeks). In keeping with the apparent blur between “delusional” and “non-delusional” BDD, there is no good evidence that “delusional BDD” responds preferentially to antipsychotics, and therefore SSRIs are seen as the first line treatment in all patients.

Several studies have provided empirical support of cognitive behavioural therapy in BDD (Veale, 2001). This might include techniques such as exposure and response prevention centred around checking one’s self in the mirror. There is also indirect evidence of benefit when psychological therapies are combined with a pharmacological approach (Fang et al., 2014).

Not surprisingly very few patients with BDD who manage to obtain surgical interventions aimed at correcting their perceived abnormalities derive long-term benefit from these (Crerand et al., 2010). With or without treatment BDD tends to be a chronic disorder with low remission rates and high rates of relapse for those who do get better (Phillips et al., 2013).

Delusional Disorders – Somatic Type

Delusional disorders were touched upon in the chapter on schizophrenia (Chapter 15), and are diagnosed when patients exhibit one (or more) delusions, but do not meet criteria for schizophrenia and apart from the impact of the delusion(s) the person’s functioning is not markedly impaired. The somatic subtype of delusional disorder is often encountered in the general hospital and applies when “the central theme of the delusion involves bodily functions or sensations” (American Psychiatric Association, 2013: 90). The symptoms associated with the delusion often bring people to seek medical help.

Perhaps the delusional disorder most commonly seen in consultation-liaison psychiatry is delusional parasitosis or Ekbom’s syndrome. In these cases sufferers believe that insects, lice, maggots or other small vermin are living in their skin (Enoch and Ball, 2001). As is typical of delusional disorders, patients may appear perfectly normal until the conversation turns to their delusional concerns, and classically patients exhibit the “matchbox sign” presenting with a small container that they believe demonstrates the desiccated body-parts of the infesting bug. Worryingly, patients may also present with conditions related to their attempts to remove the offending creatures, including pruritus from scratching to chemical burns from various homemade lotions. There is only weak evidence for the efficacy of antipsychotics (the recommended first-line treatment) in delusional parasitosis, though this is probably partially attributable to rarity of the disorder and partially to

the difficulties associated with keeping these patients on a treatment they believe to be irrelevant to their underlying problem (Lepping and Freudenmann, 2008).

Other somatic-type delusional disorders that may fall under the care of consultation-liaison are delusions that a person emits a foul odour, or delusions that a particular body part is misshapen, though the latter will usually now be classed as body dysmorphic disorder (as discussed above).

Factitious Disorders and Malingering

In factitious disorder and malingering the sick role is again central, though in these states the patient *consciously* feigns illness in order to achieve a goal. Like so many of the disorders in this chapter the definitions, and even the status of these states as “disorders”, is controversial (Bass and Halligan, 2014).

Factitious Disorders

Patients with factitious disorder consciously and deliberately falsify medical or psychological signs and symptoms, but do so for no “obvious external rewards” (American Psychiatric Association, 2013: 325). The methods of deception may range from simply lying, to falsifying documents, to unneeded venesection to induce anaemia, to amputating digits claiming they were lost in an accident. As opposed to the conscious deception in malingering (see below), in factitious disorder the sole aim of the fabrication appears to be to gain the role of a patient, though successfully achieving the deception may provide psychological reward in some cases.

Most commonly factitious disorder presents with the patient feigning one symptom or one disease. Patients with dermatitis artifacta, for example, who deliberately fabricate new skin lesions or worsen existing ones, do not usually move on to feign other conditions (Koblenzer, 2000). However in the most severe variant, dubbed Munchausen's syndrome after the fictional character in Raspe's 1785 book, patients typically make a career of their condition, moving from hospital to hospital, giving false names, detailing all manner of exotic conditions or injuries, and telling elaborate tales to substantiate their claims – pseudologia fantastica (Asher, 1951). The most pernicious version of the condition sees individuals not feigning illness in themselves, but in others, typically children in order apparently, to take on the role of a parent of a sick child. Labelled “Factitious Disorder Imposed on Another” in DSM-5-TR, this is also known as “Munchausen's via proxy” or “Meadow's syndrome” and is a form of child abuse (Meadow, 1977).

The nature of factitious disorder makes valid estimates of its prevalence almost impossible. Estimates range from 0.5 to 2%, though this depends to an extent on what degree of deceit will trigger a diagnosis, given some degree of untruthfulness is almost universal. Whatever threshold is chosen though, the

condition is almost certainly significantly under-recognised as doctors are poor lie detectors (Bass and Halligan, 2014). Feigned psychiatric symptoms are probably particularly difficult to detect. Milder factitious disorder is commoner in females and people with healthcare training are over-represented (Krahn et al., 2003). Munchausen's syndrome seems almost exclusively the domain of males.

Whether or not factitious disorder is properly regarded as a psychiatric disorder, there is no doubt that those who behave in this manner suffer significant disability. Even persons with milder manifestations may spend significant amounts of time in hospital and often risk actual disease or injury because of unneeded medical intervention. Young men with Munchausen's syndrome often live a peripatetic existence, seemingly cut off from any family or friends. Though numerous theories have been posited, the cause or causes of factitious disorder are unknown.

Assessment begins with an awareness that the condition exists that triggers a low level of suspicion when a patient's story is particularly dramatic or contains inconsistencies. Of course, factitious disorder is rare, so the drama in most histories will be real and the inconsistencies genuine. However, a lurking healthy scepticism, that precipitates a call to a previous treater or some double-checking of documentation will detect many such patients, who would otherwise have gone unnoticed. Once suspicions are properly aroused it will be important to carefully check the patient's history as delivered against all available medical records. If the diagnosis is strongly suspected or confirmed, there is no sense in "confronting" the patient as, in more severe cases, this can lead to a precipitous discharge with lines still *in situ*. Instead, ask for advice from the consultation-liaison team.

Little can be said with confidence regarding the management of factitious disorder, aside from taking obvious steps to minimise further unwarranted investigations. Anecdotally, milder cases respond and may even do quite well with supportive or dynamic psychotherapy. Typically, people with the more severe Munchausen's variant tend to simply move on, though as their efforts to create signs and symptoms intensify, they are often left with genuine conditions, such as bowel obstructions secondary to multiple operations. The management of "Factitious Disorder Imposed on Another" will inevitably include the involvement of child protection services or the police.

Malingering

Malingering involves the feigned presentation of a physical or psychological illness for one or more of a range of understandable reasons. These reasons are usually regarded as straightforwardly unethical, such as efforts to gain drug or monies, or attempts to elude responsibilities such as work or court appearance. However, in some cases, easy moral characterisation is not so simple, such as

attempts to elude military service or to gain accommodation when a person has none. DSM-5-TR notes that malingering is not a diagnosis, but it may nonetheless become a focus of clinical attention (American Psychiatric Association, 2022).

Even more than factitious disorder, the prevalence of malingering will depend upon the thresholds chosen to apply that label – few people would not, at some time in their lives, at least exaggerated some symptoms to escape a dreaded social event. While the detection of malingering requires the maintenance of a scepticism similar to that required for the detection of factitious disorders, in psychiatry malingering is probably over-diagnosed, as less experienced clinicians often attribute atypical or previously unseen phenomenology to feigned efforts to gain accommodation.

Even when malingering has been confidently established, clinicians need to recall that having previously pretended to have had pain, for example, does not immunise a person against all future actual discomfort. All new instances of symptoms need to be examined afresh, with past malingering considered but not determinative of a patient's ongoing care.

Further Reading

Stern, T. A., Fricchione, G. L., & Rosenbaum, J. F. (2010). *Massachusetts General Hospital Handbook of General Hospital Psychiatry-E-book*. Elsevier Health Sciences.

Chapter 29: Comorbidity Between Mental and Substance Use Disorders

Lauren Monds

Drug and Alcohol Services, South Eastern Sydney Local Health District, Australia, and Specialty of Addiction Medicine, The University of Sydney, Australia.

Huw Davie

Concord Drug Health Services, Sydney Local Health District, Australia, and Specialty of Psychiatry, The University of Sydney, Australia.

and

Amy Kitajima

Concord Drug Health Services, Sydney Local Health District, Australia, and Specialty of Addiction Medicine, The University of Sydney, Australia.

Introduction

Comorbidity is when an individual experiences the co-occurrence of problematic substance use and a mental health condition. Dual diagnosis refers to the situation where a person is diagnosed with both a substance use disorder and a mental disorder. Comorbidity has the potential to considerably impair a person's wellbeing, treatment progress, and overall quality of life. It can be challenging to discern the intricacies of this complex relationship as problematic substance use can be the result of a mental disorder (e.g., where an individual uses alcohol or other drugs to cope with mental health symptoms such as distress), or even the catalyst for a mental disorder (e.g., methamphetamine induced psychosis). While outside the scope of this chapter, it is important to recognise the additional possibility of "multimorbidity"; i.e. substance use and mental health disorders may also co-occur with common physical disease (e.g., liver, heart, and metabolic conditions), as well as intellectual disabilities and cognitive impairment (Bruijnen et al., 2019; Didden et al., 2020; John and Wu, 2020; Monds et al., 2017; Schulte and Hser, 2013).

When a person experiences symptoms of both mental disorder and substance use disorder simultaneously, it can pose challenges for diagnosis and treatment, as well as increase the likelihood of relapse. Therefore, it is crucial for treatment approaches to consider the interplay between these conditions to facilitate long-term recovery and overall well-being.

Prevalence

Amongst people with substance use disorders there is a very high co-occurrence of mental disorders. Some estimates indicate that up to 84% of people receiving treatment for substance use disorder meet diagnostic criteria for a mental disorder (Davis et al., 2023). Additionally, there are very high rates of problematic substance use amongst people with a mental disorder diagnosis. Based on data from the 2007 Australian National Survey of Mental Health and Wellbeing (N=8841) it was found that pre-existing mental disorders were significantly associated with increased risk of developing a substance use disorder specifically for alcohol, cannabis and stimulants (Marel et al., 2019). Rates of comorbidity for common mental disorders include:

- **Attention Deficit and Hyperactivity Disorder (ADHD):** It is thought that ADHD doubles the risk of substance use disorder and around one quarter of people with substance use disorder have co-occurring ADHD (Young et al., 2021). Lifetime prevalence rates of up to 27.7% substance use disorder have been reported in people with ADHD, with the most reported substance of concern being amphetamines (19.1%) (Anker et al., 2020).
- **Anxiety disorders (see Chapter 18):** It is thought that up to 12% of people with an anxiety disorder will have a co-occurring substance use disorder in the same 12 month period (Marel et al., 2019), with similar rates of people with a substance use disorder having an anxiety disorder (~12%; (Melchior et al., 2014)).
- **Eating disorders (see Chapter 21):** The pooled lifetime prevalence of a substance use disorder in an eating disorder population is estimated to be 21.9%, with current prevalence estimated at 7.7% (Bahji et al., 2019b).
- **Mood disorders (see Chapters 16 and 17):** A recent meta-analysis found that alcohol use disorder is the most common comorbid substance use disorder in major depressive disorder, with approximately 20% of people with major depressive disorder having alcohol use disorder, with a higher prevalence rate in males (36%); other substance use disorders were estimated at up to 12% in major depressive disorder (Hunt et al., 2020). It is thought that up to 50% of people with a bipolar disorder will also experience a substance use disorder in their lifetime (Messer et al., 2017).

- Personality Disorders (see Chapter 22): Up to 50% of people with a personality disorder also have a substance use disorder (Köck and Walter, 2018) and up to 60% of people with a substance use disorder are thought to have a personality disorder (McMain and Ellery, 2008).
- Schizophrenia (see Chapter 15): One recent review indicates that up to 90% of people with schizophrenia will be diagnosed with a substance use disorder in their lifetime including nicotine or 61.5% excluding nicotine (Ayano, 2019).
- Trauma disorders (see Chapter 20): It is estimated that 45% of people with post-traumatic stress disorder (PTSD) also have a substance use disorder (Pietrzak et al., 2011), and between 36-50% of people with substance use disorder also meet diagnostic criteria for PTSD (Gielen et al., 2012).

It is also important to note that many people present to an alcohol and other drugs service with coexisting psychiatric symptoms. Even if not meeting the full diagnostic criteria of a disorder these symptoms can still impact functioning and the treatment of the substance use disorder (Cross et al., 2018).

Models of Illness

There is a range of terminology and classification regarding comorbidity of substance use disorders and mental disorder. Commonly used terms include “comorbidity”, “dual diagnosis” and “co-occurring conditions”. “Dual diagnosis” remains commonly used within Australia, although there has been a recent shift towards “co-occurring conditions” to reflect the fact that individuals often have multiple co-occurring substance use disorders or mental disorders, and that people often present with symptoms of disorders without meeting the full diagnostic criteria (Marel et al., 2022).

A number of models have been proposed to explain the high rates of co-occurring substance use and mental health conditions, which can be broadly split into three main categories: The Common Factors Hypothesis, the Direct Causal Hypothesis, and Indirect Causal Hypothesis (Marel et al., 2022). Figure 29.1 shows a summary these models.

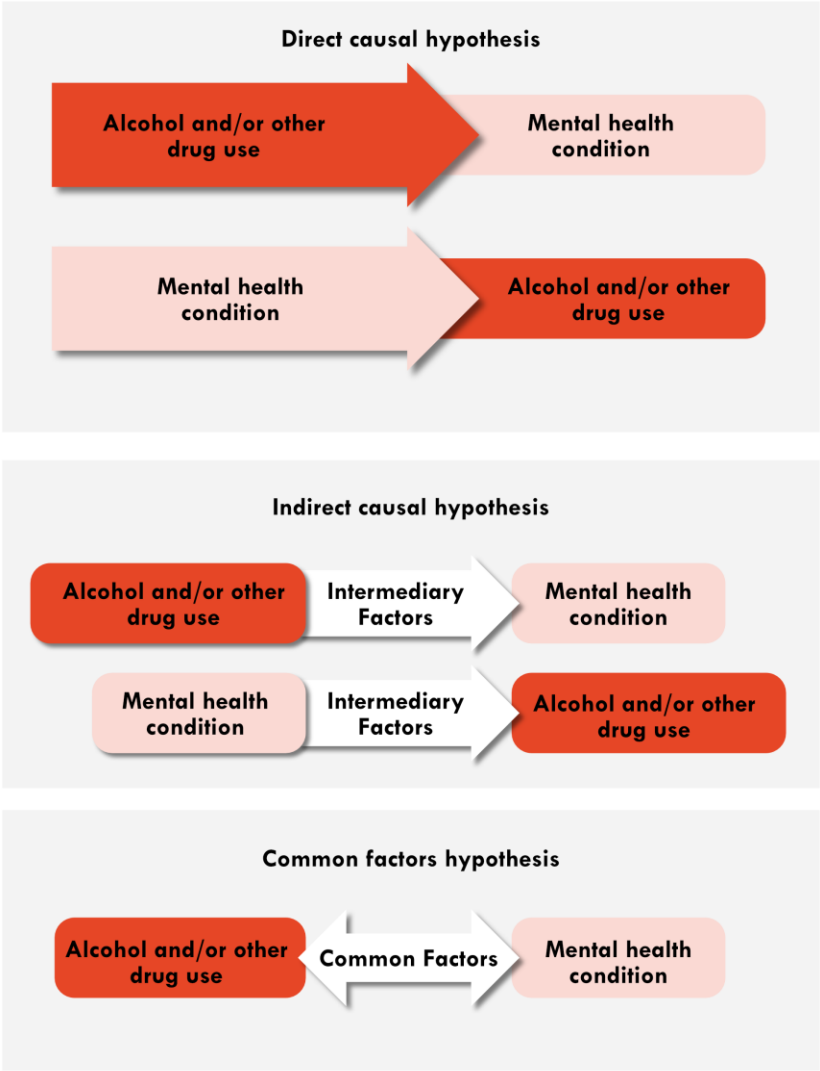


Figure 29.1. Proposed models to explain the high rates of co-occurring substance use and mental health conditions.

Common Factors Hypothesis

Substance use disorders and mental disorders share biological, psychological and social risk factors that may contribute to their co-occurrence. For example, adverse childhood experiences which are stressful and potentially

traumatic, such as abuse, neglect, housing problems or parental mental health issues, are associated with increased rates of developing both substance use disorders and mental disorders in adults (Mersky et al., 2013). Genetic risk factors may also be shared between conditions. A study investigating genetic vulnerabilities in patients with substance use disorders found a strong association between any substance use disorder diagnosis and the polygenic risk score for schizophrenia, suggesting shared polygenic liability (Hartz et al., 2017).

Direct Causal Hypothesis (Mental Health Condition Causes Substance Use Disorder)

Substances are often used to help manage symptoms of mental health conditions, and this behaviour is often referred to as “self-medicating”. For example, a person with an underlying anxiety disorder may drink alcohol to help manage their symptoms given the anxiolytic effect of alcohol. Regular use of alcohol to manage anxiety may result in larger amounts being consumed with increasing frequency with the risk of developing tolerance and dependence, ultimately resulting in a substance use disorder in addition to their underlying anxiety disorder.

Substance Use Disorder Causes Mental Health Condition (Secondary Mental Health Condition)

This model theorises that substance use disorders can directly cause the development of mental health conditions. For example, there is increasing evidence that people who experience stimulant-induced psychotic episodes are at increased risk of transitioning to a diagnosis of schizophrenia with ongoing stimulant use (Medhus et al., 2015).

Indirect Causal Hypothesis

This model posits that substance use may precipitate a mental health condition (or vice versa), but that intermediary factors are the cause, rather than a direct biological or psychological cause. For example, a person with a major depressive disorder may develop difficulties maintaining employment due to their depression, and this unemployment may trigger alcohol use (Marel et al., 2022).

Assessment

Assessing people with co-occurring substance use and mental health conditions is challenging as many signs and symptoms of substance intoxication or withdrawal are the same as those for mental health conditions. For example, symptoms of generalised anxiety disorder such as excessive anxiety, restlessness,

irritability, sleep disturbance and muscle tension, are all also symptoms of alcohol withdrawal in the absence of an underlying anxiety disorder. Ideally, patients can have a comprehensive psychiatric assessment during a period of abstinence once withdrawal symptoms have resolved, in order to minimise confounding symptoms. However, given the high risk of comorbidity, it is important not to repeatedly delay assessment and treatment of a patient's mental disorder until their substance use disorder resolves. Detailed assessment of the time course of their conditions, particularly during times of abstinence, can help determine if symptoms of mental disorder persists when patients were not acutely intoxicated or in withdrawal.

Approaches to Treatment

The approaches to treating co-occurring substance use and mental health conditions can be categorised into four methods: sequential treatment, parallel treatment, integrated treatment, and stepped care (Marel et al., 2022).

Sequential Treatment

The individual's primary condition is addressed first (typically, the substance use disorder), followed by treatment of the secondary condition.

Parallel Treatment

Drug and mental health conditions are treated simultaneously, however treatment is provided by separate drug health and mental health services.

Integrated Treatment

Drug and mental health conditions are addressed simultaneously by the same service or health provider. This allows exploration of the complex relationship between the co-occurring conditions.

Stepped Care

A flexible approach. The least intensive and expensive treatment is trialled first and, if ineffective, increased in intensity in a stepwise fashion according to the severity of the individual's condition.

While there is debate regarding the relative merits of each approach, there is a lack of robust research in this area (NSW Ministry of Health, 2015). Integrated approaches are widely endorsed for the treatment of co-occurring conditions due to intuitive advantages in providing holistic care, however further research is required

regarding treatment outcomes with this approach (Glover-Wright et al., 2023; Yule and Kelly, 2019). Clinicians need to weigh the advantages of each approach as it applies to the individual's circumstances.

Patients with comorbid conditions often find it challenging to access treatment, given the complexity of their conditions and the difficulty in determining the specialty or service to take primary responsibility. It is important to regularly assess and screen for co-occurring conditions when patients are receiving treatment for mental health conditions or substance use disorders, and provide a patient-centred and trauma informed approach to their management.

Psychosocial Interventions in Addiction

Psychosocial interventions encompass a range of therapeutic strategies which aim to enhance an individual's physical, psychological and social wellbeing. Interventions can be offered as part of a holistic treatment plan which should be developed in collaboration with the patient in relation to their unique social context (NSW Ministry of Health, 2023).

Brief Intervention

This is a short and often opportunistic counselling strategy. Clinicians raise awareness and provide education on harm reduction for patients who are identified as “at risk” substance users through screening and assessment. A commonly used structure for brief intervention is the “FRAMES” approach (Feedback, Responsibility, Advice, Menu for change, Empathy, Self-efficacy) (NSW Ministry of Health, 2023).

Motivational Interviewing

Motivational Interviewing is a non-confrontational counselling strategy which utilises an individual's own ambivalent attitudes (e.g., towards the negative impacts of substance use) as a tool to elicit motivation for change. The four steps in Motivational Interviewing are engagement, focus, evoking and planning for change (Miller and Rollnick, 2002). Motivational Interviewing has been proven to improve treatment adherence and engagement (DiClemente et al., 2017).

Cognitive Behavioural Therapy

Cognitive behavioural therapy (see Chapter 33) examines the interrelationship between thoughts, emotions and behaviours. Specific techniques are used to challenge unhelpful thoughts and maladaptive behaviours which are

thought to contribute to distress or psychological symptoms of the individual's condition (Graham, 2004). Techniques include cognitive restructuring, goal setting and event scheduling (Graham, 2004).

Relapse Prevention

Relapse prevention strategies are an integral part of treatment planning. Strategies include normalising relapse, planning for high-risk situations, enhancing commitment to change and ensuring social supports (NSW Department of Health, 2008).

Self-Help Groups

Numerous self-help groups are available and may be helpful for individuals with co-occurring conditions. This includes Alcoholics and Narcotics Anonymous, SMART Recovery as well as specific “dual diagnosis” recovery groups (Kelly et al., 2020). There can be diversity in the ethos of groups, and it is important to ensure that groups are conducted in a non-confrontational manner which re-enforces formal treatment messages. Note that the use of medications (e.g. opioid treatment programs) can be controversial in 12-step programs which encourage complete abstinence in recovery (Roth et al., 2014). It is also important to consider whether an individual suffers from social anxiety or impairments which may preclude group participation (Peros et al., 2021).

Mindfulness Training

Mindfulness is a meditative technique which encourages individuals to focus on the present, and allow thoughts and feelings to occur without judgment (Ostafin et al., 2015). This improves awareness of unhelpful patterns of thinking which may feed into cycles of substance use and mental disorder (Carmody, 2015).

Contingency Management

This involves positive re-enforcement of desired behaviour. Examples include providing vouchers in exchange for clean urine samples and treatment adherence (Davis et al., 2016). Contingency management has been proven to be particularly effective in encouraging substance abstinence (Ginley et al., 2021).

Pharmacotherapy in Addiction

Medications can be used in conjunction with psychosocial interventions to support the aims of relapse prevention and/or harm minimization.

Pharmacotherapy options for alcohol use disorder and opioid use disorder are discussed in this section. Note – for information on the use of medications and approaches to managing acute withdrawal syndromes, please refer to the NSW Health Handbook on the Management of Withdrawal from Alcohol and Other Drugs (NSW Ministry of Health, 2022).

Alcohol Use Disorder

In Australia, pharmacotherapies which are TGA-approved for the treatment of moderate to severe alcohol use disorder include acamprosate, naltrexone and disulfiram. The recommended approach is to utilize pharmacotherapies as part of a comprehensive treatment plan, which typically includes psychosocial interventions (Haber and Riordan, 2021).

- Acamprosate: a GABA analogue which is thought to modulate GABA and glutamate pathways associated with withdrawal cravings. Acamprosate is used in maintaining abstinence.
- Naltrexone: an opioid receptor antagonist, which reduces levels of dopamine (and therefore reward mechanisms) associated with alcohol consumption. Indications include maintaining abstinence and reducing excessive consumption (“controlled drinking”).
- Disulfiram: interferes with the metabolism of alcohol by inhibiting acetaldehyde dehydrogenase. This results in the accumulation of acetaldehyde, which is responsible for the unpleasant “disulfiram reaction” with exposure to alcohol. This includes flushing, dizziness, nausea and vomiting, palpitations and headaches. Given this potentially severe reaction, disulfiram has a strong deterrent effect and is indicated only for those committed to maintaining abstinence.

Other pharmacotherapies which are used off-label in alcohol use disorder include baclofen and topiramate. Evidence supporting their use is less robust, but they may be considered as second-line treatments where approved pharmacotherapies have failed or are contraindicated. Further information and specific prescribing considerations can be found in Guidelines for the Treatment of Alcohol Problems (Haber and Riordan, 2021).

Opioid Use Disorder

Opioid agonist treatment refers to the provision of long-acting opioid medications as part of a structured intervention, which involves regular review and psychosocial supports. The primary goal of opioid agonist treatment is the reduction of harm associated with illicit opioid dependence. This includes diverse short and

long-term health, economic and social harms for the individual and community (e.g., overdose, disease transmission, psychological instability, homelessness, crime, incarceration). Long-term stabilisation and engagement allow individuals to address comorbid health concerns, as well as implement lifestyle changes and strategies that can help improve control over their substance use. Over time this can lead to safer or reduced patterns of use, improved psychosocial function and health, and ultimately abstinence. (NSW Ministry of Health, 2018)

Approved opioid agonist treatments in Australia for treatment of opioid use disorder include:

- Methadone: full opioid agonist
- Sublingual buprenorphine (“Subutex”): partial opioid agonist
- Sublingual buprenorphine-naloxone (“Suboxone”): partial opioid agonist & opioid antagonist
- Long-acting injectable buprenorphine (“Buvidal” and “Sublocade”): partial opioid agonist, available in weekly and monthly formulations

Some patients may seek opioid antagonist treatment with naltrexone as a relapse prevention strategy. Although this may be cautiously considered in select circumstances, evidence supporting the safety and efficacy of naltrexone treatment interventions is limited and naltrexone is not currently PBS-approved for use in opioid use disorder. See NSW Clinical Guidelines – Treatment of Opioid Dependence (NSW Ministry of Health, 2018) and Clinical Guidelines for Use of Depot Buprenorphine (NSW Ministry of Health, 2019) for further information.

Pharmacotherapy in Co-Occurring Conditions

Medications can be indicated in the treatment of both drug and mental health conditions. Given the complexities of co-occurring presentations, there are several important factors to consider, including medication adherence, abuse potential, polypharmacy and drug interactions. See Comorbidity Guidelines for further considerations in specific co-occurring addiction and psychiatric conditions (Marel et al., 2022).

Conclusion

The management of co-occurring drug health and mental health conditions is challenging. It has been shown that individuals with co-occurring conditions have poorer health outcomes and more severe psychosocial dysfunction than those with separately occurring conditions. This includes greater severity of drug use, poorer

physical health, increased risk of suicide and self-harm, unemployment and homelessness (Marel et al., 2022). The complexity and heterogeneity of co-occurring presentations further make it difficult to draw definitive conclusions about universally effective treatment approaches.

These challenges, while significant, are not absolute barriers to meaningful engagement or progress. The general principles for providing addiction treatments to patients with co-occurring conditions emphasise individualised and comprehensive care. Adopting collaborative, client-centred and trauma-informed approaches can help facilitate engagement, build trust in the therapeutic relationship and optimise treatment outcomes (Marel et al., 2022).

Further Reading

Guidelines on the management of co-occurring alcohol and other drug and mental health conditions in alcohol and other drug treatment settings:

comorbidityguidelines.org.au/pdf/comorbidity-guideline.pdf

Chapter 30: Comorbidity of Psychiatric Illness and Physical Illness

Daniel Talbot

Westmead Hospital, Western Sydney Local Health District, Australia, and Specialty of Psychiatry, The University of Sydney, Australia.

and

Anthony Harris

Specialty of Psychiatry, Sydney Medical School, The University of Sydney, Australia, Westmead Institute for Medical Research, Australia, and Western Sydney Local Health District Mental Health Services, Australia.

Introduction

Psychiatric illnesses rarely occur in isolation; patients will often present with two or more co-occurring psychiatric illnesses. For instance, a mood disorder is often accompanied by an anxiety disorder, and substance use disorders and trauma often present hand-in-hand. This pattern of co-occurrence includes physical illness as mental and physical health are closely interconnected. For instance, chronic physical illnesses such as cardiovascular disease, diabetes, chronic pain, cancer, and autoimmune disorders often have a significant impact on mental health, while psychiatric illnesses can, in turn, affect the onset, course, and prognosis of physical conditions for example the association of schizophrenia with a substantial burden of physical illness. By understanding these interactions, we can provide more holistic and comprehensive care to individuals with comorbidities.

Epidemiology of Comorbidity

Comorbidity of Psychiatric Illnesses

Many patients will present with multiple concurrent psychiatric illnesses, however prevalence rates of comorbidity reported across populations differ. For instance, results from a North American national survey demonstrate that the

lifetime prevalence of having two or more psychiatric disorders was as high as 28%, and three or more was 17% (Kessler et al., 2005). Similar findings have been demonstrated in Australian adults ($n = 8,841$), with approximately 25% of people reporting more than one concurrent psychiatric illness (Teesson et al., 2009), whilst a German sample returned a psychiatric comorbidity prevalence rate of 12% amongst 5,318 adults in the community (Jacobi et al., 2014).

Comorbidity of mental health issues also tends to vary with age, with children demonstrating less psychiatric comorbidity compared to adults (Kessler et al., 2005). Vasileva et al. (2021) conducted a meta-analysis of 10 epidemiological studies on children aged 1-7 years ($n = 18,282$), demonstrating that 6% had more than one psychiatric illness. On the other hand, some special populations tend to have greater psychiatric comorbidity than the general population. For instance, foster children (e.g., 31% of foster children in a Norwegian sample; (Lehmann et al., 2013) and incarcerated psychiatric patients (e.g., 57% of Dutch men in psychiatric penitentiary centres; (van Buitenen et al., 2020) report far higher rates of psychiatric comorbidity compared to people in the community.

Depression and anxiety disorders (see Chapter 18), being the most prevalent mental health conditions, are most commonly comorbid with other psychiatric illnesses (Kessler et al., 2005). However, due to the complexity and the intrusiveness of symptoms, trauma-related conditions such as Post Traumatic Stress Disorder (PTSD; see Chapter 20) are also commonly comorbid with other psychiatric conditions. For example, Kang et al. (2019) systematic review ($n = 24$ studies) demonstrated that PTSD was strongly comorbid with depressive disorders (33%-52%), generalized anxiety disorder (14%-15%), and substance use disorders (2%-11%) amongst older war veterans.

Table 30.1. Commonly occurring specific psychiatric illnesses and physical health conditions.

Commonly Occurring Conditions	Description
Depression and Cardiovascular Disease	Depression is frequently comorbid with cardiovascular disease, including conditions like coronary artery disease, heart failure, and stroke. The chronic stress associated with depression can contribute to inflammation, endothelial dysfunction, and other physiological changes that increase the risk of cardiovascular problems (Hare et al., 2014).
Anxiety Disorders and Gastrointestinal Disorders	Anxiety disorders, such as generalized anxiety disorder and panic disorder, often coexist with gastrointestinal conditions like irritable bowel syndrome and inflammatory bowel disease. The exact mechanisms underlying this comorbidity are not fully understood, but stress and the gut-brain axis play significant roles (Meuret et al., 2020).
Eating Disorders and Metabolic Conditions	Eating disorders, such as anorexia nervosa, bulimia nervosa, and binge eating disorder, often have comorbid metabolic conditions, including diabetes (Winston, 2020), and metabolic syndrome (Hudson et al., 2020). The unhealthy eating patterns associated with eating disorders can disrupt metabolic regulation and lead to long-term physical health consequences.
Bipolar Disorder and Cardiovascular Disorders	Bipolar disorder has been linked to an increased risk of cardiovascular disorders, including hypertension, coronary artery disease, and metabolic syndrome (Laursen et al., 2013). Both the inherent biological factors and lifestyle behaviours, such as sedentary lifestyle and poor dietary choices, contribute to this comorbidity.
Schizophrenia and Metabolic Syndrome	Individuals with schizophrenia have a higher prevalence of metabolic syndrome, which includes obesity, dyslipidaemia, hypertension, and insulin resistance (Mitchell et al., 2013). Antipsychotic medications, particularly second-generation antipsychotics, can contribute to weight gain and metabolic disturbances, further increasing the risk of metabolic syndrome.
PTSD and Chronic Pain	Chronic pain conditions, such as fibromyalgia and chronic low back pain, are commonly comorbid with PTSD. Traumatic experiences can heighten pain perception and increase the risk of developing chronic pain conditions. Conversely, chronic pain can exacerbate PTSD symptoms and complicate recovery (Brennstuhl et al., 2015).

Comorbidity Psychiatric Illnesses and Physical Conditions

Comorbidity is not limited to psychiatric illnesses alone but also extends to physical health conditions. The presence of comorbidities significantly impacts the diagnosis, treatment, and overall well-being of individuals, requiring a comprehensive and integrated approach to patient care. At present, in the absence of such an approach, people with a mental illness have a substantially reduced lifespan of 15.9 years in men and 12.0 years in women compared to the general community (Lawrence et al., 2013). This difference is increasing.

The Australian National Survey of Mental Health and Wellbeing indicated that 1 in 8 (12%) adults with a 12-month prevalence of mental illness also reported a comorbid physical condition. Additionally, 1 in 20 (5%) reported two or more comorbid physical conditions (ABS, 2008). Further to this, the *National Health Survey: Mental Health and co-existing physical health conditions 2014-2015* indicates that 15.8% of all Australians (3.6 million people) report co-existing long-term mental and behavioural and physical health conditions. Examples of commonly occurring specific psychiatric illnesses and physical health conditions are listed in Table 30.1.

Overlapping Aetiologies - The Reason Comorbidity is so Common

A significant reason for widespread comorbidity across psychiatric illnesses is that many disorders have overlapping aetiologies, meaning that they can have common underlying causes or risk factors. This subtle interchange of aetiological processes over the developmental course in the individual such as genetics, neurobiological abnormalities, stressors from the environment, and cognitive and behavioural processes contribute to a multifinality of outcomes in which multiple disorders develop and express themselves as different psychiatric and physical conditions (Table 30.2).

It's important to note that while these overlapping aetiologies provide insight into the co-occurrence of mental health disorders, they do not fully explain the complex interactions and individual differences involved. Mental health disorders are multifaceted, and the interplay between genetic, environmental, and psychosocial factors is unique for each person. Understanding these overlapping aetiologies can help inform treatment approaches that address multiple conditions simultaneously. Also, one needs to avoid attributing physical symptoms to psychiatric illness in people with an established psychiatric diagnosis. The identification of new symptoms should trigger a careful review and investigation recognising that although the somatisation of psychological issues occurs, so does physical illness.

Table 30.2. Overlapping aetiologies of disorders.

Genetic Factors	Many mental health disorders have a genetic component, and certain genetic variations or mutations can increase the risk for multiple disorders. For instance, there may be genetic susceptibility shared between depression and anxiety disorders, or between bipolar disorder and schizophrenia (Lee et al., 2019; Smoller, 2016).
Neurobiological Abnormalities	Disruptions in brain structure, function, or neurotransmitter systems can be involved in the development of various mental health disorders. For example, autoimmune disease has been implicated in significant psychiatric and neurological illness e.g. anti-NMDA encephalitis (Pollak et al., 2020).
Environmental Stressors	Adverse life events, such as trauma, abuse, or chronic stress, can contribute to the development of different mental health disorders. Traumatic experiences, are needed to develop post-traumatic stress disorder (PTSD) but also increase the risk of developing schizophrenia (Bailey et al., 2018). Substance abuse can exacerbate both physical and mental illness.
Cognitive and Behavioural Processes	Certain cognitive and behavioural processes may be common to multiple mental health disorders. Negative cognitive biases, impaired emotion regulation, or maladaptive coping strategies can be observed in conditions like depression, anxiety disorders, and eating disorders (American Psychiatric Association, 2013).

A similar set of closely related factors can be described for the interaction of psychiatric illness and physical illness. Particularly for chronic illnesses there is substantial overlap in illnesses (see Figure 30.1). People with psychiatric illnesses are more likely to smoke, abuse other substances, eat poorly and be more sedentary than other people in the community worsening both their physical and mental health. Our health system addresses the intersection of physical and mental health poorly, with an unclear demarcation of responsibility for the physical health care needs of people with a psychiatric illness and a loss of general health care skills in specialist services. The pharmacological treatments of severe mental illness in particular is associated with weight gain, sedation and other adverse effects that are causally related to the development of cardiovascular disease, type 2 diabetes mellitus, obesity and metabolic syndrome (Firth et al., 2019). These illnesses are related in their own right with an increased risk of high prevalence psychological disorders such as depression and anxiety.

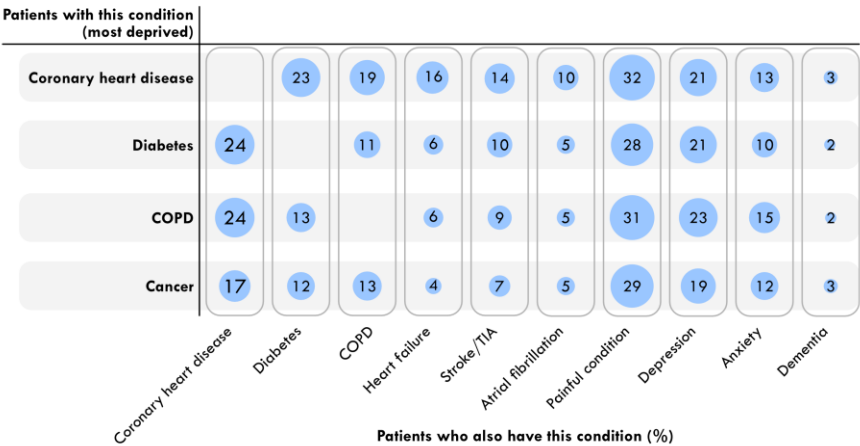


Figure 30.1. Selected comorbidities in people with four common, important disorders. COPD=chronic obstructive pulmonary disease. TIA=transient ischaemic attack (Barnett et al., 2012).

Mutual Maintenance Model of Comorbidity

There are several models that have been proposed to explain the occurrence of comorbidity. One of the most well-established models is the Mutual Maintenance Model (Sharp and Harvey, 2001). The Mutual Maintenance Model is a theoretical framework that seeks to understand the complex relationship between different disorders and how they can influence and exacerbate each other. It suggests that comorbidity is not merely a coincidence but rather often leads to a vicious cycle where the presence of one disorder worsens the other and vice versa. Notably, this model has been applied to the occurrence of PTSD and chronic pain, and proposes that the two disorders exacerbate one another through several mechanisms. First, attentional and cognitive biases toward potentially threatening sensations may lead to overestimations, heightened expectations, and negative interpretations of both fear- and pain-evoking stimuli. Second, pain itself may act as a cue for trauma, triggering fear and hyperarousal, and vice versa. Last, depressive symptoms, avoidance, and fatigue could lead to inactivity, physical deconditioning, and disability - all perpetuating further avoidance and preventing fear extinction (Asmundson et al., 2002).

Treatment Implications

Given the significant prevalence of psychiatric and physical comorbidities, it is essential to consider the implications comorbidities have for treatment.

Look For Comorbidities in Assessment

Although psychiatric conditions initially may present alone, they frequently develop into a range of inter-related pathologies. Common aetiologies and the effects of many physical illnesses or complaints on the individual mean that psychiatric illness is frequently experienced. Anticipating the presence of depression and or anxiety in an individual with chronic pain, is as important as looking for hypertension in an individual with schizophrenia.

Be Part of a Treating Team

Coordinated care involving the general practitioner and whatever specialist care is required will provide the best chance of good quality care. The person in the best place to provide this coordination is usually the general practitioner as long as they feel confident in the treatment of someone with a mental illness. A multidisciplinary approach to care is indicated for many physical health conditions, such as diabetes, integrating mental health care into this matrix will improve patient outcomes.

Early Intervention and Careful Treatment Choice

Prevention is key to the reduction of disease burden and lifestyle interventions around substance use, weight and exercise are central to that. Past reluctance or pessimism around the use of such interventions in people with a mental illness is not warranted with effective interventions developed (Firth et al., 2019), however these programs are still often not generally available.

Treatment for psychiatric illness should acknowledge the interaction of physical and mental illness. Non-pharmacological treatments are frequently effective and have fewer physical adverse effects than pharmacological interventions. For example exercise is an effective treatment for depression and many forms of anxiety, and has an important role to play in the maintenance of our general health (Singh et al., 2023). This effect is bi-directional with improved mental health through exercise assisting in the management of physical illnesses. Improved nutrition helps avoid obesity and metabolic disorder while contributing to mental health (Jacka et al., 2012).

The recognition that many pharmacological treatments have significant adverse effects should also guide initial treatment selection towards psychological interventions for mental illnesses such as anxiety or depression. If pharmacological treatment is indicated due to symptom severity or patient choice, then consideration of possible adverse effects should contribute to medication selection. Avoiding medications associated with significant weight gain is important.

Related to the common aetiology of many psychiatric illnesses is the effectiveness of psychological and pharmacological treatments across diagnostic categories. As an example, antidepressants such as selective serotonin uptake inhibitors (SSRIs) are effective for the treatment of both anxiety and depression.

Screen, Intervene, and Advocate!

Due to the wide range of physical comorbidities that can arise in the population of people with a mental illness it is important to make sure that not only are standard monitoring guidelines adhered to, for example thyroid function tests for individuals being treated with lithium due to the propensity of lithium to alter thyroid function, but more generally that the person with a mental illness, is being seen for their general health. This goes beyond the role of the treating mental health team and needs to involve the whole range of health professionals anchored around the person's general practitioner. Routine health screening, immunisation and interventions for substance use should occur. It is also not sufficient to ensure that blood pressure, weight and waist circumference are being monitored, but that any abnormalities identified are effectively treated and followed up.

Unfortunately, the stigma that attaches to psychiatric illness is still associated with far less assertive care for people with a psychiatric illness resulting in fewer investigations, fewer interventions, and less treatment of physical illness in people with a psychiatric illness (Mitchell et al., 2012). This is even though people with a psychiatric illness are as adherent to their physical health medication as other people in the community (Gorczyński et al., 2017). This contributes to the large gap in life expectancy between people with a psychiatric illness and the general community. Hence the need for advocacy for people with a mental illness when they are in the health system or important treatment choices are being made.

Conclusions

An understanding of comorbidity is central to the development of effective management plans for people with a mental illness. This comorbidity crosses over from psychiatric illness to include substance use and physical illness and is responsible for a significant gap in longevity between people with a mental illness and the general population. The basis of this comorbidity lies in common genetic and other aetiological roots of illness, and its treatment is best addressed by a multidisciplinary health team that can anticipate and then treat the multifaceted problems that our patients present with.

Further Reading

Firth, J., Siddiqi, N., Koyanagi, A. I., Siskind, D., Rosenbaum, S., Galletly, C., ... & Stubbs, B. (2019). The Lancet Psychiatry Commission: a blueprint for protecting physical health in people with mental illness. *The Lancet Psychiatry*, 6(8), 675-712.

TREATMENTS



Artist: Anne Naylor

Title: *The Lithium Membrain*

Medium: Acrylic Paint on Canvas

Dimensions: 1500 x 1000 mm

Chapter 31: Sleep Wake Cycle Management

Lisa Lampe

School of Medicine and Public Health, The University of Newcastle, Australia.

Introduction

Problems with sleep are common in the community. A telephone survey of a representative national sample determined that about 20-35% of those surveyed reported some type of sleep difficulty a few times a week or more, and estimated that about 7% of respondents had a severe clinical insomnia and about 5% had sleep apnoea (Hillman and Lack, 2013). Inadequate sleep has personal and broader health and economic consequences, both direct and indirect. This section introduces strategies to assist in managing primary insomnia, that is, insomnia not secondary to medical or other causes. Apart from the psychological distress and daytime sleepiness it can cause, one analysis suggested that 2.9% of depression and 3.9% of workplace injuries might be attributable to primary insomnia (Deloitte Access Economics, 2011).

DSM-5-TR identifies *Insomnia Disorder* when an individual experiences one or more of: difficulty initiating sleep, difficulty maintaining sleep or early morning awakening with inability to return to sleep; and when these problems cause distress or impairment, occur at least 3 nights per week and have done so for at least 3 months (American Psychiatric Association, 2022). The diagnosis cannot be made where the individual is voluntarily restricting their sleep or where the insomnia can be attributed to another disorder (e.g., depression, anxiety disorder) or a substance, or where it is better explained by another sleep-wake disorder (e.g., narcolepsy). (American Psychiatric Association, 2013: xli).

Assessment

An assessment of sleep problems should include the following:

1. Nature of the problem and phase of sleep (e.g., initial, middle, terminal, trouble getting off to sleep or staying asleep)

2. Duration of the problem and any possible precipitants

3. Concern about the problem – what worries the person most about their sleep problem?

4. Careful history around sleep hygiene including:

- pattern and frequency of sleep problems, for example, how many times per week, triggered by stress etc.
- pattern of day time activities that may interfere with sleep, such as exercise or large meals too close to retiring
- any ‘napping’ through the day
- time to bed
- any ‘rituals’ before retiring (e.g., checking for noise, having a specific snack or drink)
- activities in bed: especially reading, watching TV, using mobile devices
- time taken to fall asleep: average, minimum, maximum
- any awakenings through the night, their duration and responses to them (e.g., go to toilet, have a snack, take medication or herbal preparations)
- nightmares and abnormal sleep behaviours such as sleepwalking
- time of waking
- time of rising

The history should clarify whether the pattern differs on weekdays and weekends. Information about the sleep environment is also important, for example, noise, light and heat, physical discomforts, as well as details about caffeine, alcohol, and other substance use.

5. Factors that promote good quality sleep such as exercise and relaxation.

6. Beliefs about the causes and consequences of insomnia: here we are particularly interested in perceived risks and adverse consequences of poor sleep that may contribute to anxiety about not sleeping, which in turn may heighten arousal around bedtime. Examples of such beliefs include fears of losing control the next day (e.g., panicking, having angry outbursts) or being unable to function (e.g., making too many mistakes at work and being fired, being too tired to go to work or study). There may be beliefs that poor sleep will cause physical or mental harm, or impact adversely on relationship or work functioning. You should consider whether there is evidence of overestimation of the cost or likelihood of any feared consequences ('catastrophising').

7. Medical and psychiatric history aiming to identify any causal or contributing factors including illnesses/conditions and their treatment. Remember to ask about painful conditions and restless legs. Consider the possibility of sleep apnoea, especially in the presence of risk factors such as obesity and hypertension.

Factors contributing to insomnia will usually be evident from the history. However, it may be useful to collect additional data. For example, to ask the individual to keep a sleep diary for a week or two. The sleep diary should cover 24 hours each day, and ask the person to note times when they get into bed and out of bed, and when they are asleep. Additional factors such as exercise, stresses or any factors the patient believes may be relevant may also be noted. As 'clock watching' can contribute to anxiety about sleep, the diary keeping period is kept short.

There are also numerous mobile applications for monitoring sleep. Recent data suggest that wearable technology that relies on accelerometer data may be reasonably accurate, although possibly more so for good rather than poor sleepers (Lorenz and Williams, 2017). Nevertheless, it is widely accessible and may avoid the problem of 'clock watching'.

Management

A comprehensive management plan is based on careful assessment. The treatment package should consider biological and psychological elements, including cognitive and behavioural interventions. A stepped approach is often helpful, beginning with psychoeducation about the biology and psychology of sleep, and sleep hygiene. The two-process model of sleep-wake regulation (circadian, or Process C, and homeostatic sleep drive, or Process S) can be explained in an amount of detail and technicality appropriate to the patient's background and desire for information. What is essential is to convey information that the patient will be able to understand

and apply to their situation. Good sleep hygiene is the foundation of an effective sleep-wake cycle management program.

Common patterns of insomnia

- The individual who typically falls asleep in front of the television but then feels wide awake as soon as they get into bed. There is typically associated anxiety (performance anxiety) about being able to sleep.
- Initial insomnia (also referred to as increased sleep onset latency) associated with going to bed too early relative to rising time: typically, sleeping in then trying to go to sleep again within less than 16 hours or so, leaving inadequate time for sleep pressure (via Process S) to build.
- Waking at daybreak or with the rest of the family, for example, to get children ready for school, then going back to bed and sleeping for another few hours, followed by initial insomnia that night.
- Restless or broken sleep with awakenings to go the toilet or in response to noise from partner or children.

Evidence Base

CBT for insomnia (CBT-I) is usually delivered as a treatment package consisting of a number of elements including psychoeducation, sleep hygiene, cognitive challenging, relaxation and behavioural strategies such as stimulus control and sleep restriction. Several meta-analyses of randomised controlled studies reported medium to large effect sizes for both subjective and objective measures of sleep quality, maintained until at least 12 month follow-up (Geiger-Brown et al., 2015; Okajima et al., 2011). Psychological measures also improved, and it was noted that total sleep time showed improvement from post-treatment to follow-up.

Fundamentals of Good Sleep Hygiene

The fundamentals of good sleep hygiene are shown in the box below. Other suggestions such as a tryptophan-rich snack or drink (milk, bananas) and a warm shower or bath (avoid overheating) have no evidence to support them, but some people may find these helpful before bed, perhaps especially if such activities come to be associated with ‘winding down’ towards sleep.

It is appreciated that many people without insomnia do not follow all the sleep hygiene recommendations. However, the health professional can encourage people who are experiencing troubling insomnia to try to implement all these strategies fairly strictly in order to have the best chance of creating change. Once the insomnia has improved, then if they strongly desire, for example, to read in bed or sleep in on weekends, the individual can try a cautious introduction of these activities and see whether they can maintain their healthy sleep patterns.

If psychological factors such as excessive generalised worry, or specific fears around the causes or consequences of insomnia are identified, then cognitive interventions may also be helpful. Depending on the degree of improvement following implementation of sleep hygiene principles over at least a few weeks, it may be advisable to proceed to other levels of intervention. These include *stimulus control* and *sleep restriction*.

Fundamentals of good sleep hygiene

1. Have a regular rising time (most important in regulating Process S) ideally following normal circadian rhythms and aligned to natural cycles of light and dark (to set Process C). The most important factor in setting (entraining) the circadian clock is light exposure in the morning. To entrain a 24-hour regular sleep wake cycle requires getting up in the morning, at the same time every day, including weekends. After a late night or a poor night's sleep, the individual should get up at the usual time and go to bed around the usual time the next night. Ideally the person spends daytime awake and night-time in bed.
2. A regular bedtime is desirable (though not as important as a regular rising time).
3. Manage light exposure: early morning sunlight is helpful, and night-time bright and blue light (such as that emitted by electronic devices such as phones and tablets) is best avoided.
4. Restrict activities in bed to sleep and sex (i.e. no reading, use of electronic devices, watching TV).
5. Spend only the amount of time in bed that is required for sleep, plus 15-20 minutes to fall asleep.
6. Wakefulness can be affected by physical and mental activity levels, fasting/feeding, social interactions, lighting conditions and posture. Therefore, it is helpful to 'wind down' in the hour or two before bedtime: avoid stressful or activating activities, heavy meals and strenuous exercise.
7. Manage alerting substances: caffeine has a long half-life and those experiencing insomnia should at least trial avoiding it after mid-afternoon.
8. The bedroom should be quiet, dark and a comfortable temperature, without excessive bedding likely to cause overheating.
9. Avoid daytime naps longer than 10-20 minutes (the advised time limit varies).

Stimulus Control

Stimulus control refers to strategies that aim to break conditioned arousal and strengthen the bed and bedroom as stimuli for sleep. It is effective in treating sleep onset and sleep maintenance insomnia. The key strategies are:

1. Go to bed when sleepy (not too early, but also not delaying once feeling sleepy). It is important to distinguish physical fatigue from sleepiness.
2. If not asleep within 15-20 minutes, get up out of bed and sit quietly without engaging in stimulating activities or exposure to excessive light. Go back to bed when again feeling sleepy.

Stimulus control should be combined with sleep hygiene principles, particularly avoidance of non-sleep related activities in bed (apart from sex), setting a regular rising time, and avoidance of napping.

This strategy can also be employed to assist in breaking habits such as waking repeatedly to toilet where this is not necessary. Many people habitually go to the toilet when they wake up, and this can of itself present a stimulus to wake. Most people can retrain their bladder to last the night by refraining from immediately getting up to go to the toilet when they wake up. Rather, they might try lying quietly and not focussing on bladder sensations. If not asleep again within 15-20 minutes they should get up. A health assessment for causes of urinary frequency or lowered bladder capacity may be indicated for some patients.

Sleep Restriction

Sleep restriction is more accurately a restriction of time in bed. Many individuals attempt to compensate for poor quality or inadequate sleep during the night by going to bed earlier and earlier. In taking a history it is not uncommon to find that a person is spending 10 hours or more in bed. Most people have a sense of how many hours sleep they need to feel rested and in good health. For adults, this will often be around 6-8 hours, depending on age. If 7 hours sleep would be 'normal' for a person, but they spend 10 hours in bed, it is highly likely that they will be awake for 3 hours during the night. This time may occur through frequent awakenings, or in long stretches. This is an important element of psychoeducation, and based on this information many patients can make immediate changes to their sleep-related behaviours.

More formally, average time asleep and 'sleep efficiency' (proportion of time in bed that the person is asleep) can be calculated by means of a sleep diary or electronic monitoring. A rising time is determined based on factors such as work or

study routines, and normal circadian rhythms. Working backwards, a retiring time is calculated based on the average hours of sleep and allowing 15-20 minutes to fall asleep. Limiting time in bed to the reported average sleep time increases the homeostatic drive to sleep. It is not recommended to restrict time in bed to less than 5-6 hours. When the average sleep efficiency is greater than 0.85 for a week, time in bed can be increased by 15 minutes (Siebern and Manber, 2010). The 'sleep window' ideally will match normal circadian rhythms, for example, going to bed when the sleep drive is highest and the activating process lowest.

Tips for Clinical Practice

Sleep restriction is very effective and improvements can often be seen within a week. However, it requires a significant degree of motivation and commitment from the patient. Careful attention needs to be paid to making the rising and retiring times realistic. As an example, for a patient who has been routinely going to bed at 0100 hours, lying awake for a couple of hours each night, and then sleeping in until 1100, setting a new rising time of 0600 is unlikely to be realistic or consistently achievable. It can also be hard to get up and sit out of the bed when the house is cold in winter. A stepped model of intervention, beginning with the easier strategies to employ, may be a practical approach.

Further Reading

Grima, N. A., Bei, B., & Mansfield, D. (2019). Insomnia management. *Australian Journal of General Practice*, 48(4), 198-202.

Sleep Health Foundation: sleephealthfoundation.org.au/fact-sheets.html

Chapter 32: Structured Problem Solving and Behavioural Activation

Lisa Lampe

School of Medicine and Public Health, The University of Newcastle, Australia.

Introduction

This chapter introduces CBT techniques that have a good evidence basis, are widely applicable and suitable for use in a range of settings; they are well suited to situations in which there is little time available for each session, and in which the total number of sessions that can be offered is relatively small. In general, they are also techniques that may be mastered relatively quickly by therapists without specialist mental health qualifications, and so are particularly well suited to general medical settings.

Problem Solving Therapies (PST) and Structured Problem Solving (SPS)

Problems and challenges are an inevitable part of life, and individuals need effective strategies for navigating them. Being faced with dilemmas and difficulties in life is usually at least somewhat stressful. Whilst emotion-focussed coping (focussing on, and managing, emotional responses) has an important role in self-management, it is also important to be able to take a problem-focussed approach when faced with a potentially modifiable stressor. The ability to effectively problem solve is seen as an important contributor to social competence (D'Zurilla, 1988). Social competence refers to the ability of an individual to function independently and deal with the demands of everyday life; it requires social skills, behavioural competencies and adaptive coping strategies. The ability to solve problems in real-life settings is sometimes referred to as *social problem solving*, which is said to include elements of social learning and self-management, as well as being a general coping strategy (D'Zurilla, 1988). Problem solving therapies (PST) are an example of problem-focussed coping strategies. There was a rapid expansion of PST in the

1970s and 1980s, with the development of a number of structured approaches, including Structured Problem Solving (SPS).

Rationale for SPS

Numerous studies have shown a link between perceived, or actual, poor problem-solving ability and poorer physical and mental health, as well as a tendency to avoid problems rather than deal with them directly. Conversely, those who perceive themselves as effective problem solvers appear more likely to tackle problems directly, have a greater expectation of success, and have a more positive self-concept. Better problem-solving is postulated to play an important role in both mediating, and moderating, the relationship between stressful life events and well-being (Bell and D'Zurilla, 2009). More effective problem solving may reduce the burden of stress from life problems, but also contribute to enhanced confidence and sense of self-efficacy.

Evidence Base

PST has been shown to be more effective for depression than a treatment that involved discussion of current life problems with a problem-solving goal, but without explicit training in problem-solving skills (Nezu, 1986). The same study found more positive changes in behaviours and attitudes towards problem solving in the PST group, providing support for the view that these skills can be learned. A meta-analysis of 31 studies of PST across all types of mental or physical health problems (Malouff et al., 2007) including depression, dysthymia, obesity, conduct disorder, substance misuse and suicidal ideation, found effect sizes of the order of 1.4 compared to waitlist or no treatment; 0.5 against treatment as usual and attention-placebo; and 0.2 against other experimental treatments. This analysis found that PST that included training in problem-orientation and homework was associated with a larger effect size (Malouff et al., 2007). A meta-analysis focussing on depressive disorders reported equivalence for medication ($d = -0.13$) and alternative psychosocial therapies ($d = 0.17$), and superiority to supportive therapy and attention control ($d = 0.45$). PST in anxiety has been shown to be effective, but to date has generally not been shown to be superior to other forms of treatment (Kendrick et al., 2005; Provencher and Ladouceur, 2004).

Problem Solving

Problem-solving in real-life social settings includes two processes: problem orientation and problem-solving strategies. Problem orientation refers to beliefs and attributions around problems and problem-solving in general, for example, beliefs about the causes of problems, general views about the likelihood of finding solutions,

beliefs about personal problem-solving ability, and locus of control (the degree to which an individual feels they can control events and outcomes in their lives). The major goals of PST are to facilitate a positive problem orientation and enhance problem-solving skills and ability.

Positive Problem Orientation

Aspects of positive problem orientation include perceiving problems as challenges that can be dealt with effectively, seeing a potential to benefit from the situation, having a positive view of one's own ability to problem-solve, and being realistic about the time and effort involved, and about outcomes (for example, it is unlikely that there will be a 'perfect' solution to any problem).

Structured Problem Solving

Problem solving ability can be enhanced by taking a rational and structured approach to problem solving. There are four main problem solving skills taught in PST (D'Zurilla, 1988). These are: problem definition and formulation, generation of alternative solutions, decision-making, solution implementation and verification. These may be further subdivided into the following steps:

1. Problem definition and formulation
2. Generation of alternative solutions
3. Evaluation of pros and cons of each possible solution
4. Choice of solution (or combination of solutions)
5. Planning for and implementing chosen solution(s)
6. Review of the outcome

Problem definition is a key step, as it formulates the issue in a way that is amenable to the problem-solving approach. Many individuals find this difficult, tending to present broad issues, emotional distress or decision-making dilemmas, for example, 'I'm too anxious' or 'Should I leave my job?'. Steps 5 and 6 are also important to focus on: many people can generate ideas about how to tackle their problems, but either fail to make concrete plans, or generate plans that are not realistic. Many people do not review the outcome, and so may miss the opportunity to learn from success, or to fine tune their approach to further improve the outcome. Without a review there is also a risk that individuals may see a suboptimal outcome as final, and fail to appreciate that they can use what they learned from this initial attempt to solve the problem to inform their next cycle of problem-solving. It is important to realise that many problems in adult life are complex, and typically associated with uncertainties such that the first attempt at problem-solving may not achieve an ideal outcome. It does not reflect a failure of the individual or an insoluble problem, but rather realistic challenges.

In clinical practice SPS can be conducted with a patient in one long session, which is likely to require 45-60 minutes. This will only be possible where all the necessary information is to hand – sometimes there is a need to obtain further information to assist in generating options or weighing them up. In a community setting, including general practice, a series of 15 minute appointments based around each problem solving step, with ‘homework’ for the patient between visits, can work well.

Clinical Example

Tahlee is a 26 year old single mother with two boys aged 2 years and 4 years. She presents to her GP in distress asking: *‘What should I do? I don’t know whether to move to Gosford or stay in Sydney!’* Tahlee presents the problem as though it is a decision-making dilemma with a choice of two options. Exploration is important in order to define and formulate the real problem.

Doctor: *“Can you tell me about why you are thinking of moving?”*

Tahlee: *“My mother lives in Gosford. She could help with the children.”*

Doctor: *“What kind of help do you need with the children?”*

Tahlee: *“I want to get a job. There’s no-one to care for the kids while I would be working.”*

Doctor: *“So getting a job is a priority for you?”*

Tahlee: *“Yes! But I need childcare to do that.”*

Doctor: *“It seems like getting a job and finding childcare are both problems. Which would you like to focus on as the main problem?”*

Tahlee: *“I think finding childcare is my main problem.”*

The problem is now more clearly defined and more amenable to problem-solving. Tahlee can now be introduced to the principles of structured problem solving, and the doctor can support her in working through the steps. It is most helpful if Tahlee can generate as many options as possible. In achieving this, the doctor should avoid making suggestions. Patients can be asked to canvass others for ideas, or to imagine what others might do. These tasks can be set as homework between sessions. It is then important for Tahlee to be realistic about the pros and cons of each option. For example, her list of possible solutions might look like this:

1. Move to Gosford to be closer to Mum.
2. Ask ex-partner’s mother if she can help.
3. Find out more about eligibility for government childcare subsidy.
4. Start a family day care business so I can work at home.

5. Look for other ways to work from home or be able to have the kids with me.
6. Wait until the kids are a bit older.

Tahlee's GP encourages her to consider the pros and cons of each possible solution in turn. For example:

1. Gosford

<i>Pros</i>	<i>Cons</i>
Mother happy to babysit	Very few jobs available
Cheaper to live in Gosford	Don't know anyone up there except Mum

2. Ask ex-partner's mother to help

<i>Pros</i>	<i>Cons</i>
I get on quite well with her	Don't really want to see more of ex-partner, though he doesn't live there
She has offered to help before	
Can stay in Sydney where there are more jobs	

And so on, until all options are considered. The health professional has an important role in supporting patients to be realistic in their assessments. Once all options have been assessed, the patient chooses the best option or combination of options and makes a specific implementation plan, covering aspects such as 'who, what, when, where, how'. In the example above, Tahlee might choose to approach her ex-partner's mother for help, but find out more about the government childcare subsidy at the same time. In the medium term she might also explore options around ways to work from home.

In the final step, a review is undertaken, either by the patient alone or supported by the health professional. How well did the option work? Is there a residual problem to be addressed? It will sometimes be the case that the problem may be partially resolved and could benefit from another cycle of problem solving.

The long-term aim is for patients to develop independent problem-solving skills. Along the way they may encounter more problems with which they need support to apply this technique. The health professional's attitude of faith in the individual's ability to solve their own problems, together with avoidance of telling

them what to do or giving advice, will promote the acquisition of this skill and confidence in using it.

Behavioural Activation

Behavioural activation (BA) is designed as a brief, time-limited intervention to increase activity and engagement in behaviours that are associated with positive emotional consequences. It is most often employed in depression, since depressed individuals particularly have been shown to engage in less positively reinforced behaviour but also to employ avoidance behaviours, and strategies such as rumination and complaining that have been described as escape behaviours (Moulds and Kandris, 2007). However, BA may be of value wherever inactivity and withdrawal have become prominent problems.

Behavioural activation therapy for depression is based on the ‘behavioural analytic theory’ of depression, a model that proposes that individuals can become depressed when there is an imbalance in reward and punishment in their environment; namely, too much punishment and inadequate reward. Behavioural activation therapy aims to reconnect individuals with sources of positive reinforcement in their environment by scheduling activities into their day that provide them with a sense of pleasure or achievement. The first therapy package developed from this model is attributed to Lewinsohn and colleagues in the 1970s, who developed Behavioural Activation as an intervention aimed at increasing engagement in pleasurable activity. A key consideration of the behaviour analytic model is that the purpose or goal of a behaviour is more important than the form it takes. For example, spending all day indoors reading may be an avoidance behaviour for one individual, a pleasure rarely afforded to another, or a necessity of student life for a third. For the first person, reading functions as avoidance, for the second it is a pleasure in itself, and for the third it is a means to a desired end.

With the advent of cognitive therapy in the 1980s, behavioural activation was deemed inadequate as a treatment for depression as there was no consideration of cognitive factors. However, renewed research interest in behavioural activation found it to be equally effective as a full cognitive behavioural treatment package for depression at post-treatment (Jacobson et al., 1996) and prevention of relapse at 2-years follow-up (Gortner et al., 1998). In a more naturalistic setting of existing clinical services (both general practice and psychological therapy practices), BA delivered by junior mental health workers was found to be non-inferior to CBT delivered by accredited CBT therapists (Richards et al., 2016). BA lends itself well to manualisation, and the principles of delivering this therapy can be mastered quickly. It is therefore an ideal therapeutic intervention for non-specialists. BA typically includes psychoeducation about depression and the rationale for focusing

on activity, identification of activities that bring a sense of pleasure or achievement (or that have this potential), and a diary for planning activities and monitoring the results, for example, rating the sense of pleasure and/or achievement gained. It is important not to limit the focus to pleasurable activities, but also to target those that may bring a sense of achievement. A BA program may also include baseline monitoring of activity and identification of behavioural goals (e.g. (Lejuez et al., 2001)). When using the technique to treat a specific condition, for example, anxiety or depression, baseline ratings of the target symptoms, together with progress monitoring should be undertaken. For example, use of rating scales such as the Depression, Anxiety and Stress Scales (DASS; (Lovibond, 1995)). In most cases, BA can be effectively delivered within eight sessions.

BA may be combined with ‘graded task assignment’, whereby behavioural goals that are initially too difficult or demanding are broken down into smaller steps that themselves become targets for achievement within a BA plan.

Tips For Clinical Practice

Many patients with psychological problems are highly self-critical and may tend to set perfectionistic goals and targets. A key element of BA is mastery, so a good rule of thumb is to set goals for which there is a 90% likelihood of achievement, in order that the patient predominantly experiences success. Discussion with the patient to assist them to set realistic (rather than idealistic) goals and targets can be an important element of therapy. It is better to set small and easily accomplished goals with the experience of success than to add to experiences of failure. For example, asked to identify a goal that would bring a sense of achievement a patient may nominate ‘completing my overdue tax return’. Setting this as a goal may be unrealistic, and it may be better to employ graded task assignment here, by breaking up this goal into a series of smaller and more manageable tasks. This is best negotiated with the patient so it can take into account their specific circumstances, but might include goals such as ‘sort through the pile of papers on my desk to identify those that may be relevant for tax’, ‘see if I have my group certificates’, ‘obtain group certificates’, following which the patient might begin the tax return by completing one page at a time of the online form, or one category of expenses at a time if on paper, for example.

Since lack of motivation and problems with initiating behaviour are the problems that BA aims to treat, the health professional will need strategies to increase the likelihood that the patient will actually plan and complete tasks. This is most likely to be the case when at least some of the planning is undertaken within the session, rather than simply presenting information and advice then leaving the patient to implement it all at home. Taking enough time to ensure the patient has

engaged with the therapy – that is, that they appreciate the rationale and agree that it may have some benefit for them – is also important. This may mean spending more than one session on the introduction to and preparation for BA. See (Lejuez et al., 2001) for an example of this.

A sense of personal achievement and confidence will build motivation, and may be enhanced by supporting the patient to periodically review and acknowledge their achievements. For patients who feel as though nothing brings pleasure, it can also be helpful to sensitively guide them to attend to positive ratings, which might also indicate activities worth repeating. Examples of recording forms may be found on various websites or in (Lejuez et al., 2001).

Conclusions

Problem-solving therapies and behavioural activation are therapeutic interventions that may be mastered by generalist clinicians without specialist mental health qualifications, and which can be implemented over relatively brief time periods. They may be as effective as other psychological treatments and in some cases as effective and better tolerated than pharmacotherapy. They may be used alone or together with pharmacological or other psychological interventions. The selection of therapeutic intervention is best guided by an assessment of the individual's presenting problems.

Further Reading

- Cassar, J., Ross, J., Dahne, J., Ewer, P., Teesson, M., Hopko, D., & Lejuez, C. W. (2016). Therapist tips for the brief behavioural activation therapy for depression-revised (BATD-R) treatment manual practical wisdom and clinical nuance. *Clinical Psychologist*, 20(1), 46-53.
- Lampe L. (2008) Take Control of Your Worry. Chapter 9 “Structured Problem Solving” pp100-117. University of Sydney Press. Sydney, NSW.
- Lejuez, C. W., Hopko, D. R., LePage, J. P., Hopko, S. D., & McNeil, D. W. (2001). A brief behavioral activation treatment for depression. *Cognitive and Behavioral Practice*, 8(2), 164-175.
- Pierce, D. (2012). Problem solving therapy: Use and effectiveness in general practice. *Australian Family Physician*, 41(9), 676-679.

Chapter 33: CBT and the Cognitive Behavioural Model

Juliette Drobny

Specialty of Psychiatry, The University of Sydney, Australia.

Introduction

Cognitive Behaviour Therapy or CBT is a psychological skills-based treatment for a wide variety of mental disorders. As its name suggests, it is based on a combination of cognitive and behavioural principles to assist individuals in identifying unhelpful and irrational thoughts and behaviours and replace them with more realistic and adaptive ones. Unlike traditional long-term psychodynamic psychotherapies that often require years, CBT is a short to medium term therapy focussed on current symptoms and problems, designed to equip patients with a range of skills to enable them to become their own therapist. To understand how CBT works, it is important to be familiar with the underlying theoretical principles.

Principles of Behaviour Therapy

There is a common misconception about what behavioural treatments are. Behaviour therapy is often misunderstood as a treatment that simply involves *behaving* in a certain way or *doing* certain things. However, behaviour therapy is based on learning theory involving classical conditioning (sometimes known as Pavlovian conditioning) and operant conditioning.

Classical Conditioning

Around the turn of the 20th century, Ivan Pavlov, a Russian physiologist, was the first to discover and study the phenomenon of classical conditioning, a type of associative learning (i.e., learning through association). While conducting experiments on the digestive processes of dogs, he observed that when presented with food (unconditioned stimulus) dogs would naturally salivate (unconditioned response). However, the dogs also salivated to neutral stimuli present at the time food was delivered, such as hearing the footsteps of the assistant who fed them. He

ran experiments where he used neutral stimuli such as ringing a bell immediately before delivering food. After several pairings, the dogs learned the bell signalled the delivery of food such that the bell on its own triggered the dogs to salivate (see Figure 33.1). The bell became a conditioned stimulus and salivation to the bell was the conditioned response.

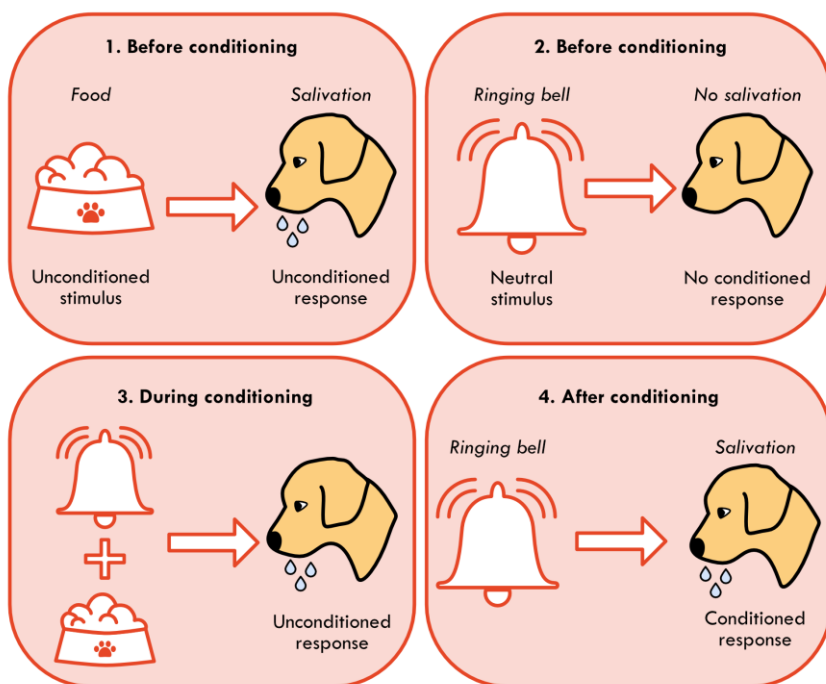


Figure 33.1. Pavlovian conditioning.

Classical Conditioning and Human Fear

Watson and Raynor (Watson and Rayner, 1920) demonstrated that human fears could be learned through Pavlovian conditioning principles with the case of Little Albert. Little Albert was an 11-month-old boy with no known fears, who was conditioned to fear a white rat. A loud noise (unconditioned stimulus) triggered a startle response and crying (unconditioned response). After repeated pairings of the white rat with the loud noise, the white rat alone triggered a conditioned fear response. The fear also generalised to include other furry animals and toys. Thus, classical conditioning is relevant to fear acquisition and the development of anxiety disorders, most notably post-traumatic stress disorder.

Operant Conditioning

Operant Conditioning was first introduced by the highly influential American psychologist and behaviourist, B.F. Skinner (Skinner and Vargas, 1957). It refers to modifying an individual's behaviour through providing consequences to either increase or decrease a behaviour. Operant conditioning is another type of associative learning where learning occurs through rewards and punishments for behaviour. In operant conditioning, the learner makes an association between a behaviour and its consequence. For example, if you tell a joke and everybody laughs, you're likely to tell that joke again. Here, people laughing is the reward or reinforcement which increases the joke telling behaviour. However, if you tell a joke and no one laughs, you're unlikely to tell that joke again. In this way, nobody laughing acts as a punishment and decreases the behaviour.

Components of Operant Conditioning

The components of operant conditioning involve reinforcement and punishment. A reinforcer is any event that strengthens or increases the behaviour, while a punishment is any adverse event or outcome that causes a decrease in the behaviour. Reinforcers and punishments can either be positive or negative. In operant conditioning, positive refers to the *addition* of a stimulus while negative refers to the *removal* of a stimulus. So, positive reinforcement involves adding a desirable stimulus after the behaviour, an example of which is praising children for putting away their toys. Here, praise is used as a positive reinforcer to increase good behaviour.

Negative reinforcement is often mistakenly confused with punishment. In negative reinforcement, a bad outcome or feeling is removed after the behaviour. For example, imagine a person with a dog phobia who feels very anxious around dogs. Whenever he avoids a dog, his anxiety rapidly reduces. The reduction in anxiety feels good and as such is reinforcing which then serves to strengthen or increase his avoidance behaviour around dogs.

Punishment can also be positive or negative. In positive punishment an aversive stimulus is added after the behaviour, for example receiving a speeding fine after speeding. In negative punishment, a desirable stimulus is removed after the behaviour, such as confiscating a favourite toy after bad behaviour. Both positive and negative punishment serve to decrease the frequency of the behaviour.

Negative Reinforcement and Avoidance

Negative reinforcement is extremely relevant to the maintenance of anxiety disorders and depression. Avoidance or escape is a ubiquitous response to anxiety provoking or distressing situations. As shown in the example of avoidance in a dog phobic displayed in Figure 33.2, distress rapidly rises in response to the dog. The distress is a highly unpleasant experience that triggers the urge to avoid or escape. In the short-term, avoiding the dog results in a rapid reduction in distress and therefore becomes very reinforcing. Through negative reinforcement, the person has learned avoidance helps to reduce their distress and so continues to use avoidance strategies to manage their distress. Avoidance therefore serves to maintain distress in the long-term.

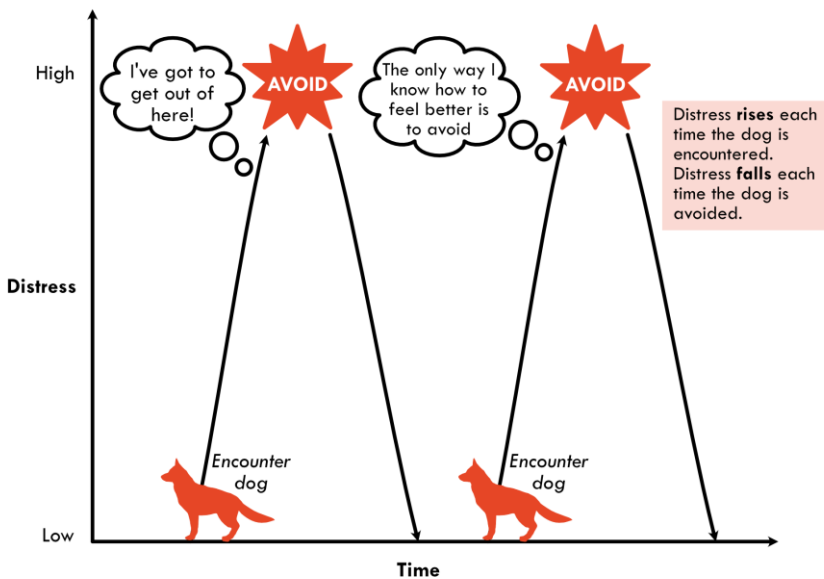


Figure 33.2. Role of avoidance in the maintenance of distress.

Additionally, it is imperative to recognise that by avoiding, the person never learns what happens if they remained in the situation. Would the dog actually bite? Avoidance prevents people from learning whether or not their fears come true, therefore perpetuating such fears, potentially over a lifetime.

Avoidance behaviours are critical to the maintenance of anxiety and depressive disorders. Though not always labelled as avoidance, behaviours designed to reduce distress and/or prevent feared outcomes can be considered a form of avoidance. In Obsessive Compulsive Disorder (OCD), such behaviours are termed compulsions or rituals (e.g., handwashing, checking). Safety seeking behaviours are another form of avoidance used in many psychological conditions. They refer to any behaviour the

person uses to minimise their feared outcome from eventuating. Consider the following safety seeking behaviours across different disorders and their impact on feared outcomes:

- A person with social anxiety disorder goes to a party. He fears others will think he is boring. He minimises speaking and keeps his answers brief.
- A person with post-traumatic stress disorder goes to a restaurant. He fears being harmed. He sits with his back to the wall, facing the entrance and constantly scans the environment for threatening people.
- A person with depression is in a new relationship. They believe they are unlovable and fear being rejected. They avoid expressing their opinions and always agree with their partner's requests.

In all these examples, the safety seeking behaviours function to reduce the person's distress in the short-term and prevent validation of the underlying fears, however they also prevent new learning and therefore directly contribute to the maintenance of such fears and beliefs over time.

Learning Theory

Different aspects of learning theory, namely classical and operant conditioning, have been highly influential in understanding the acquisition and maintenance of psychological disorders. Learning theory has also shaped the development of behavioural treatments for anxiety and depressive disorders, namely exposure therapy and behavioural activation, respectively. Exposure based treatments are based on unlearning learned associations from classical and operant conditioning principles, while behavioural activation is based purely on operant conditioning principles. Concepts involved in weakening learned associations will firstly be covered.

Extinction

When a conditioned stimulus is repeatedly presented *without* the unconditioned stimulus, it results in a reduction in the conditioned response. Returning to Pavlov's dogs, during the initial trial of an extinction program, the dog will continue to salivate to the bell. However, after repeated trials of hearing the bell without delivery of food, the dog learns the bell no longer signals food and salivation to the bell extinguishes.

Habituation

Habituation is a simple type of non-associative learning that leads to reduced behavioural and physiological responses to repeated or prolonged stimuli. Shown in every animal species, it involves learning to ignore non-essential stimuli that are neither helpful nor harmful. Habituation does not require conscious awareness. Habituation is experienced in daily life, an example of which is hearing a loud jack hammer across the street. Initially, the sound grabs your attention and you find it distracting and annoying. Over the next few days, the sound continues at a constant pace such that eventually you end up tuning it out. You have habituated to the sound.

Exposure Therapy

Principles of learning theory described above lead to the development of behavioural treatments for human fears, namely exposure therapy. During exposure therapy, the patient is exposed to the feared stimulus in a safe environment. In lay terms, exposure therapy involves facing one's fears.

Imaginal Versus In Vivo Exposure

Exposure therapy can be conducted in imagination or in real life (in vivo). In imaginal exposure, the patient confronts the feared stimuli in their imagination whereas in in vivo exposure the patient interacts with the situation in reality, for example driving, catching a train, heights. In vivo exposure yields faster results and better transfer to the actual situation. However, imaginal exposure may be preferable when:

- It is difficult or impractical to obtain access to real life cues (e.g., flying, dogs)
- Anxiety provoking cues are internal (e.g., thoughts, traumatic memories, intrusive images)
- Anxiety to the real cue is too great

Whether imaginal or in vivo exposure is used, the procedures involved are largely identical.

Treatment Rationale

As confronting feared stimuli elicits some level of discomfort, the first and perhaps most important step involves providing a thorough treatment rationale (Addis and Jacobson, 2000). Patients should be introduced to the role of avoidance behaviours in the maintenance of anxiety and how graded and repeated exposure to

feared situations leads to reduced anxiety over time and the opportunity to allow disconfirmation of fears. The use of everyday examples can be helpful to demonstrate principles of habituation (e.g., repeatedly watching a horror movie scene, learning to drive). The importance of emphasising the *graded* nature of exposure therapy cannot be underscored enough. There is always a way to decrease the degree of difficulty to safe and manageable levels to engage even the most fearful patient.

Exposure Hierarchy

An exposure hierarchy of around 8 to 12 feared situations is constructed collaboratively with the patient who is the expert on the nature of their fears. Specific factors associated with their fears should be elicited and incorporated into the hierarchy. Situations are ranked from least to most anxiety provoking on a scale from 0 (no anxiety) to 100 (Extreme anxiety). Safety seeking behaviours and rituals may be incorporated into the exposure hierarchy in lower steps but should gradually be withdrawn and removed by completion of the hierarchy. Incorporating such safety seeking behaviours in the final step of an exposure hierarchy artificially reduces anxiety and prevents disconfirmation of fears, contributing to the maintenance of situational fears.

Table 33.1 displays an example hierarchy for a patient with agoraphobia wanting to catch an express train to the city. The factors that influence the level of anxiety include:

- Number of stations
- Alone vs accompanied with friend
- Presence of safety seeking behaviours (e.g., mobile phone, water bottle)
- Crowdedness of the train (e.g. peak hour, weekend)
- All stops vs express train

Table 33.1. Example in vivo exposure hierarchy.

Anxiety Rating	Traveling to the city by express train
100	Travel to city on an express train in peak hour alone without mobile phone or water bottle
90	Travel to city on an express train in peak hour alone with mobile phone and water bottle
80	Travel to city (6 stations) alone on a weekday in peak hour without mobile phone or water bottle
70	Travel to the city (6 stations) on the weekend alone without mobile phone or water bottle
60	Travel to work in peak hour (4 stations) alone without mobile phone or water bottle
50	Travel on a train to work in peak hour (4 stations) alone with mobile phone and water bottle
40	Travel on a train to work in peak hour (4 stations) with friend without mobile phone or water bottle
30	Travel on a train (2 stations) on a weekday but not in peak hour with friend, without mobile phone or water bottle
20	Travel on a train (2 stations) on weekend with friend, mobile phone and water bottle
10	Sit at the train station alone and read the timetable

Graded Exposure

Following construction of the exposure hierarchy, an initial item of mild to moderate anxiety (rated around 20-30) is selected for exposure, either imaginally or in vivo. Starting with lower-level items is useful to demonstrate the procedure and ensure early successful experiences to enhance confidence in the procedure and themselves. When conducting exposure, three conditions are essential:

- Exposure to the feared situation must be prolonged to enable reduction of anxiety.
- Avoidance, safety seeking behaviours or rituals must be prevented.
- Exposure is repeated and conducted in a variety of settings to facilitate new learning.

During imaginal exposure, the patient sits comfortably in their chair with their eyes closed (or initially with eyes open if too difficult) and the feared scenario is described in detail with the patient prompted to imagine and describe their sensory experiences:

- What can you see?
- What can you hear?
- What can you smell?
- What does it feel like?
- What's happening in your body?

When conducting imaginal and in vivo exposure, patients are asked to rate their level of anxiety throughout the exposure using the Subjective Units of Distress Scale (SUDS), where 0 represents no anxiety, 5 represents moderate anxiety and 10 represents extreme anxiety. The exposure task is repeated until the SUDS ratings decrease to low levels. Habituation of anxiety is typically achieved within around 40 minutes, though some patients require longer. Patients are encouraged to repeat the exposure task for homework between sessions. Once the exposure task elicits low levels of anxiety (or at least 50% reduction of the initial anxiety), mastery is considered to be achieved and the next step in the hierarchy is introduced. If the next item feels too difficult, flexibility is required and an intermediary step can be introduced (e.g., initially incorporating a safety seeking behaviour). The procedure is repeated until all steps in the hierarchy have been completed. Progression from imaginal to in vivo exposure (where appropriate) should then occur.

Figure 33.3 depicts the effect of exposure therapy on anxiety over time. In the first exposure trial, anxiety rises sharply and the patient has a strong urge to avoid in some way. However, by remaining in the situation, anxiety initially remains high but then gradually reduces *without* engaging in avoidance. During the second trial, anxiety continues to rise but not as high as the first time and takes less time to reduce. As the exposure trials progress, initial anxiety ratings decrease further and anxiety levels reduce faster.

In OCD (see Chapter 19), in vivo exposure is referred to as exposure plus response prevention (ERP). During ERP, the patient is exposed to a situation that elicits anxiety (e.g., using a stove) and is then prevented from engaging in the neutralising ritual or compulsion (e.g., checking). As with in vivo exposure, ERP adopts a graded approach, such that some rituals are initially permitted but are gradually reduced as the patient progresses through the hierarchy. For example, checking rituals may gradually be reduced from 10 checks, then 6 checks, then 4, 2, 1 and until no checking is performed.

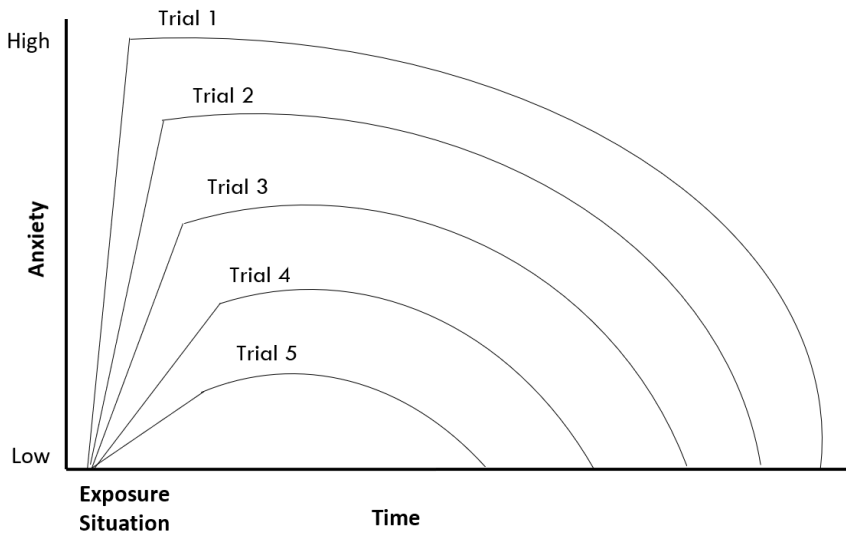


Figure 33.3. Effect of exposure therapy on anxiety over time.

The origins of exposure therapy date back to the 1920s, however it continues to remain a powerful intervention used in the treatment of a variety of disorders, particularly where fear and avoidance behaviours are prominent.

Behavioural Therapy for Depression: Behavioural Activation

The Behavioural Model of Depression

The behavioural model of depression is based on operant conditioning principles (Ferster, 1973). In brief, it suggests that individuals become depressed when there is an imbalance of reward and punishment in their environment, specifically, when there are low rates of reward and high rates of punishment. In this context punishment refers to events, stressors or circumstances that are aversive, controlling or stressful. Examples include social isolation, financial hardship, workplace bullying, marital tension, physical health problems and caring for a parent with dementia. In contrast, reward refers to any activity or experience that provides pleasure, achievement, meaning or fulfilment, such as spending time with a friend, watching a movie, going out to dinner or playing sport. The behavioural treatment of depression is referred to as *Behavioural Activation*. Behavioural activation is a relatively simple treatment that emphasizes the association between increased activity and improvement in mood (Lewinsohn and Graf, 1973). It encourages patients to engage with sources of reward in their environment and to reduce

potential sources of punishment. The following describes the major components of behavioural activation.

The Depression Cycle

When conducting behavioural activation, it is important to introduce the Depression Cycle depicted in Figure 33.4 which underscores the strong relationship between activity level and mood. The depression cycle starts with feeling depressed, which produces symptoms of decreased motivation, lack of interest and reduced energy. These symptoms promote avoidance behaviours whereby depressed individuals begin to avoid activities or withdraw from people or situations, such that they start doing less. They do fewer activities that give them pleasure and/or a sense of achievement, resulting in reduced rates of reward. It follows that the fewer activities individuals engage in, the less *opportunity* they have for experiencing pleasure and achievement, resulting in them *experiencing* less reward, thereby maintaining their depression. To break the cycle, individuals are encouraged to *increase* their level of activity to increase opportunities for experiencing pleasure and achievement as a means of improving mood.

A primary objective of behavioural activation is to highlight that behaviour can be changed before mood improves. Indeed, waiting for one's mood to change (e.g., "I'll go for a walk when I have the motivation") is likely to keep the person trapped in the cycle of depression. Instead, patients are encouraged to engage in the activity even if they are "not in the mood". Despite there being real biological and cognitive changes during depression, the individual is still physically capable of performing most behaviours. For example, in order to go for a walk, an individual must select and put on appropriate clothing, fill a water bottle, open the front door, walk outside etc., all of which can be completed during depression.

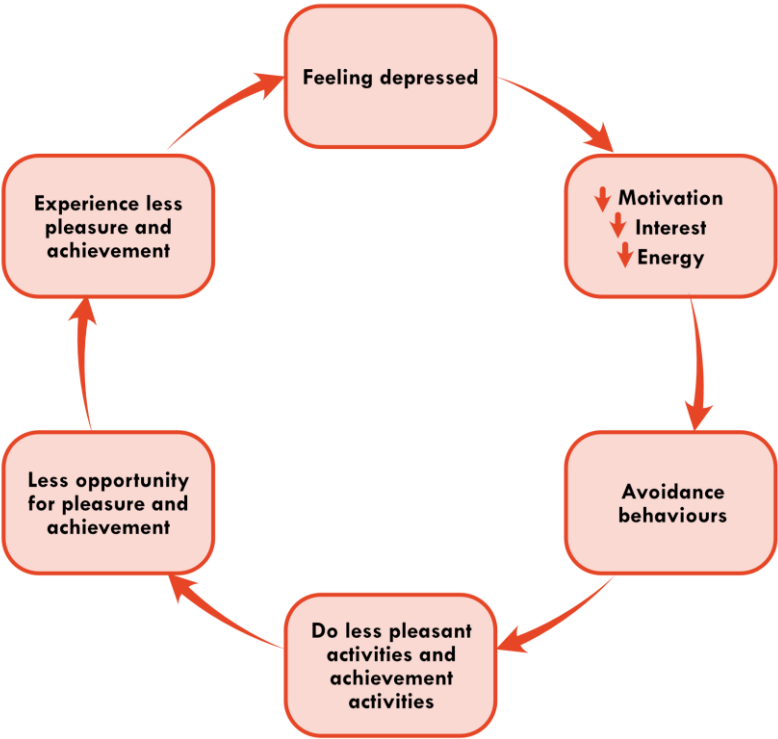


Figure 33.4. Behavioural cycle of depression.

Role of Avoidance in Depression

The role of avoidance in maintaining depression also needs to be highlighted to patients. When individuals become depressed, avoidance and escape behaviours are frequently used to reduce distress or avoid difficult interpersonal interactions. Avoidance behaviours can be conceptualised as a coping response, as illustrated in the following example.

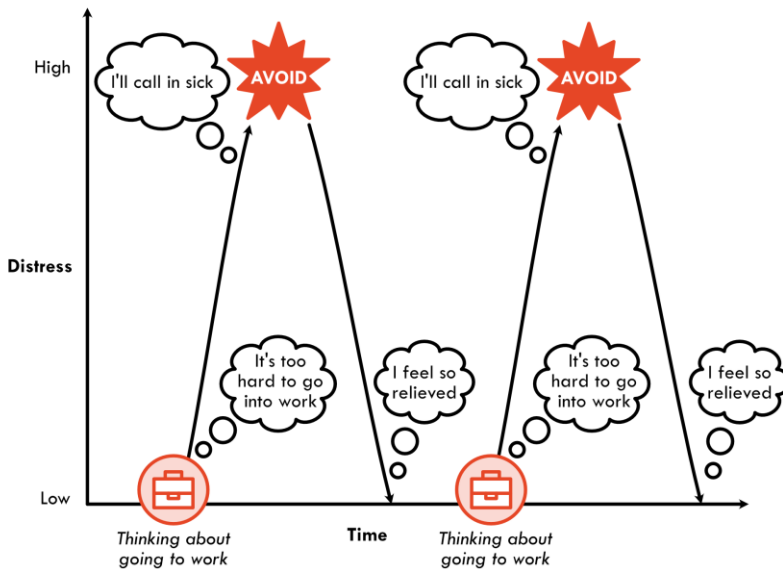


Figure 33.5. Role of avoidance in depression.

Diana is a 36-year-old lady with depression. She wakes in the morning feeling overwhelmed and filled with dread at the thought of having to go to work. As depicted in Figure 33.5, this situation rapidly increases her level of distress. In an attempt to cope with her negative feelings, she calls in sick (i.e., avoids going to work) and takes to bed. Instantly, her distress levels decrease as she feels relieved, she does not have to face the demands and pressures of going in. So, in the short-term, avoidance makes her feel better by taking her bad feelings away (negative reinforcement), and if something is reinforcing it increases the likelihood that the behaviour will be repeated. Hence the next time she wakes feeling overwhelmed at the thought of going to work, she is likely to call in sick again.

The problem with Diana's avoidance is that although it makes her feel better in the short term, it takes her away from naturally occurring sources of positive reinforcement in her environment. By avoiding work, she misses out on opportunities to experience a sense of achievement from her work or having enjoyable social interactions with co-workers. Additionally, her avoidance can contribute to secondary problems, such as an increased workload, time pressure, reduced income, financial pressures and even relationship tensions with her boss, serving to increase sources of punishment in her environment. The reduction in positive reinforcement and increase in punishment maintains the depression and promotes further avoidance. Countering avoidance with adaptive coping behaviours is an important component of behavioural activation.

Activity Monitoring

After presenting the depression cycle and role of avoidance, patients are instructed to complete activity monitoring forms where they are asked to record their level of activity and corresponding mood ratings to make connections between the two. Having the patient record their pleasure and achievement ratings for each activity can also be useful for selection of future activities, as high pleasure and achievement ratings suggest activities that should be encouraged, whereas low ratings suggest the activity may be less useful or may indicate the presence of maladaptive behaviours within the activity such as rumination.

Selecting Activities

The next step involves selecting rewarding activities for the person to engage in and scheduling them into their day. The choice of activities must be tailored to each patient's specific interests and circumstances. A useful method for selecting activities is to start by identifying specific goals in different life domains, such as family and relationships, work, study, health, fitness, spirituality, leisure and so on. Goals should be selected in relation to the patient's values for each domain. For each domain, a range of activities are developed and ranked from easiest to most difficult. For each activity, the frequency and duration are collaboratively discussed with the therapist.

Scheduling Activities

Once a range of activities has been identified, they need to be scheduled into a timetable. It is critical to schedule activities in advance, at least the night before. This way, patients are not relying on their mood to dictate their activity for the day. It is also imperative that patients write down their activities rather than mentally tell themselves what they are going to do the next day. Writing the activity into a timetable or diary serves as a 'contract' that maximises the likelihood of completing the activity. Patients are encouraged to tick off the activity after it has been completed which provides an additional sense of achievement. Patients often comment that by writing it down, it encourages them to complete the activity as they don't want to let themselves down.

Writing it down also minimises the chance of forgetting. With concentration and memory problems being commonplace in depression, forgetting to complete an activity contributes to unintentional treatment non-compliance and deprives the patient of a possible opportunity for positive reinforcement.

A range of pleasure and achievement-based activities should be selected when scheduling activities with a balance of each scheduled into each day. Previously avoided activities (e.g., replying to emails, completing tax returns) can be scheduled as achievement-based activities which should also help prevent secondary problems from developing. Again, patients are encouraged to record their level of pleasure and achievement for each task shortly after completing it to provide an accurate representation of their experience at the time.

When scheduling activities, the therapist wants to maximise success and minimise failure. Selecting an activity that is too challenging or overwhelming will result in either intentional non-compliance or a sense of failure, further perpetuating low mood. Breaking the task down into a series of smaller steps can be very helpful. The patient is encouraged to simply complete only the first or second steps. In this way, the activity is no longer viewed as overwhelming and the chance of success is maximised which serves to enhance mood.

Subsequent Sessions

In behavioural activation, the therapist and patient collaboratively work together in selecting and scheduling activities for the patient to complete between sessions as homework. Subsequent sessions involve reviewing how the patient went, drawing conclusions about the relationship between mood and activity level, identifying ongoing sources of avoidance as well as tackling obstacles to compliance.

After reviewing homework, activities are selected for the following week. In some instances, additional techniques may be incorporated to assist the patient in completing the activity. For example, a socially isolated patient wishing to increase his social contact may lack the necessary social skills to establish or maintain relationships. As such, social skills training may be incorporated. The primary objective, however, is to move patients from an avoidance to an active-based lifestyle where they are in contact with ongoing sources of positive reinforcement.

Key Aspects to Behavioural Activation

Routine Disruptions

Depressed patients frequently exhibit routine disruptions which serve to maintain their depression. Treatment needs to identify such routine disruptions and develop new, adaptive routines for basic activities such as sleeping, eating, showering, work etc. Often establishing a new routine in itself can provide a significant improvement in mood.

For example, a patient with a long history of depression developed a habit of playing computer games after coming home from work, often playing until the early hours of the morning, resulting in chronic sleep deprivation. He would typically sleep in, causing him to rush to work. When at work, he felt tired throughout the day, was irritable and unfriendly with co-workers which contributed to social exclusion and isolation and thus furthered his use of computer games. As part of treatment, he was initially encouraged to restrict using computer games during the week, engage in alternative sources of entertainment and experiment with going to bed at an earlier time. This change in his routine was effective in reducing his fatigue and irritability which also improved his interactions with his co-workers and lifted his mood.

Role of Choice

A basic tenet of behavioural activation involves highlighting the role of ‘choice’ in maintaining depression. Although patients do not choose to be depressed, it is important to highlight they can *choose* to engage in behaviours that will likely make them feel better, or they can choose to use avoidance behaviours and stay depressed. Patients are encouraged to take responsibility for the behavioural choices they make as well as the consequences such behaviours bring.

To illustrate, a depressed patient agreed to help her elderly neighbour go shopping but shortly after regretted doing so. She acknowledged that calling to cancel their arrangements would instantly give her a sense of relief, but would subsequently make her feel guilty and worthless, fuelling her depression which she would then manage by taking to bed and avoiding her neighbour, a lady whom she often enjoyed socialising with. Alternatively, she could recognise she was not in the mood to go, but take her neighbour shopping anyway, which, based on previous experience, always resulted in her feeling better as she enjoyed knowing she had helped someone and she typically enjoyed the social contact. Discussing her choices and the consequences in this way enhanced the patient’s motivation to engage in active, healthy behaviour.

Rewarding Depressed Behaviours

Consistent with behavioural principles of operant conditioning, therapists and patients need to be vigilant for ways in which the patient may be accessing positive and negative reinforcement for *depressed* behaviour. As previously discussed, avoidance behaviours are often negatively reinforcing, in that they temporarily alleviate distress or reduce responsibilities. However, some depressed behaviours may also attract *positive* reinforcement, for example, in the form of sympathy or increased attention from others.

Troubleshooting Techniques

An issue in behavioural activation that often comes up early in treatment is when patients' depressive symptoms (e.g., passivity, lethargy, poor motivation) interfere in their ability to implement the treatment. If this occurs, a number of strategies can be considered.

Acceptance of the Treatment Rationale

Firstly, it is essential patients are on board with the treatment rationale. If they do not believe that their inactivity and avoidance is causing them problems, they are unlikely to comply with homework exercises to increase their activity levels (Addis and Jacobson, 2000). In such instances, patients are encouraged to adopt an empirical perspective. Having them record their activity and mood ratings for homework and discussing these ratings in session can help them see the connection between their behaviour and their feelings. In addition, encouraging a “wait and see” approach can be helpful where they implement some small changes and observe the effect it has on their mood.

Between-Session Contact

To enhance motivation and compliance, increased contact with the patient can be incorporated between sessions. This can be done through several methods such as calling the patient, or having the patient call in or email after the activity was due to be completed. As patients often do not want to disappoint the therapist or let them down, this can be quite effective in increasing their compliance. Of course, this technique should only be used early in treatment so as not to foster dependency, however it can also be briefly brought back in at other times when there has been a setback and the patient is struggling to make changes.

Use of Rewards and Punishments

Another approach to enhancing compliance involves using operant techniques, that is, using rewards for compliance or punishment for non-compliance. For example, Jeannette, a patient who was passionate about animal welfare said she would donate \$50 to the National Hunters' Association as a punishment if she did not complete her assigned treatment activity.

Rumination

Finally, sometimes patients report that they completed their assigned activation task but it failed to improve their mood. For example, a patient was encouraged to do some gardening, having previously derived great pleasure and achievement from her garden. After weeding the entire garden bed, she reported her mood remained low. When questioned on what she was thinking about while gardening, she reported ruminating about her marriage breakdown and was therefore only partially engaged with the activity. If rumination occurs during the assigned activity, patients should be encouraged to focus their attention onto the task at hand and their surrounding environment, becoming fully aware of their sensory experiences, including sounds, smells, colours, tastes etc. This is similar to mindfulness practices which have been shown to be helpful in reducing rumination (van der Velden et al., 2023).

Rumination can also be conceptualised as a form of avoidance (Moulds et al., 2007). By ruminating on their problems they are not taking active steps in solving or reducing such problems. A husband who was ruminating about an argument he had with his wife, was not actually doing anything productive to improve the relationship. Therefore, assessing the presence and function of rumination can be critical in enhancing the success of behavioural activation treatments.

Effectiveness of Behavioural Activation

Behavioural activation is an effective treatment for individuals with major depression and compares well to other empirically validated treatments for depression (Uphoff et al., 2020). Mazzuchelli and colleagues (Mazzucchelli et al., 2009) conducted a large meta-analysis of studies examining the effectiveness of behavioural activation. They found behavioural activation to be superior to control conditions comprising waiting lists and treatment as usual (Hedges $g = 0.78$, $p < .001$) and equally effective as cognitive therapy (Hedges $g = -0.01$, $p > .05$). In comparison to antidepressant medication, a randomised controlled trial by Dimidjian and colleagues in 2006 (Dimidjian et al., 2006) comparing behavioural activation (BA) with the antidepressant (AD) paroxetine found behavioural activation to be at least as effective as the antidepressant in treating adults with mild depression (remission rates of 44% for BA and 42% for AD, $p > .05$) and more effective in improving remission rates for adults with severe depression (BA = 56%, AD = 23%, $p = .002$) (Dimidjian et al., 2006).

Advantages of Behavioural Activation

Behavioural activation as a treatment is relatively easy for patients to understand, and combined with its effectiveness, makes it a suitable first-line treatment. Meta-analyses on behavioural activation included participants from a wide range of populations, finding it to be a suitable treatment for mild to severely depressed patients, depressed older adults, children and adolescents, and even individuals with dementia and intellectual disabilities.

Unlike cognitive therapy, behavioural activation does not require patients to be psychologically minded or possess high verbal skills. As a non-pharmacological intervention, it does not carry the side-effects commonly experienced with antidepressant medication such as nausea, insomnia, sedation and sexual dysfunction, and has also been found to protect against relapse after antidepressant discontinuation (Dobson et al., 2008).

Finally, due to its simplicity, it is easy to disseminate. It has been effectively delivered in a group therapy format, as well as brief and longer-term individual therapy. It is also suitable for self-help approaches, including bibliotherapy and Internet-based delivery, though it is likely that some therapist input would be necessary due to the potential for depressive symptoms to promote dropout or interfere with compliance. Behavioural activation therefore has the potential to improve the accessibility of effective treatments for depression, helping to reduce the burden of depression for the individual and greater community.

Cognitive Theory

Cognitive therapy is based on the cognitive model of emotion, often referred to as the ABC model. In the ABC model, A refers to a situation or *Activating Event*, and C refers to the feeling or *Emotional Consequence*. Throughout life, people typically make the erroneous assumption that A leads to C, that is, that situations cause feelings. However, according to the ABC model, the situation triggers thoughts, interpretations or *Beliefs* (B) which are responsible for driving the emotional response ($A \rightarrow B \rightarrow C$; see Figure 33.6).

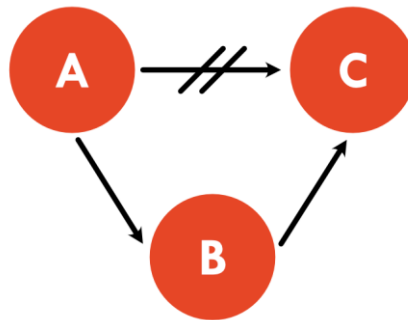


Figure 33.6. The ABC or cognitive model of emotion.

To illustrate, Sarah is lying in bed at 3 am only to be awoken by a loud noise outside (A). She thinks, “There’s a burglar outside!” (B) and feels scared (C). Her neighbour Mary hears the same noise and thinks, “The neighbour’s dogs are going through the bins and have made a mess” causing her to feel annoyed. This example highlights that the situation cannot cause their emotion, as two different emotions were experienced from the same situation. Instead, their *thoughts* are the real driver of their emotional responses. The ABC model can also be extended to explain differing behavioural responses. Upon hearing the noise, Sarah gets out of bed, turns on the light and checks outside, while Mary simply rolls over and falls asleep. Their differing behaviours were directly influenced by their individual cognitions. Thus, the cognitive model highlights the importance of our thoughts in influencing our emotional and behavioural responses. The primary purpose of cognitive therapy is to assist individuals in challenging irrational and unhelpful thoughts and beliefs and replace them with more balanced, realistic ones.

Negative Thoughts That Drive Anxiety and Depression

Anxiety and related emotions such as feeling frightened, scared, worried or nervous are driven by thoughts about danger or threat. Common themes involve threats to health, safety, finances, property as well as psychological threats such as negative evaluation and rejection. While anxiety is obviously the primary emotion in anxiety disorders, it is also a feature of many other psychiatric conditions such as obsessive-compulsive disorder, post-traumatic stress disorder, eating disorders, body dysmorphic disorder, and even psychosis. Consider the corresponding diagnosis for the following threat-based cognitions.

- “If I have a panic attack I could have a heart attack and die.”

- “People might think I’m boring and stupid.”
- “If I make a mistake I could get fired.”
- “The balcony might collapse.”
- “People will see my cheeks are not symmetrical and will think I’m ugly and abnormal.”
- “I have pain in my stomach. I think it might be cancer.”
- “The bank could get robbed again.”
- “I touched the door handle. I might get HIV.”
- “I just ate a banana. I’m going to get fat.”
- “People in the street were laughing at me. My phone calls have been posted on the Internet.”

Depression and sadness are driven by negative thoughts about the self, the world or the future, often referred to as the *cognitive triad*. Common depressive cognitions include:

- “I’m worthless.”
- “I’m a failure.”
- “I’m not good enough.”
- “I’m unlovable.”
- “People are selfish and untrustworthy.”
- “The world is unfair.”
- “Things never work out for me.”
- “My circumstances won’t change.”
- “My future is hopeless.”

Cognitive Therapy

Cognitive therapy, as discussed above, is based on the notion that thoughts mediate the relationship between situations and feelings. For anxiety and depressive disorders, it is argued that the thoughts driving and maintaining those negative emotional states are in some way irrational, unrealistic or unhelpful. To change feelings, the thoughts causing those feelings need to be challenged and replaced with more realistic and helpful thoughts. However, for thoughts to be changed, they must firstly be identified. Indeed, a primary aim in cognitive therapy is to assist individuals in identifying the thoughts or beliefs driving their emotions.

Identifying Negative Thoughts

Thought Monitoring

Based on the ABC model described above, thought monitoring records are commonly employed to help individuals identify their thoughts. For homework, individuals may be instructed to write down the situation, thoughts and emotions when feeling distressed. As the thoughts identified are present when the emotion is present, monitoring thoughts in this way is a good place to start. However, the thoughts recorded may not always identify the *causal thought*, namely the thought that is directly responsible for causing the experienced emotion.

Downward Arrow Technique

Once individuals have some thoughts from their thought monitoring, they can apply the downward arrow technique to identify likely causal thoughts. Like peeling back the layers of an onion to get at the core, the downward arrow technique is a useful method to peel back the layers of thoughts to identify the core or causal thought.

The downward arrow technique involves asking the question, “What would be so bad about that?” Upon receiving the answer, the question is repeated, “If that were true, what would be so bad about that?” and continues to be repeated until the causal thought is identified. There is no set number of times the question needs to be repeated. Typically, the individual will have a ‘gut reaction’ when they have identified the causal thought. The following example illustrates the technique.

Diane is a 21-year-old university student referred for cognitive therapy for depression. Her therapist asked her to complete some thought monitoring based on the ABC model for homework.

Therapist: *“Let’s start by having a look at your thought monitoring form. The situation was you lying in bed thinking about your exam. The emotion was feeling depressed. And the thought?”*

Diane: *“I failed my exam.”*

Therapist: *“Applying the downward arrow technique, if you did fail your exam, what would be so bad about that?”*

Diane: *“Then I’d have to redo the subject again next semester.”*

Therapist: *“And if that were true, what would be so bad about that?”*

Diane: *“I’d be overloaded next semester.”*

Therapist: *“And if you were overloaded next semester, what would be so bad about that?”*

Diane: *"Then I'd probably fail more subjects."*
Therapist: *"So if you failed more subjects, what would be so bad about that?"*
Diane: *"Then my academic transcript would look horrible."*
Therapist: *"And if that were true, what would be so bad about that?"*
Diane: *"If my academic transcript was horrible, I wouldn't get a decent job and I'd amount to nothing."*

By using the downward arrow technique, it can be seen that behind the initial thought about failing one exam, there are very negative beliefs about her future ("I'll never get a decent job") and herself ("I'll amount to nothing").

Working Backwards from Behaviour

Another method of identifying thoughts involves examining an individual's behaviours for clues as to their causal thoughts. A patient with panic attacks who always carries a plastic bag is more likely to be worried about vomiting than having a heart attack. A patient with OCD with cleaning compulsions may say they clean because they like things to look perfect, however their use of expensive antibacterial cleaning products suggests they are fearful of germs and illness.

Assisting patients to develop skills in identifying their thoughts is a crucial first step in cognitive therapy as identification of their causal thought lays the foundation for thought challenging. If the causal thought fails to be accurately identified, subsequent thought challenging will be ineffective in changing their mood.

Cognitive Challenging

Cognitive challenging involves asking questions to undermine the individual's belief in the negative thought. There are five key methods of thought challenging which will be described below.

Challenging the Probability

A common thinking distortion that drives distress is overestimating the probability or likelihood of the negative event (e.g., "I'm having a heart attack", "Everyone will think I'm boring", "I'm going to get fired", "People are out to hurt me"). Inflated perceptions of threat are responsible for maintaining many psychological disorders and cognitive therapy in the form of challenging the probability is aimed at helping the individual establish more realistic estimates of threat. There are several different methods for challenging the probability.

Jury Method

Patients are asked to imagine their thought is on trial in front of a jury and generate evidence to present to the jury so the jury can determine whether the thought is true or untrue. Patients are often very good at generating evidence that supports their negative thought. The jury method helps them to develop a more balanced perspective by considering evidence that may disconfirm the thought as well as encouraging them to question the quality and veracity of the evidence they use in support of the thought.

Thought: “My husband could have a car accident driving home from work and be seriously injured or killed”

Evidence For	Evidence Against
He’s driving at night He’s tired after work Driver fatigue kills more than drink driving I won’t be there to point out potential hazards on the road	He’s an experienced driver He has had zero serious car accidents He’s driven home from work for the last 15 years, i.e. over 3,500 trips at night with only 1 minor accident (rear ended, no injury) Even when I’m not in the car he still gets home safely every day He’s only driving home from work for 30 minutes, not long-distance interstate driving where driver fatigue is most relevant as being dangerous.

Step Method

Asking the individual what steps would be necessary in order for the thought to come true can also be a useful way of challenging a negative thought. Using the previous example of Diane who worried she would amount to nothing if she failed her exam, from the starting point of failing one exam to the end point of amounting to nothing, what steps would need to occur in between? Each step has its own unique probability. By multiplying the probability of each step, a more accurate estimate of the feared outcome can be obtained. In order to amount to nothing, each one of those steps must be 100% guaranteed to come true. If even just one of those steps does not come true, the chain of events is broken (i.e., multiplying a number by 0 equals 0) and therefore the end result cannot occur. Often by identifying all the necessary steps, individuals are able to recognise that certain steps are very unlikely, and in this way they can realise that the situation is not as dire or likely as they feared.

World Method

Another method for examining the likelihood of a thought is to ask what

the world would look like if the thought was true. If failing an exam meant a person would amount to nothing, what would the world look like? Presumably, all people who had failed even one exam would be unemployed and unemployable. This is definitely not the case. Moreover, after failing an exam, students would immediately drop out of university rather than repeat the subject, as according to the thought, repeating the subject would be pointless; they would inevitably amount to nothing and repeating would just be a waste of time and money. Again, this is not the case. Many students who fail an exam will go on to repeat the subject and pass, ultimately completing their degree and going on to have successful careers. By challenging the probability in these ways, individuals are able to start questioning the validity of their thoughts and in doing so, their anxiety reduces and mood starts to improve.

Challenging the Cost

Another thinking distortion individuals frequently make involves overestimating the cost or badness of a negative event. When worrying about a negative event, or even after a negative event has occurred, there is a tendency to view the situation as worse or more costly than it is in reality, often referred to as *catastrophising*. When challenging the cost, patients are encouraged to focus on the realistic consequences (i.e., non-emotional consequences) of the negative event to determine its true degree of badness. Comparing the realistic consequences of their feared negative event with the consequences of other negative events (e.g., head-cold, burglary, house fire, paraplegia, terminal illness) can help them put things in perspective. The distress associated with a negative event should be proportionate to its cost; an event with short-term trivial consequences (e.g., bad haircut) should therefore be less distressing than events with long-term significant deleterious consequences (e.g., paraplegia). For example, a patient with social anxiety worried a woman he spoke to at a party thought he was stupid, resulting in high anxiety and low mood. He was asked to consider the realistic consequences of this woman's opinion of him, that is, will her opinion cause him to get fired, cause him to get cancer, prevent him from driving, buying his groceries or holidaying in Fiji? Recognising there were no realistic consequences to her opinion was helpful in lowering his cost estimates and reducing his distress.

In situations where there are realistic negative consequences, asking individuals to look at the consequences over time can be helpful. For example, how bad will this be in a week, a month, a year, in 5 years and so on. Of course, challenging the cost of high-cost events (e.g., plane crash, death etc.) should be avoided, in which case other thought challenging methods should be considered.

Questioning Control

Questioning control involves asking individuals whether they are worrying about an outcome beyond their control. If they have control over an outcome, then a solution should be implemented. For example, a high-school student worried she would perform poorly in her public speaking presentation. Does she have any control over her performance? Yes. She could practice her speech, seek feedback, perform in front of her family etc. However, it is common for individuals to worry about things they cannot control or over which they have little control (e.g., “*What if I get cancer?*” “*What if the plane crashes?*” “*What if my wife leaves me?*”).

Going back to Diane who thinks she has failed her exam. Perhaps she did fail. However, the examination is now complete. At this point in time, she has no control over the results. What will be, will be. Continuing to worry and feel depressed about the potential failure is going to lead to weeks or months of unnecessary distress and suffering until the results are eventually released. A useful mantra to keep in mind is, “I’ll cross that bridge when I come to it”. If there is no control over the event, let the thought go and deal with the problem if or when it arises. Learning to let go of things out of one’s control is an important part of cognitive restructuring. This point ties in strongly with the concept of helpfulness.

Challenging the Helpfulness

Challenging the helpfulness involves encouraging the individual to consider the advantages and disadvantages of their thought. “What is the thought doing for you? Is it helpful in any way?” Individuals frequently believe there are advantages in thinking the way they do (Wells, 2005). Commonly endorsed positive beliefs about worry include:

- Worrying helps me solve problems.
- Worrying helps me be organized.
- Worrying helps me nip problems in the bud.
- Worrying helps me emotionally prepare.
- If I worry about it, it’s less likely to happen.
- Worrying shows I’m a caring person.

Where individuals endorse benefits to worrying, it’s important to help them consider the downside to worrying. Are there negative impacts on sleep, concentration, muscle tension, productivity, mood, irritability, anxiety, health, relationships? Do the benefits of worrying outweigh these disadvantages? Are the perceived benefits able to be achieved in other, more helpful ways? Encouraging

individuals to challenge the helpfulness of thoughts can move them in the right direction. When the thought re-occurs, they can acknowledge it is unhelpful to engage with and choose to redirect their thinking to more productive pursuits.

External Perspectives

A lot of individuals with anxiety and depression can be very good at advising others. They know what others should be thinking, yet they do not apply it to themselves. Asking them, “What would you tell a friend to help them if they had the thought?” is a useful question to encourage a healthier way of thinking. Individuals can often be very good at generating these sorts of alternative thoughts if it is for someone else. Therefore, externalising the situation to others can be a very useful method for changing an individual’s thought. After externalising the situation to a friend, Diane says, “Ok, you failed the exam. Getting depressed about it won’t help the situation. You’re better off looking at what went wrong in the first instance and correcting those problems. Did you not leave enough time to study because you were spending all your time with your boyfriend? Did you not understand the information and were too embarrassed to ask for help? Just get back in there, make the necessary changes and move on. Thinking you’re a failure and feeling sorry for yourself is not going to help anyone!”

Another useful question commonly used in cognitive restructuring is, “What would your most relaxed and rational friend or family member say?” If thoughts drive emotion, and negative thoughts drive anxiety and depression, it follows that relaxed, rational people must have very realistic, evidence-based thinking. They do not jump to irrational, negative or threat-based conclusions. By imagining how a rational friend would view the same situation can help individuals adopt a more balanced and helpful perspective about their thoughts.

Conclusion

Put simply, cognitive therapy encourages individuals to stop believing the irrational or unhelpful thoughts in their head. As human beings, we have a strong tendency to believe the thoughts that arise in our mind. In depressed individuals, those thoughts and beliefs have caused them great despair and suffering, restriction of activities and strained relationships with family and friends. Cognitive therapy, through the use of these thought challenging methods, is designed to undermine this human tendency.

Behavioural Experiments

What are Behavioural Experiments?

Despite its name, behavioural experiments are a very powerful method for testing and challenging negative thoughts. Their purpose is to test the validity or accuracy of the patient's belief system and help construct more adaptive, realistic beliefs. Behavioural experiments involve planned experiential activities, typically based on either experimentation or observation. Patients' negative thoughts or beliefs are elicited and then an experiment is set up to test out their thoughts. Results from the experiment are gathered and then compared to patients' initial predictions of what they thought would occur. Results that contradict their predictions help to decrease their belief in the negative thought, which can then assist in reducing distress and increasing self-confidence.

A key advantage of behavioural experiments is that they can be tailored to patients' unique cognitions. An experiment is set up to directly target a specific thought and patients experience the results first-hand, making the evidence highly relevant and convincing.

Conducting a Behavioural Experiment

A number of steps are required to implement and conduct an effective behavioural experiment (Bennett-Levy et al., 2004). The first and most important step is defining the target cognition or belief to be tested. Specifying the cognition in this way allows an experiment to be tailored to the patient's belief. The second step involves planning an experiment to conduct in order to test out the patient's belief. This is done collaboratively with the patient. Step 3 involves identifying what the patient *predicts* will occur in the experiment and rating the degree of belief (0-100%) in the prediction. The prediction must be operationalised in an observable, testable way. Predictions involving mind-reading (e.g., "He will not like me") are not suitable. Instead, behavioural markers of the prediction must be identified (e.g., He will look at his watch and make excuses to leave within 15 seconds of talking to me. He will not smile or laugh while talking to me).

The next step involves the patient conducting the experiment and gathering the results. It is essential to define the results in accordance with the prediction (e.g., "We talked for 10 minutes) rather than the patient's subjective experience of the experiment (e.g., "I felt anxious the whole time). After collecting the results, the patient needs to reflect on the conclusion of the experiment. What was learned from the experiment? What does this say about the validity of the original belief? Do the results of the experiment support or disconfirm the belief? Finally, how much does

the patient now believe this thought? Ideally, post-experiment belief ratings should decrease, however if they remain unchanged or even increase, factors that might be contributing to this should be explored.

Types of Behavioural Experiments

There are several different types of behavioural experiments, although active experiments and observational experiments are the most common.

Active Experiments

Active experiments are very useful for combating the avoidance behaviours responsible for the maintenance of negative thoughts. An experiment is set up to test patients' negative cognitions or beliefs. Their predictions or hypotheses of what will occur during the experiment are identified and operationalised. They are then asked to behave in a way that is different from how they would usually act (typically some form of avoidance behaviour). The results of the experiment are recorded and they are encouraged to reflect on the implications of the experiment on their original thought or underlying belief.

To illustrate using Sarah, a patient with panic disorder, her cognition is, "If I panic at the supermarket, I'll collapse". To prevent collapsing, she holds onto her trolley for support (safety behaviour). To test her belief, she was encouraged *not* to hold onto the trolley when experiencing panic sensations to enable her to learn that even if she does nothing to save herself, she does not collapse. In this way, the active experiment reduces her belief about the dangerousness of panic symptoms such that she can experience the sensation without anxiety. Active behavioural experiments operate as a form of exposure therapy but with a strong cognitive component.

Observational Experiments

Observational experiments, as the name suggests, are where patients gather data to test their cognitions or beliefs. This can be done through direct observation or through conducting a survey. Indeed, a variety of creative approaches can be employed to test patients' beliefs. Raj, a patient with body dysmorphic disorder believed he was hideously ugly but in reality was of normal appearance. A survey was used to test his belief which involved placing a photo of his face amongst a selection of 15 photos of other faces from a similar ethnic background, in this case, Indian. Each face was labelled from 1 to 16, with Raj's face as number 9. The photos were shown to a group of people and each person was asked to identify the face they believed was the ugliest. The patient predicted more people would select number 9. The results revealed that no one chose 9 as the ugliest face. Indeed, 9 received

spontaneous compliments such as “9 is cute” and “9 has a nice smile”. The survey was useful to highlight that others did not perceive him to be ugly which made him feel less anxious and improved his functioning.

Case Study

Kaye was a 46-year-old single mother with a teenage son employed full-time as a sales manager in a department store. Premorbidly, she was a high functioning, extremely independent, competent, capable, assertive woman. At the time she came to treatment, she was experiencing a major depressive episode that started 8 months prior in the context of watching her mother die from cancer, and her mood deteriorated further after her mother’s death. Part of Kaye’s depression was a natural grief reaction, however 5 months after her mother died she had a 3-week admission to a private psychiatric hospital for severe depression. She was discharged on an antidepressant and a benzodiazepine to assist with anxiety symptoms. She was referred by her GP for psychological treatment.

Her presenting symptoms, despite the medications, were common depressive symptoms of pervasive low mood, marked anhedonia, impaired sleep and poor appetite. She also experienced severe anxiety, waking in the morning with her stomach in knots and continuous churning sensations, though she did not have an anxiety disorder. The other striking feature of her presentation, given her high premorbid functioning, was her marked avoidance behaviour. Kaye had worked as a manager in the same company for 20 years, acknowledged she was good at her job, but had stopped working for 3 months. Moreover, she moved out of home to live with a cousin for the last 6 months believing she was unable to cope on her own. Her cognition driving this avoidance behaviour was, “I’ve lost my confidence, I can’t cope”.

In the first session, the rationale for how avoidance maintains negative beliefs was presented. Kaye related very well to the way in which avoidance was maintaining her negative belief about not coping and was on board with the idea of testing it out using an active behavioural experiment. Returning to work was collaboratively selected as the initial target for treatment. Kaye’s target cognition was, *“I have lost all self-confidence. I will not be able to cope with going back to work”*. Fortunately, she had a supportive boss and arranged to return to work in a graded capacity, initially starting with just three hours. She was asked to interact with staff and customers. Her prediction was, *“I will not be able to cope. I will not know what to say to customers and will have a breakdown and start crying on the shop floor”*. Her belief in this prediction was 80%.

A week later in her second session she reported the results of the experiment as, *"I helped customers. They were satisfied with my service and seemed happy when they left. I did not have a breakdown or cry at all"*. When asked to reflect on the conclusion of her experiment, she said, *"I am good at my job. I know what I'm doing and can cope"*. Her post-experiment belief rating reduced to 20%. Repetition of the experiment was required for homework, incorporating longer shifts and increased levels of responsibility.

Within the second session, a second behavioural experiment was selected aimed at helping her return home. Previously she reported enjoying living at home, appreciating the privacy and her own space and company. The target cognition for this second experiment was, *"I will not be able to cope being at home on my own"*. The behavioural experiment involved returning home on the weekend with her son. Her prediction was, *"I will feel so anxious at home I will not be able to tolerate it and will feel paralysed with anxiety. I will not be able to function"*. Her belief rating was 90%. As she had previously returned home on numerous occasions for short periods to water the plants and do some simple cleaning, it was considered reasonable for her to test her belief by staying overnight.

The following week, Kaye attended the session looking bright, smiling and wearing make-up. She had returned home that weekend, felt mildly anxious initially, unpacked her belongings, did some cleaning and cooked dinner. She said she enjoyed watching TV and slept well in her own bed. For her conclusion, she said, *"I coped being at home. It was actually good to be in my own home again"*. Her post-experiment belief rating had dropped to zero. Kaye's case provides an example of a patient with a severe depressive episode, who with the use of some behavioural experiments, was able to test her negative cognitions and tackle her avoidance behaviour, which was effective in improving her mood and functioning within a short time frame.

A Cognitive or Behavioural Technique?

There is some debate amongst theorists as to whether behavioural experiments are a cognitive or behavioural technique. From a cognitive perspective, behavioural experiments are considered a cognitive method as they are designed to test the validity of negative thoughts. However, from a behavioural model, behavioural experiments help reduce the depressive avoidance behaviours thereby increasing access to sources of reward while decreasing sources of punishment. Moreover, they often involve exposure to feared or avoided situations, and exposure therapy is at the core of behavioural treatment. Behavioural experiments are perhaps best conceptualized as a true cognitive-behavioural treatment. They can provide rapid results, hence for patients with testable thoughts, behavioural experiments are an extremely useful therapeutic technique.

Effectiveness of CBT

CBT has repeatedly been demonstrated to be an effective treatment across the entire range of anxiety and depressive disorders. It has shown effectiveness for adults, as well as child and adolescent populations and older adults (James et al., 2020). Effect sizes for CBT (in comparison to waitlist controls and treatment as usual) across anxiety and depressive disorders are large, with no significant differences between cognitive and behavioural treatment packages. CBT has demonstrated long-term treatment gains, with follow-up periods up to 19 years (Benjamin et al., 2013).

CBT can be effectively delivered in individual, group and internet-based formats (Olthuis et al., 2016; Young-Southward et al., 2020). Internet-based CBT offers a cost-effective alternative to traditional face-to-face CBT with research finding equivalent outcomes, provided some level of support is available (Olthuis et al., 2016).

CBT is frequently combined with antidepressant medication in clinical practice for both anxiety and depressive disorders (Andrews et al., 2018; Malhi et al., 2021a). Randomised controlled trials comparing CBT alone, medication alone or CBT plus medication are few, with most studies finding CBT plus medication slightly superior to medication alone but offers little advantage over CBT alone at both post-treatment and follow-up (Dardas et al., 2023). CBT helps reduce risk of relapse after antidepressant discontinuation.

Despite the effectiveness of CBT, the majority of patients continue to experience residual symptoms (Brown and Barlow, 1995), hence there is considerable room for improvement in treatment response. The development of ‘third wave’ CBT treatments such as mindfulness-based CBT (MCBT) or Acceptance and Commitment Therapy (ACT) offer promising alternatives, however meta-analytic comparisons between these newer treatments and standard CBT are yet to show significant differences (Hunot et al., 2013).

Further Reading

- Andrews, G. (2003). *The treatment of anxiety disorders: Clinician guides and patient manuals*. Cambridge University Press.
- Andrews, G., Bell, C., Boyce, P., Gale, C., Lampe, L., Marwat, O., ... & Wilkins, G. (2018). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of panic disorder, social anxiety disorder and generalised anxiety disorder. *Australian & New Zealand Journal of Psychiatry*, 52(12), 1109-1172.
- Beck, A. T., Rush, A. J., Shaw, B. F., Emery, G., DeRubeis, R. J., & Hollon, S. D. (2024). *Cognitive therapy of depression*. Guilford Publications.
- Bennett-Levy, J., Butler, G., Fennell, M., Hackman, A., Mueller, M., and Westbrook, D (2004). *Cognitive behaviour therapy: science and practice series*. London: Routledge.
- Lewinsohn, P. M., & Clarke, G. N. (1984). Group treatment of depressed individuals: the 'coping with depression' course. *Advances in Behaviour Research and Therapy*, 6(2), 99-114.
- Malhi, G. S., Bell, E., Bassett, D., Boyce, P., Bryant, R., Hazell, P., ... & Murray, G. (2021). The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian & New Zealand Journal of Psychiatry*, 55(1), 7-117.
- McSweeney, F. K., & Murphy, E. S. (2014). *The Wiley Blackwell Handbook of Operant and Classical Conditioning*.

Chapter 34: Psychotherapy

Anthony Korner

Westmead Psychotherapy Program, Western Sydney Local Health District, and Specialty of Psychiatry, The University of Sydney, Australia.

“I can only find myself in and between me and my fellows in a human conversation”

Robert Hobson, 1985

The Psychotherapeutic Attitude

Holistic care requires doctors to attend to the psychosocial needs of patients as well as to their medical condition. Everyday human functioning involves a complex coordination of skills and social roles that carry each person’s sense of identity and value. Effective clinical intervention always needs to consider the maintenance and support of the patient’s dignity and self-worth. Every time a person meets with a physician there is a communicative exchange that impacts the patient’s well-being and function (Korner, 2020). As medical professionals we strive to make this exchange helpful to the patient. Clinicians rely on patients’ inherent resilience while attempting to effect constructive change, always fostering hope. Consideration also needs to be given to the patient’s level of understanding, social resources and cultural background. Good clinical practice is underpinned by this biopsychosocial approach.

The term psychotherapy refers to treating emotional and mental disorders, problems and suffering through psychological means. It is commonly thought of as “talking treatment”, although it may also encompass psychological elements other than speaking. Communication has an emotional impact that is *felt*, in addition to its factual content (Korner, 2020). A therapeutic interaction aims not only to help the patient towards improved health but also to feel better. A psychological perspective will be maintained if it is remembered that doctors are committed to the value of all human lives, understanding that every human interaction has meaning for its participants. A therapeutic attitude involves being non-judgmental, genuine, showing unconditional positive regard and maintaining a warm, empathic, patient-centred position (Rogers, 1995). While there are many types of therapy, an emotionally connected, confiding relationship, a healing setting, a rationale for the

therapeutic process, the provision of hope and some element of “ritual”, requiring active participation by both patient and therapist, have been identified as factors common to successful intervention (Frank and Frank, 1993).

Psychiatric presentations form a major part of general medical practice (Davies, 2003). For many patients the only health care they will receive for their mental disorder is through a primary care practitioner. Therefore, General Practitioners (hereafter termed GPs) need to have familiarity with psychological approaches to treatment. This chapter will focus on psychotherapy as it might be employed in primary care, with an emphasis on supportive psychotherapy and options for specialist referral.

Psychological Well-Being

Medical doctors are orientated towards the identification of pathology. In contrast, patients generally seek something like “restoration of well-being”. Hence it is important to have a concept of psychological well-being underpinning a recovery focus in psychological intervention. Attention is devoted to interests and relationships. The Harvard Longitudinal Study lends scientific weight to the idea that good relationships keep us happier and healthier (Vaillant, 2012). Freud³ is said to have defined psychological health as the capacity to *love* and *work*. These days we might add *play* to love and work, recognizing the importance of recreation to maintaining a sense of satisfaction in life. We also need *rest*, often disturbed in patients with psychiatric disorders. When well, we expect adults to function in a self-organizing way. The goal of medical intervention is usually to restore this capacity, helping people to “get back on track”. Medical and psychiatric interventions should provide information that allows the patient to make informed choices about their own care.

Normative Development

Humans are born into a world of continual communicative interaction and relationship. Human development is prolonged beyond any other species. Growth and learning occur, initially, in the asymmetric interaction between the immature infant and others (caregivers) with greater experience in life.

³ The statement “to love and to work” as a description of mental health is attributed to Sigmund Freud. There is a similar quote in one of his later works, “Civilisation and its Discontents”: “The communal life of human beings had, therefore, a two-fold foundation: the compulsion to work, which was created by external necessity, and the power of love... ”.

Infants are *communicants* with whom carers interact from birth (Bullock, 1979). They have capacities for turn-taking and affective expression enabling a level of communication and relationship (Malloch and Trevarthen, 2009). Even though the infant's participation is initially instinctive, rather than reflecting conscious understanding, patterns of relating become established that involve differential processing of positive and negative affect. The infant is dependent on the carer for relief of distress which is important in providing the infant with a basic sense of security and trust.

When the infant displays positive affect, through gaze, smiles and vocalizations, he or she will typically be responded to in a more playful manner, setting up exchanges that have been described as “the proto-conversation” (Trevarthen, 2018). When the carer takes the cues of the infant and provides an attuned response, a condition is created where the infant begins experiencing life as having value, generating “*a particular kind of pleasure that neither could gain alone*” (Meares, 2016).

At around 6 or 7 months of age infants start to exhibit separation anxiety. This is thought to relate to the emerging sense of distinctness from others, particularly the primary caregiver. For the infant, this brings with it fears of abandonment. Provided the caregiver remains fairly consistent, this phase will usually be negotiated without disturbance of the developmental trajectory. Mobility and exploration increase and communication becomes more differentiated within the dyad. There is an increase in the specificity of meaning understood within the infant-carer relationship (Halliday, 1975).

With the acquisition of language in the second and third years of life, the child becomes a participant within a broader community. A secure child will go through a phase where play is sometimes carried on alone, often accompanied by chatter with the child seemingly talking to him- or her- self, while using objects (e.g. toys) to represent stories and actions, often narrating or treating the objects as if they were alive and could communicate. This kind of activity is termed symbolic play (Piaget, 2013). This form of externalized self-talk is probably a prelude to being able to think in words without the need for vocalization.

Language acquisition and the growth of other cognitive capacities means that mental life becomes structured into awareness of present, past and future, freeing the individual to think beyond the immediate present (Korner, 2020). The emergence of episodic (autobiographical) memory around the age of three also contributes to the establishment of an inner world of associations and a sense of agency that allows for greater independence.

The child now has an inner zone of private experience, although he or she must also be continually adapting to the external world. The private domain of self emerges in the public space of relationships and physical contingencies. Personality consists of the private self, on the one hand, and public identity on the other.

As the child grows, roles and expectations change. The infant is expected to be a recipient of care. Expectations for the assumption of responsibility and self-organization increase as the child matures. The formation of a distinct identity, the development of skills that allow for gainful employment and establishment of a peer network, become necessary for entry as an autonomous individual into the adult world. As adults we face the further challenges of establishing a home, being able to care for others and contribute to communal life. Consideration of the individual's stage of life and its attendant challenges (Erikson, 1993) is essential to appropriate psychological engagement.

Trauma and Loss

Early childhood adversity such as neglect, physical, sexual or emotional abuse, can lead to disturbance in the development of personality. Various types of insecure early attachment relationship have been shown to predict adult pathology [attachment has four main types: secure; insecure-avoidant; insecure-ambivalent; disorganised (Ainsworth et al., 2015; Bowlby, 1982; Main et al., 1985); see Chapter 13]. Individuals growing in the context of chronically inadequate, insecure or absent relationships are severely disadvantaged. Childhood loss, injury or illness may be important factors in developmental adversity. While many of these people show great resilience and make constructive adaptations to life, some will require psychotherapy, sometimes with complex needs requiring specialist services.

The extent and impact of childhood trauma, abuse, and neglect has been increasingly recognized in recent decades (Felitti et al., 2019). Recognition of the significance of trauma and loss to the individual is an important aspect of establishing a good therapeutic relationship, contributing to the process of healing. Trauma-informed care accompanied by a recovery focus has become an expectation of all mental health care (Herman, 2015).

Formulation using a Biopsychosocial Approach

Diagnosis in relation to mental disorders depends upon adequate assessment of clinical symptoms, mental state and history relating to the onset of the condition. This provides a generic assessment, often with limited information about the individual. A more detailed history is required to individualize the assessment,

considering both presenting condition and preceding developmental context. This encourages recognition of difficulties faced and adaptations made, over the lifespan.

A comprehensive approach to formulation necessarily involves biological, psychological and social dimensions (see Chapter 7). Mental disorders typically reflect a loss of capacity to coordinate responses across these domains. In a given case one or more of these areas of assessment may be more critical than another. Some points to consider in making a formulation are listed below:

- What factors account for the patient presenting *now*? Often this relates to changed circumstances, illness, relational problems, financial stresses, etc.
- Early life experience and quality of attachment relationships.
- Childhood experience including ability to relate to other children, abilities at school and other areas of competence or deficit.
- Markers of the ability to create an independent life, including occupational functioning, educational attainment and the capacity to develop and sustain intimate relationships.
- The quality of adult relationships.

Having considered these areas, many clinicians find it useful to organize factors identified into *precipitating*, *predisposing*, *protective* and *perpetuating* categories. This helps guide clinical decision-making. Formulation using a biopsychosocial model serves two primary purposes:

1. It allows sharing of the clinician's overall assessment, engaging the patient and helping them understand the rationale for treatment.
2. It informs an individualized management plan, providing interventions specific to the person and circumstances.

In the context of psychotherapeutic intervention, adequate formulation also helps the patient feel understood. People are constantly formulating actions and responses in relation to the demands of everyday life. In the psychotherapeutic context the clinician additionally seeks to incorporate developmental understanding and the role of unconscious factors in the clinical presentation. One of the goals is to help develop a shared narrative, which often has the effect of enhancing therapeutic connection.

Case illustrations are used to show the application of formulation to everyday practice:

Jenny is a 31-year-old mother of three who has had a decline in function over the last three years with frequent suicide attempts, self-harm, unstable mood, persecutory ideation and distressing internal imagery. There have been repeated admissions to hospital. This decline was associated with loss of a job at which she had performed well. She always had difficulty with the parental role, leaving these responsibilities to her husband, who was on a full-time carer's benefit since the birth of their first child. While she felt guilty about not being a better mother, she believed her husband was a good carer. Jenny reported learning difficulties in school. Her mother thought a severe febrile illness involving seizures in the first year of life had affected her. She had been told that she had been behind her siblings in pre-school development, with delayed language acquisition. She was second youngest of five. Family relationships had not been overtly traumatic although she always felt her mother was critical and had favoured her siblings. A diagnosis of Borderline Personality Disorder was made with an additional note of intellectual function in the low average range.

Jenny presents with a change in function that relates to a loss of employment (social factor), significantly affecting her sense of identity (psychological factor). There are indications that her baseline functional level had been affected by an illness characterized by seizures associated with delayed development and relative intellectual impairment (biological factor). This deficit may have been compounded by a relatively invalidating family environment (psychological factor) and difficulty establishing a sense of competence as she always felt she was deficient relative to her peers. The birth of her children was experienced as anxiety-provoking with an adaptation that involved giving up parental responsibility (avoidant adaptation) while gaining a sense of self-worth by contributing to the family through work (psychosocial adaptation).

Brief on Jenny's case comment: although a range of managements, including pharmacological and psychological intervention were provided, the key intervention, in terms of leading to sustained recovery, was a social intervention centring on supporting Jenny in a return to work.

Joe, a 35-year-old man, lives with his wife and four children. He came to hospital after the death of his cat, sobbing uncontrollably, with a wish to commit suicide. It emerged that he had also lost a friend and had suffered an assault in the preceding six months. He felt disorientated and ashamed of his behaviour. When his wife and children visited, he did not want to see them.

Joe had never been close to his parents. His father was a violent alcoholic, while his mother was passive and emotionally cold. They separated when Joe was 9 years old. Both remarried and had other children.

When he first met his wife, he was surprised at her level of interest and love. However, when they had children, she became more interested in them. The person he felt closest to was a male friend, who had died of cancer recently. Since then, he had continued to go to work, come home and talk to his cat, watch television and go to sleep. Six months ago, he was assaulted while attending to an injured man in the course of his work. He was upset by the lack of support from management.

Shared (spoken) formulation.

"I don't know if this is right, but it seems to me that it is no wonder that you were inconsolable when your cat died, as she was the only thing that you could safely talk to. Because your dad was unsafe when you were a kid, and your mum was distant, you never felt that anyone really cared for you, and you learnt that when things go bad, like your parents separating, you just have to look after yourself. At first your wife was like a breath of fresh air, but when the children came, you seem to have lost the feeling of closeness with her. The only person that seemed to matter was your good mate, which probably made his death doubly painful. You lost him, and there was no one to talk to about your sadness. I guess you dealt with this by pulling away from everybody, spending a bit of time with your cat, and just following a routine of work and sleep. The situation got worse after you were assaulted, particularly as the lack of concern from management seems to have dovetailed with that feeling that nobody cares, which you have lived with all your life."

Brief comment on Joe's case: the shared understanding conveyed in this formulation was found helpful by the patient who engaged subsequently in psychotherapy with a positive outcome. He declined offers of alternative treatment with medication. (Case 2 from (Korner et al., 2010)).

Psychotherapy in Primary Care

Primary care is often the first point of contact for people suffering mental distress or disorder. GPs need to be able to recognize major mental disorders and prescribe appropriate treatment. However, they also need to have flexibility in responding to a range of distress and adjustment disorders for which the most appropriate form of intervention is psychological, often involving brief psychotherapeutic intervention. Judicious intervention may minimize the development of more intractable disorders. The type of intervention varies depending on the skills of the clinician. All doctors need to be able to provide adequate psychoeducation and supportive psychotherapy. Attending to the psychological well-being of patients through appropriate supportive psychotherapy is an element of all medical and psychiatric care, even when there is a focus on other interventions such as pharmacological measures or surgical procedures. Elements include active listening, adequate explanation of condition and treatment, allowing time to answer questions, considering the patient's emotional response, reaching agreement about a treatment plan and facilitating any necessary additional referrals or external support. For some patients scheduling additional sessions to allow sufficient time for talking and working through emotional problems is a necessary part of treatment.

Supportive Psychotherapy

The aim of supportive psychotherapy is to optimize the patient's psychological functioning, making use of the available psychosocial network. This involves using the therapeutic relationship to provide an emotionally safe environment for the patient where problems can be identified and worked through. The focus is primarily on the present, with a practical "here and now" orientation. As with all clinical work, an adequate assessment and formulation is required. Problems need to be prioritized and the person's social, financial and occupational resources considered. If the clinician is a GP, consideration needs to be given as to whether referral is required or whether it is possible to provide adequate support within the practice. A treatment plan should be negotiated. In many cases this will involve organizing appointments of sufficient length to allow adequate articulation and emotional processing of the problem.

Some elements of supportive psychotherapy are listed below

Create an Appropriate Framework

Primary care includes a lot of crisis support requiring flexibility on the part of the practitioner. When a psychotherapeutic intervention is required, scheduling sessions with a fixed time at regular intervals is helpful. This should be mutually

negotiated and then every effort should be made to keep to the agreed times. It should be made clear that the sessions are for talking through problems. Other interventions may also be agreed upon (see below). It needs to be made clear that the work is conducted within professional boundaries.

Active Listening

It is often difficult to express distress or disturbances of mental state clearly. The doctor needs to listen for emotional inflection and expression as well as word content. Patients experience benefit from simply being heard. The doctor needs to share what has been understood, attempting to see things from the patient's perspective and check whether there is agreement. Maintaining attention, summarising, seeking clarification and reflecting back what has been said are all elements of effective therapeutic communication.

The Therapeutic Relationship

A compassionate, trusting, responsive relationship is the basis of much healing practice.

Ventilation and Emotional Expression

If patients are in a state of emotional distress, then it is essential to allow sufficient time for expression and processing. Catharsis, the expression of strong emotion that has often been suppressed in other settings, may provide real relief for patients struggling with feelings of vulnerability or other strong emotions. Therapists need to be able to tolerate and sometimes encourage emotional expression.

Reassurance, Encouragement and Explanation

These kinds of response are characteristic of helping relationships and often contain anxiety, allowing a more rational appraisal of the situation.

Coupling and Amplification

When patients are in a fragile emotional state, it is often helpful to make simple responses, like “uh-huh” or repeating a phrase of the patient's that seems significant. This “coupling” with what the patient expresses often provides a sense of connection. Where the therapist senses something of significance for the patient, this can be amplified by showing interest in what has been said, encouraging the person to expand, often enlivening the communication (Meares, 2012).

Cognitive and Behavioural Techniques

Depending on the skills of the practitioner, these may be applied flexibly in a supportive psychotherapy. *Problem-solving* helps clarify the nature of the problem and generate alternative strategies to address it. *Re-framing* involves providing an alternative appraisal, often one with less prejudicial connotations that helps normalize a particular issue. *Mindfulness* exercises may promote greater self-awareness, helping to decrease reactivity if practised regularly. Other forms of relaxation exercise may be beneficial, increasing the person's sense of agency in being able to manage anxiety (e.g., progressive muscular relaxation; slow diaphragmatic breathing).

Lifestyle

Social and lifestyle interventions should always be considered. These may include encouraging scheduling of activities that are positive for the individual, consideration and attention to sleep hygiene, regular exercise, attention to a healthy diet and consideration of work stresses and the need for leave. For some patients, substance and alcohol abuse will need to be addressed. Cessation of smoking and modification of caffeine intake may be helpful.

Family Involvement

Many patients feel isolated and have difficulties communicating with family members, sometimes bringing problems to a professional instead. Where a matter has been kept secret, it may have had a significant impact on family relationships. Sometimes bringing something into the open with the family can be a great relief, although it is a matter of judgement that needs to be considered carefully with the patient. Family members may also be enlisted in providing practical support, helping engage the patient in a greater range of activities. Sometimes intervention in relation to a specific dysfunctional relationship may be required (e.g., referral for couples counselling).

Case Example

A 48-year-old man, Bob, presents to his GP with what he describes as “extreme stress”. His workload has been increased in the context of other staff being laid off. He has noticed increased irritability and is concerned that this is affecting his work and family. He has been getting home late, not seeing his children regularly, and sleeping poorly. This has worsened over two weeks, since the changes at work. He is an ambitious and somewhat driven man. He doesn’t have low mood but is anxious and shaky at assessment. He describes feeling “at the end of his tether”, although this refers to his emotional state rather than to suicidal ideation. He is otherwise in good health.

The doctor agrees with the patient’s characterisation of severe stress, pointing out he has also become extremely anxious. After assessing his physical state and reassuring Bob that there doesn’t seem to be any physical problem, the doctor suggests taking a day or two off work, scheduling a further appointment to address the anxiety symptoms he is getting. With initial reluctance Bob agrees to take a day off and come back the following day. When reviewed Bob expresses a degree of relief and was pleased to have seen his children that morning. The doctor guides Bob in a progressive muscular relaxation exercise. After this Bob becomes quite emotional, saying he’d never realized how tense he was until he’d experienced this relaxed state. He talked at some length about the sense of being burdened by multiple responsibilities and gave further background to his symptoms. Bob felt further relief from this expression of emotion, feeling he was well enough to go back to work the next day. The doctor was somewhat concerned he may be pushing himself too soon but provided a work certificate for one day, making a further appointment for the following week. There was a discussion of work-life balance and the need to schedule positive activities with the family. The following week Bob reported improved function at work, feeling that having been able to open up about his problems had made a big difference.

Liaison With Other Services

Involving other specialized services may be necessary (e.g., Psychologist, Anxiety Disorder Program, Community Mental Health Team, Drug & Alcohol services; Social Welfare Services). For many patients the GP will be the main case manager for coordination of services. It has become standard practice for GPs to develop Mental Health Care Plans in conjunction with psychologists and other Mental Health Practitioners, enabling access to psychotherapeutic interventions for patients who may otherwise be unable to afford them.

Psychoeducation

Mental disorders and disturbances in emotional or mental state are all embodied processes. As such it is relevant to educate patients regarding underlying physiological processes, using simple language supplemented by supportive written material where possible (there are many Mental Health-related websites with readily accessible information – for example Beyond Blue; Black Dog Institute; Project Air, etc).

It is helpful for patients to recognize that symptoms aren't simply imaginary but have a basis in bodily processes. While it is beyond the scope of this section to address psychoeducation in relation to specific mental disorders, the body's response to stress and some of the physiology underpinning anxiety and emotion are briefly described, as well as some physiological processes that support mental well-being.

Stress Response (see Chapter 18: Anxiety Disorders)

In humans, it is the perception of threat that elicits the stress response, sometimes referred to as the “fear cascade”. *Threat* includes perceived danger to personal integrity, self, or relationships. The source of threat may not always be obvious even to the person experiencing it. Emotional responses often bypass conscious cortical processing systems, involving rapid non-conscious pathways in central parts of the brain (e.g., amygdala). Other parts of the nervous and endocrine systems (involving production of stress hormones, cortisol and adrenaline) get activated with recruitment of the *fight-flight* response, or the more drastic *freeze* responses, perhaps better termed *tonic immobility* and *collapsed immobility* (Kozłowska et al., 2015b).

Normal functioning, supporting social engagement and higher neural functioning, is regulated by the *parasympathetic* part of the autonomic nervous system, operating outside conscious control. In response to threat, the system mobilizes and orients to appraise the threat. If the threat continues, a full *fight-flight* response will ensue. This involves considerable physiological arousal sometimes associated with bodily symptoms of panic. If the level of threat is perceived as life-threatening, a different physiological response may be elicited involving a relative shut-down of many bodily systems. In other species this is associated with a “death-feigning” response, protective in some circumstances. In humans *collapsed immobility* may be associated with numbing and marked reduction in cognitive and motor function or even loss of consciousness. Emotional accompaniments can include a sense of helplessness or emotional distress (Kozłowska et al., 2015b; Porges, 2011).

Models of Mental Life and the Brain-Mind System

Mental life and personal experience are supported by a complex coordination of interacting neural networks that give rise to consciousness and the sense of personal existence. Some areas seem particularly important in supporting the experience of emotion, relational interaction, and sense of self. Three areas of the brain are emerging as important in this regard (Knox, 2010): a) the *mirror neurone system*, closely related to the cortical motor system, is activated while observing movement in others and seems to be important in the sense of relationship, participation and connection with others; b) the *Central Midline Structures* (CMS), including the limbic system, a relatively ancient part of the central nervous system in evolutionary terms, are critical to the experience of emotion, understood as a whole person response to environmental interaction; and, c) the *default network*, a widely distributed network in the brain, characteristically active when the person is **not** involved in particular tasks (i.e. states of relative rest; internal focus). Indications are that this area is important in the experience of thoughts, feelings, processing of memory and other forms of “self-related processing”.

Bodily Bases of Well-Being

Adequate rest, recreation, sleep and nutrition are all important to mental well-being. In humans, as social beings, the capacity to express ourselves and share with others, coordinating ourselves in social and work networks is supported by the parasympathetic nervous system (through the vagal nerve), helping maintain the body in a relaxed, socially engaged mode (Porges, 2011). Social connection and relationships with others are important to mental balance throughout life. Processes such as meditation and relaxation techniques support well-being by slowing breathing, leading to stronger vagal regulation of the heart and cardiovascular system, supporting higher mental functioning. Creative expression may be an important aspect of well-being for many, giving representation to the complexity of human lives.

Appropriate Referral

GPs are involved in referral to acute psychiatric services in relation to presentations such as psychosis, the suicidal patient, severe mood disturbances and specific conditions covered elsewhere. Doctors generally need to familiarize themselves with available specialist services. The focus here is on referral for psychotherapy.

A crucial factor is that the doctor recognizes professional limitations. Highly complex presentations require a psychiatric review, often involving multidisciplinary work. Specific services will be indicated for particular counselling

needs: drug and alcohol services, bereavement counsellors, sexual assault services, structured psychotherapeutic services or combined psychotherapeutic, psychiatric and behavioural approaches (e.g., Dialectical Behaviour Therapy, Anxiety Disorder clinics, youth-oriented services, such as *Headspace*). With milder presentations the GP may be optimally placed to provide advice and support.

Attempts to treat complex patients alone may lead GPs to become overinvolved which may result in boundary violations such as developing inappropriate relationships. However, the GP often continues to have an important role in the provision of community care with challenging patients, in conjunction with other mental health services.

Some patients present with lifelong difficulties, often falling into diagnostic categories of treatment resistant depression, personality disorder, addictive disorder, post-traumatic disorder, somatisation disorder, dissociative disorder or others. Where trauma is shown to have been significant in development, the terms “complex post-traumatic stress disorder” or “developmental trauma disorder” have been used. (Herman, 2015; Van der Kolk, 2014).

For those who are dysfunctional but motivated to seek intensive help, more specialized intervention can be sought. There is a confusing array of available psychotherapies. For simplicity these are considered under the categories of, 1) Psychodynamic therapies; 2) Cognitive-Behavioural Therapy (see Chapter 33); and 3) Structured Therapies. For purposes of referral, the doctor should understand that psychodynamic therapies are in the psychoanalytic tradition, acknowledging that unconscious factors influence well-being. They rely upon sustained engagement in a therapeutic relationship oriented to empathic understanding of the patient’s inner world, emotional life and patterns of coping. Aims include working through trauma and the development of greater autonomy and flexibility. These approaches require extensive training and supervision. The most intensive therapies (i.e., psychoanalysis four or five times a week) are emotionally challenging and require a reasonably high level of function and motivation on the part of the patient, with a capacity for reflective and symbolic thought. Many psychodynamic practitioners work in frameworks of once or twice a week, face-to-face sessions with most patients. This is a more realistic and affordable option for many patients.

Additionally, doctors can augment the supportive care they provide through online resources and involvement with self-help groups. Even when patients are referred on, the GP maintains a supportive role when episodes of care with other services and therapists have been completed.

Psychodynamic Therapies

Psychodynamic therapies are oriented to helping people make sense of experience. This includes exploring unconscious and traumatic influences, and considering experience within the therapeutic relationship. People with long-established emotional difficulties, recurrent self-defeating patterns of behaviour and complex experiences of trauma and loss may benefit. Psychodynamic therapies typically involve a sustained engagement over at least 12 months. A sense of demoralization or lack of fulfilment may be common factors for patients seeking therapy, transcending diagnostic categories (Frank and Frank, 1993).

All people experience life through the lens of past experience, albeit usually without realizing how past experience is shaping present interaction. Individual modes of coping are developed in early life through experiences with caregivers and significant others. These adaptations are established while the infant is in a pre-verbal state. Such habitual patterns are not accessible to conscious awareness but rather happen automatically (unconsciously), being manifest in behaviour, particularly in relational life. The emerging experience of self is affected by relational interactions. Not only is there “no such thing as an infant” (Winnicott, 1986) but there is no sense of meaning for an individual in isolation from his or her environment.

Psychodynamic therapies all employ the technique of *free association* which encourages patients to express whatever is felt, following the drift of internal images. This is a key to understanding patterns of interest and growth, traumatic disruptions and avoidances, inhibitions and self-protective measures that have become established. While there are many competing theories, there has been a trend towards recognizing the centrality of relationship-seeking in development, with increasing emphasis on applying understanding from normal developmental interactions to the situation of therapy. Another change of emphasis has been from “neurosis”, where individual anxiety was often seen as having an irrational basis in infantile relationships, to the increasing recognition of trauma, often in the form of an early relational environment involving abuse, neglect, or mismatches between infant and carer that have contributed to failures in development (Meares, 2004b).

Growth requires an empathic and responsive relationship that recognizes the patient’s distinct individuality and value while maintaining clear boundaries. Apart from basic needs for safety and security we also need new experience in order to develop (Korner, 2020). In a successful therapy there will be a broadening of adaptive capacities and improved tolerance of affect with a greater sense of the person being able to take responsibility for his or her life. Some brief orienting comments are made below regarding three central aspects of psychodynamic

therapies: framework and boundaries; transference and countertransference; defence and adaptation.

Freud is credited with developing the technique of free association, although as Jones points out: "There can be no exact date for the discovery of the 'free association' method... it evolved very gradually between 1892 and 1895, becoming steadily refined and purified from the adjuvants - hypnosis, suggestion, pressing, and questioning - that accompanied it at its inception" (Jones, 2019).

Therapeutic Framework and Boundaries

The intention of the psychodynamic therapist is to provide an emotionally safe space, a "secure base" from which the patient may be able to safely explore his or her experience. The regularity of meetings in the same place at specific times is an important part of establishing a sense of reliability. Patients also need to be clear that information they disclose will be held confidentially. Consistency of sessions in time, place and person, a confiding relationship and understanding of the limits of contact and alternative options (e.g., for crisis care) constitute the therapeutic framework, often referred to as "the frame".

Ethically, breaches of confidentiality should only relate to questions of safety of the patient or others (e.g., when patient is suicidal; when patient is making credible threats against others; when information is disclosed that reveals a child is at risk). The frame also needs to include some explanation of therapeutic process, involving encouragement of the patient to speak openly about his or her inner life and experience, with a view to improving self-awareness, understanding and facilitating the realization of personal goals.

Patients will sometimes behave in ways that put pressure on the therapist to deviate from the framework (e.g., seeking for a session to be extended or seeking an alternate time). While the therapist needs to have some flexibility, major changes to the framework should be kept to a minimum. The task is to understand, with the patient, the nature of any deviation to the frame, and to bring the patient back to the normal routine of sessions.

Transference and Countertransference

All people develop expectations based on past experience. This is a normative process with predictive value that begins from birth. The earliest patterns of expectation emerge in relation to significant others (carers, siblings) during a period where memory is recorded in procedural and perceptual systems outside conscious awareness. This is to say that interactional patterns employed in

communication and social behaviour are influenced by early experience in a relatively automatic way. In clinical situations the patient unconsciously displaces behavioural patterns and emotional reactions from past relationships onto the therapist, the phenomenon of *transference* (Freud, 1997). While unconscious displacement of past experience occurs, it is also important to recognise that the patient's experience is of feelings in the present, relating to the immediate interaction. Transference involves the feeling experienced in the therapeutic relationship by the patient towards the therapist. Working with transference implies not only understanding the patient's condition but also reflecting upon experience in the therapeutic relationship.

Clinical circumstances where patients present with hostility, gift-giving, seductive behaviour, intense demands, or incoherence may all reflect transference influences. See the box below for an illustration of this kind of presentation.

A young man presents to his psychiatrist with unbridled hostility, verbal abuse, and threats. This follows a previous encounter where he had disclosed an extensive history of abuse from a sadistic and controlling father. The psychiatrist sensed that this was a man for whom any expression of vulnerability had met with violent responses during his development. When a link was made, recognizing his courage in talking about past abuse, the man calmed considerably.

Later experience modifies perceptions and expectations and is more accessible to consciousness. In therapy patterns of behaviour and interactional response become evident in the therapeutic conversation (see the box below for an example).

Establishing an emotionally responsive therapeutic relationship creates a situation where the thoughts and feelings being experienced in the therapeutic relationship can be reflected upon, often with recognition that there are parallels with other, earlier relationships. Understanding transference has potential to put into words matters that had been relatively unconscious to the individual. The young woman mentioned above eventually discovered that the therapist wasn't going to react with hostility and became more able to share her inner experience.

A young woman with an eating disorder finds it difficult to speak in therapy. It emerges that whenever she made attempts to pursue her interests her father would react harshly, brooking no opposition. Her mother was employed as the father's agent in vetoing the daughter's initiatives. Silence at home often reflected anger on the part of her father. The therapist is aware that prolonged silence would be experienced as hostile by this patient. Accordingly, there is an effort to provide a responsive, turn-taking environment, different from the atmosphere of her home environment.

In her original environment, this patient could not establish a sense of agency. It was expected this would take time to emerge in therapy because of her anxiety that whatever she puts forward would be squashed by the “other”.

This isn't always a straightforward process. Often transference experiences are associated with strong emotion, reflecting earlier traumatic experience. When therapy continues over relatively long periods, an increased intensity of emotionally-charged interactions may develop with some patients. However, if safety in the therapeutic relationship has been established, there will be gradual integration of these powerful affects.

Just as the patient has responses with a background in early experience, so do therapists. At times therapists will also struggle with powerful emotional responses. Some of these will be directly related to the patient's behaviour. However, there is often a component that relates to the therapist's past. These responses, occurring within the therapist, are broadly referred to as the **countertransference**. While distortions related to the therapist's background may sometimes interfere with therapy, countertransference can also be a source of new information. An evoked response in the therapist, quite outside the range of his own previous experience, influences the therapeutic interaction in the example provided in the box below.

A young woman, in therapy for some months, sits in stony-eyed silence, refusing to respond to the therapist's comments. The therapist is uncomfortable, feeling a cold sense of threat in the relationship. Giving voice to this discomfort, by saying that “something doesn't feel quite right”, leads to the patient disclosing a detailed story involving an imagined killing and suicide. The sense of lethality in the silence informed the therapist's response.

The full range of emotions may be evoked in patient and / or therapist, at times with significant intensity. This may encompass hostility, anger, love, hatred, envy, sexual feelings, etc. The therapist needs to contain strong evoked emotion and help the patient to contain anxiety and distress, so that it becomes possible to reflect on interactions. The therapist strives to keep emotional arousal within tolerable limits, sometimes referred to as working within the “window of tolerance”. The therapeutic relationship is also an emotional journey where the sense of connection is often rewarding for both patient and therapist. A positive sense of connection is probably necessary to the integration of trauma.

Supervision enables the therapeutic interaction to be seen with greater clarity. Learning about one’s own responses is also facilitated by having a personal therapy. This has been a traditional part of psychodynamic training. A modern understanding of transference and countertransference is that *both* patient and therapist contribute to this dimension of the therapeutic relationship.

Defence and Adaptation

Given that humans always live in communities and cultures, learning to find one’s way as a social participant in life is a complex task. The sense of self is dependent upon feeling on the one hand and language, relationship and culture on the other. Strong feelings can be difficult to contain. Traumatic experiences, like an assault or an unexpected shock, disrupt our sense of self and actually narrow our consciousness. This kind of experience involves *dissociation*, impairing the capacity to function in a socially responsive way, often associated with a sense of fragmentation, hypervigilance, numbing or depersonalisation.

In everyday life there are many things that we would prefer “not to know about”. Uncomfortable feelings are avoided. We change our mindset to think about less unpleasant things or alternatively we might become argumentative, criticizing whatever may have caused offense. Given the opportunity for reflection, people may come to see that they are behaving “defensively” and that this affects social interaction. One person may react to conflict by withdrawing and ruminating on feelings of guilt about an interaction, an *internalizing* strategy. Another person may respond angrily and blame others for his discomfort, an *externalizing* (or *projective*) strategy. Sometimes the experience of reality may be so distressing as to provoke reactions involving *disavowal* or *denial*.

Identification is a more complex form of adaptation in which a person emulates the characteristics of another as a whole, including behaviour, style and demeanour. It may be conscious or unconscious. When it occurs early in life it is invariably unconscious. Some people may perceive complex states as emanating

from an external source (*projection*) and, simultaneously and unconsciously, *identify* these complex personal characteristics, felt to be intolerable or alien to self, in a particular other person. This complex phenomenon is referred to as *projective identification* (Klein, 1946).

When this occurs in therapy there is likely to be a sense of intensity, perhaps a sense for the therapist that the patient is “getting under my skin”, which can be confusing and disturbing. The patient is likely to attempt to control or coerce the other (therapist) in the case of *projective identification* because there is, at some level, awareness that the other represents an aspect of self that should be under control. Sometimes defensively projected feelings may be induced in the therapist. For example, the psychotherapist begins to feel the sadness or rage that the patient denies or from which they have dissociated. If understood, this then provides a way of it being communicated and returned to the patient. This would be a case of the therapist processing an experience that had arisen in the countertransference.

Similar unconscious processes may be at play when a patient acts impulsively or dangerously instead of talking about problems, the process of *acting out*, which may sometimes be induced in psychotherapy. For instance, a patient who feels abandoned during a break in therapy may self-harm. The therapist tries to maintain a reflective position, understanding the meaning of the behaviour. In order to manage these difficult situations, the therapist needs to find a reflective space in supervision or peer review. This assists in processing and containment, fostering development of the therapeutic response.

Many patients, notably those in the group of Cluster B (extroverted) Personality Disorders (such as Borderline Personality Disorder) have established an organization of experience into “me” and “not-me”, or “good” and “bad” from an early, pre-verbal age. This leads to a failure to see the world and the people in it as whole entities with good and bad qualities. This is a form of dissociation known as *splitting* and is typically an adaptation to disorganized attachment. A common manifestation of this division into “good” and “bad” in clinical practice is that, where there is a team of people involved in the care of such a patient, there will be a polarization of attitudes, sometimes leading to conflict over the treatment approach.

Patterns of response develop as part of an individual’s repertoire in relational interaction in the same kind of way as has been described for transference. Some kinds of adaptation to the environment are operative from the earliest phases of life (e.g. dissociation). Others depend upon later development and verbal capacity (e.g. intellectualization). The reflective space created in therapy may allow the person to “see” the ways in which they interact and identify what is helpful, or self-defeating. Often the best way of coping early in life may no longer be helpful as the

person matures. However, it should be remembered that we all need to cope in some way and that all defences serve a protective function in some circumstances.

Psychodynamic Therapy: Summary

The agent of change in psychotherapy is generally considered to be an empathic therapeutic relationship. This involves the therapist's capacity to imagine the other's point of view, sense their strengths, engage with shared understanding and engender new possibilities.

Some psychodynamic approaches have been developed for working with patients once considered “unanalyzable”, such as those with Borderline Personality Disorder. One such form of therapy is the Conversational Model (Hobson, 1985; Meares, 2005), for which there is a manual (Meares, 2012). Transference-Focused Psychotherapy (Clarkin et al., 2007) is another evidence-based psychodynamic approach for borderline personality disorder.

The processes of psychodynamic therapy include exploring emotions, examining avoidances, identifying recurring patterns, discussing past experience, focusing on relationships, examining the patient-therapist relationship and valuing fantasy life (Shedler, 2010). With respect to the valuing of ‘fantasy’ life, engaging the person's imagination and dreams provides great potential for assisting the person to understand his or her needs and desires, facilitating a more generative life that fits with the individual as opposed to simply meeting external expectations. For people willing to commit to a process of collaborative self-examination, psychodynamic therapy can be a life-changing experience.

Group Therapies and 12-Step Programs/Structured Therapies

Doctors need to familiarize themselves with available resources. A detailed account of the vast array of psychotherapeutic modalities is beyond the scope of this chapter. However, available resources typically include a range of counselling and self-help services (e.g. drug and alcohol groups, day programs for mental health, sexual assault services, eating disorder programs). Availability is variable across public and private health services in Australia. So-called “12 step” programs such as Alcoholics Anonymous, Narcotics Anonymous, Gamblers Anonymous and GROW (for people with mental disorders) are widely available. Mindfulness training has grown in popularity (e.g. Mindfulness Based Stress Reduction). Some practices, such as meditation or yoga may provide psychological benefit without being explicitly “psychotherapeutic”.

A brief description is given of two structured, manualized therapies which have been supported as evidence-based treatments for people with personality disorder. The best known of these is *Dialectical Behaviour Therapy* (DBT), developed over the last few decades as a treatment for borderline personality disorder, particularly for those with affect instability who also self-harm (Linehan, 2014). This has a structured approach with individual therapy and skills-based group therapy, as well as provision of out of hours support for patients. When first introduced it was described as a variant of cognitive behaviour therapy (Linehan et al., 1994). It is based on skills that centre on emotion regulation, differentiating “rational mind” from “emotion mind”. The underpinning philosophy has its roots in Buddhist teaching, emphasizing mindfulness: becoming aware of mental and emotional life while striving to minimize reactivity. The program is one year and involves contracts around attendance and committing to cessation of self-harm. DBT has demonstrated benefit in patients with borderline personality disorder and is widely, though not uniformly, available in both private and public sectors in Australia.

Mentalization-Based Therapy (MBT) was developed in the United Kingdom and has stronger links to psychodynamic therapy (Bateman and Fonagy, 2016). It also involves a combined group and individual approach. There is a focus on staying in empathic relation to the patient while working on promotion of “mentalizing” (reflective thought). Three types of mental state that often interfere with mentalizing are identified: 1) “psychic equivalence” where the patient has certainty of the truth of their own position and an incapacity to tolerate other perspectives; 2) “pretend mode”, referring to states where people are operating on the basis of fulfilling other’s expectations without a sense of genuineness; and 3) “teleological mode” which is action driven, with the person focused on results (‘action not words’). MBT cautions that hyperactivation of the attachment system is a real danger in therapy and can sometimes create problems that prove difficult to resolve. In part for this reason, MBT cautions against prolonged therapies, arguing that 12 – 18 months would usually be an appropriate episode of care. MBT has limited availability in Australia although is likely to become more widely practised.

Stepped Care

It is difficult to provide adequate resources to cater for everyone that might benefit from psychodynamic and structured therapies. Despite this there is evidence that such interventions can be cost-effective (Hall et al., 1999). State projects such as SPECTRUM in Victoria, and Project Air in NSW aim to provide improved access to therapy for those relying on public mental health services. Consumer groups as well as health professionals have been involved in designing these programs which have an online component as well as an on the ground presence. They are organized through a stepped process where initial access to services is facilitated. Patients are

able to access brief psychological intervention quickly. If required, further intervention is organized (medium-term psychotherapy or group therapy). Longer interventions, such as DBT, MBT or longer psychodynamic therapies are reserved for those who have continuing difficulties in recovery. While coordination of such services remains aspirational in many areas, the GP who has good awareness of available resources may have an important role in coordinating such a stepped care approach, selecting from resources available in public and private sectors.

Further Reading

- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14(4), 245-258.
- Shedler, J. (2010). The efficacy of psychodynamic psychotherapy. *American Psychologist*, 65(2), 98.

Chapter 35: Basic Principles of Psychopharmacology

Philip Boyce

Specialty of Psychiatry, Westmead Clinical School, University of Sydney, Westmead Institute of Medical Research, Westmead, Australia.

and

Anthony Harris

Westmead Hospital, Wentworthville, Australia, Westmead Institute for Medical Research, Westmead, Australia, and University of Sydney, Camperdown, Australia.

The introduction of lithium (1948), antipsychotics (chlorpromazine - 1952), antidepressants (imipramine - 1957), and benzodiazepines (chlordiazepoxide - 1960) heralded a dramatic change in the fate of people with severe mental illness. Prior to the discovery of these treatments, the mainstay of management was in essence removal from society and containment in asylums - with little reprieve from the severe symptoms themselves, although, with time some may have spontaneously remitted from their illness. Only a few sedative medications were in use, such as paraldehyde, barbiturates and morphine to provide some relief. However, they had no effect upon the course of the illness. The only biological treatments available were electroconvulsive therapy and psychosurgery, both widely used and administered beyond their present-day indications because of the paucity of alternatives. The widespread uptake of the new psychopharmacological treatments led to a dramatic fall in the population of people housed in asylums and contributed to the emergence of de-institutionalisation. Today, two of the top 10 prescribed drugs by volume in Australia are antidepressants (Editor, 2023).

Although there is a neuroscience-based nomenclature available for defining the class of action of psychotropic medications (<https://nbn2r.com/>) this is rarely used in practice. We will describe the medications by their principal action, although their use in practice goes beyond that. Psychotropic medications have specific actions, for example, the antipsychotics block post-synaptic dopamine receptors or selective serotonin receptor inhibitors increase serotonin availability by limiting its reuptake into the presynaptic cell. These actions suggest

neurotransmitter hypotheses that provide simplistic and incomplete explanations for how medications work. Psychotropic medications all influence many different neurotransmitters (they are ‘dirty’ drugs) but also other pathways such as immunological systems. Their actions are complex and are not fully understood.

A Reminder of the Importance of Pharmacokinetics

All medications achieve a clinical effect by balancing several factors. Firstly, the medication must have an affinity for the pharmacological site of action that is deemed to be therapeutic. In other words, does it act at the receptors which are important for the action on the disease? An example of this is the degree of binding of antipsychotic medications to the dopamine D₂ receptor. Secondly, the drug needs to be able to reach a suitable concentration at the site of action within the brain – its bioavailability needs to be such that it will engage target receptors. An example of this critical first step is the low bioavailability of the antipsychotic asenapine if administered orally because of its high first-pass metabolism. In this case not enough of the medication reaches the brain. Then the half-life of the medication, as determined by its metabolism and excretion, needs to allow the medication to reach a concentration that can sustain an action. Some medications such as quetiapine or venlafaxine are usually administered in extended-release compounds because of the rapid metabolism of the compound. And finally, individual factors such as age, genetics and preexisting renal or hepatic impairment need to be taken account of when assessing the risks as against benefits of the medication, such as the need to be very careful in the monitoring of lithium in the presence of renal impairment (see Figure 35.1)

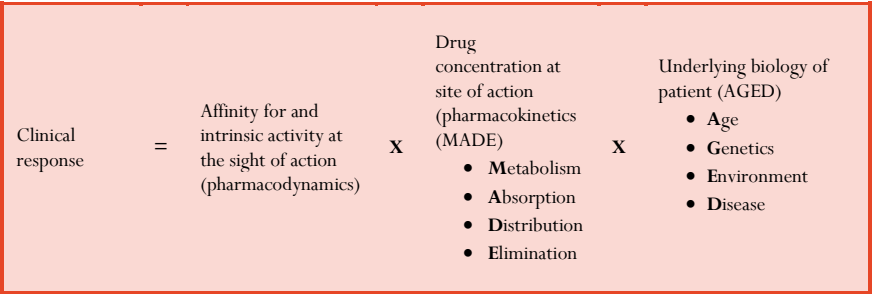


Figure 35.1. Factors that contribute to the achievement of a clinical response. Adapted from Preskorn (2012).

Antipsychotics

Chlorpromazine, the first of the antipsychotics, was first synthesised in 1950 and was immediately noted to cause detachment and calm in patients in

surgical and psychiatric settings. Jean Delay and Pierre Deniker in 1952 were some of the first psychiatrists to trial the medication and they reported that it was of benefit in controlling manic and psychotic agitation. Within a year it was widely available for use in psychiatry throughout France (Seeman, 2021). The discovery that drugs that block dopamine transmission (D_2 receptor) have an antipsychotic effect led to the development of the dopamine hypothesis for schizophrenia (Seeman, 2021).

Table 35.1. Antipsychotic Medication

Class	Commonly used medications available in Australia	Major indications and application in clinical practice
First generation antipsychotics (typical)	<i>Chlorpromazine, flupentixol, haloperidol, zuclopenthixol</i>	Schizophrenia Mania Psychosis
Second generation antipsychotics (atypical antipsychotics)	<i>amisulpride, asenapine, clozapine, lurasidone, olanzapine, paliperidone, quetiapine, risperidone, ziprasidone</i>	Schizophrenia Psychosis Mania As adjunctive treatment for MDD Bipolar depression
Third generation antipsychotics	<i>aripiprazole, brexpiprazole, cariprazine</i>	Schizophrenia Psychosis Mania As adjunctive treatment for MDD Bipolar depression

Note: MDD = Major Depressive Disorder

The ‘first generation’ antipsychotic medications all have a common action in blocking the D_2 receptor in the mesolimbic and mesocortical system. There are other dopaminergic pathways in the brain, and blocking dopamine in these areas leads to adverse effects including extrapyramidal symptoms (EPS) due to dopamine blockade in the nigrostriatal system and increased prolactin release due to dopamine blockade in the tuberoinfundibular system. In the 1990s a second generation of antipsychotics became available. These antipsychotics, based on the action of clozapine, also acted on the $5HT_{2A}$ serotonin receptors (along with other receptors) which reduced the risk of EPS. More recently a third generation of antipsychotic medications which act as partial agonists at the dopamine $D_{2/3}$ receptors have been introduced. Notably, there has been little advance in terms of

efficacy, between the first and most recent generation antipsychotics. The difference between the various agents lies predominantly with their tolerability and adverse effect profiles. Table 35.1 shows an overview of antipsychotic medications and Table 35.2 lists the typical side-effects these medications may cause.

Table 35.2. Propensity for adverse effects from antipsychotic medication

Class	Sedation	Weight	EPS	Chol	Prolactin	QTc
1st Generation Antipsychotics						
chlorpromazine	+++	+++	++	+++	+++	++
haloperidol	+	++	+++	+	+++	++
zuclopenthixol	+++	++	++	++	+++	-
2nd generation - D₂ & 5HT₂ antagonists						
asenapine	++	++	+	-	+	+
clozapine	++++	+++	-	XXX	-	+
lurasidone	++	+	+	-	+	-
olanzapine	+++	+++	-	++	+	+
paliperidone	+	++	+	-	+++	+
quetiapine	++	++	+	-	+	++
risperidone	+	++	++	-	+++	+
ziprasidone	+	+/-	+	-	-	++
D₂ & D₃ antagonist						
amisulpride	+	+	++	-	+++	++
3rd Generation – D₂ or D₂/D₃ partial agonists						
aripiprazole	+	+	-	-	-	-
brexpiprazole	+	+	-	-	-	-
cariprazine	+	+	++	-	-	-

Note: - = no effect; +mild; ++ = moderate; +++ = large effect; XXX – highly anticholinergic, paradoxical increase salivation. EPS = Extrapyramidal Side Effects, Chol = anticholinergic effect, prolactin = increased prolactin secretion, QTc = prolongation of QTc interval. (Galletly et al., 2016)

Antipsychotic medication can be administered as oral, sublingual or intramuscular preparations. Intramuscular preparations can aim for short term effect (e.g. zuclopenthixol acetate) or as long acting injectables (LAI), sometimes known as “depot” medications, which aim to improve treatment adherence over the longer term. These preparations can be given from every 2 weeks to up to 6-monthly.

Antidepressant Medications

The introduction of imipramine, a tricyclic antidepressant (TCA) and the monoamine oxidase inhibitors (MAOIs) led to a dramatic change in the treatment of severe depression. Prior to this, the only effective treatment for severe depression were ECT and for some psychosurgery. Psychotherapy had limited success for most severe forms of depression and in particular melancholia – largely because patients were too unwell to engage in meaningful therapy. A monoamine hypothesis of depression was formulated in response to these initial discoveries in the same way that the simplistic dopamine hypothesis of schizophrenia was developed after the introduction of antipsychotics (Hirschfeld, 2000).

The first generation of antidepressants, the tricyclics and MAOIs, while effective as antidepressants were limited by their significant adverse effect burden and toxicity at high doses, which made them dangerous in both intentional and inadvertent overdosing. The tricyclics caused anticholinergic adverse effects (dry mouth, blurred vision, postural hypotension, urinary retention [for males] and worsening of glaucoma) and the MAOIs required patients to be on a low tyramine diet to avoid the risk of a hypertensive crisis. These were significant limitations. The introduction of drugs that targeted serotonin – namely, Selective Serotonin Reuptake Inhibitors (SSRIs) in the 1990s, with fluoxetine (Prozac), completely changed this.

The SSRIs were easier to prescribe than the TCAs, as they had fewer adverse effects and were safer in overdose. This quickly led to an upsurge in prescribing antidepressant medications to a broader population. At the same time, and akin to existing antidepressants, the SSRIs were found to be effective in the treatment of anxiety disorders. Anxiety and depression are frequently seen together and changes in the serotonergic system are common to both (Pourhamzeh et al., 2022). However, neither set of disorders should be seen simply as a disease of the serotonergic system, rather a far more complex process extending beyond this single neurotransmitter system is thought to be responsible (see chapter 25). Table 35.3 shows an overview of antidepressant medication.

Table 35.3. Antidepressant Medications.

Class	Commonly used medications available in Australia	Major indications
Tricyclic antidepressants (TCA)	<i>amitriptyline, clomipramine, dosulepin, imipramine, nortriptyline</i>	MDD Anxiety disorders OCD (clomipramine only) Pain
Noradrenergic and specific serotonergic antidepressant (NaSSA)	<i>mirtazapine</i>	MDD
Selective Serotonin Reuptake Inhibitors (SSRIs)	<i>citalopram, escitalopram, fluoxetine, fluvoxamine, sertraline,</i>	MDD Anxiety disorders OCD
Serotonin and Noradrenaline Reuptake Inhibitors (SNRIs)	<i>venlafaxine, desvenlafaxine, duloxetine</i>	MDD Anxiety disorders OCD
Monoamine oxidase Inhibitors (MAOI)	<i>phenelzine, tranylcypromine,</i>	MDD Atypical depression Anxiety disorders
Reversible inhibitor of monoamine oxidase (RIMA)	<i>moclobemide</i>	MDD
Noradrenaline reuptake inhibitor	<i>reboxetine</i>	MDD
Selective catecholamine (Noradrenaline & Dopamine) reuptake inhibitor	<i>bupropion</i>	MDD Smoking cessation
Serotonin Modulator	<i>vortioxetine</i>	MDD
Melatonergic	<i>agomelatine</i>	MDD Generalised anxiety disorder

Note: MDD = Major Depressive Disorder, OCD = Obsessive Compulsive Disorder

Anxiolytics, Hypnotics and Sedatives

Relieving anxiety, calming fears or helping to induce sleep have long been important aims for medicine or traditional health care from earliest times, and substances used to achieve this have included alcohol, cannabis, kava, opioids and many other herbal agents. Treatments for anxiety and insomnia were some of the earliest psychoactive substances synthesised and included bromide salts, chloral hydrate paraldehyde and barbiturates. In the 20th century, the barbiturates were widely used, both as sleeping pills and antianxiety agents. However, there were significant problems with them, particularly the high risk of dependence and their lethality in overdose.

The introduction of the benzodiazepines (initially chlordiazepoxide) was a considerable advance as they were safe in overdose as compared to antidepressants and yet highly effective in reducing anxiety and calming individuals. At the time the potential for drug dependence was not known but this became a significant problem as well the experience of severe withdrawal symptoms upon cessation. Dependence caused patients to seek prescribers ‘doctor shopping’ as prescribing became restricted. This is particularly a risk in benzodiazepines with a short half-life, such as alprazolam, which has led to their use being restricted. While they were originally first line treatments for anxiety disorders, this role has been superseded by the SSRIs.

Benzodiazepines are also commonly used as sedatives or hypnotics. Again, due to the risk of dependence the use of these medications for insomnia is not recommended beyond the short term. Finally, a benzodiazepine such as midazolam, which can be used parenterally, has become invaluable for the short-term management of patients who are highly agitated in emergency situations. Diazepam, clonazepam and lorazepam may also be used parenterally. Table 35.3 shows an overview of anxiolytics, and hypnotics and sedatives.

Benzodiazepines, zopiclone and barbiturates, along with many anaesthetic agents and anticonvulsants, all act as allostatic modulators of the GABA-A receptors, increasing GABAergic activity and contributing to tonic inhibition across the brain (Koh et al., 2023; Kim et al., 2020).

Table 35.4. Anxiolytics, Hypnotics and Sedatives

Medication type	Class	Commonly used medications available in Australia	Major indications
Anxiolytics	Anxiolytics	<i>alprazolam,</i> <i>bromazepam,</i> <i>clobazam,</i> <i>clonazepam,</i> <i>diazepam,</i> <i>lorazepam,</i> <i>oxazepam</i>	Anxiety, alcohol withdrawal, catatonia, epilepsy, muscle spasm, panic attack
Hypnotics and sedatives	Benzodiazepine hypnotics	<i>flunitrazepam,</i> <i>midazolam,</i> <i>nitrazepam,</i> <i>temazepam,</i>	Anaesthesia, insomnia
	Benzodiazepine related hypnotics	<i>zopiclone</i>	Insomnia

Lithium and Anticonvulsant Drugs

Lithium, a simple salt, was found to have marked antimanic properties by the Australian psychiatrist John Cade, making it one of the most specifically targeted medication we have in psychiatry (see also; Finding Sanity by Greg De Moore and Ann Westmore, Allen and Unwin, 2016) (Malhi et al., 2024). It is thought to achieve this by acting upon multiple steps of intracellular signalling and other cellular processes in a highly complex web of activity promoting neuroprotection and neural plasticity (Alda, 2015).

Lithium transformed the management of manic-depressive illness - the earlier term for bipolar disorder and led the pharmacological era in psychiatry. It was found to have a prophylactic mood stabilizing effect, that prevented bipolar relapses to either pole of the illness. It is also used as an augmenting agent in major depressive disorder. Lithium is a simple molecule that is excreted in the urine unmetabolized – however it has a therapeutic window, which needs to be maintained to achieve efficacy and avoid both acute and long-term adverse effects such as thirst, tremor, thyroid changes, renal dysfunction and gastrointestinal effects. Details regarding the therapeutic range and monitoring can be found in the RANZCP Clinical Practice Guidelines for Mood Disorders (Malhi et al., 2016b)

Certain anticonvulsants have also been found to have mood stabilizing effects, including carbamazepine, sodium valproate and lamotrigine. These medications were heavily marketed by the pharmaceutical industry for use in bipolar disorder. This contributed to lithium falling out of fashion, despite being the most effective mood stabilizer and antimanic agent. These anticonvulsants are also considered to be mood stabilizers (as are the second-generation antipsychotics), but they don't have the unique properties lithium has. Sodium valproate is contraindicated for women of childbearing age as it has teratogenic effects and can lead to infants born with intellectual impairment. It is also not recommended for males who are planning to father, as their offspring are at increased risk of neurodevelopmental disorders too. Table 35.5 shows an overview of mood stabilizers.

Table 35.5. Mood stabilizers.

Class	Commonly used medications available in Australia	Major indications
	<i>Lithium</i> ¹	Bipolar disorder, adjunctive treatment for MDD
Anticonvulsant mood stabilizers	<i>Carbamazepine, Lamotrigine, Sodium valproate,</i>	Bipolar disorder, epilepsy

¹ Lithium is the only mood stabilizer that has the only long-term prophylactic effect and has genuine effect on both poles of the disorder.

Stimulant Medications

Stimulants are primarily prescribed for the treatment of attention deficit hyperactivity disorder (ADHD). They are also used for the treatment of sleep disorders such as narcolepsy, binge eating disorder and treatment resistant depression. Due to the risk of misuse and diversion the supply of these medications has additional restrictions. There has been a doubling in the prescription of stimulants since 2018 in Australia (Australian Institute of Health and Welfare, 2024b). This has paralleled an increased awareness of ADHD and demand for stimulants. A careful approach to the assessment and diagnosis of people with ADHD along with the consideration of behavioural and family treatments is recommended (see Chapter 25) Table 35.6 shows an overview of stimulants.

Table 35.6. Stimulants.

Class	Commonly used medications available in Australia	Major indications
Stimulants	Armodafinil, dexamfetamine, lisdexamfetamine, methylphenidate, modafinil	ADHD Narcolepsy Binge eating disorder
Non-stimulant medication	Atomoxetine, guanfacine	ADHD

Key Principals

A number of considerations need to be thought through when prescribing psychotropic medication - we want to prescribe a medication that is effective, well tolerated and safe.

Make a Diagnosis

First, there needs to be a clear diagnosis of the patient’s disorder. This does not negate the importance of a biopsychosocial and lifestyle formulation, which will guide the comprehensive management of the patient. The diagnosis (along with the presenting symptoms) guides the clinician as to which medication is appropriate for the patient, if indeed medication is necessary. Non-pharmacological options should always be considered and, even if medication is prescribed, non-pharmacological and psychosocial options should also be used.

Patient Preference

A key consideration in selecting a medication is patient preference. Poor adherence to medication is frequent in mental health with up to 44-56% of people with a severe mental illness being non-adherent (Semahegn et al., 2020). This makes poor treatment adherence one of the commonest reasons why treatment with medication fails. Adherence is improved if the patient is involved in the decision about which treatment to take and this is best done in a shared decision-making model (Hoffmann et al., 2014). This includes a discussion about the expected benefits and potential adverse effects of the medication. Be aware that patients might have misinformation, from the internet or through word of mouth, about the adverse effects of medications. In such situations, it is essential to listen to the patient’s concerns and present them with clear and accurate information

about the drug, including providing them with consumer fact sheets. Personal, familial and cultural factors need to be taken account of.

A discussion of the risks and benefits of a medication, taking into account patient preference, is an essential part of prescribing and part of the process of obtaining consent from the patient. Even though in psychiatry people can be compelled under the Mental Health Act to take a particular medication, their concerns, especially about adverse effects, need to be considered.

Treatment adherence can be improved by a wide range of evidence-based interventions. This includes simplifying when medication is taken, making this only once in the 24-hour cycle, preferably at night as many psychotropic medications can be sedating; or how it is dispensed, multiple medications can be pre-packaged by the pharmacist. Using prompts such as an alert in the person's mobile phone or leaving the medication next to the toothbrush that is used every night can be effective as can finding other ways of including taking medication in the regular routine of going to bed. Enrolling the patient in a formal program for treatment adherence can be useful. These programs have education, community support and automatic prompts to remind patients to refill prescriptions. They often involve the pharmacist more actively in the patient's care. Cognitive behavioural therapy programs have been developed specifically to improve adherence. Some antipsychotic medication can be given as a long-acting injectable preparation. Sometimes this route of administration is the patient's choice. When medication compliance will be a problem, long-acting injectable antipsychotic medication may need to be mandated as part of a community treatment order under the Mental Health Act. Finally, electronic medication monitoring systems that can check on actions such as opening a pill bottle, are being used in pharmacological trials and increasingly, will be available for general community use (Kini and Ho, 2018). Despite all these interventions, the basic task of explaining why someone should take a medication remains an essential part of the role of a doctor.

Efficacy

When we prescribe a medication for a patient, we need to ensure that the medication is effective in treating the patient's principal diagnosis or symptoms profile. All psychotropic drugs will have been tested in randomised controlled trials to test their efficacy compared with placebo, the gold standard clinical trial. All medications must have demonstrated efficacy in pivotal clinical trials before they are approved for use for a particular indication, and before they can be prescribed. In Australia, such approval comes from the Therapeutic Goods Administration (TGA) so they can be listed on the Pharmaceutical Benefits Scheme (PBS).

In trying to determine the efficacy of a particular treatment we need to look at individual randomised controlled trials (RCTs) and the systematic reviews and meta-analyses performed when RCTs are brought together. These will provide the best sources of evidence for the efficacy of a medication. A recent development has been to compare the efficacy of drugs using network meta-analyses, that allow comparison between various drugs being used to treat a particular condition. Generally, drugs are not tested against each other in clinical trials, but they will all have been compared to placebo; in a network meta-analysis it is possible to extrapolate the comparative efficacy by examining how each drug compares with placebo.

A problem with RCTs is that the participants are generally not typical of the patients seen in routine clinical practice. Participants are recruited based on having a straightforward diagnosis, with no comorbidities, no underlying personality disorder, no physical health problems and being reliable in taking medication. Women of childbearing age, unless they are on contraception, children or adolescents and the elderly are excluded from efficacy studies. By contrast, large scale effectiveness studies recruit ‘real world’ patients with fewer exclusion criteria and provide better evidence of the effectiveness of psychotropic medication. However, there are too few such studies.

Psychotropic medications also have a range of actions that are not addressed by the general categories of “antidepressant” and “antipsychotic” (Moncrieff and Cohen, 2009), for example antipsychotics have been used in the treatment of depression. While there are arguments about how the medications work, the key consideration is how effective the drug is in the treatment of a particular disorder or symptom.

Tolerability

An effective medication is not going to benefit a patient if it is not taken as prescribed. Poor tolerability is one of the main reasons patients do not take their medication. All medications have some side-effects (or adverse effects), of varying degrees of severity. These adverse effects arise in part because of the pharmacodynamics of the drug (or what the drug does to the body). Psychotropic drugs have effects beyond their primary targeted action, with many different neurotransmitter systems affected which contribute to the adverse effects. For example, the therapeutic effect of the first-generation antipsychotics is through dopaminergic blockade in the mesolimbic system, but these same medications also block dopamine in the nigrostriatal system causing extrapyramidal symptoms and

in the tuberoinfundibular system leading to prolactin release causing gynaecomastia and sexual side-effects.

Antidepressants have a range of different adverse effects and in determining the most appropriate medication for the patient there needs to be a discussion with the patient about the side-effects the patient does not want to potentially have, as shown in Figure 35.2.

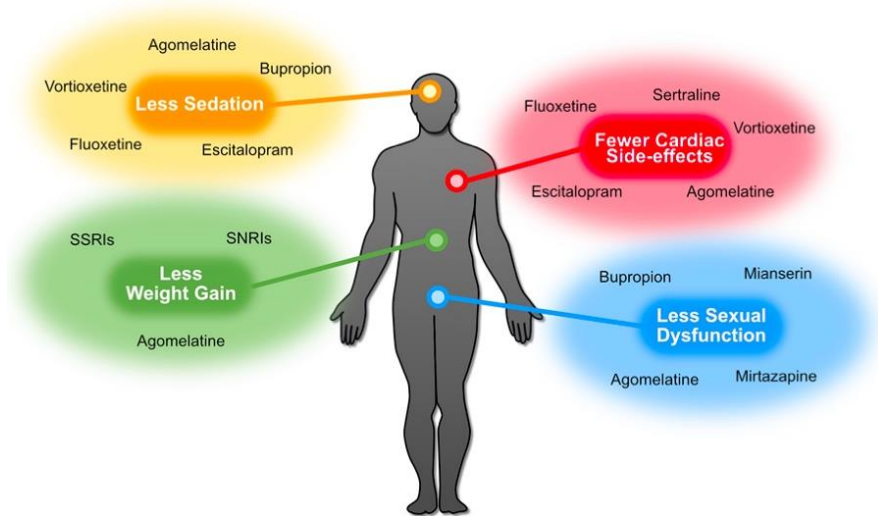


Figure 35.2. Adverse effect profiles of the antidepressants. Adapted from Malhi et al. (2021b).

How the drug is dealt with by the body, its absorption, distribution, metabolism and excretion (pharmacokinetics) influence serum levels of the active drug and will also contribute to the development of adverse effects. The pharmacokinetics of the drug, such as their propensity to induce or inhibit the cytochrome system (CYP450 enzymes) can also contribute to interactions with other medications the patient may be taking. Adverse effects can arise as well from psychological factors, what the patient expects the medication to do. It is noteworthy that in RCTs, patients randomised to placebo will generally report some adverse effects (a nocebo effect).

How to Use Medications

Clinical Practice Guidelines, such as those developed for the Royal Australian and New Zealand College of Psychiatrists (RANZCP), bring together

the various clinical trials and meta-analyses to provide guidance on the most efficacious medications to be used for the major psychiatric disorders. The RANZCP has produced guidelines for the Anxiety disorders, Mood disorders, Schizophrenia and Eating disorders (<https://www.ranzcp.org/clinical-guidelines-publications>). Guidelines produced by the National Institute for Health and Care Excellence (NICE) in the UK (<https://www.nice.org.uk/about/what-we-do/our-programmes/nice-guidance/nice-guidelines>) complement and extend College guidelines across a far broader range of disorders. Practical information about indications, dose and adverse effects is available through eTherapeutic Guidelines (Judd et al., 2021) or the Maudsley Prescribing Guidelines in Psychiatry (Taylor et al., 2021). Both resources are available online.

Conclusion

The number of medications available for treatment of psychiatric disorders has grown steadily over the past three decades and effectiveness and evidence base for these treatments is now comparable to other areas of medicine (Leucht et al., 2012). Their use in clinical practice is enhanced by the wide range of psychological interventions that are sometimes as effective or even more so for some psychiatric conditions – such as anxiety disorders (Huhn et al., 2014). As clinicians, we need to have a working knowledge on the pharmacodynamics, pharmacokinetics and adverse effects of the medications to allow us to communicate to our patients why we are suggesting they take a particular medication. This will allow you to give the best advice for your patients as well as being able to anticipate any adverse effects. Finally, medications only have an effect if they are taken, and working with your patient to establish the best course of treatment is essential.

Further Reading

- Kini, V., & Ho, P. M. (2018). Interventions to improve medication adherence: a review. *JAMA*, 320(23), 2461-2473.
- Taylor, D. M., Barnes, T. R., & Young, A. H. (2021). *The Maudsley prescribing guidelines in psychiatry*. John Wiley & Sons.
- Stahl SM. (2021) *Essential Psychopharmacology*, Cambridge: Cambridge University Press.

Chapter 36: Electroconvulsive Therapy and Other Brain Stimulation Techniques

Prashanth Mayur

Specialty of Psychiatry, The University of Sydney, Australia, and Cumberland Hospital, Western Sydney Local Health District, Australia.

Electroconvulsive Therapy

Introduction

Electroconvulsive therapy (ECT) was first used by Ugo Cerletti and Lucio Bini in Italy in 1938 (Cerletti, 1950). Since then, it has been employed continuously for the treatment of a range of conditions, most importantly severe depression. ECT is perhaps the most controversial treatment in psychiatry. It is associated with varying degrees of misinformation, prejudice and stigma (with inaccurate portrayals in popular movies). However, at its core, the value of ECT as one of the principle therapeutic tools to treat severe mental illnesses has remained unchanged for the past 80 years. In recent decades, there have been many systematic refinements to the technique of ECT that retain clinical effectiveness while reducing adverse effects.

Indications

In contemporary practice, the primary indications for ECT (American Psychiatric Association, 2008) are as follows:

- Major Depressive Disorder in severe, melancholic and/or psychotic subtypes (see Chapter 16)
- Schizophrenia particularly catatonic subtype (see Chapter 15)
- Bipolar disorder- severe manic episode, mixed affective episode (see Chapter 17)
- Catatonia due to other causes

The criteria for ECT among these diagnostic conditions are *primary* when there is a need for the rapid resolution of symptoms. This occurs typically in clinical situations of acute or imminent suicide risk or severe self-neglect - not eating and drinking. The criteria for ECT are *secondary* when there is treatment resistance or intolerance with medications. The secondary indications for ECT are as follows:

- Treatment-resistant Major Depressive Disorder.
- Intolerance to antidepressant medication
- Treatment resistant schizophrenia.
- Treatment resistant severe manic episode, mixed affective episode.

Is ECT Effective?

ECT has the disadvantage of being a treatment that has been used for many decades when it comes to assembling the evidence of its effectiveness and safety as treatment practices and research methodology have varied over this period. Nevertheless, there is strong evidence for the effectiveness of ECT in the treatment of major depression when tested against sham ECT - in which participants were anaesthetised but not given ECT – with a standardised effect size (ES) of -0.91, (95% CI -1.27 to -0.54) and against antidepressant pharmacotherapy ES 0.80, (95% CI -1.29 to -0.29) (The UK ECT Review Group, 2003). There is evidence for its effectiveness in bipolar disorder, for both the treatment of the depressive phase of bipolar disorder (Bahji et al., 2019a) and the manic phase (Elias et al., 2021). There is also clear evidence for the use of ECT as a treatment for schizophrenia both for the acute phase of the illness (Tharyan and Adams, 2009) and as an augmenting therapy with clozapine for treatment resistant schizophrenia (Lally et al., 2016).

Patient Selection

Conditions for which ECT are indicated are outlined above (Fink, 1993). Patient preference is important when considering ECT. Good prognostic indicators (van Diermen et al., 2018) of a response to a course of ECT include:

- elderly patients with severe delusional depression.
- a past history of response to ECT

Patients with a primary personality disorder or anxiety disorders including obsessive compulsive disorder are unlikely to benefit from ECT.

Contraindications

There are many *relative* contraindications to ECT (American Psychiatric Association, 2008). These carry a greater risk of harm to the patient, which precludes ECT as an immediate option until the risk is addressed. Some of the contraindications are as follows:

- acute myocardial infarction
- acute stroke
- raised intracranial pressure
- cardiac failure
- acute lower respiratory tract infection
- uncontrolled hypertension
- narrow angle glaucoma.

Ideally, management of these medical high-risk conditions should take place before ECT is commenced (Abrams, 2002). As an example, the commencement of ECT should be delayed for 3 months following a myocardial infarction. Similarly, commencement of ECT should be delayed until an acute lower respiratory infection is treated. Implicit in this discussion is the absence of *absolute* contraindications and that the choice of ECT is based on relative risks and benefits.

Mechanism of Action

The mechanisms of action of ECT in treating a myriad of seemingly different and contrasting disorders are complex and multimodal involving not only the plasticity of brain networks but also receptor systems, catecholamine-neurochemistry, brain peptides and genes. While the ‘macro effects’ can be studied at least partially in human subjects, the molecular effects are mostly limited to studies on rodent brains.

A useful starting point is the clinical observation that eliciting a generalised seizure is a prerequisite for clinical improvement whereas ‘sham ECT’ with only anaesthesia will not produce a response (Ottosson, 1996). During an ECT treatment an ictal-electroencephalographic (ictal-EEG) typically shows an early phase of high frequency low amplitude activity followed by a mid-seizure phase of spike and slow wave activity. The EEG at the end of the seizure is characterised by post-ictal electrical silence or suppression. The degree of post-ictal suppression (Suppes et al., 1996) and the measurement of ‘regularity’ of the mid-seizure delta waves (Krystal et al., 1997) are predictive of antidepressant response. These changes to electrical activity of the brain are hypothetically linked to massive surges of GABA interneuron activity that has occurred due to electrical brain stimulation.

Positron Emission Tomography studies during ECT have shown that there are distinct increases in regional cerebral blood flow in basal ganglia, amygdala and frontal lobes compared to before ECT (Takano et al., 2007). The inter-ECT brain changes using PET studies indicate a reduction in cerebral metabolic rate of glucose in the frontal lobes and cingulate cortex and such changes are correlated with antidepressant responses (Nobler et al., 2001). More recent structural and functional magnetic resonance imaging studies have also identified a network of structures including the subgenual cingulate cortex, dorsolateral prefrontal cortex, ventromedial prefrontal cortex and hippocampus that are implicated in depression and the expression of emotion (Argyelan et al., 2023).

Electroshock (ECS) on rodent brains have revealed many distinct neurochemical effects on catecholamines (serotonin, norepinephrine and dopamine). The most studied effect is the sensitization of the post synaptic serotonin receptor and a consequent increase in serotonin transmission (Burnet et al., 1999). ECS may enhance post-synaptic noradrenaline neurotransmission and pre-synaptic dopamine neurotransmission and induces gene expression in frontal and hippocampal areas (Altar et al., 2004). The ECS-regulated genes - a notable gene being Brain Derived Neurotrophic Factor (BDNF) - are involved in a wide array of changes that essentially support neuroplastic changes in the brain through neurogenesis and altered neurotransmitter signalling.

ECT- Process and Technique

A course of ECT is composed of ECT sessions 2-3 times per week. The total number of sessions during an acute course can vary from shorter courses of 3-6 sessions to treat mania to 8-16 sessions to treat severe depression. Following an acute course, ECT may be administered less frequently for up to 6 months to prevent a relapse (continuation ECT) and beyond 6 months to prevent a new episode or prophylaxis (maintenance ECT).

ECT is typically administered using a short-acting anaesthesia (thiopentone, propofol) followed by a muscle relaxant (suxamethonium). Oxygenation (100% O₂ positive pressure) is maintained throughout the procedure from the onset of anaesthesia until spontaneous respiration, except during the period of electrical stimulation. Basic standards of ECT mandate routine monitoring of physiological parameters (EEG, ECG, pulse oximetry).

With each treatment (ECT session) an electrical stimulus is given via electrodes placed on the scalp to deliver a prescribed dose of electrical charge to the brain. The charge is delivered in brief pulses. The pulses vary in width (duration)

from 0.3 to 1 millisecond. Pulse widths shorter than 0.5 milliseconds are called ‘ultra-brief’ and have clinical benefit while minimising memory adverse effects. On contemporary ECT machines the dose of electrical charge delivered is determined by adjusting current (amplitude), width of the individual pulses, frequency of the pulses and the stimulus train length which typically varies from 0.5 to 8 seconds.

There are three commonly used electrode positions to deliver the stimulus dose: 1) bitemporal, 2) unilateral and 3) bifrontal. Bitemporal ECT is where the electrode is placed 1 inch above the midpoint of the line drawn between the tragus and the outer canthus of the eye. Unilateral ECT is where the electrode is placed 1 inch lateral (right or left) to the intersection of the line connecting the auditory tragi and the line joining the nasion to the inion. Bifrontal ECT is where the electrode is placed 5cm above the outer canthus of the eye on a bony ridge at the lateral edge of the forehead where the forehead meets the temple.

During the first ECT session, an essential step is the determination of seizure threshold, which is the minimum stimulus dose that is required to produce a generalised seizure of at least 20 seconds. Thereafter, from the second ECT session, the stimulus dose is administered at a higher stimulus intensity, usually a multiple of the seizure threshold. The determination of this multiple varies with the electrode position to deliver the stimulus. The multiple is smaller (Letemendia et al., 1993; Sackeim et al., 2000) (1.5-2.5 times threshold) for bitemporal and bifrontal electrode position and larger (Sackeim et al., 2000) (5-6 times threshold) for a unilateral electrode position. A higher stimulus intensity above the threshold has demonstrated a better clinical efficacy particularly in unilateral ECT (Sackeim et al., 2000).

Preparing the Patient

ECT is a medical procedure. Similar to other medical procedures, patients will need to be prepared for their sessions. Written consent is sought from the patient. In circumstances where ECT is considered essential and the patient does not have the capacity to provide consent, or if they are an involuntary patient, then an order for involuntary treatment with ECT can be made under the appropriate Mental Health Act.

A focused clinical examination is a basic requirement. At a minimum, a bedside clinical examination of the chest, a neurological examination, examination of blood pressure and an examination of the state of the dentition to make sure that there are no loose teeth are required. A chest X ray and an ECG are routinely ordered. Brain imaging is not typically required. Patients are instructed to fast for at least 6 hours before the procedure. Most medications except anti-hypertensive

medications are withheld before the commencement of ECT and given following the completion of the procedure. An anaesthetic review is recommended particularly in patients who are medically high-risk.

Is ECT Safe?

Modern ECT as described above is safe. Death due to ECT is extremely rare (one in 30000 treatments) and is more often related to anaesthetic events (e.g. aspiration) than the ECT stimulus itself. On the other hand, adverse effects such as headache or muscle aches (related to muscle relaxant) are commonly encountered during ECT.

Memory effects are the most clinically significant adverse effect associated with ECT. Memory effects are more pronounced with bitemporal ECT compared to unilateral ECT (Squire, 1977). There are broadly two types of memory disturbances: anterograde amnesia and retrograde amnesia. Anterograde amnesia is almost always seen in varying degrees with ECT. It could be mild in the form of forgetting some of what the doctors discussed with the patient to severe post ECT confusion or delirium. Anterograde amnesia is fully reversible within 2-4 weeks after the completion of the course of ECT (Semkovska and McLoughlin, 2010). Retrograde amnesia is sometimes seen during ECT. Retrograde amnesia is of two types: non-personal memory (memory of public events) and personal or autobiographical memory. It is important to note that ECT produces less personal memory loss compared to non-personal memory loss. Personal memory when affected is usually restricted to memories formed over the past 6 months to 2 years (Lisanby et al., 2000). Unfortunately, personal memory loss can be permanent.

Legal and Ethical Issues

The administration of ECT may sometimes pose difficult ethical dilemmas. They are encountered typically when a patient is incapable of providing consent for the treatment due to their severe mental illness. Some examples are a severely depressed patient may seek (to avoid) ECT so that he or she could die or experience hell, a catatonic patient may not be able to discuss the pros and cons of ECT because he is mute and unresponsive. In such situations, beneficence and non-maleficence direct one to prescribe the most effective form of ECT with the least number of adverse effects. One should not forget the *moral* underpinnings of the treatment that should afford the patient the right to be treated with respect and dignity, right for a second opinion and the right to have ones next of kin treated with consideration.

Many patients voluntarily consent to have ECT when ECT is recommended. The question of 'mental competence' may arise even in such

situations. The prescriber should then ask whether the patients understands the nature of his condition, understand and *appreciate* the nature of ECT and consequences of accepting or refusing ECT (Clark, 1985).

Transcranial Magnetic Stimulation

Introduction

Transcranial magnetic stimulation (TMS) is the induction of small currents in the superficial layers of the brain through applying rapidly alternating magnetic fields to the head. An electromagnetic coil, which emits pulsed magnetic fields is held on the scalp, which induces electrical current in the brain. TMS stimulation paradigms include single pulse TMS where stimulation is one pulse at a time and repetitive pulse TMS (rTMS) where stimulation is repeated at regular intervals ranging from 1-25Hz. In contrast to ECT, TMS delivers a more focal electrical stimulation of the brain. In addition, a course of TMS can be entirely administered as an outpatient procedure in contrast to a typical acute course of ECT.

Indications

TMS is indicated in the treatment of Major Depressive Disorders. There have been trials conducted over a number of decades which have showed a modest effect size comparable to that of antidepressants but less than that of ECT (Bares et al., 2009). The role of TMS in the treatment of schizophrenia is uncertain. While little benefit appears to be gained in using TMS for the treatment of positive symptoms of schizophrenia, in particular auditory hallucinations (Marzouk et al., 2019), more benefit has been seen in the use of TMS for the treatment of negative symptoms (Lorentzen et al., 2022). More experience is needed before the indications of TMS in schizophrenia are clear.

Contraindications

Presence of metal particles in the head, cochlear implants, implanted neural stimulators or medical pumps are absolute contraindications. Risk of seizures although rare should be considered during the administration of TMS.

Patient selection

TMS is generally administered to less severely depressed patients and those who are relatively less medication-resistant in contrast to ECT. Patient choice is also a factor in decisions to commence ECT or TMS.

Mechanism of Action

TMS generates electrical currents oriented perpendicular to the magnetic field called ‘eddy currents’ in the superficial areas of the cortex. This can trigger functional neural circuits and cause observable behavioural effects. At a single motor neuron level, when stimulus is applied over the motor cortex area of the scalp at sufficient intensity, there is a twitch of contralateral hand muscles. In single-pulse stimulation, the twitch is instantaneous and short lasting. In rTMS stimulation, functional neural circuits are stimulated to produce lasting changes in cortical excitability at the site of the stimulation and increased connectivity and plasticity in the stimulated areas (Jannati et al., 2023). Thus, alteration or correction of focal abnormalities in cortical excitability, posited in psychiatric conditions such as depression, is considered to be a mechanism of action of rTMS.

TMS - Process and Technique in Depression

TMS is given to an awake patient and does not require anaesthesia. It is therefore a non-invasive form of brain stimulation. Screening for metal particles in the head, past seizures or head injuries should occur before commencement of TMS. TMS is administered under medical supervision. At the beginning the resting motor threshold (RMT) is determined by single pulse stimulation of the motor cortex that will produce twitching of the contralateral thumb muscles. Determination of RMT allows the determination of the stimulus intensity generally below the RMT (80-90%) or above the RMT (110-120%). In the treatment of depression, stimulation is given with either left sided high frequency (10Hz) stimulation or right sided low frequency (1Hz) stimulation. A scalp position 5 cm forward from the motor site in the sagittal plane allows for the determination of the coil position over the dorsolateral prefrontal cortex. One session will generally involve multiple trains of stimulation, a typical recommendation is 75 trains in a session (Fitzgerald and Daskalakis, 2013). Each train duration should be limited to 4 seconds if the intensity is at 120% to avoid provoking a seizure. Newer treatment techniques are personalising placement of the coil on the basis of functional and structural brain anatomy.

Clinical recommendation of first line rTMS treatment for depression should involve a course of 4 weeks of rTMS (at least 5 sessions a week) with left sided high frequency stimulation at 110-120% stimulus intensity (Fitzgerald and Daskalakis, 2013).

Is rTMS safe?

rTMS is generally well tolerated. Syncope or seizure can rarely occur during stimulation. During stimulation, ear plugs are recommended to minimise

impact on hearing. There is no evidence to suggest that rTMS administered through standard protocols produces cognitive impairment. This is a major difference with respect to that of ECT.

Other Brain Stimulations

There are several other brain stimulation techniques including transcranial Direct Current Stimulation (tDCS), Vagal Nerve Stimulation (VNS), and Deep Brain Stimulation (DBS). Direct current stimulation using tDCS (applied to the scalp using saline soaked electrodes) is based on polarization of tissue thereby affecting the firing rate of neurons. It is a neuromodulation technique that has experimental value in the treatment of depression. The technique delivers 1-3 mA of DC current to the scalp, is safe and well tolerated. It is also purported to be a technique to enhance cognition although this remains to be proven.

Vagal Nerve Stimulation is the stimulation of the left cervical vagal nerve by a subcutaneously implanted pulse stimulator. This technique has been approved by the Food and Drug Administration (FDA) in the United States for the treatment of treatment-resistant chronic depression although only two randomised controlled trials have been conducted (Bottomley et al., 2020).

Deep Brain Stimulation using implantable electrodes to stimulate specific areas of the brain has promise to treat intractable depression, OCD and Tourette syndrome (Williams and Okun, 2013).

Conclusion and Future Directions

The field of brain stimulation contains established treatments such as ECT and rTMS and emerging treatments such as transcranial Direct Current Stimulation, Vagal Nerve Stimulation and Deep Brain Stimulation. The evolving application of these techniques to a range of psychiatric conditions offers exciting possibilities that have the potential to enhance the biological treatment toolbox for psychiatric conditions. It is likely that even if they are not stand-alone treatments, they have the potential to be integrated with existing pharmacological and psychological approaches to treat chronic and medication-resistant conditions in psychiatry.

Further Reading

- Abrams, R. (2002). *Electroconvulsive therapy*. Oxford University Press.
- Weiss, A., Hussain, S., Ng, B., Sarma, S., Tiller, J., Waite, S., & Loo, C. (2019). Royal Australian and New Zealand College of Psychiatrists professional practice guidelines for the administration of electroconvulsive therapy. *Australian & New Zealand Journal of Psychiatry*, 53(7), 609-623.

References

- Abramowitz JS, Whiteside SP and Deacon BJ (2006) The effectiveness of treatment for pediatric obsessive-compulsive disorder: A meta-analysis. *Behavior Therapy* 36(1): 55-63.
- Abrams R (2002) *Electroconvulsive therapy*. Oxford University Press.
- Abrams RC and Horowitz SV (1999) Personality disorders after age 50: meta-analytic review of the literature. In: Rosowsky E, Abrams RC, Zweig RA, et al. (eds) *Personality disorders in older adults: emerging issues in diagnosis and treatment*. : Lawrence Earlbaum Associates, 55-68.
- Accinni T, Papadogiannis G and Orso L (2021) De-escalation techniques in various settings. *Empathy, Normalization and De-escalation: Management of the Agitated Patient in Emergency and Critical Situations*. 65-91.
- Adams GC, Wrath AJ and Meng X (2018) The relationship between adult attachment and mental health care utilization: A systematic review. *The Canadian Journal of Psychiatry* 63(10): 651-660.
- Adams KA and McGuire EK (2022) *Research methods, statistics, and applications*. Sage Publications.
- Adams Y, Drew NM and Walker R (2014) Principles of practice in mental health assessment with Aboriginal Australians. In: Dudgeon P, Milroy H, & Walker R (eds). *Working together: Aboriginal and Torres Strait Islander mental health and wellbeing principles and practice*. Telethon Kids Institute, Kulunga Aboriginal Research Development Unit, Department of the Prime Minister and Cabinet (Australia).
- Addis ME and Jacobson NS (2000) A closer look at the treatment rationale and homework compliance in cognitive-behavioral therapy for depression. *Cognitive Therapy and Research* 24: 313-326.
- Agar MR, Quinn SJ, Crawford GB, et al. (2016) Predictors of mortality for delirium in palliative care. *Journal of Palliative Medicine* 19(11): 1205-1209.
- Agarkar S, Hurt SW and Young RC (2018) Speed of antidepressant response to electroconvulsive therapy in bipolar disorder vs. major depressive disorder. *Psychiatry Research* 265: 355-359.
- Ainsworth MD (1964) Patterns of attachment behavior shown by the infant in interaction with his mother. *Merrill-Palmer Quarterly of Behavior and Development* 10(1): 51-58.
- Ainsworth MD (1972) Attachment and dependency: A comparison.
- Ainsworth MDS, Blehar M, Waters E, et al. (1978) Patterns of attachment: Observations in the strange situation and at home. *Hillsdale, N: Erlbaum*.
- Ainsworth MDS, Blehar MC, Waters E, et al. (2015) *Patterns of attachment: A psychological study of the strange situation*. Psychology Press.

- Aizenstein HJ, Baskys A, Boldrini M, et al. (2016) Vascular depression consensus report—a critical update. *BMC Medicine* 14: 1-16.
- Akobeng AK (2005) Understanding randomised controlled trials. *Archives of Disease in Childhood* 90(8): 840-844.
- Alda M (2015) Lithium in the treatment of bipolar disorder: pharmacology and pharmacogenetics. *Molecular Psychiatry* 20(6): 661-670.
- Alexopoulos GS, Abrams RC, Young RC, et al. (1988) Cornell scale for depression in dementia. *Biological Psychiatry* 23(3): 271-284.
- Alexopoulos GS, Meyers BS, Young RC, et al. (1997) 'Vascular depression'hypothesis. *Archives of General Psychiatry* 54(10): 915-922.
- Alexopoulos GS, Streim JE, Carpenter D, et al. (2004) Expert consensus guidelines for using antipsychotic agents in older patients. *The Journal of Clinical Psychiatry* 65(Suppl 2): 1-105.
- Altar CA, Laeng P, Jurata LW, et al. (2004) Electroconvulsive seizures regulate gene expression of distinct neurotrophic signaling pathways. *Journal of Neuroscience* 24(11): 2667-2677.
- Altman N and Krzywinski M (2017) Points of significance: interpreting P values. *Nature Methods* 14(3): 213-215.
- American Psychiatric Association (1980) *Diagnostic and statistical manual of mental disorders*. Washington, D.C: American Psychiatric Association.
- American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV)*. Washington DC: American Psychiatric Press.
- American Psychiatric Association (2008) *The practice of electroconvulsive therapy: recommendations for treatment, training, and privileging (A task force report of the American Psychiatric Association)*. American Psychiatric Pub.
- American Psychiatric Association (2013) *The Diagnostic and Statistical Manual for Mental Disorders DSM 5*. Arlington, VA: American Psychiatric Publishing.
- American Psychiatric Association (2022) *Diagnostic and statistical manual of mental disorders : DSM-5-TR*. Washington, DC: American Psychiatric Association Publishing.
- Amrhein V, Greenland S and McShane B (2019) Scientists rise up against statistical significance. *Nature* 567(7748): 305-307.
- Andreasen NC (2007) DSM and the Death of Phenomenology in America: An Example of Unintended Consequences. *Schizophrenia Bulletin* 33(1): 108-112.
- Andrews G, Bell C, Boyce P, et al. (2018) Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of panic disorder, social anxiety disorder and generalised anxiety disorder. *Australian & New Zealand Journal of Psychiatry* 52(12): 1109-1172.

- Angst J, Gamma A, Rössler W, et al. (2009) Long-term depression versus episodic major depression: Results from the prospective Zurich study of a community sample. *Journal of Affective Disorders* 115(1-2): 112-121.
- Angst J, Rössler W, Ajdacic-Gross V, et al. (2019) Differences between unipolar mania and bipolar-I disorder: Evidence from nine epidemiological studies. *Bipolar Disorders* 21(5): 437-448.
- Anker E, Haavik J and Heir T (2020) Alcohol and drug use disorders in adult attention-deficit/hyperactivity disorder: prevalence and associations with attention-deficit/hyperactivity disorder symptom severity and emotional dysregulation. *World Journal of Psychiatry* 10(9): 202.
- Arevalo-Rodriguez I, Smailagic N, i Figuls MR, et al. (2015) Mini-Mental State Examination (MMSE) for the detection of Alzheimer's disease and other dementias in people with mild cognitive impairment (MCI). *Cochrane Database of Systematic Reviews*.(3).
- Argyelan M, Deng Z-D, Ousdal OT, et al. (2023) Electroconvulsive therapy-induced volumetric brain changes converge on a common causal circuit in depression. *Molecular Psychiatry* 29(2), 229-237.
- Asher R (1951) Munchausen's Syndrome. *The Lancet*.(Feb 10): 339-341.
- Asmundson GJ, Coons MJ, Taylor S, et al. (2002) PTSD and the experience of pain: research and clinical implications of shared vulnerability and mutual maintenance models. *The Canadian Journal of Psychiatry* 47(10): 930-937.
- Atkinson J (2002) *Trauma trails, recreating song lines: The transgenerational effects of trauma in Indigenous Australia*. Spinifex Press.
- Atkinson J (2017) The Value of Deep Listening - The Aboriginal Gift to the Nation. TEDxSydney.
- Atkinson J, Nelson J, Brooks R, et al. (2014) Addressing individual and community transgenerational trauma. *Working together: Aboriginal and Torres Strait Islander mental health and wellbeing principles and practice* 2: 289-307.
- Austin MP and Marce Society Position Statement Advisory C (2014) Marce International Society position statement on psychosocial assessment and depression screening in perinatal women. *Best Pract Res Clin Obstet Gynaecol* 28(1): 179-187.
- Australian Bureau of Statistics (2015) *Australian Demographic Statistics*. Available at: abs.gov.au (accessed 30th March).
- Australian Bureau of Statistics (2019) *National Aboriginal and Torres Strait Islander Health Survey*. Available at:
<https://www.abs.gov.au/statistics/people/aboriginal-and-torres-strait-islander-peoples/national-aboriginal-and-torres-strait-islander-health-survey/latest-release>.

- Australian Bureau of Statistics (2022) *Causes of Death, Australia*. Available at: <https://www.abs.gov.au/statistics/health/causes-death/causes-death-australia/latest-release>.
- Australian Bureau of Statistics (2023) *Prisoners in Australia*. Available at: <https://www.abs.gov.au/statistics/people/crime-and-justice/prisoners-australia/latest-release>.
- Australian Health Ministers' Advisory Council (2013) A national framework for recovery-oriented mental health services: Guide for practitioners and providers.
- Australian Health Practitioner Regulation Agency (AHPRA) (2020) AHPRA and National Boards Guidelines: Mandatory notifications about registered students.
- Australian Indigenous HealthInfoNet (2024) *Social and Cultural Determinants*. Available at: <https://healthinfolnet.ecu.edu.au/learn/determinants-of-health/social-cultural-determinants/>.
- Australian Institute of Health and Welfare (2011) Substance use among Aboriginal and Torres Strait Islander people. Canberra: Australian Institute of Health and Welfare.
- Australian Institute of Health and Welfare (2016) Australia's health 2016. Australia's health series no. 15. Cat. no. AUS 199. Canberra: Australian Institute of Health and Welfare.
- Australian Institute of Health and Welfare (2022a) *Admitted Patients*. Available at: <https://www.aihw.gov.au/reports-data/myhospitals/sectors/admitted-patients>.
- Australian Institute of Health and Welfare (2022b) Australian Burden of Disease Study: impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2018. Canberra: Australian Government.
- Australian Institute of Health and Welfare (2024a) *Involuntary Treatment in Mental Health Care*. Available at: <https://www.aihw.gov.au/mental-health/topic-areas/involuntary-treatment> (accessed 20th September 2024).
- Australian Institute of Health and Welfare (2024b) *Mental health-related prescriptions*. Available at: <https://www.aihw.gov.au/mental-health/topic-areas/mental-health-prescriptions> (accessed 7th October 2024).
- Australian Institute of Health and Welfare A (2022c) Suicide and intentional self-harm.
- Australian Institute of Health and Welfare A (2023) Prevalence and impact of mental illness. <https://www.aihw.gov.au/mental-health/overview/mental-illness>.

- Ayano G (2019) Co-occurring medical and substance use disorders in patients with schizophrenia: a systematic review. *International Journal of Mental Health* 48(1): 62-76.
- Aydemir O and Malhi GS (2007) Aretaeus of Cappadocia. *Acta Neuropsychiatrica* 19(1): 62-63.
- Badura-Brack AS, Naim R, Ryan TJ, et al. (2015) Effect of attention training on attention bias variability and PTSD symptoms: Randomized controlled trials in Israeli and US combat veterans. *American Journal of Psychiatry* 172(12): 1233-1241.
- Bagaric M, Touyz S, Heriseanu A, et al. (2020) Are bulimia nervosa and binge eating disorder increasing? Results of a population-based study of lifetime prevalence and lifetime prevalence by age in South Australia. *European Eating Disorders Review* 28(3): 260-268.
- Bagot RC and Meaney MJ (2010) Epigenetics and the biological basis of gene x environment interactions. *Journal of the American Academy of Child & Adolescent Psychiatry* 49(8): 752-771.
- Bahji A, Hawken ER, Sepehry AA, et al. (2019a) ECT beyond unipolar major depression: systematic review and meta-analysis of electroconvulsive therapy in bipolar depression. *Acta Psychiatrica Scandinavica* 139(3): 214-226.
- Bahji A, Mazhar MN, Hudson CC, et al. (2019b) Prevalence of substance use disorder comorbidity among individuals with eating disorders: A systematic review and meta-analysis. *Psychiatry Research* 273: 58-66.
- Bailey T, Alvarez-Jimenez M, Garcia-Sanchez AM, et al. (2018) Childhood Trauma Is Associated With Severity of Hallucinations and Delusions in Psychotic Disorders: A Systematic Review and Meta-Analysis. *Schizophrenia Bulletin* 44(5): 1111-1122.
- Baker EF (2022) *Psychiatric Emergencies*. Oxford University Press.
- Bandelow B, Sher L, Bunevicius R, et al. (2012) Guidelines for the pharmacological treatment of anxiety disorders, obsessive-compulsive disorder and posttraumatic stress disorder in primary care. *International journal of psychiatry in clinical practice* 16(2): 77-84.
- Bar-Haim Y, Holoshitz Y, Eldar S, et al. (2010) Life-threatening danger and suppression of attention bias to threat. *American Journal of Psychiatry* 167(6): 694-698.
- Bares M, Kopecek M, Novak T, et al. (2009) Low frequency (1-Hz), right prefrontal repetitive transcranial magnetic stimulation (rTMS) compared with venlafaxine ER in the treatment of resistant depression: a double-blind, single-centre, randomized study. *Journal of Affective Disorders* 118(1-3): 94-100.

- Barnett K, Mercer SW, Norbury M, et al. (2012) Epidemiology of multimorbidity and implications for health care, research, and medical education: a cross-sectional study. *Lancet* 380: 37-43.
- Bass C and Halligan P (2014) Factitious disorders and malingering: challenges for clinical assessment and management. *Lancet* 383(9926): 1422-1432.
- Bateman A and Fonagy P (1999) Effectiveness of partial hospitalization in the treatment of borderline personality disorder: a randomised controlled trial. *The American Journal of Psychiatry* 158: 36-42.
- Bateman A and Fonagy P (2016) *Mentalization based treatment for personality disorders: A practical guide*. Oxford University Press.
- Bauchner H, Fontanarosa PB, Flanagin A, et al. (2018) Scientific misconduct and medical journals. *JAMA* 320(19): 1985-1987.
- Beaglehole R, Bonita R and Kjellström T (1993) *Basic epidemiology*. World Health Organization Geneva.
- Beekman AT, De Beurs E, Van Balkom AJ, et al. (2000) Anxiety and depression in later life: co-occurrence and communality of risk factors. *American Journal of Psychiatry* 157(1): 89-95.
- Bell AC and D'Zurilla TJ (2009) Problem-solving therapy for depression: a meta-analysis. *Clinical Psychology Review* 29(4): 348-353.
- Bell E, Boyce P, Porter RJ, et al. (2021a) Irritability in Mood Disorders: Neurobiological Underpinnings and Implications for Pharmacological Intervention. *CNS Drugs* 35(6): 619-641.
- Bell E, Bryant RA, Boyce P, et al. (2021b) Irritability through Research Domain Criteria: an opportunity for transdiagnostic conceptualisation. *BJPsych Open* 7(1): e36.
- Benjamin CL, Harrison JP, Settapani CA, et al. (2013) Anxiety and related outcomes in young adults 7 to 19 years after receiving treatment for child anxiety. *Journal of Consulting and Clinical Psychology* 81(5): 865.
- Bennett-Levy J, Butler G, Fennell M, et al. (2004) Cognitive behaviour therapy: Science and practice series. Oxford guide to behavioural experiments in cognitive therapy.
- Bennett HA, Einarson A, Taddio A, et al. (2004) Prevalence of depression during pregnancy: systematic review. *Obstetrics and Gynecology* 103(4): 698-709.
- Bergink V, Boyce P and Munk-Olsen T (2015a) Postpartum psychosis: a valuable misnomer. *Australian & New Zealand Journal of Psychiatry* 49(2): 102-103.
- Bergink V, Burgerhout KM, Koorengevel KM, et al. (2015b) Treatment of psychosis and mania in the postpartum period. *American Journal of Psychiatry* 172(2): 115-123.
- Bermudez MB, Costanzi M, Macedo MJA, et al. (2019) Improved quality of life and reduced depressive symptoms in medical students after a single-session intervention. *Brazilian Journal of Psychiatry* 42: 145-152.

- Bernstein GA and Shaw K (1997) Practice parameters for the assessment and treatment of children and adolescents with anxiety disorders. American Academy of Child and Adolescent Psychiatry. *Journal of the American Academy of Child & Adolescent Psychiatry* 36(10 Suppl): 69s-84s.
- Beunders AJ, Orhan M and Dols A (2023) Older age bipolar disorder. *Curr Opin Psychiatry* 36(5): 397-404.
- Bighelli I, Rodolico A, García-Mieres H, et al. (2021) Psychosocial and psychological interventions for relapse prevention in schizophrenia: a systematic review and network meta-analysis. *The Lancet Psychiatry* 8(11): 969-980.
- Binder EB, Bradley RG, Liu W, et al. (2008) Association of FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults. *JAMA* 299(11): 1291-1305.
- Bird S (2011) Capacity to consent to treatment. *Australian Family Physician* 40(4): 249-250.
- Bisson JI, Wright LA, Jones KA, et al. (2021) Preventing the onset of post traumatic stress disorder. *Clinical Psychology Review* 86: 102004.
- Black DW, Blum N, McCormick B, et al. (2013) Systems Training for Emotional Predictability and Problem Solving (STEPPS) group treatment for offenders with borderline personality disorder. *Journal of Nervous & Mental Disease* 201(2): 124-129.
- Blanchard EB, Hickling EJ, Forneris CA, et al. (1997) Prediction of remission of acute posttraumatic stress disorder in motor vehicle accident victims. *Journal of Traumatic Stress* 10: 215-234.
- Blanchflower DG and Oswald AJ (2008) Is well-being U-shaped over the life cycle? *Social Science & Medicine* 66(8): 1733-1749.
- Blazer DG (2003) Depression in late life: review and commentary. *The journals of gerontology series A: Biological sciences and medical sciences* 58(3): M249-M265.
- Bohus M, Stoffers-Winterling J, Sharp C, et al. (2021) Borderline personality disorder. *The Lancet* 398(10310): 1528-1540.
- Bonanno GA, Ho SM, Chan JC, et al. (2008) Psychological resilience and dysfunction among hospitalized survivors of the SARS epidemic in Hong Kong: a latent class approach. *Health Psychology* 27(5): 659.
- Bonanno GA, Romero SA and Klein SI (2015) The temporal elements of psychological resilience: An integrative framework for the study of individuals, families, and communities. *Psychological Inquiry* 26(2): 139-169.
- Borkovec T, Ray WJ and Stober J (1998) Worry: A cognitive phenomenon intimately linked to affective, physiological, and interpersonal behavioral processes. *Cognitive Therapy and Research* 22(6): 561-576.

- Boschloo L, Schoevers RA, Beekman ATF, et al. (2014) The four-year course of major depressive disorder: The role of staging and risk factor determination. *Psychotherapy and Psychosomatics* 83(5): 279-288.
- Bottomley JM, LeReun C, Diamantopoulos A, et al. (2020) Vagus nerve stimulation (VNS) therapy in patients with treatment resistant depression: A systematic review and meta-analysis. *Comprehensive Psychiatry* 98: 152156.
- Bowlby J (1951) Maternal care and mental health. *Bulletin of the World Health Organisation*, 3: 355-533.
- Bowlby J (1969) Attachment and loss New York: Basic.
- Bowlby J (1982) Attachment and loss: retrospect and prospect. *American journal of Orthopsychiatry* 52(4): 664.
- Bowlby J and Bowlby R (2012) *The making and breaking of affectional bonds*. Routledge.
- Bowtell M, McGorry P and O'Donoghue B (2018) Is a higher dose of antipsychotic medication required to treat a relapse following discontinuation in first episode psychosis? *Schizophrenia Research* 204, 407-408.
- Boyce P and Barriball E (2010) Puerperal psychosis. *Arch Womens Ment Health* 13(1): 45-47.
- Boyce P, Galbally M and Gregoire A (2018) Pregnancy: The earliest opportunity for prevention and early intervention for mental disorders. In: Bhugra D, Bhui K, Wong S, et al. (eds) *Oxford Textbook of Public Mental Health*. Oxford: Oxford University Press, pp.383-387.
- Boyce P and Hadzi-Pavlovic D (2011) Risks risky and not so risky. *Acta Neuropsychiatrica* 23(1): 43-45.
- Boyce P and Hickey A (2005) Psychosocial risk factors to major depression after childbirth. *Social Psychiatry & Psychiatric Epidemiology* 40(8): 605-612.
- Boyce P, Hickie I and Parker G (1991) Parents, partners or personality? Risk factors for post-natal depression. *Journal of Affective Disorders* 21(4): 245-255.
- Bradley R, Greene J, Russ E, et al. (2005) A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry* 162(2): 214-227.
- Brennstuhl MJ, Tarquinio C and Montel S (2015) Chronic pain and PTSD: Evolving views on their comorbidity. *Perspectives in Psychiatric Care* 51(4).
- Breslau N, Davis GC, Andreski P, et al. (1991) Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Archives of General Psychiatry* 48(3): 216-222.
- Breslau N, Davis GC, Peterson EL, et al. (1997) Psychiatric sequelae of posttraumatic stress disorder in women. *Archives of General Psychiatry* 54(1): 81-87.
- Brewerton TD (2007) Eating disorders, trauma, and comorbidity: Focus on PTSD. *Eating Disorders* 15(4): 285-304.

- Brewin CR, Andrews B and Valentine JD (2000) Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology* 68(5): 748.
- Brodaty H, Draper B, Saab D, et al. (2001) Psychosis, depression and behavioural disturbances in Sydney nursing home residents: prevalence and predictors. *International Journal of Geriatric Psychiatry* 16(5): 504-512.
- Brodaty H, Luscombe G, Parker G, et al. (1997) Increased rate of psychosis and psychomotor change in depression with age. *Psychological Medicine* 27(5): 1205-1213.
- Brooks III JO and Hoblyn JC (2005) Secondary mania in older adults. *American Journal of Psychiatry* 162(11): 2033-2038.
- Broome MR, Harland R, Owen GS, et al. (2013) *The Maudsley Reader in Phenomenological Psychiatry*. Cambridge University Press.
- Brophy L, Edan V, Kisely S, et al. (2021) The Urgent Need to Review the use of CTOs and Compliance with the UNCRPD Across Australian Jurisdictions. *International Journal of Mental Health and Capacity Law* 28: 1-75.
- Brown TA and Barlow DH (1995) Long-term outcome in cognitive-behavioral treatment of panic disorder: clinical predictors and alternative strategies for assessment. *Journal of consulting and clinical psychology* 63(5): 754.
- Bruce AN, Battista A, Plankey MW, et al. (2015) Perceptions of gender-based discrimination during surgical training and practice. *Medical Education Online* 20(1): 25923.
- Bruch H (2001) *The golden cage: The enigma of anorexia nervosa*. Harvard University Press.
- Brugger SP and Howes OD (2017) Heterogeneity and Homogeneity of Regional Brain Structure in Schizophrenia: A Meta-analysis. *JAMA Psychiatry* 74(11): 1104-1111.
- Bruijnen CJ, Dijkstra BA, Walvoort SJ, et al. (2019) Prevalence of cognitive impairment in patients with substance use disorder. *Drug and Alcohol Review* 38(4): 435-442.
- Bryant RA (2003) Early predictors of posttraumatic stress disorder. *Biological Psychiatry* 53(9): 789-795.
- Bryant RA (2010) Acute stress disorder as a predictor of posttraumatic stress disorder: a systematic review. *The Journal of Clinical Psychiatry* 71(2): 381.
- Bryant RA, Friedman MJ, Spiegel D, et al. (2011) A review of acute stress disorder in DSM-5. *FOCUS* 9(3): 335-350.
- Bryant RA and Guthrie RM (2007) Maladaptive self-appraisals before trauma exposure predict posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology* 75(5): 812.
- Bryant RA and Harvey AG (1997) Attentional bias in posttraumatic stress disorder. *Journal of Traumatic Stress* 10: 635-644.

- Bryant RA, Harvey AG, Dang ST, et al. (1998) Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. *Journal of Consulting and Clinical Psychology* 66(5): 862.
- Bryant RA, Harvey AG, Guthrie RM, et al. (2000) A prospective study of psychophysiological arousal, acute stress disorder, and posttraumatic stress disorder. *Journal of Abnormal Psychology* 109(2): 341.
- Bryant RA, Moulds ML, Guthrie RM, et al. (2008) A randomized controlled trial of exposure therapy and cognitive restructuring for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology* 76(4): 695.
- Bryant RA, Moulds ML, Guthrie RM, et al. (2005) The additive benefit of hypnosis and cognitive-behavioral therapy in treating acute stress disorder. *Journal of Consulting and Clinical Psychology* 73(2): 334.
- Bryant RA, Nickerson A, Creamer M, et al. (2015) Trajectory of post-traumatic stress following traumatic injury: 6-year follow-up. *The British Journal of Psychiatry* 206(5): 417-423.
- Bryant RA, O'Donnell ML, Creamer M, et al. (2013) A multisite analysis of the fluctuating course of posttraumatic stress disorder. *JAMA Psychiatry* 70(8): 839-846.
- Buckley PF, Miller BJ, Lehrer DS, et al. (2009) Psychiatric comorbidities and schizophrenia. *Schizophrenia Bulletin* 35: 383-402.
- Buckley TC, Blanchard EB and Neill WT (2000) Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review* 20(8): 1041-1065.
- Buhlmann U, Glaesmer H, Mewes R, et al. (2010) Updates on the prevalence of body dysmorphic disorder: a population-based survey. *Psychiatry Research* 178(1): 171-175.
- Buist AE, Barnett BEW, Milgrom J, et al. (2002) To screen or not to screen--that is the question in perinatal depression. *Medical Journal of Australia* 177 Suppl: S101-105.
- Bullowa M (1979) *Before speech: The beginning of interpersonal communication*. Cambridge University Press.
- Burnet PW, Sharp T, LeCorre SM, et al. (1999) Expression of 5-HT receptors and the 5-HT transporter in rat brain after electroconvulsive shock. *Neuroscience Letters* 277(2): 79-82.
- Burns A and Iliffe S (2009) Alzheimer's disease. *BMJ* 338(feb05 1): b158-b158.
- Burns K, Jayasinha R, Tsang R, et al. (2012) Behaviour management-a guide to good practice: managing behavioural and psychological symptoms of dementia. *Sydney: Dementia Collaborative Research Centre*.
- Burns T, Catty J, Dash M, et al. (2007) Use of intensive case management to reduce time in hospital in people with severe mental illness: systematic review and meta-regression. *BMJ* 335(7615): 336.

- Burry L, Mehta S, Perreault MM, et al. (2018) Antipsychotics for treatment of delirium in hospitalised non-ICU patients. *Cochrane Database of Systematic Reviews*.(6).
- Byrne M, Agerbo E, Ewald H, et al. (2003) Parental age and risk of schizophrenia: a case-control study. *Archives of General Psychiatry* 60(7): 673-678.
- Cahalan S (2020) *The Great Pretender: The Undercover Mission that Changed our Understanding of Madness*. Canongate Books.
- Callaghan S and Ryan CJ (2016) An evolving revolution: evaluating Australia's compliance with the Convention on the Rights of Persons with Disabilities in mental health law. *University of New South Wales Law Journal* 39(2): 596-624.
- Cantu-Weinstein A, Branning R, Alamir M, et al. (2024) Diagnosis and treatment of Wernicke's encephalopathy: A systematic literature review. *General Hospital Psychiatry*.
- Cardno AG and Gottesman II (2000) Twin studies of schizophrenia: From bow-and-arrow concordances to Star Wars Mx and functional genomics. *American Journal of Medical Genetics* 97(1): 12-17.
- Carmody J (2015) Mindfulness as a general ingredient of successful psychotherapy. *Handbook of mindfulness and self-regulation*. 235-248.
- Casey P and Kelly B (2007) *Fish's Clinical Psychopathology: Signs and Symptoms in Psychiatry*. London: The Royal College of Psychiatrists.
- Caspi A, Moffit TE, Cannon M, et al. (2005) Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene: Longitudinal evidence of a gene x environment interaction. *Biological Psychiatry* 57: 1117-1127.
- Cassidy J and Shaver PR (1999) *Handbook of attachment: Theory, research, and clinical applications*. Rough Guides.
- Catts SV, O'Toole BI, Carr VJ, et al. (2010) Appraising evidence for intervention effectiveness in early psychosis: conceptual framework and review of evaluation approaches. *Australian & New Zealand Journal of Psychiatry* 44(3): 195-219.
- Catts V, Catts S, O'Toole B, et al. (2008) Cancer incidence in patients with schizophrenia and their first-degree relatives—a meta-analysis. *Acta Psychiatrica Scandinavica* 117(5): 323-336.
- Centers for Disease Control and Prevention (2012) *Principles of Epidemiology in Public Health Practice*. Atlanta, GA: United States Department of Health and Human Services.
- Cerletti U (1950) Old and new information about electroshock. *American Journal of Psychiatry* 107(2): 87-94.

- Chakrabarty T, Torres IJ, Su WW, et al. (2021) Cognitive subgroups in first episode bipolar I disorder: Relation to clinical and brain volumetric variables. *Acta Psychiatrica Scandinavica* 143(2): 151-161.
- Chan JKN, Correll CU, Wong CSM, et al. (2023) Life expectancy and years of potential life lost in people with mental disorders: a systematic review and meta-analysis. *EClinicalMedicine* 65: 102294.
- Chaudron LH (2013) Complex challenges in treating depression during pregnancy. *The American Journal of Psychiatry* 170(1): 12-20.
- Chen M, Fitzgerald HM, Madera JJ, et al. (2019) Functional outcome assessment in bipolar disorder: A systematic literature review. *Bipolar Disorders* 21(3): 194-214.
- Chenoweth L, King MT, Jeon Y-H, et al. (2009) Caring for Aged Dementia Care Resident Study (CADRES) of person-centred care, dementia-care mapping, and usual care in dementia: a cluster-randomised trial. *The Lancet Neurology* 8(4): 317-325.
- Chisolm MS and Payne JL (2016) Management of psychotropic drugs during pregnancy. *BMJ* 332: h5918.
- Chu DA, Bryant RA, Gatt JM, et al. (2019) Cumulative childhood interpersonal trauma is associated with reduced cortical differentiation between threat and non-threat faces in posttraumatic stress disorder adults. *Australian & New Zealand Journal of Psychiatry* 53(1): 48-58.
- Cicchetti D and Rogosch FA (1996) Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology* 8(04): 597-600.
- Cioffi I, Ponzio V, Pellegrini M, et al. (2021) The incidence of the refeeding syndrome. A systematic review and meta-analyses of literature. *Clinical Nutrition* 40(6): 3688-3701.
- Clark C (1985) Report of the electro-convulsive therapy review committee. Toronto: Ontario Ministry of Health.
- Clark DM (1986) A cognitive approach to panic. *Behaviour Research and Therapy* 24(4): 461-470.
- Clarkin J, Yeomans F and Kernberg O (2006) *Psychotherapy of Borderline Personality Disorder, Focusing on Object Relations*. Washington, DC.: American Psychiatric Publishing.
- Clarkin JF, Levy KN, Lenzenweger MF, et al. (2007) Evaluating three treatments for borderline personality disorder: A multiwave study. *American Journal of Psychiatry* 164(6): 922-928.
- Coen RF, Robertson DA, Kenny RA, et al. (2016) Strengths and limitations of the MoCA for assessing cognitive functioning: findings from a large representative sample of Irish older adults. *Journal of Geriatric Psychiatry and Neurology* 29(1): 18-24.

- Coleman K, Norris S and Weston A (2009) National Health and Medical Research Council. *NHMRC Additional Levels of Evidence and Grades for Recommendations for Developers of Guidelines: Stage 2 Consultation; Early 2008–End Jun 2009*.
- Colijn MA, Nitta BH and Grossberg GT (2015) Psychosis in later life: a review and update. *Harvard Review of Psychiatry* 23(5): 354-367.
- Corderoy A, Large MM, Ryan C, et al. (2024) Factors associated with involuntary mental healthcare in New South Wales, Australia. *BJPsych Open* 10(2): e59.
- Correll CU, Galling B, Pawar A, et al. (2018) Comparison of Early Intervention Services vs Treatment as Usual for Early-Phase Psychosis: A Systematic Review, Meta-analysis, and Meta-regression. *JAMA Psychiatry* 75(6): 555-565.
- Cox JL, Holden JM and Sagovsky R (1987) Detection of postnatal depression: development of the 10-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry* 150: 782-786.
- Creavin ST, Wisniewski S, Noel-Storr AH, et al. (2016) Mini-Mental State Examination (MMSE) for the detection of dementia in clinically unevaluated people aged 65 and over in community and primary care populations. *Cochrane Database of Systematic Reviews*.(1).
- Crerand CE, Menard W and Phillips KA (2010) Surgical and minimally invasive cosmetic procedures among persons with body dysmorphic disorder. *Annals of Plastic Surgery* 65(1): 11-16.
- Cross SP, Scott JL, Hermens DF, et al. (2018) Variability in clinical outcomes for youths treated for subthreshold severe mental disorders at an early intervention service. *Psychiatric Services* 69(5): 555-561.
- Cummings J (2023) Anti-amyloid monoclonal antibodies are transformative treatments that redefine Alzheimer's disease therapeutics. *Drugs* 83(7): 569-576.
- Cummings JL (1992) Depression and Parkinson's disease: a review. *The American Journal of Psychiatry*.
- Cuthbert BN (2020) The role of RDoC in future classification of mental disorders. *Dialogues in Clinical Neuroscience* 22(1): 81-85.
- D'Zurilla T (1988) Problem-Solving Therapies. In: Dobson KS (ed) *Handbook of Cognitive-Behavioral Therapies*. New York: The Guilford Press, pp.85-135.
- Dadds MR, El Masry Y, Wimalaweera S, et al. (2008) Reduced eye gaze explains "fear blindness" in childhood psychopathic traits. *Journal of the American Academy of Child & Adolescent Psychiatry* 47(4): 455-463.
- Dahlin M, Joneborg N and Runeson B (2005) Stress and depression among medical students: A cross-sectional study. *Medical Education* 39(6): 594-604.
- Dalton SO, Mellekjær L, Thomassen L, et al. (2005) Risk for cancer in a cohort of patients hospitalized for schizophrenia in Denmark, 1969–1993. *Schizophrenia Research* 75(2-3): 315-324.

- Damian AM, Jacobson SA, Hentz JG, et al. (2011) The Montreal Cognitive Assessment and the mini-mental state examination as screening instruments for cognitive impairment: item analyses and threshold scores. *Dementia and Geriatric Cognitive Disorders* 31(2): 126-131.
- Dardas LA, Xu H, Franklin MS, et al. (2023) Cognitive behavioural therapy and medication for treatment of adolescent depression: a network meta-analysis. *Behavioural and Cognitive Psychotherapy* 51(3): 230-245.
- Davies J (2003) *A manual of mental health care in general practice*. Commonwealth Department of Health and Ageing, Mental Health and Suicide, Commonwealth of Australia.
- Davis A, McMaster P, Christie DC, et al. (2023) Psychiatric comorbidities of substance use disorders: does dual diagnosis predict inpatient detoxification treatment outcomes? *International Journal of Mental Health and Addiction* 21(6): 3785-3799.
- Davis DR, Kurti AN, Skelly JM, et al. (2016) A review of the literature on contingency management in the treatment of substance use disorders, 2009–2014. *Preventive Medicine* 92: 36-46.
- De Gucht V and Heiser W (2003) Alexithymia and somatisation. A quantitative review of the literature. *Journal of Psychosomatic Research* 54: 425-434.
- De J and Wand AP (2015) Delirium screening: a systematic review of delirium screening tools in hospitalized patients. *The Gerontologist* 55(6): 1079-1099.
- De Leo D, Draper BM, Snowden J, et al. (2013) Contacts with health professionals before suicide: missed opportunities for prevention? *Comprehensive Psychiatry* 54(7): 1117-1123.
- De Leo D, Scocco P and Meneghel G (1999) Pharmacological and psychotherapeutic treatment of personality disorders in the elderly. *International Psychogeriatrics* 11(2): 191-206.
- de Monticelli R (2019) Edmund Husserl. Oxford University Press.
- Delahanty DL, Raimonde AJ and Spoonster E (2000) Initial posttraumatic urinary cortisol levels predict subsequent PTSD symptoms in motor vehicle accident victims. *Biological Psychiatry* 48(9): 940-947.
- Deloitte Access Economics (2011) Re-awakening Australia. The economic cost of sleep disorders in Australia, 2010. (accessed 27 June 2016).
- Dennis C and Hodnett ED (2009) Psychosocial and psychological interventions for treating postpartum depression [Systematic Review]. *Cochrane Database of Systematic Reviews*. 4.
- Dennis CL, Falah-Hassani K and Shiri R (2017) Prevalence of antenatal and postnatal anxiety: systematic review and meta-analysis. *Br J Psychiatry* 210(5): 315-323.
- Department of Health and Aged Care.(2013) National practice standards for the mental health workforce 2013.

- Department of the Prime Minister and Cabinet (2017) National Strategic Framework for Aboriginal and Torres Strait Islander peoples' mental health and social and emotional wellbeing 2017–2023. Australian Government.
- deRoos-Cassini TA, Mancini AD, Rusch MD, et al. (2010) Psychopathology and resilience following traumatic injury: a latent growth mixture model analysis. *Rehabilitation Psychology* 55(1): 1.
- Di Forti M, Iyegbe C, Sallis H, et al. (2012) Confirmation that the AKT1 (rs2494732) Genotype Influences the Risk of Psychosis in Cannabis Users. *Biological Psychiatry* 72: 811-816.
- DiClemente CC, Corno CM, Graydon MM, et al. (2017) Motivational interviewing, enhancement, and brief interventions over the last decade: A review of reviews of efficacy and effectiveness. *Psychology of Addictive Behaviors* 31(8): 862.
- Didden R, VanDerNagel J, Delforterie M, et al. (2020) Substance use disorders in people with intellectual disability. *Current Opinoin Psychiatry* 33(2): 124-129.
- Diefenbach GJ and Goethe J (2006) Clinical interventions for late-life anxious depression. *Clinical Interventions in Aging* 1(1): 41-50.
- Diesfeld K (2003) Insight: unpacking the concept in mental health law. *Psychiatry, Psychology and Law* 10(1): 63-70.
- Dillard-Wright D (2007) Sympathy and the Non-human: Max Scheler's Phenomenology of Interrelation. *Indo-Pacific Journal of Phenomenology* 7(2): 1-9.
- Dimidjian S, Hollon SD, Dobson KS, et al. (2006) Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology* 74(4): 658.
- Dobson KS, Hollon SD, Dimidjian S, et al. (2008) Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the prevention of relapse and recurrence in major depression. *Journal of Consulting and Clinical Psychology* 76(3): 468.
- Dombrovski AY and Mulsant BH (2007) ECT: the preferred treatment for severe depression in late life. *International Psychogeriatrics* 19(1): 10-14.
- Dörr O (2013) Hermeneutical and Dialectical Thinking in Psychiatry and the Contribution of Karl Jaspers. New York, NY: Springer New York, pp.19-32.
- Draaisma D (2000) *Metaphors of Memory: A history of ideas about the mind*. Cambridge: Cambridge University Press.
- Drayer RA, Mulsant BH, Lenze EJ, et al. (2005) Somatic symptoms of depression in elderly patients with medical comorbidities. *International Journal of*

- Geriatric Psychiatry: A journal of the psychiatry of late life and allied sciences* 20(10): 973-982.
- Dubois B, Slachevsky A, Litvan I, et al. (2000) The FAB: a frontal assessment battery at bedside. *Neurology* 55(11): 1621-1626.
- Dudgeon P, Cox A, Walker R, et al. (2014) Voices of the peoples: The national empowerment project research report 2015. Perth: National Empowerment Project, University of Western Australia.
- Duffy M, Gillespie K and Clark DM (2007) Post-traumatic stress disorder in the context of terrorism and other civil conflict in Northern Ireland: randomised controlled trial. *BMJ* 334(7604): 1147.
- Duncan LE, Ratanatharathorn A, Aiello AE, et al. (2018) Largest GWAS of PTSD (N= 20 070) yields genetic overlap with schizophrenia and sex differences in heritability. *Molecular Psychiatry* 23(3): 666-673.
- Dunmore E, Clark DM and Ehlers A (1999) Cognitive factors involved in the onset and maintenance of posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy* 37(9): 809-829.
- Dunmore E, Clark DM and Ehlers A (2001) A prospective investigation of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy* 39(9): 1063-1084.
- Dyrbye LN, Massie FS, Eacker A, et al. (2010) Relationship between burnout and professional conduct and attitudes among US medical students. *JAMA* 304(11): 1173-1180.
- Early Psychosis Guidelines Writing Group and EPPIC National Support Program (2016) Australian Clinical Guidelines for Early Psychosis. 2nd ed. Melbourne: Orygen, The National Centre of Excellence in Youth Mental Health.
- Editor (2023) Top 10 drugs 2022-23. *Australian Prescriber* 46(4): 93.
- Eisenberg L (1986) Mindlessness and brainlessness in psychiatry. *British Journal of Psychiatry* 148 497-508.
- Elias A, Thomas N and Sackeim HA (2021) Electroconvulsive Therapy in Mania: A Review of 80 Years of Clinical Experience. *American Journal of Psychiatry* 178(3): 229-239.
- Engel GL (1977) The need for a new medical model: a challenge for biomedicine. *Science* 196(4286): 129-136.
- Engels GI, Duijsens IJ, Haringsma R, et al. (2003) Personality disorders in the elderly compared to four younger age groups: a cross-sectional study of community residents and mental health patients. *Journal of Personality Disorders* 17(5): 447-459.
- Enoch D and Ball H (2001) *Uncommon Psychiatric Syndromes*. Oxford University Press.
- Erikson EH (1993) *Childhood and society*. WW Norton & Company.

- Fagiolini A, Chengappa KN, Soreca I, et al. (2008) Bipolar disorder and the metabolic syndrome: causal factors, psychiatric outcomes and economic burden. *CNS Drugs* 22(8): 655-669.
- Fairburn CG, Bailey-Straebl S, Basden S, et al. (2015) A transdiagnostic comparison of enhanced cognitive behaviour therapy (CBT-E) and interpersonal psychotherapy in the treatment of eating disorders. *Behaviour Research and Therapy* 70: 64-71.
- Fanelli D (2009) How many scientists fabricate and falsify research? A systematic review and meta-analysis of survey data. *PloS one* 4(5): e5738.
- Fang A, Matheny NL and Wilhelm S (2014) Body Dysmorphic Disorder. *Psychiatric Clinics of North America* 37(3): 287-300.
- Fässberg MM, Cheung G, Canetto SS, et al. (2016) A systematic review of physical illness, functional disability, and suicidal behaviour among older adults. *Aging & Mental Health* 20(2): 166-194.
- Feinstein A (2011) Conversion disorder: advances in our understanding. *Canadian Medical Association Journal* 183(8): 915-920.
- Felitti VJ, Anda RF, Nordenberg D, et al. (2019) Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The adverse childhood experiences (ACE) study. *American Journal of Preventive Medicine*.
- Fertleman C and Carroll W (2013) Protecting students and promoting resilience. British Medical Journal Publishing Group.
- Fink M (1993) Who should get ECT? The Clinical Science of Electroconvulsive Therapy. Washington DC: American Psychiatric Press.
- Firth J, Siddiqi N, Koyanagi A, et al. (2019) The Lancet Psychiatry Commission: a blueprint for protecting physical health in people with mental illness. *The Lancet Psychiatry* 6(8): 675-712.
- Fitzgerald PB and Daskalakis ZJ (2013) *Repetitive transcranial magnetic stimulation treatment for depressive disorders: a practical guide*. Springer Science & Business Media.
- Foa EB, Ehlers A, Clark DM, et al. (1999) The posttraumatic cognitions inventory (PTCI): Development and validation. *Psychological Assessment* 11(3): 303.
- Foa EB, Liebowitz MR, Kozak MJ, et al. (2007) Randomized, Placebo-Controlled Trial of Exposure and Ritual Prevention, Clomipramine, and Their Combination in the Treatment of Obsessive-Compulsive Disorder. *FOCUS* 5(3): 368-380.
- Foa EB, Rothbaum BO, Riggs DS, et al. (1991) Treatment of posttraumatic stress disorder in rape victims: a comparison between cognitive-behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology* 59(5): 715.

- Foa EB, Steketee G and Rothbaum BO (1989) Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy* 20(2): 155-176.
- Folstein M, Folstein S and McHugh P (1975a) Mini-Mental State Examination (MMS, MMSE)[Database record]. *PsycTESTS Dataset* 10.
- Folstein MF, Folstein SE and McHugh PR (1975b) “Mini-mental state”: a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research* 12(3): 189-198.
- Fong TG, Tulebaev SR and Inouye SK (2009) Delirium in elderly adults: diagnosis, prevention and treatment. *Nature Reviews Neurology* 5(4): 210-220.
- Forbes D, Fletcher S, Parslow R, et al. (2011) Trauma at the hands of another: Longitudinal study of differences in the posttraumatic stress disorder symptom profile following interpersonal compared with noninterpersonal trauma. *The Journal of Clinical Psychiatry* 72(3): 21208.
- Forbes D, Lockwood E, Phelps A, et al. (2013) Trauma at the hands of another: Distinguishing PTSD patterns following intimate and nonintimate interpersonal and noninterpersonal trauma in a nationally representative sample. *The Journal of Clinical Psychiatry* 74(2): 21205.
- Fornaro M, Solmi M, Perna G, et al. (2016) Lisdexamfetamine in the treatment of moderate-to-severe binge eating disorder in adults: systematic review and exploratory meta-analysis of publicly available placebo-controlled, randomized clinical trials. *Neuropsychiatric Disease and Treatment*. 1827-1836.
- Foulds G and Bedford A (1975) Hierarchy of classes of personal illness. *Psychological Medicine* 5(2): 181-192.
- Frank JD and Frank JB (1993) *Persuasion and healing: A comparative study of psychotherapy*. JHU Press.
- Freeston MH, Rhéaume J, Letarte H, et al. (1994) Why do people worry? *Personality and Individual Differences* 17(6): 791-802.
- Freud S (1958) The disposition to obsessional neurosis, a contribution to the problem of the choice of neurosis. *The Standard Edition of the Complete Psychological Works of Sigmund Freud, Volume XII (1911-1913): The Case of Schreber, Papers on Technique and Other Works*. pp.311-326.
- Freud S (1997) *Dora: An analysis of a case of hysteria*. Simon and Schuster.
- Friedhoff AJ and Winkle EV (1962) Isolation and characterization of a compound from the urine of schizophrenics. *Nature* 194(4831): 897-898.
- Frings MS (1974) *Max Scheler (1874-1928) : centennial essays*. The Hague: Springer.
- Frings MS and Scheler M (2012) *Person and Self-Value: Three Essays*. Springer.
- Fritz K, Russell AMT, Allwang C, et al. (2017) Is a delay in the diagnosis of bipolar disorder inevitable? *Bipolar Disorders* 19(5): 396-400.
- Frommberger UH, Stieglitz R-D, Nyberg E, et al. (1998) Prediction of posttraumatic stress disorder by immediate reactions to trauma: a

- prospective study in road traffic accident victims. *European Archives of Psychiatry and Clinical Neuroscience* 248: 316-321.
- Galatzer-Levy IR, Andero R, Sawamura T, et al. (2017) A cross species study of heterogeneity in fear extinction learning in relation to FKBP5 variation and expression: Implications for the acute treatment of posttraumatic stress disorder. *Neuropharmacology* 116: 188-195.
- Galatzer-Levy IR, Huang SH and Bonanno GA (2018) Trajectories of resilience and dysfunction following potential trauma: A review and statistical evaluation. *Clinical Psychology Review* 63: 41-55.
- Galbally M, Snellen M, Walker S, et al. (2010) Management of antipsychotic and mood stabilizer medication in pregnancy: recommendations for antenatal care. *Australian & New Zealand Journal of Psychiatry* 44(2): 99-108.
- Galletly C, Castle D, Dark F, et al. (2016) Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the management of schizophrenia and related disorders. *Australian & New Zealand Journal of Psychiatry* 50(5): 410-472.
- Gaughran F, Dineen S, Dineen M, et al. (1997) Stress in medical students. *Irish Medical Journal* 90(5): 184-185.
- GBD 2019 Mental Disorders Collaborators (2022) Global, regional, and national burden of 12 mental disorders in 204 countries and territories, 1990-2013;2019: a systematic analysis for the Global Burden of Disease Study 2019. *The Lancet Psychiatry* 9(2): 137-150.
- Gee G, Dudgeon P, Schultz C, et al. (2014) Social and emotional wellbeing and mental health: an Aboriginal perspective. *Working together: Aboriginal and Torres Strait Islander mental health and wellbeing principles and practice*. 55-68.
- Geiger-Brown JM, Rogers VE, Liu W, et al. (2015) Cognitive behavioral therapy in persons with comorbid insomnia: A meta-analysis. *Sleep Medicine Reviews* 23: 54-67.
- Gelpin E, Bonne O, Peri T, et al. (1996) Treatment of recent trauma survivors with benzodiazepines: a prospective study. *The Journal of Clinical Psychiatry* 57(9): 390-394.
- Gerrits RG, Jansen T, Mulyanto J, et al. (2019) Occurrence and nature of questionable research practices in the reporting of messages and conclusions in international scientific Health Services Research publications: a structured assessment of publications authored by researchers in the Netherlands. *BMJ Open* 9(5): e027903.
- Ghaemi SN, Sachs GS, M. Chiou A, et al. (1999) Is bipolar disorder still underdiagnosed? Are antidepressants overutilized? *Journal of Affective Disorders* 52(1): 135-144.
- Gielen N, Havermans R, Tekelenburg M, et al. (2012) Prevalence of post-traumatic stress disorder among patients with substance use disorder: it is

- higher than clinicians think it is. *European Journal of Psychotraumatology* 3(1): 17734.
- Ginley MK, Pfund RA, Rash CJ, et al. (2021) Long-term efficacy of contingency management treatment based on objective indicators of abstinence from illicit substance use up to 1 year following treatment: A meta-analysis. *Journal of Consulting and Clinical Psychology* 89(1): 58.
- Glover-Wright C, Coupe K, Campbell AC, et al. (2023) Health outcomes and service use patterns associated with co-located outpatient mental health care and alcohol and other drug specialist treatment: A systematic review. *Drug and Alcohol Review* 42(5): 1195-1219.
- Goodman WK, Price LH, Rasmussen SA, et al. (1989) The Yale-Brown Obsessive Compulsive Scale. I. Development, use, and reliability. *Archives of General Psychiatry* 46(11): 1006-1011.
- Gorczynski P, Firth J, Stubbs B, et al. (2017) Are people with schizophrenia adherent to diabetes medication? A comparative meta-analysis. *Psychiatry Research* 250: 17-24.
- Gordovez FJA and McMahon FJ (2020) The genetics of bipolar disorder. *Molecular Psychiatry* 25(3): 544-559.
- Gortner ET, Gollan JK, Dobson KS, et al. (1998) Cognitive-behavioral treatment for depression: Relapse prevention. *Journal of consulting and clinical psychology* 66: 377-384.
- Graham HL (2004) *Cognitive-behavioural integrated treatment (C-BIT): a treatment manual for substance misuse in people with severe mental health problems*. John Wiley & Sons.
- Granholm E, Holden J, Link PC, et al. (2013) Randomized controlled trial of cognitive behavioral social skills training for older consumers with schizophrenia: defeatist performance attitudes and functional outcome. *The American Journal of Geriatric Psychiatry* 21(3): 251-262.
- Gray R, Bressington D, Ivanecka A, et al. (2016) Is adherence therapy an effective adjunct treatment for patients with schizophrenia spectrum disorders? A systematic review and meta-analysis. *BMC Psychiatry* 16(1): 90.
- Grigoriadis S, Vonderporten EH, Mamisashvili L, et al. (2013) Antidepressant exposure during pregnancy and congenital malformations: is there an association? a systematic review and meta-analysis of the best evidence. *Journal of Clinical Psychiatry* 74(4): e293-308.
- Grilo CM, Stout RL, Markowitz JC, et al. (2010) Personality disorders predict relapse after remission from an episode of major depressive disorder: a 6-year prospective study. *Journal of Clinical Psychiatry* 71(12): 1629-1635.
- Grinshpoon A, Barchana M, Ponizovsky A, et al. (2005) Cancer in schizophrenia: is the risk higher or lower? *Schizophrenia Research* 73(2-3): 333-341.
- Gunderson J, Masland S and Choi-Kain L (2018) Good psychiatric management: a review. *Current Opinion Psychology* 21: 127-131.

- Gupta A, Harris S and Naina H (2015) To sit or stand during the medical interview: a poll of caucasian patients. *Medical Practice Management* 31(3): 110-112.
- Gurrera RJ, Simpson JC and Tsuang MT (2007) Meta-analytic evidence of systematic bias in estimates of neuroleptic malignant syndrome incidence. *Comprehensive Psychiatry* 48(2): 205-211.
- Guthrie E, Moorey J, Margison F, et al. (1999) Cost-effectiveness of brief psychodynamic-interpersonal therapy in high utilizers of psychiatric services. *Archives of General Psychiatry* 56: 519-526.
- Haber PS and Riordan BC (2021) *Guidelines for the Treatment of Alcohol Problems (4th ed.)*. Specialty of Addiction Medicine, Faculty of Medicine and Health, The University of Sydney.
<https://alcoholtreatmentguidelines.com.au/pdf/guidelines-for-the-treatment-of-alcohol-problems.pdf>
- Hall J, Caleo S, Stevenson J, et al. (1999) *Economic analysis of psychotherapy for borderline personality disorder patients*. Citeseer.
- Halliday MAK (1975) Learning how to mean. *Foundations of Language Development*. Elsevier, pp.239-265.
- Hamilton BA (2008) Assessment of at-risk mental states for psychosis in young aboriginal and non-aboriginal people using the CAARMS. Unpublished Ph.D. Thesis: University of Sydney.
- Hammarberg K, Kirkman M and De Lacey S (2016) Qualitative research methods: when to use them and how to judge them. *Human Reproduction* 31(3): 498-501.
- Hans-Christoph S (2002) The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry* 159(8): 1284-1293.
- Hare DL, Toukhsati SR, Johansson P, et al. (2014) Depression and cardiovascular disease: a clinical review. *European heart journal* 35(21): 1365-1372.
- Harkness LL (1993) Transgenerational transmission of war-related trauma. *International Handbook of Traumatic Stress Syndromes*. Springer, pp.635-643.
- Harrison BJ, Soriano-Mas C, Pujol J, et al. (2009) Altered corticostriatal functional connectivity in obsessive-compulsive disorder. *Archives of General Psychiatry* 66(11): 1189-1200.
- Harrison P (1999) The neuropathology of schizophrenia. A critical review of the data and their interpretation. *Brain* 122: 593-624.
- Hartz SM, Horton AC, Oehlert M, et al. (2017) Association between substance use disorder and polygenic liability to schizophrenia. *Biological Psychiatry* 82(10): 709-715.
- Hawkins N, Richards PS, Granley HM, et al. (2004) The impact of exposure to the thin-ideal media image on women. *Eating Disorders* 12(1): 35-50.
- Hay P, Chinn D, Forbes D, et al. (2014) Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of

- eating disorders. *Australian & New Zealand Journal of Psychiatry* 48(11): 977-1008.
- Hay PJ and Claudino AM (2012) Clinical psychopharmacology of eating disorders: a research update. *International Journal of Neuropsychopharmacology* 15(2): 209-222.
- Hazell P (2011) Depression in children and adolescents. *BMJ Clinical Evidence* 2011.
- Hazell P (2015) Depression in children and adolescents: complementary therapies. *BMJ Clin Evid* 2015.
- Hazell P and Jairam R (2012) Acute treatment of mania in children and adolescents. *Current Opinion Psychiatry* 25(4): 264-270.
- Hazell PL and Stuart JE (2003) A randomized controlled trial of clonidine added to psychostimulant medication for hyperactive and aggressive children. *Journal of the American Academy of Child & Adolescent Psychiatry* 42(8): 886-894.
- Heald A (2010) Physical health in schizophrenia: a challenge for antipsychotic therapy. *European Psychiatry: the Journal of the Association of European Psychiatrists* 25 Suppl 2: S6-11.
- Health NCCfM (2005) Post-traumatic stress disorder: The management of PTSD in adults and children in primary and secondary care.
- Health NCCfM (2009) The NICE Guideline on Diagnosis and Management of ADHD in Children, Young People and Adults. London: The British Psychological Society and The Royal College of Psychiatrists.
- Heckers S and Walther S (2023) Catatonia. *New England Journal of Medicine* 389(19): 1797-1802.
- Heinrichs RW and Zakzanis KK (1998) Neurocognitive deficit in schizophrenia: a quantitative review of the evidence. *Neuropsychology* 12: 426-445.
- Helfand BK, D'Aquila ML, Tabloski P, et al. (2021) Detecting delirium: a systematic review of identification instruments for non-ICU settings. *Journal of the American Geriatrics Society* 69(2): 547-555.
- Henderson M, Brooks SK, Del Busso L, et al. (2012) Shame! Self-stigmatisation as an obstacle to sick doctors returning to work: a qualitative study. *BMJ Open* 2(5): e001776.
- Hendrickson RC and Raskind MA (2016) Noradrenergic dysregulation in the pathophysiology of PTSD. *Experimental Neurology* 284: 181-195.
- Hendriks SM, Spijker J, Licht CM, et al. (2016) Long-term disability in anxiety disorders. *BMC Psychiatry* 16: 1-8.
- Hengartner MP (2020) Is there a genuine placebo effect in acute depression treatments? A reassessment of regression to the mean and spontaneous remission. *BMJ Evidence - Based Medicine* 25(2): 46.
- Herman JL (2015) *Trauma and recovery: The aftermath of violence--from domestic abuse to political terror*. Hachette uK.

- Hertsgaard L, Gunnar M, Erickson MF, et al. (1995) Adrenocortical responses to the strange situation in infants with disorganized/disoriented attachment relationships. *Child Development* 66(4): 1100-1106.
- Hesse E and Main M (2000) Disorganized infant, child, and adult attachment: Collapse in behavioral and attentional strategies. *Journal of the American Psychoanalytic Association* 48(4): 1097-1127.
- Hillman DR and Lack LC (2013) Public health implications of sleep loss: the community burden. *Medical Journal of Australia* 199(8, Supplement): S7-S10.
- Hirschfeld RM (2000) History and evolution of the monoamine hypothesis of depression. *Journal of Clinical Psychiatry* 61(6): 4-6.
- Hjorthøj CD, Stürup AEMD, McGrath JJP, et al. (2017) Years of potential life lost and life expectancy in schizophrenia: a systematic review and meta-analysis. *The Lancet Psychiatry* 4(4): 295-301.
- Hobson R (1985) *Forms of Feeling.*, London. Tavistock Publications.
- Hoffmann TC, Légaré F, Simmons MB, et al. (2014) Shared decision making: what do clinicians need to know and why should they bother? *Medical Journal of Australia* 201(1): 35-39.
- Hollander E (1993) *Obsessive-compulsive-related disorders*. Amer Psychiatric Pub Inc.
- Howard LM, Molyneaux E, Dennis CL, et al. (2014) Non-psychotic mental disorders in the perinatal period. *The Lancet* 384(9956): 1775-1788.
- Howes OD, McCutcheon R, Owen MJ, et al. (2017) The Role of Genes, Stress, and Dopamine in the Development of Schizophrenia. *Biological Psychiatry* 81(1): 9-20.
- Hsieh S, Schubert S, Hoon C, et al. (2013) Validation of the Addenbrooke's Cognitive Examination III in frontotemporal dementia and Alzheimer's disease. *Dementia and Geriatric Cognitive Disorders* 36(3-4): 242-250.
- Hudson JI, Javaras KN and Pope Jr HG (2020) The challenges of metabolic syndrome in eating disorders. *Psychiatric Annals* 50(8): 346-350.
- Huhn D and Nikendei C (2018) International students—support and integration initiatives at Medical Faculties in Germany. *GMS Journal for Medical Education* 35(5).
- Huhn M, Nikolakopoulou A, Schneider-Thoma J, et al. (2019) Comparative efficacy and tolerability of 32 oral antipsychotics for the acute treatment of adults with multi-episode schizophrenia: a systematic review and network meta-analysis. *The Lancet*, 394(10202), 939-951.
- Huhn M, Tardy M, Spineli LM, et al. (2014) Efficacy of Pharmacotherapy and Psychotherapy for Adult Psychiatric Disorders: A Systematic Overview of Meta-analyses. *JAMA Psychiatry* 71(6): 706-715.
- Hunot V, Moore TH, Caldwell DM, et al. (2013) 'Third wave' cognitive and behavioural therapies versus other psychological therapies for depression. *Cochrane Database of Systematic Reviews*.(10): Cd008704.

- Hunt GE, Malhi GS, Lai HMX, et al. (2020) Prevalence of comorbid substance use in major depressive disorder in community and clinical settings, 1990–2019: Systematic review and meta-analysis. *Journal of Affective Disorders* 266: 288-304.
- Hunter E (1998) Ways of seeing: changing directions in the health of Indigenous populations. *Clinical Child Psychology and Psychiatry* 3(4): 519-530.
- Husain MM, Rush AJ, Sackeim HA, et al. (2005) Age-related characteristics of depression: a preliminary STAR* D report. *The American Journal of Geriatric Psychiatry* 13(10): 852-860.
- Husebo BS, Ballard C, Sandvik R, et al. (2011) Efficacy of treating pain to reduce behavioural disturbances in residents of nursing homes with dementia: cluster randomised clinical trial. *BMJ* 343.
- Ilchef R (1997) Neuroleptic-induced laryngeal dystonia can mimic anaphylaxis. *Australian & New Zealand Journal of Psychiatry* 31(6): 877-879.
- Inouye SK (2006) Delirium in older persons. *New England Journal of Medicine* 354(11): 1157-1165.
- Inouye SK and Charpentier PA (1996) Precipitating factors for delirium in hospitalized elderly persons: predictive model and interrelationship with baseline vulnerability. *JAMA* 275(11): 852-857.
- Insel T, Cuthbert B, Garvey M, et al. (2010) Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *American Journal of Psychiatry* 167(7): 748-751.
- Insel TR and Quirion R (2005) Psychiatry as a clinical neuroscience discipline. *JAMA* 294(17): 2221-2224.
- Inskip HM, Harris EC and Barraclough B (1998) Lifetime risk of suicide for affective disorder, alcoholism and schizophrenia. *British Journal of Psychiatry* 172: 35-37.
- IsHak W, Nikraves R, Lederer S, et al. (2013) Burnout in medical students: a systematic review. *The clinical teacher* 10(4): 242-245.
- Jääskeläinen E, Juola P, Hirvonen N, et al. (2012) A Systematic Review and Meta-Analysis of Recovery in Schizophrenia. *Schizophrenia Bulletin*. 39(6), 1296-130.
- Jablensky A (2015) Schneider, Kurt (1887–1967).
- Jablensky A and Lawrence D (2001) Schizophrenia and cancer: is there a need to invoke a protective gene? *Archives of General Psychiatry* 58(6): 579-580.
- Jacka FN, Mykletun A and Berk M (2012) Moving towards a population health approach to the primary prevention of common mental disorders. *BMC Medicine* 10: 149.
- Jacka FN, Ystrom E, Brantsaeter AL, et al. (2013) Maternal and early postnatal nutrition and mental health of offspring by age 5 years: a prospective cohort study. *Journal of the American Academy of Child & Adolescent Psychiatry* 52(10): 1038-1047.

- Jacobi F, Höfler M, Siegert J, et al. (2014) Twelve-month prevalence, comorbidity and correlates of mental disorders in Germany: the Mental Health Module of the German Health Interview and Examination Survey for Adults (DEGS1-MH). *International Journal of Methods in Psychiatric Research* 23(3): 304-319.
- Jacobson NS, Dobson KS, Truax PA, et al. (1996) A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology* 64: 295-304.
- James AC, Reardon T, Soler A, et al. (2020) Cognitive behavioural therapy for anxiety disorders in children and adolescents. *Cochrane Database of Systematic Reviews*.(11).
- Jannati A, Oberman LM, Rotenberg A, et al. (2023) Assessing the mechanisms of brain plasticity by transcranial magnetic stimulation. *Neuropsychopharmacology* 48(1): 191-208.
- Jansson L (2019) First-Rank Symptoms of Schizophrenia. Oxford University Press.
- Jaspers K (1963) *General psychopathology*. Manchester: Manchester University Press.
- Jaspers K (1968) The Phenomenological Approach in Psychopathology. *British Journal of Psychiatry* 114(516): 1313-1323.
- Jehel L, Paterniti S, Brunet A, et al. (2003) Prediction of the occurrence and intensity of post-traumatic stress disorder in victims 32 months after bomb attack. *European Psychiatry* 18(4): 172-176.
- Jerónimo J, Queirós T, Cheniaux E, et al. (2018) Formal thought disorders—historical roots. *Frontiers in Psychiatry* 9: 572.
- John WS and Wu L-T (2020) Chronic non-cancer pain among adults with substance use disorders: prevalence, characteristics, and association with opioid overdose and healthcare utilization. *Drug and Alcohol Dependence* 209: 107902.
- Jones E (2019) *The life and work of Sigmund Freud*. Plunkett Lake Press.
- Jones I, Chandra PS, Dazzan P, et al. (2014) Bipolar disorder, affective psychosis, and schizophrenia in pregnancy and the post-partum period. *The Lancet* 384(9956): 1789-1799.
- Jones I and Craddock N (2001) Familiality of the puerperal trigger in bipolar disorder: results of a family study. *American Journal of Psychiatry* 158(6): 913-917.
- Josephson AM (2007) Practice parameter for the assessment of the family. *Journal of the American Academy of Child & Adolescent Psychiatry* 46(7): 922-937.
- Judd F, Ahmed R, Blashki G, et al. (2021) eTherapeutic Guidelines: Psychotropic. Melbourne, Australia: Therapeutic Guidelines.
- Judd LL and Akiskal HS (2003) Depressive episodes and symptoms dominate the longitudinal course of bipolar disorder. *Current Psychiatry Reports* 5(6): 417-418.

- Kagan J, Reznick JS, Clarke C, et al. (1984) Behavioral inhibition to the unfamiliar. *Child Development*. 2212-2225.
- Kalish VB, Gillham JE and Unwin BK (2014) Delirium in older persons: evaluation and management. *American Family Physician* 90(3): 150-158.
- Kang B, Xu H and McConnell ES (2019) Neurocognitive and psychiatric comorbidities of posttraumatic stress disorder among older veterans: a systematic review. *International Journal of Geriatric Psychiatry* 34(4): 522-538.
- Karno M, Golding JM, Sorenson SB, et al. (1988) The epidemiology of obsessive-compulsive disorder in 5 United-States communities. *Archives of General Psychiatry* 45(12): 1094-1099.
- Karstoft K-I, Galatzer-Levy IR, Statnikov A, et al. (2015) Bridging a translational gap: using machine learning to improve the prediction of PTSD. *BMC Psychiatry* 15: 1-7.
- Katzman MA, Bleau P, Blier P, et al. (2014) Canadian clinical practice guidelines for the management of anxiety, posttraumatic stress and obsessive-compulsive disorders. *BMC Psychiatry* 14(1): 1-83.
- Kelly BD (2019) Variations in involuntary hospitalisation across countries. *The Lancet Psychiatry* 6(5): 361-362.
- Kelly JF, Humphreys K and Ferri M (2020) Alcoholics Anonymous and other 12-step programs for alcohol use disorder. *Cochrane Database of Systematic Reviews* 3(3): Cd012880.
- Kendall J (2003) Designing a research project: randomised controlled trials and their principles. *Emergency Medicine Journal* 20(2): 164-168.
- Kendell RE (1975) Psychiatric diagnosis in Britain and the United States. *British Journal of Psychiatry Spec No 9*: 453-461.
- Kendell RE, Wainwright S, Hailey A, et al. (1976) The influence of childbirth on psychiatric morbidity. *Psychological Medicine* 6(2): 297-302.
- Kendler KS, Gardner CO, Annas P, et al. (2008) A longitudinal twin study of fears from middle childhood to early adulthood: evidence for a developmentally dynamic genome. *Archives of General Psychiatry* 65(4): 421-429.
- Kendler KS, McGuire M, Gruenberg AM, et al. (1993) The Roscommon Family Study. III. Schizophrenia-related personality disorders in relatives. *Archives of General Psychiatry* 50(10): 781-788.
- Kendler KS, Neale MC, Kessler RC, et al. (1992) Major depression and generalized anxiety disorder: same genes, (partly) different environments? *Archives of General Psychiatry* 49(9): 716-722.
- Kendrick T, Simons L, Mynors-Wallis L, et al. (2005) A trial of problem-solving by community mental health nurses for anxiety, depression and life difficulties among general practice patients. The CPN-GP study. *Health Technology Assessment* 9(37): 1-+.

- Kennedy H (1969) Young Children in Brief Separation. By James and Joyce Robertson. *British Journal of Medical Psychology* 42(2): 191-193.
- Keramatian K, Chakrabarty T, Saraf G, et al. (2021) Grey matter abnormalities in first-episode mania: A systematic review and meta-analysis of voxel-based morphometry studies. *Bipolar Disorders* 23(3): 228-240.
- Keramatian K, Pinto JV, Schaffer A, et al. (2022) Clinical and demographic factors associated with delayed diagnosis of bipolar disorder: Data from Health Outcomes and Patient Evaluations in Bipolar Disorder (HOPE-BD) study. *Journal of Affective Disorders* 296: 506-513.
- Kessler RC, Berglund P, Demler O, et al. (2005) Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry* 62(6): 593-602.
- Kessler RC, Sonnega A, Bromet E, et al. (2013) Posttraumatic stress disorder in the National Comorbidity Survey. *Fear and Anxiety*. Routledge, pp.22-34.
- Kim-Cohen J, Caspi A, Moffitt TE, et al. (2003) Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. *Archives of General Psychiatry* 60(7): 709-717.
- Kim JJ, Gharpure A, Teng J, et al. (2020) Shared structural mechanisms of general anaesthetics and benzodiazepines. *Nature* 585(7824): 303-308.
- Kini V and Ho PM (2018) Interventions to Improve Medication Adherence: A Review. *JAMA* 320(23): 2461-2473.
- Kirmayer LJ, Robbins JM, Dworkind M, et al. (1993) Somatization and the recognition of depression and anxiety in primary care. *American Journal of Psychiatry* 150: 734-741.
- Kisely S, McMahon L and Siskind D (2023) Benefits following community treatment orders have an inverse relationship with rates of use: meta-analysis and meta-regression. *BJPsych Open* 9(3): e68.
- Kisely S, Yu D, Maehashi S, et al. (2021) A systematic review and meta-analysis of predictors and outcomes of community treatment orders in Australia and New Zealand. *Australian & New Zealand Journal of Psychiatry* 55(7): 650-665.
- Kitayama N, Quinn S and Bremner JD (2006) Smaller volume of anterior cingulate cortex in abuse-related posttraumatic stress disorder. *Journal of Affective Disorders* 90(2-3): 171-174.
- Kleim B, Ehlers A and Glucksman E (2007) Early predictors of chronic post-traumatic stress disorder in assault survivors. *Psychological Medicine* 37(10): 1457-1467.
- Kleim B, Grey N, Wild J, et al. (2013) Cognitive change predicts symptom reduction with cognitive therapy for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology* 81(3): 383.
- Klein DA, Goldenring JM and Adelman WP (2014) HEEADSSS 3.0: the psychosocial interview for adolescents updated for a new century fueled

by media. *Contemporary Paediatrics*. Retrieved 15th October 2024, from <https://www.contemporarypediatrics.com/view/heedadsss-30-psychosocial-interview-adolescents-updated-new-century-fueled-media>

- Klein M (1946) Notes on some schizoid mechanisms. *The International Journal of Psycho-analysis* 27: 99.
- Knox J (2010) *Self-Agency in Psychotherapy: Attachment, Autonomy, and Intimacy* (Norton Series on Interpersonal Neurobiology). WW Norton & Company.
- Koblenzer CS (2000) Dermatitis artefacta. Clinical features and approaches to treatment. *American Journal of Clinical Dermatology* 1(1): 47-55.
- Köck P and Walter M (2018) Personality disorder and substance use disorder—An update. *Mental Health & Prevention* 12: 82-89.
- Koenen KC, Fu QJ, Ertel K, et al. (2008) Common genetic liability to major depression and posttraumatic stress disorder in men. *Journal of Affective Disorders* 105(1-3): 109-115.
- Koenen KC, Moffitt TE, Poulton R, et al. (2007) Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort. *Psychological Medicine* 37(2): 181-192.
- Koh W, Kwak H, Cheong E, et al. (2023) GABA tone regulation and its cognitive functions in the brain. *Nature Reviews Neuroscience* 24(9): 523-539.
- Korner A (2020) *Communicative exchange, psychotherapy and the resonant self: Roads to realization*. Routledge.
- Korner A, Benditt N, Ptak U, et al. (2010) Formulation, conversation and therapeutic engagement. *Australasian Psychiatry* 18(3): 214-220.
- Kornør H, Winje D, Ekeberg Ø, et al. (2008) Early trauma-focused cognitive-behavioural therapy to prevent chronic post-traumatic stress disorder and related symptoms: a systematic review and meta-analysis. *BMC Psychiatry* 8: 1-8.
- Kozłowska K, Palmer DM, Brown KJ, et al. (2015a) Reduction of autonomic regulation in children and adolescents with conversion disorders. *Psychosomatic Medicine* 77(4): 356-370.
- Kozłowska K, Walker P, McLean L, et al. (2015b) Fear and the defense cascade: clinical implications and management. *Harvard Review of Psychiatry*. 23(4), 263-287.
- Kozłowska K, Walker P, McLean L, et al. (2015c) Fear and the defense cascade: clinical implications and management. *Harvard Review of Psychiatry* 23(4): 263-287.
- Krahn LE, Li H and O'Connor MK (2003) Patients who strive to be ill: Factitious disorder with physical symptoms. *American Journal of Psychiatry* 160: 1163-1168.

- Krishnan KRR, Delong M, Kraemer H, et al. (2002) Comorbidity of depression with other medical diseases in the elderly. *Biological Psychiatry* 52(6): 559-588.
- Krystal AD, Zaidman C, Greenside HS, et al. (1997) The largest Lyapunov exponent of the EEG during ECT seizures as a measure of ECT seizure adequacy. *Electroencephalography and Clinical Neurophysiology* 103(6): 599-606.
- Kuehner C (2017) Why is depression more common among women than among men? *The Lancet Psychiatry* 4(2): 146-158.
- Kurrie S, Brodaty H and Hogarth R (2012) *Physical comorbidities of dementia*. Cambridge University Press.
- Ladouceur R, Talbot F and Dugas MJ (1997) Behavioral expressions of intolerance of uncertainty in worry: Experimental findings. *Behavior Modification* 21(3): 355-371.
- Laidlaw K, Davidson K, Toner H, et al. (2008) A randomised controlled trial of cognitive behaviour therapy vs treatment as usual in the treatment of mild to moderate late life depression. *International Journal of Geriatric Psychiatry* 23(8): 843-850.
- Lally J, Ajnakina O, Stubbs B, et al. (2017) Remission and recovery from first-episode psychosis in adults: systematic review and meta-analysis of long-term outcome studies. *British Journal of Psychiatry* 211(6): 350-358.
- Lally J, Tully J, Robertson D, et al. (2016) Augmentation of clozapine with electroconvulsive therapy in treatment resistant schizophrenia: A systematic review and meta-analysis. *Schizophrenia Research* 171: 215-224.
- Lampe L and Malhi GS (2018) Avoidant personality disorder: current insights. *Psychology Research and Behavior Management*. 55-66.
- Large MM and Ryan CJ (2014) Suicide risk categorisation of psychiatric inpatients: what it might mean and why it is of no use. *Australasian Psychiatry* 22(4): 390-392.
- Laursen TM, Plana-Ripoll O, Andersen PK, et al. (2019) Cause-specific life years lost among persons diagnosed with schizophrenia: Is it getting better or worse? *Schizophrenia Research* 206: 284-290.
- Laursen TM, Wahlbeck K, Hällgren J, et al. (2013) Life expectancy and death by diseases of the circulatory system in patients with bipolar disorder or schizophrenia in the Nordic countries. *PloS One* 8(6): e67133.
- Laver K, Cumming R, Dyer S, et al. (2016) Clinical practice guidelines for dementia in Australia.
- Lawrence D, Hancock KJ and Kisely S (2013) The gap in life expectancy from preventable physical illness in psychiatric patients in Western Australia: retrospective analysis of population based registers. *BMJ* 346: f2539.

- Lee PH, Anttila V, Won H, et al. (2019) Genomic Relationships, Novel Loci, and Pleiotropic Mechanisms across Eight Psychiatric Disorders. *Cell* 179(7): 1469-1482.e1411.
- Lee SY, Fisher J, Wand AP, et al. (2020) Developing delirium best practice: a systematic review of education interventions for healthcare professionals working in inpatient settings. *European Geriatric Medicine* 11: 1-32.
- Lehmann S, Havik OE, Havik T, et al. (2013) Mental disorders in foster children: a study of prevalence, comorbidity and risk factors. *Child and Adolescent Psychiatry and Mental Health* 7: 1-12.
- Lejuez CW, Hopko DR and Hopko SD (2001) A brief behavioral activation treatment for depression. *Behavior Modification* 25(2): 255-286.
- Lele K, Leung KC and Peisah C (2023) A reflective practice group tutorial series for medical students. *Australasian Psychiatry* 31(4).
- Lepping P and Freudenmann RW (2008) Delusional parasitosis: a new pathway for diagnosis and treatment. *Clinical and Experimental Dermatology* 33(2): 113-117.
- Lesch K-P, Bengel D, Heils A, et al. (1996) Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science* 274(5292): 1527-1531.
- Letemendia F, Delva N, Rodenburg M, et al. (1993) Therapeutic advantage of bifrontal electrode placement in ECT. *Psychological Medicine* 23(2): 349-360.
- Leucht S, Hierl S, Kissling W, et al. (2012) Putting the efficacy of psychiatric and general medicine medication into perspective: review of meta-analyses. *British Journal of Psychiatry* 200(2): 97-106.
- Lewinsohn PM and Graf M (1973) Pleasant activities and depression. *Journal of Consulting and Clinical Psychology* 41(2): 261.
- Lewis G and Pelosi AJ (1990) The case-control study in psychiatry. *British Journal of Psychiatry* 157(2): 197-207.
- Lichtermann D, Ekelund J, Pukkala E, et al. (2001) Incidence of cancer among persons with schizophrenia and their relatives. *Archives of General Psychiatry* 58(6): 573-578.
- Light E (2019) Rates of use of community treatment orders in Australia. *International Journal of Law and Psychiatry* 64: 83-87.
- Lim GY, Tam WW, Lu Y, et al. (2018) Prevalence of Depression in the Community from 30 Countries between 1994 and 2014. *Scientific Reports* 8(1): 2861.
- Linehan M (2014) *DBT? Skills training manual*. Guilford Publications.
- Linehan MM, Armstrong HE, Suarez A, et al. (1991) Cognitive-behavioral treatment of chronically parasuicidal borderline patients. *Archives of General Psychiatry* 48(12): 1060-1064.

- Linehan MM, Tutek DA, Heard HL, et al. (1994) Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. *American Journal of Psychiatry* 151(12): 1771-1775.
- Lisanby SH, Maddox JH, Prudic J, et al. (2000) The effects of electroconvulsive therapy on memory of autobiographical and public events. *Archives of General Psychiatry* 57(6): 581-590.
- Liu H, Petukhova MV, Sampson NA, et al. (2017) Association of DSM-IV posttraumatic stress disorder with traumatic experience type and history in the World Health Organization World Mental Health Surveys. *JAMA Psychiatry* 74(3): 270-281.
- Lonergan E, Luxenberg J and Sastre AA (2009) Benzodiazepines for delirium. *Cochrane Database of Systematic Reviews*.(4).
- Lorentzen R, Nguyen TD, McGirr A, et al. (2022) The efficacy of transcranial magnetic stimulation (TMS) for negative symptoms in schizophrenia: a systematic review and meta-analysis. *Schizophrenia* 8(1): 35.
- Lorenz CP and Williams AJ (2017) Sleep apps: what role do they play in clinical medicine? *Current Opinion in Pulmonary Medicine* 23(6): 512-516.
- Lothgren M. (2004). Economic evidence in psychotic disorders: a review. *European Journal of Health Economics*, 5(Suppl 1), S67-74.
- Lovibond, S.H., & Lovibond, P.F. (1995). *Manual for the Depression Anxiety Stress Scales [Kit]*. Sydney, NSW: Psychology Foundation of Australia.
- Lynch DL, Stern AE, Kim Oates R, et al. (1993) Who participates in child sexual abuse research? *Journal of Child Psychology and Psychiatry* 34(6): 935-944.
- Madden S, Miskovic-Wheatley J, Clarke S, et al. (2015) Outcomes of a rapid refeeding protocol in adolescent anorexia nervosa. *Journal of Eating Disorders* 3(1): 1-8.
- Maenner MJ (2021) Prevalence and characteristics of autism spectrum disorder among children aged 8 years—autism and developmental disabilities monitoring network, 11 sites, United States, 2018. *MMWR. Surveillance Summaries* 70.
- Maglione JE, Thomas SE and Jeste DV (2014) Late-onset schizophrenia: do recent studies support categorizing LOS as a subtype of schizophrenia? *Current Opinion Psychiatry* 27(3): 173.
- Mai F (2004) Somatisation Disorder: a practical review. *Canadian Journal of Psychiatry* 49: 652-662.
- Main M (2000) The organized categories of infant, child, and adult attachment: Flexible vs. inflexible attention under attachment-related stress. *Journal of the American Psychoanalytic Association* 48(4): 1055-1096.
- Main M, Kaplan N and Cassidy J (1985) Security in infancy, childhood, and adulthood: A move to the level of representation. *Monographs of the Society for Research in Child Development*. 66-104.

- Malhi GS (2010) History of the Introduction of Lithium into Medicine and Psychiatry. *Acta Neuropsychiatrica* 22(3): 161.
- Malhi GS, Bassett D, Boyce P, et al. (2015) Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian & New Zealand Journal of Psychiatry* 49(12): 1087-1206.
- Malhi GS and Bell E (2019) Fake views: Schizoaffective disorder is not ‘SAD’, just bad. *Australian & New Zealand Journal of Psychiatry* 53(5): 481-484.
- Malhi GS and Bell E (2023) Mood Disorders. In: Tyrer P (ed) *Making Sense of the ICD-11: For Mental Health Professionals*. Cambridge: Cambridge University Press, pp.39-58.
- Malhi GS, Bell E, Bassett D, et al. (2021a) The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian & New Zealand Journal of Psychiatry* 55(1): 7-117.
- Malhi GS, Bell E, Bassett D, et al. (2021b) The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian & New Zealand Journal of Psychiatry* 55(1): 7-117.
- Malhi GS, Bell E, Bassett D, et al. (2023) Positioning of psychodynamic psychotherapy in the treatment of depression: A comparison of the RANZCP 2020 and NICE 2022 guidelines. *Australasian Psychiatry*. 31(3), 322-325.
- Malhi GS, Bell E, Boyce P, et al. (2020a) The 2020 Royal Australian and New Zealand College of psychiatrists clinical practice guidelines for mood disorders: Bipolar disorder summary. *Bipolar Disorders* 22(8): 805-821.
- Malhi GS, Bell E, Boyce P, et al. (2022) NICE guideline for depression ranks short-term psychodynamic psychotherapy (STTP). *Bipolar Disorders* 24(5): 467-471.
- Malhi GS, Bell E, Singh AB, et al. (2020b) The 2020 Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders: Major depression summary. *Bipolar Disorders* 22(8): 788-804.
- Malhi GS, Byrow Y, Boyce P, et al. (2016a) Why the hype about subtype? Bipolar I, bipolar II-it's simply bipolar, through and through! *Australian & New Zealand Journal of Psychiatry* 50(4): 303-306.
- Malhi GS, Das P, Bell E, et al. (2019) Modelling resilience in adolescence and adversity: a novel framework to inform research and practice. *Translational Psychiatry* 9(1): 316.
- Malhi GS, Das P, Outhred T, et al. (2018a) Understanding suicide: Focusing on its mechanisms through a lithium lens. *Journal of Affective Disorders* 241: 338-347.
- Malhi GS, Davey C and Bell E (2024) John Cade's lithium: A granular look at his achievement. *Australian & New Zealand Journal of Psychiatry* 58(9): 733-737.
- Malhi GS, Gershon S and Outhred T (2016b) Lithiummeter: Version 2.0. *Bipolar Disorders* 18(8): 631-641.

- Malhi GS, Green M, Fagiolini A, et al. (2008) Schizoaffective disorder: diagnostic issues and future recommendations. *Bipolar Disorders* 10(1p2): 215-230.
- Malhi GS, Irwin L, Hamilton A, et al. (2018b) Modelling mood disorders: An ACE solution? *Bipolar Disorders* 20: 4-16.
- Malhi GS and Mann JJ (2018) Depression. *The Lancet* 392(10161): 2299-2312.
- Malhi GS, Outhred T, Hamilton A, et al. (2018c) Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders: major depression summary. *Medical Journal of Australia* 208(4): 175-180.
- Malloch S and Trevarthen C (2009) Musicality: Communicating the vitality and interests of life. In: *Communicative musicality: Exploring the basis of human companionship* 1: 1-10.
- Malouff JM, Thorsteinsson EB and Schutte NS (2007) The efficacy of problem solving therapy in reducing mental and physical health problems: a meta-analysis. *Clinical Psychology Review* 27(1): 46-57.
- Marel C, Siedlecka E, Fisher A, et al. (2022) Guidelines on the management of co-occurring alcohol and other drug and mental health conditions in alcohol and other drug treatment settings (3rd edition). Sydney, Australia: Matilda Centre for Research in Mental Health and Substance Use, The University of Sydney.
- Marel C, Sunderland M, Mills KL, et al. (2019) Conditional probabilities of substance use disorders and associated risk factors: Progression from first use to use disorder on alcohol, cannabis, stimulants, sedatives and opioids. *Drug and Alcohol Dependence* 194: 136-142.
- Marmot M (2015) The health gap: the challenge of an unequal world. *The Lancet* 386(10011): 2442-2444.
- Martin PM and Martin-Granel E (2006) 2,500-year evolution of the term epidemic. *Emerging Infectious Diseases* 12(6): 976.
- Marx W, Penninx BWJH, Solmi M, et al. (2023) Major depressive disorder. *Nature Reviews Disease Primers* 9(1): 44.
- Marzouk T, Winkelbeiner S, Azizi H, et al. (2019) Transcranial Magnetic Stimulation for Positive Symptoms in Schizophrenia: A Systematic Review. *Neuropsychobiology* 79(6): 384-396.
- Matias-Guiu JA, Valles-Salgado M, Rognoni T, et al. (2017) Comparative diagnostic accuracy of the ACE-III, MIS, MMSE, MoCA, and RUDAS for screening of Alzheimer disease. *Dementia and Geriatric Cognitive Disorders* 43(5-6): 237-246.
- Matsunaga H, Kiriike N, Matsui T, et al. (2001) Taijin kyofusho: a form of social anxiety disorder that responds to serotonin reuptake inhibitors? *The International Journal of Neuropsychopharmacology* 4(03): 231-237.
- Maunder R and Hunter J (2021) *Damaged: childhood trauma, adult illness, and the need for a health care revolution*. University of Toronto Press.

- Maunder RG and Hunter JJ (2001) Attachment and psychosomatic medicine: developmental contributions to stress and disease. *Psychosomatic Medicine* 63(4): 556-567.
- Maunder RG and Hunter JJ (2009) Assessing patterns of adult attachment in medical patients. *General Hospital Psychiatry* 31(2): 123-130.
- Maust DT, Kim HM, Seyfried LS, et al. (2015) Antipsychotics, other psychotropics, and the risk of death in patients with dementia: number needed to harm. *JAMA Psychiatry* 72(5): 438-445.
- Mazzucchelli T, Kane R and Rees C (2009) Behavioral activation treatments for depression in adults: a meta-analysis and review. *Clinical Psychology: Science and Practice* 16(4): 383-411.
- McDonagh A, Friedman M, McHugo G, et al. (2005) Randomized trial of cognitive-behavioral therapy for chronic posttraumatic stress disorder in adult female survivors of childhood sexual abuse. *Journal of Consulting and Clinical Psychology* 73(3): 515.
- McGaugh JL (2000) Memory--a century of consolidation. *Science* 287(5451): 248-251.
- McGrath JJ, Al-Hamzawi A, Alonso J, et al. (2023) Age of onset and cumulative risk of mental disorders: a cross-national analysis of population surveys from 29 countries. *The Lancet Psychiatry* 10(9): 668-681.
- McIntyre RS, Berk M, Brietzke E, et al. (2020) Bipolar disorders. *The Lancet* 396(10265): 1841-1856.
- McIntyre RS, Cha DS, Soczynska JK, et al. (2013) Cognitive deficits and functional outcomes in major depressive disorder: Determinants, substrates, and treatment interventions. *Depression and Anxiety* 30(6): 515-527.
- McKeith IG, Dickson DW, Lowe J, et al. (2005) Diagnosis and management of dementia with Lewy bodies: third report of the DLB Consortium. *Neurology* 65(12): 1863-1872.
- McLean L (2012) The pilgrim road goes on: Psychotherapy and spirituality continue to journey together towards healing and human flourishing. *Spirituality: Theory, Praxis and Pedagogy*. Brill, pp.181-190.
- McLean L and Korner A (2014) Dreaming the (Lost) Self in Psychotherapy: Beings in Bodyspacetime in Collision, Confusion and Connection. *Exploring Bodies in Time and Space*. 251.
- McMain S and Ellery M (2008) Screening and assessment of personality disorders in addiction treatment settings. *International Journal of Mental Health and Addiction* 6: 20-31.
- McNally GP and Westbrook RF (2003) Anterograde amnesia for Pavlovian fear conditioning and the role of one trial overshadowing: Effects of preconditioning exposures to morphine in rat. *Journal of Experimental Psychology: Animal Behavior Processes* 29(3): 222.

- McWhirter L, Stone J, Sandercock P, et al. (2011) Hoover's sign for the diagnosis of functional weakness: a prospective unblinded cohort study in patients with suspected stroke. *Journal of Psychosomatic Research* 71(6): 384-386.
- Meador KJM, Baker GAP, Browning NP, et al. (2012) Effects of fetal antiepileptic drug exposure: Outcomes at age 4.5 years. *Neurology* 78(16): 1207-1214.
- Meadow R (1977) Munchausen syndrome by proxy. The hinterland of child abuse. *The Lancet* 310(8033): 343-345.
- Meagher DJ, McLoughlin L, Leonard M, et al. (2013) What do we really know about the treatment of delirium with antipsychotics? Ten key issues for delirium pharmacotherapy. *The American Journal of Geriatric Psychiatry* 21(12): 1223-1238.
- Meares R (2004a) The conversational model: an outline. *American Journal of Psychotherapy* 58(1): 51-66.
- Meares R (2004b) From Neurosis to Trauma. *Meanjin* 63(4): 16-25.
- Meares R (2005) *The metaphor of play: Origin and breakdown of personal being*. Routledge.
- Meares R (2012) *Borderline Personality Disorder and the Conversational Model: A Clinician's Manual (Norton Series on Interpersonal Neurobiology)*. WW Norton & Company.
- Meares R (2016) *The poet's voice in the making of mind*. Routledge.
- Mechanic D (1961) The concept of illness behaviour. *Journal of Chronic Diseases* 15: 189-194.
- Medda P, Toni C, Mariani MG, et al. (2015) Electroconvulsive therapy in 197 patients with a severe, drug-resistant bipolar mixed state: treatment outcome and predictors of response. *The Journal of clinical psychiatry* 76(9): 1168-1173.
- Medhus S, Rognli EB, Gossop M, et al. (2015) Amphetamine-induced psychosis: Transition to schizophrenia and mortality in a small prospective sample. *The American Journal on Addictions* 24(7): 586-589.
- Medical Deans Australia and New Zealand. (2020). Medical Deans: 2020 Student Statistics Snapshot. Medical Deans of Australia and New Zealand Inc. Retrieved 15th October 2024 from <https://medicaldeans.org.au/md/2021/11/MDANZ-Student-Statistics-Report-2020.pdf>
- Mehler PS and Brown C (2015) Anorexia nervosa—medical complications. *Journal of Eating Disorders* 3: 1-8.
- Melchior M, Prokofyeva E, Younès N, et al. (2014) Treatment for illegal drug use disorders: the role of comorbid mood and anxiety disorders. *BMC Psychiatry* 14: 1-9.
- Mellman TA, Bustamante V and Fins AI (2002) Hypnotic medication in the aftermath of trauma. *The Journal of Clinical Psychiatry* 63(12): 8400.

- Menzies K (2019) Understanding the Australian Aboriginal experience of collective, historical and intergenerational trauma. *International Social Work* 62(6): 1522-1534.
- Menzies RG and Clarke JC (1995) The etiology of phobias: A nonassociative account. *Clinical Psychology Review* 15(1): 23-48.
- Menzies School of Health Research (2008) Stay Strong Plan.
- Mersky JP, Topitzes J and Reynolds A (2013) Impacts of adverse childhood experiences on health, mental health, and substance use in early adulthood: A cohort study of an urban, minority sample in the US. *Child Abuse & Neglect* 37(11): 917-925.
- Messer T, Lammers G, Müller-Siecheneder F, et al. (2017) Substance abuse in patients with bipolar disorder: A systematic review and meta-analysis. *Psychiatry Research* 253: 338-350.
- Meuret AE, Tunnell N, & Roque A (2020). Anxiety disorders and medical comorbidity: Treatment implications. In *Anxiety Disorders: Rethinking and Understanding Recent Discoveries*. pp. 237–261.
- Miettinen OS (2007) Theoretical developments. *The development of modern epidemiology: Personal reports from those who were there*. 231-340.
- Miller WR and Rollnick S (2002) *Motivational Interviewing: Preparing People to Change Addictive Behaviour*. New York, US: Guildford Press.
- Mitchell AJ, Lord O and Malone D (2012) Differences in the prescribing of medication for physical disorders in individuals with v. without mental illness: meta-analysis. *British Journal of Psychiatry* 201(6): 435-443.
- Mitchell AJ, Vancampfort D, Sweers K, et al. (2013) Prevalence of metabolic syndrome and metabolic abnormalities in schizophrenia and related disorders—a systematic review and meta-analysis. *Schizophrenia Bulletin* 39(2): 306-318.
- Mitchell PB, Slade T and Andrews G (2004) Twelve-month prevalence and disability of DSM-IV bipolar disorder in an Australian general population survey. *Psychological Medicine* 34(5): 777-785.
- Moafmashhadi P and Koski L (2013) Limitations for interpreting failure on individual subtests of the Montreal Cognitive Assessment. *Journal of Geriatric Psychiatry and Neurology* 26(1): 19-28.
- Modini M, Tan L, Brinchmann B, et al. (2016) Supported employment for people with severe mental illness: Systematic review and meta-analysis of the international evidence. *British Journal of Psychiatry* 209(1): 14-22.
- Moher D, Shamseer L, Clarke M, et al. (2015) Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Systematic Reviews* 4: 1-9.
- Moncrieff J and Cohen D (2009) How do psychiatric drugs work? *Bmj* 338: b1963.

- Moncrieff J, Cooper RE, Stockmann T, et al. (2022) The serotonin theory of depression: a systematic umbrella review of the evidence. *Molecular Psychiatry*. 28(8), 3243-3256.
- Monds L, Ridley N, Rivas C, et al. (2017) Cognition and adaptive functioning in older people attending drug and alcohol services. *International Psychogeriatrics* 29(5): 815-823.
- Montes JM, Alegria A, Garcia-Lopez A, et al. (2013) Understanding bipolar disorder in late life: clinical and treatment correlates of a sample of elderly outpatients. *The Journal of Nervous and Mental Disease* 201(8): 674-679.
- Montroux L and Rees C (2017) *Healthcare Professionalism*. West Sussex: John Wiley and Sons.
- Morgan C, Dazzan P, Lappin J, et al. (2021) Rethinking the course of psychotic disorders: modelling long-term symptom trajectories. *Psychological Medicine*. 52(13), 2641-2650.
- Morgan VA, Waterreus A, Jablensky A, et al. (2012a) People living with psychotic illness in 2010: The second Australian national survey of psychosis. *Australian & New Zealand Journal of Psychiatry* 46(8), 735-752.
- Morgan VA, Waterreus A, Jablensky A, et al. (2011) People living with psychotic illness 2010. In: Department of Health and Ageing (ed). Canberra ACT 2601: Commonwealth of Australia,.
- Morgan VA, Waterreus A, Jablensky A, et al. (2012b) People living with psychotic illness in 2010: The second Australian national survey of psychosis. *Australian & New Zealand Journal of Psychiatry* 46(8): 735-752.
- Morris J, Belfer M, Daniels A, et al. (2011) Treated prevalence of and mental health services received by children and adolescents in 42 low-and-middle-income countries. *Journal of Child Psychology & Psychiatry & Allied Disciplines* 52(12): 1239-1246.
- Morselli E and Jerome L (2001) Dymorphophobia and taphephobia: two hitherto undescribed forms of insanity with fixed ideas. *History of Psychiatry* xii: 103-114.
- Moulds ML and Kandris E (2007) The nature and role of avoidance in depression. In: Einstein DA (ed) *Innovations and Advances in Cognitive Behaviour Therapy*. Bowen Hills, QLD.: Australian Academic Press, pp.215-223.
- Moulds ML, Kandris E, Starr S, et al. (2007) The relationship between rumination, avoidance and depression in a non-clinical sample. *Behaviour Research and Therapy* 45(2): 251-261.
- Munk-Olsen T, Laursen TM, Meltzer-Brody S, et al. (2012) Psychiatric disorders with postpartum onset: possible early manifestations of bipolar affective disorders. *Arch Gen Psychiatry* 69(4): 428-434.

- Murphy E, Kapur N, Webb R, et al. (2012) Risk factors for repetition and suicide following self-harm in older adults: multicentre cohort study. *The British Journal of Psychiatry* 200(5): 399-404.
- Murray C and Lopez AD (1996) Summary: The global burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. *Geneva and Boston: World Health Organization and Harvard School of Public Health*.
- Murray G (2018) Adjunctive psychosocial interventions for bipolar disorder: Some psychotherapeutic context for the Canadian Network for Mood and Anxiety Treatments (CANMAT) & International Society for Bipolar Disorders (ISBD) guidelines. *Bipolar Disorders* 20(5): 494-495.
- Murray J, Ehlers A and Mayou RA (2002) Dissociation and post-traumatic stress disorder: Two prospective studies of road traffic accident survivors. *The British Journal of Psychiatry* 180(4): 363-368.
- Myers MF and Gabbard GO (2008) *The physician as patient: a clinical handbook for mental health professionals*. American Psychiatric Pub. Washington, DC, USA.
- Nagendra J and Snowden J (2020) An Australian study of delusional disorder in late life. *International Psychogeriatrics* 32(4): 453-462.
- Naim R, Abend R, Wald I, et al. (2015) Threat-related attention bias variability and posttraumatic stress. *American Journal of Psychiatry* 172(12): 1242-1250.
- Nasir BF, Toombs MR, Kondalsamy-Chennakesavan S, et al. (2018) Common mental disorders among Indigenous people living in regional, remote and metropolitan Australia: a cross-sectional study. *BMJ Open* 8(6): e020196.
- Nasreddine ZS, Phillips NA, Bédirian V, et al. (2005) The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society* 53(4): 695-699.
- National Institute for Health and Clinical Excellence (2013) National Institute for Health and Care Excellence: Clinical Guidelines. *Antisocial Behaviour and Conduct Disorders in Children and Young People: Recognition, Intervention and Management*. The British Psychological Society & The Royal College of Psychiatrists, 2013: National Institute for Health and Clinical Excellence.
- National Mental Health Survey of Doctors and Students (2019) National mental health survey of doctors and students, October 2013, Updated 2019. Beyond Blue Australia.
- Nestadt G, Di C, Samuels JF, et al. (2010) The stability of DSM personality disorders over twelve to eighteen years. *Journal of Psychiatric Research* 44(1): 1-7.
- Neufeld KJ, Yue J, Robinson TN, et al. (2016) Antipsychotic medication for prevention and treatment of delirium in hospitalized adults: a systematic

- review and meta-analysis. *Journal of the American Geriatrics Society* 64(4): 705-714.
- Neuner F, Schauer M, Klaschik C, et al. (2004) A comparison of narrative exposure therapy, supportive counseling, and psychoeducation for treating posttraumatic stress disorder in an african refugee settlement. *Journal of Consulting and Clinical Psychology* 72(4): 579.
- New South Wales Ministry of Health (2020) *NSW Aboriginal Mental Health and Wellbeing Strategy 2020-2025*. Available at: **<https://www.health.nsw.gov.au/mentalhealth/resources/Publications/aborig-mh-wellbeing-2020-2025.pdf>**.
- Nezu AM (1986) Efficacy of a social problem-solving therapy approach for unipolar depression. *Journal of Consulting and Clinical Psychology* 54(2): 196-202.
- NICE (2022) *Depression in adults: treatment and management*. London, UK: National Institute for Health Care Excellence,.
- Nobler MS, Oquendo MA, Kegeles LS, et al. (2001) Decreased regional brain metabolism after ECT. *American Journal of Psychiatry* 158(2): 305-308.
- Nordsletten AE, Reichenberg A, Hatch SL, et al. (2013) Epidemiology of hoarding disorder. *The British Journal of Psychiatry*. bjp. bp. 113.130195.
- Nordstrom K, Vilke GM and Wilson MP (2016) Psychiatric emergencies for clinicians: emergency department management of serotonin syndrome. *Journal of Emergency Medicine* 50(1): 89-91.
- NSW Department of Health (2008) *Drug and Alcohol Psychosocial Interventions Professional Practice Guidelines*. North Sydney, Australia.
- NSW Health (2022) *Assessment and Management of Behaviours and Psychological Symptoms associated with Dementia (BPSD): Summary Handbook*.
- NSW Ministry of Health (2014) *Wellbeing later life your guide to programs and activities*.
- NSW Ministry of Health (2015) *Effective Models of Care for Comorbid Mental Illness and Illicit Substance Use: Evidence Check Review*. North Sydney, Australia.
- NSW Ministry of Health (2018) *NSW Clinical Guidelines: Treatment of Opioid Dependence – 2018*. NSW Government.
- NSW Ministry of Health (2019) *Clinical guidelines for use of depot buprenorphine (Buvidal and Sublocade) in the treatment of opioid dependence*. NSW Government.
- NSW Ministry of Health (2022) *Management of Withdrawal from Alcohol and Other Drugs Handbook*. NSW Government.
- NSW Ministry of Health (2023) *Alcohol and Other Drugs Psychosocial Interventions: Practice Guide*. NSW Government

- O'Connor DW, Ames D, Gardner B, et al. (2009) Psychosocial treatments of psychological symptoms in dementia: a systematic review of reports meeting quality standards. *International Psychogeriatrics* 21(2): 241-251.
- O'Hara MW and McCabe JE (2013) Postpartum depression: current status and future directions. *Annual Review of Clinical Psychology* 9: 379-407.
- O'Toole BI (2000) Screening for low prevalence disorders. *Australian & New Zealand Journal of Psychiatry* 34(1_suppl): A39-A46.
- O'Toole BI, Catts SV, Outram S, et al. (2009) The physical and mental health of Australian Vietnam veterans 3 decades after the war and its relation to military service, combat, and post-traumatic stress disorder. *American journal of epidemiology* 170(3): 318-330.
- O'Toole BI, Marshall RP, Grayson DA, et al. (1996) The Australian Vietnam veterans health study: I. Study design and response bias. *International Journal of Epidemiology* 25(2): 307-318.
- O'Brien BC, Harris IB, Beckman TJ, et al. (2014) Standards for reporting qualitative research: a synthesis of recommendations. *Academic Medicine* 89(9): 1245-1251.
- O'Bryant SE, Humphreys JD, Smith GE, et al. (2008) Detecting dementia with the mini-mental state examination in highly educated individuals. *Archives of Neurology* 65(7): 963-967.
- O'Neill N and Peisah C (2021) Capacity and the Law. 4 ed.: Australasian Legal Information Institute (AustLII) Communities.
- Okajima I, Komada Y and Inoue Y (2011) A meta-analysis on the treatment effectiveness of cognitive behavioral therapy for primary insomnia. *Sleep and Biological Rhythms* 9(1): 24-34.
- Olthuis JV, Watt MC, Bailey K, et al. (2016) Therapist-supported Internet cognitive behavioural therapy for anxiety disorders in adults. *Cochrane Database of Systematic Reviews*.(3).
- Orr SP, Pitman RK, Lasko NB, et al. (1993) Psychophysiological assessment of posttraumatic stress disorder imagery in World War II and Korean combat veterans. *Journal of Abnormal Psychology* 102(1): 152.
- Ostafin BD, Robinson MD and Meier BP (2015) *Handbook of mindfulness and self-regulation*. Springer.
- Ottosson J-O (1996) Experimental studies of the mode of action of electroconvulsive therapy. *The Journal of ECT* 12(3): 172-194.
- Owen-Smith A, Stewart C, Green C, et al. (2016) Adherence to common cardiovascular medications in patients with schizophrenia vs. patients without psychiatric illness. *General Hospital Psychiatry* 38: 9-14.
- Oyeboode F (2023) *Sims' symptoms in the mind : textbook of descriptive psychopathology*. Philadelphia: Elsevier.

- Ozer EJ, Best SR, Lipsey TL, et al. (2003) Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychological Bulletin* 129(1): 52.
- Palmer BA, Pankratz VS and Bostwick JM (2005) The Lifetime Risk of Suicide in Schizophrenia: A Reexamination. *Archives of General Psychiatry* 62(3): 247-253.
- Paolucci S, Gandolfo C, Provinciali L, et al. (2006) The Italian multicenter observational study on post-stroke depression (DESTRO). *Journal of Neurology* 253: 556-562.
- Parker G, Eyers K and Boyce P (2014) *Overcoming Baby Blues: A comprehensive guide to perinatal depression*. Sydney: Allen & Unwin.
- Parnas J and Bovet P (2014) *Psychiatry made easy: operation(al)ism and some of its consequences*. Oxford University Press.
- Parnas J, Sass LA and Zahavi D (2013) Rediscovering psychopathology: the epistemology and phenomenology of the psychiatric object. *Schizophrenia Bulletin* 39(2): 270-277.
- Parnas J and Zahavi D (2002) *The Role of Phenomenology in Psychiatric Diagnosis and Classification*. Chichester, UK: John Wiley & Sons, Ltd, pp.137-162.
- Parsons T (1951) *The Social System*. London: Routledge & K. Paul.
- Parsons T (1975) The sick role and the role of the physician reconsidered. *The Milbank Memorial Fund Quarterly. Health and Society* 53(5): 257-278.
- Paton MB, Large MM and Ryan CJ (2014) Debate: Clinical risk categorisation is valuable in the prevention of suicide and severe violence – No. *Australasian Psychiatry* 22(1): 10-12.
- Peacock J and Peacock P (2011) *Oxford handbook of medical statistics*. Oxford university press.
- Peisah C (2006) Practical application of family and systems theory in old age psychiatry: three case reports. *International Psychogeriatrics* 18(2): 345-353.
- Peisah C (2017) Student Support. Sydney: Sydney University Medical Society Mental Health Seminar.
- Peisah C (2020) Medical Welfare & Culture: Time for Change. Sydney: Nepean Hospital Grand Rounds.
- Peisah C, Forlenza O and Chiu E (2009) Ethics, capacity, and decision-making in the practice of old age psychiatry: an emerging dialogue. *Current Opinion Psychiatry* 22(6): 519-521.
- Peisah C, and Haysom G (2021). Professional Capacity. In: N. O'Neill & C. Peisah (Eds.), *Capacity and the Law (14 ed.)*. Australasian Legal Information Institute.
- Peisah C and Skladzien E (2014) *The use of restraints and psychotropic medications in people with dementia*. Alzheimer's Australia.

- Penders KA, Peeters IG, Metsemakers JF, et al. (2020) Personality disorders in older adults: A review of epidemiology, assessment, and treatment. *Current Psychiatry Reports* 22: 1-14.
- Peros OM, Webb L, Fox S, et al. (2021) Conducting exposure-based groups via telehealth for adolescents and young adults with social anxiety disorder. *Cognitive and Behavioral Practice* 28(4): 679-689.
- Perugi G, Medda P, Toni C, et al. (2017) The Role of Electroconvulsive Therapy (ECT) in Bipolar Disorder: Effectiveness in 522 Patients with Bipolar Depression, Mixed-state, Mania and Catatonic Features. *Current Neuropsychopharmacology* 15(3): 359-371.
- Phillips KA, Didie ER, Menard W, et al. (2006) Clinical features of body dysmorphic disorder in adolescents and adults. *Psychiatry Research* 141(3): 305-314.
- Phillips KA, Menard W, Quinn E, et al. (2013) A 4-year prospective observational follow-up study of course and predictors of course in body dysmorphic disorder. *Psychological Medicine* 43(5): 1109-1117.
- Piaget J (2013) *Play, dreams and imitation in childhood*. Routledge.
- Pietrzak RH, Goldstein RB, Southwick SM, et al. (2011) Prevalence and Axis I comorbidity of full and partial posttraumatic stress disorder in the United States: Results from Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Anxiety Disorders* 25(3): 456-465.
- Pilowsky I (1969) Abnormal illness behaviour. *British Journal of Medical Psychology* 42: 347-351.
- Pitman RK, Sanders KM, Zusman RM, et al. (2002) Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. *Biological Psychiatry* 51(2): 189-192.
- Platz C and Kendell RE (1988) A matched-control follow-up and family study of 'puerperal psychoses'. *British Journal of Psychiatry* 153: 90-94.
- Pollak TA, Lennox BR, Müller S, et al. (2020) Autoimmune psychosis: an international consensus on an approach to the diagnosis and management of psychosis of suspected autoimmune origin. *The Lancet Psychiatry* 7(1): 93-108.
- Porges SW (2011) *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation (Norton series on interpersonal neurobiology)*. WW Norton & Company.
- Porta M (2014) *A dictionary of epidemiology*. Oxford university press.
- Porter R (1997) *The Greatest Benefit to Mankind*. London: Harper Collins.
- Post RM, Altshuler LL, Kupka R, et al. (2016) Age of onset of bipolar disorder: Combined effect of childhood adversity and familial loading of psychiatric disorders. *Journal of Psychiatric Research* 81: 63-70.

- Pourhamzeh M, Moravej FG, Arabi M, et al. (2022) The Roles of Serotonin in Neuropsychiatric Disorders. *Cellular and Molecular Neurobiology* 42(6): 1671-1692.
- Preskorn SH (2012) Clinically important differences in the pharmacokinetics of ten newer “atypical” antipsychotics: Part1. *Journal of Psychiatric Practice* 18: 199-204.
- Prince M, Stewart R, Ford T, et al. (2003) *Practical Psychiatric Epidemiology*. Oxford University Press Oxford.
- Provencher MD and Ladouceur R (2004) Efficacy of problem-solving training and cognitive exposure in the treatment of generalized anxiety disorder: A case replication series. *Cognitive and Behavioral Practice* 11(4): 404-414.
- Rapee RM and Heimberg RG (1997) A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy* 35(8): 741-756.
- Rauch SL and Drevets WC (2009) neuroimaging and neuroanatomy of stress-induced and fear circuitry disorders. In: Andrews G, Charney DS, Sirovatka PJ, et al. (eds) *Stress-induced and fear circuitry disorders: Refining the research agenda for DSM-V*. Arlington, VA: American Psychiatric Association, pp.215-254.
- Rees E, O'Donovan MC and Owen MJ (2015) Genetics of schizophrenia. *Current Opinion in Behavioral Sciences* 2: 8-14.
- Reichborn-Kjennerud T (2010) Genetics of personality disorders. *Clinics in Laboratory Medicine* 30(4): 893-910.
- Reinhardt MM and Cohen CI (2015) Late-life psychosis: diagnosis and treatment. *Current Psychiatry Reports* 17: 1-13.
- Resources for Research Ethics Education (1999-2016) *What is Research Ethics?* Available at: <http://research-ethics.org/introduction/what/>.
- Ressler KJ, Berretta S, Bolshakov VY, et al. (2022) Post-traumatic stress disorder: clinical and translational neuroscience from cells to circuits. *Nature Reviews Neurology* 18(5): 273-288.
- Rhee TG, Olfson M, Nierenberg AA, et al. (2020) 20-Year Trends in the Pharmacologic Treatment of Bipolar Disorder by Psychiatrists in Outpatient Care Settings. *American Journal of Psychiatry* 177(8): 706-715.
- Richards DA, Ekers D, McMillan D, et al. (2016) Cost and Outcome of Behavioural Activation versus Cognitive Behavioural Therapy for Depression (COBRA): a randomised, controlled, non-inferiority trial. *The Lancet* 388(10047): 871-880.
- Richman JA and Flaherty JA (1990) Gender differences in medical student distress: contributions of prior socialization and current role-related stress. *Social Science & Medicine* 30(7): 777-787.
- Riffenburgh RH (2012) *Statistics in Medicine*. Academic press.

- Roberts D Clin Psy NP, Kitchiner NJ, Kenardy J, et al. (2009) Systematic review and meta-analysis of multiple-session early interventions following traumatic events. *American Journal of Psychiatry* 166(3): 293-301.
- Robertson J and Bowlby J (1952) Responses of young children to separation from their mothers. *Courrier du Centre International de l'Enfance* 2: 131-142.
- Robins LN (1981) National Institute of Mental Health Diagnostic Interview Schedule. *Archives of General Psychiatry* 38: 381-389.
- Robinson KH, Bansel P, Denson N, et al. (2014) *Growing up queer: Issues facing young Australians who are gender variant and sexuality diverse*.
- Rocca E and Anjum RL (2020) Complexity, Reductionism and the Biomedical Model. In: Anjum RL, Copeland S and Rocca E (eds) *Rethinking Causality, Complexity and Evidence for the Unique Patient: A CauseHealth Resource for Healthcare Professionals and the Clinical Encounter*. Cham: Springer International Publishing, pp.75-94.
- Rogers CR (1995) *On becoming a person: A therapist's view of psychotherapy*. Houghton Mifflin Harcourt.
- Rosenhan DL (1973) On Being Sane in Insane Places. *Science (American Association for the Advancement of Science)* 179(4070): 250-258.
- Rosfort R (2019) *Phenomenology and Hermeneutics*. Oxford University Press.
- Rossell SL, Harrison BJ and Castle D (2015) Can understanding the neurobiology of body dysmorphic disorder (BDD) inform treatment? *Australasian Psychiatry* 23(4): 361-364.
- Roth JD, White WL and Kelly JF (2014) *Broadening the base of addiction mutual support groups*. Routledge.
- Rothbaum BO, Kearns MC, Reiser E, et al. (2014) Early intervention following trauma may mitigate genetic risk for PTSD in civilians: a pilot prospective emergency department study. *The Journal of Clinical Psychiatry* 75(12): 5292.
- Rowland JT, Basic D, Storey JE, et al. (2006) The Rowland Universal Dementia Assessment Scale (RUDAS) and the Folstein MMSE in a multicultural cohort of elderly persons. *International Psychogeriatrics* 18(1): 111-120.
- Royal Australian and New Zealand College of Psychiatrists (2017) *Involuntary commitment and treatment - mental health legislation*. Available at: <https://www.ranzcp.org/clinical-guidelines-publications/in-focus-topics/mental-health-legislation/involuntary-commitment-and-treatment> (accessed 20th September 2024).
- Royal Australian New Zealand College of Psychiatrists (2014) Ethical Guideline 11: Principles and Guidelines for Aboriginal and Torres Strait Islander Mental Health.
- Rush AJ, Trivedi MH, Wisniewski SR, et al. (2006) Acute and Longer-Term Outcomes in Depressed Outpatients Requiring One or Several Treatment

- Steps: A STAR*D Report. *American Journal of Psychiatry* 163(11): 1905-1917.
- Ryan C, Callaghan S and Peisah C (2015a) The capacity to refuse psychiatric treatment: A guide to the law for clinicians and tribunal members. *Australian & New Zealand Journal of Psychiatry* 49(4): 324-333.
- Ryan C, Nielssen O, Paton M, et al. (2010) Clinical decisions in psychiatry should not be based on risk assessment. *Australasian Psychiatry* 18(5): 398-403.
- Ryan CJ and Callaghan S (2016) The impact on clinical practice of the 2015 reforms to the NSW Mental Health Act. *Australasian Psychiatry*. (in press).
- Ryan CJ, Callaghan S and Peisah C (2015b) The capacity to refuse psychiatric treatment – a guide to the law for clinicians and tribunal members. *Australian & New Zealand Journal of Psychiatry* 49(4): 324-333.
- Ryan CJ, Large M, Gribble R, et al. (2015c) Assessing and managing suicidal patients in the emergency department. *Australasian Psychiatry* 23(5): 513-515.
- Rytwinski NK, Scur MD, Feeny NC, et al. (2013) The co-occurrence of major depressive disorder among individuals with posttraumatic stress disorder: A meta-analysis. *Journal of Traumatic Stress* 26(3): 299-309.
- Sackeim HA, Prudic J, Devanand D, et al. (2000) A prospective, randomized, double-blind comparison of bilateral and right unilateral electroconvulsive therapy at different stimulus intensities. *Archives of General Psychiatry* 57(5): 425-434.
- Saha S, Chant D, Welham J, et al. (2005) A systematic review of the prevalence of schizophrenia. *PLoS Medicine* 2(5): e141.
- Said D, Kypri K and Bowman J (2013) Risk factors for mental disorder among university students in Australia: findings from a web-based cross-sectional survey. *Social Psychiatry and Psychiatric Epidemiology* 48: 935-944.
- Sajatovic M, Calabrese JR and Mullen J (2008) Quetiapine for the treatment of bipolar mania in older adults. *Bipolar Disorders* 10(6): 662-671.
- Sajatovic M, Gyulai L, Calabrese JR, et al. (2005) Maintenance treatment outcomes in older patients with bipolar I disorder. *The American Journal of Geriatric Psychiatry* 13(4): 305-311.
- Salkovskis PM (1999) Understanding and treating obsessive-compulsive disorder. *Behaviour Research & Therapy* 37 Suppl 1: S29-52.
- Sansone RA and Sansone LA (2012) Antidepressant adherence: are patients taking their medications? *Innovations in clinical neuroscience* 9(5-6): 41.
- Sartor CE, McCutcheon V, Pommer N, et al. (2011) Common genetic and environmental contributions to post-traumatic stress disorder and alcohol dependence in young women. *Psychological Medicine* 41(7): 1497-1505.
- Savla GN, Vella L, Armstrong CC, et al. (2013) Deficits in domains of social cognition in schizophrenia: a meta-analysis of the empirical evidence. *Schizophrenia Bulletin* 39(5): 979-992.

- Sawyer M, Arney FM, Baghurst PA, et al. (2000) *Child and Adolescent Component of the National Survey of Mental Health and Wellbeing*. Available at: <http://www.health.gov.au/internet/main/publishing.nsf/content/mental-pubs-m-young> (accessed 28.3.2016).
- Schioldann J (2009) Introduction pages. *History of the Introduction of Lithium into Medicine and Psychiatry: Birth of Modern Psychopharmacology 1949*. Adelaide: Adelaide Academic Press and Brascoe Publishing, pp.xix-xxv.
- Schioldann J (2013) From guinea pigs to manic patients: Cade's 'story of lithium'. *Australian & New Zealand Journal of Psychiatry* 47(5): 484-486.
- Schmidt NB, Lerew DR and Jackson RJ (1997) The role of anxiety sensitivity in the pathogenesis of panic: prospective evaluation of spontaneous panic attacks during acute stress. *Journal of Abnormal Psychology* 106(3): 355.
- Schnurr PP, Friedman MJ, Foy DW, et al. (2003) Randomized trial of trauma-focused group therapy for posttraumatic stress disorder: Results from a Department of Veterans Affairs cooperative study. *Archives of General Psychiatry* 60(5): 481-489.
- Schönfeldt-Lecuona C, Kuhlwilm L, Cronemeyer M, et al. (2020) Treatment of the neuroleptic malignant syndrome in international therapy guidelines: a comparative analysis. *Pharmacopsychiatry* 53(02): 51-59.
- Schuckit MA (2014) Recognition and management of withdrawal delirium (delirium tremens). *New England Journal of Medicine* 371(22): 2109-2113.
- Schulte MT and Hser Y-I (2013) Substance use and associated health conditions throughout the lifespan. *Public health reviews* 35: 1-27.
- Schulz KF, Altman DG and Moher D (2010) CONSORT 2010 statement: updated guidelines for reporting parallel group randomised trials. *Journal of Pharmacology and Pharmacotherapeutics* 1(2): 100-107.
- Schünemann H (2013) Bro ek J, Guyatt G, Oxman A, editors. GRADE Working Group. GRADE handbook for grading quality of evidence and strength of recommendations.
- Schünemann H, Vist G, Higgins J, et al. (2022) Chapter 15: Interpreting results and drawing conclusions. In: Higgins J, Thomas J, Chandler J, et al. (eds) *Cochrane Handbook for Systematic Reviews of Interventions*. Cochrane.
- Scogin F, Rohen N and Bailey E (2000) Geriatric Depression Scale. In: Maruish ME (ed) *Handbook of psychological assessment in primary care settings*. Lawrence Erlbaum Associates Publishers.
- Scotton WJ, Hill LJ, Williams AC, et al. (2019) Serotonin syndrome: pathophysiology, clinical features, management, and potential future directions. *International Journal of Tryptophan Research* 12: 1178646919873925.
- Seeman MV (2021) History of the dopamine hypothesis of antipsychotic action. *World Journal of Psychiatry* 11(7): 355-364.

- Selvaraj S and John V (2020) Taking care of medical students: the pillars of future healthcare. *SciELO Brasil*, 4-5.
- Semahegn A, Torpey K, Manu A, et al. (2020) Psychotropic medication non-adherence and its associated factors among patients with major psychiatric disorders: a systematic review and meta-analysis. *Systematic Reviews* 9(1): 17.
- Semkovska M and McLoughlin DM (2010) Objective cognitive performance associated with electroconvulsive therapy for depression: a systematic review and meta-analysis. *Biological Psychiatry* 68(6): 568-577.
- Serfaty MA, Haworth D, Blanchard M, et al. (2009) Clinical effectiveness of individual cognitive behavioral therapy for depressed older people in primary care: a randomized controlled trial. *Archives of General Psychiatry* 66(12): 1332-1340.
- Setkowski K, Palantza C, van Ballegooijen W, et al. (2023) Which psychotherapy is most effective and acceptable in the treatment of adults with a (sub)clinical borderline personality disorder? A systematic review and network meta-analysis. *Psychological Medicine* 53(8): 3261-3280.
- Sevy S, Nathanson K, Visweswarajah H, et al. (2004) The relationship between insight and symptoms in schizophrenia. *Comprehensive Psychiatry* 45(1): 16-19.
- Shalev A, Liberzon I and Marmar C (2017) Post-traumatic stress disorder. *New England Journal of Medicine* 376(25): 2459-2469.
- Shalev AY, Ankri Y, Israeli-Shalev Y, et al. (2012) Prevention of posttraumatic stress disorder by early treatment: results from the Jerusalem Trauma Outreach And Prevention study. *Archives of General Psychiatry* 69(2): 166-176.
- Shalev AY and Freedman S (2005) PTSD following terrorist attacks: a prospective evaluation. *American Journal of Psychiatry* 162(6): 1188-1191.
- Shalev AY, Freedman S, Peri T, et al. (1998) Prospective study of posttraumatic stress disorder and depression following trauma. *American Journal of Psychiatry* 155(5): 630-637.
- Sharp TJ and Harvey AG (2001) Chronic pain and posttraumatic stress disorder: mutual maintenance? *Clinical Psychology Review* 21(6): 857-877.
- Shedler J (2010) The efficacy of psychodynamic psychotherapy. *American Psychologist* 65(2): 98.
- Sheerin CM, Lind MJ, Bountress KE, et al. (2017) The genetics and epigenetics of PTSD: overview, recent advances, and future directions. *Current Opinion in Psychology* 14: 5-11.
- Sheridan Rains L, Zenina T, Dias MC, et al. (2019) Variations in patterns of involuntary hospitalisation and in legal frameworks: an international comparative study. *The Lancet Psychiatry* 6(5): 403-417.

- Shine JM, Müller EJ, Munn B, et al. (2021) Computational models link cellular mechanisms of neuromodulation to large-scale neural dynamics. *Nature Neuroscience* 24(6): 765-776.
- Shneidman E (1999) Perturbation and lethality: A psychological approach to assessment and intervention.
- Shorter E (2009) The history of lithium therapy. *Bipolar Disorders* 11(s2): 4-9.
- Shulman KI, Rochon P, Sykora K, et al. (2003) Changing prescription patterns for lithium and valproic acid in old age: shifting practice without evidence. *BMJ* 326(7396): 960-961.
- Siebern AT and Manber R (2010) Insomnia and its effective non-pharmacologic treatment. *Medical Clinics of North America* 94: 581-591.
- Silove D, Alonso J, Bromet E, et al. (2015) Pediatric-onset and adult-onset separation anxiety disorder across countries in the World Mental Health Survey. *American Journal of Psychiatry* 172(7): 647-656.
- Singh B, Hughes AJ, Mehta G, et al. (2016) Efficacy of prazosin in posttraumatic stress disorder: a systematic review and meta-analysis. *The Primary Care Companion for CNS Disorders* 18(4): 26306.
- Singh B, Olds T, Curtis R, et al. (2023) Effectiveness of physical activity interventions for improving depression, anxiety and distress: an overview of systematic reviews. *British Journal of Sports Medicine*. 57(18), 1203-1209.
- Siskind D, Orr S, Sinha S, et al. (2022) Rates of treatment-resistant schizophrenia from first-episode cohorts: systematic review and meta-analysis. *The British Journal of Psychiatry* 220(3): 115-120.
- Skinner BF and Vargas J (1957) Verbal Behavior. Acton, MA. Copley Publishing Group. *Journal of Behavioral Education* 12: 185-206.
- Slade A, Holland ML, Ordway MR, et al. (2020) Minding the Baby®: Enhancing parental reflective functioning and infant attachment in an attachment-based, interdisciplinary home visiting program. *Development and Psychopathology* 32(1): 123-137.
- Slater E (1965) Diagnosis of "Hysteria". *British Medical Journal*.(1): 1395-1399.
- Smith GC and Pell JP (2003) Parachute use to prevent death and major trauma related to gravitational challenge: systematic review of randomised controlled trials. *BMJ* 327(7429): 1459-1461.
- Smith GW, Ruiz-Sancho A and Gunderson JG (2001) An intensive outpatient program for patients with borderline personality disorder. *Psychiatric Services* 52(4): 532-533.
- Smoller JW (2016) The genetics of stress-related disorders: PTSD, depression, and anxiety disorders. *Neuropsychopharmacology* 41(1): 297-319.
- Snowdon J, Draper B, Brodaty H, et al. (2010) Prevalence and treatment of late life depression. *Australian & New Zealand Journal of Psychiatry* 44(11): 1054-1054.

- Snowdon J, Galanos D and Vaswani D (2011a) A 2009 survey of psychotropic medication use in Sydney nursing homes. *The Medical Journal of Australia* 194(5): 270-271.
- Snowdon J, Phillips J, Zhong B, et al. (2017) Changes in age patterns of suicide in Australia, the United States, Japan and Hong Kong. *Journal of Affective Disorders* 211: 12-19.
- Snowdon J, Rosengren D, Daniel F, et al. (2011b) Australia's use of the Cornell scale to screen for depression in nursing homes. *Australasian Journal on Ageing* 30(1): 33-36.
- Soares-Weiser K, Maayan N, Bergman H, et al. (2015) First rank symptoms for schizophrenia. *Cochrane Database Systematic Reviews* 1(1): Cd010653.
- Solmi M, Fiedorowicz J, Poddighe L, et al. (2021) Disparities in Screening and Treatment of Cardiovascular Diseases in Patients With Mental Disorders Across the World: Systematic Review and Meta-Analysis of 47 Observational Studies. *American Journal of Psychiatry* 178(9): 793-803.
- Solmi M, Radua J, Olivola M, et al. (2022) Age at onset of mental disorders worldwide: large-scale meta-analysis of 192 epidemiological studies. *Molecular Psychiatry* 27(1): 281-295.
- Solmi M, Seitidis G, Mavridis D, et al. (2023) Incidence, prevalence, and global burden of schizophrenia-data, with critical appraisal, from the Global Burden of Disease (GBD) 2019. *Molecular Psychiatry* 28(12): 5319-5327.
- Somers JM, Goldner EM, Waraich P, et al. (2006) Prevalence and incidence studies of anxiety disorders: a systematic review of the literature. *The Canadian Journal of Psychiatry* 51(2): 100-113.
- Southwick SM, Bremner JD, Rasmusson A, et al. (1999) Role of norepinephrine in the pathophysiology and treatment of posttraumatic stress disorder. *Biological Psychiatry* 46(9): 1192-1204.
- Southwick SM, Krystal JH, Bremner JD, et al. (1997) Noradrenergic and serotonergic function in posttraumatic stress disorder. *Archives of General Psychiatry* 54(8): 749-758.
- Spadaro A, Scott KR, Koyfman A, et al. (2022) High risk and low prevalence diseases: Serotonin syndrome. *The American Journal of Emergency Medicine* 61: 90-97.
- Speranza AM, Nicolais G, Maggiora Vergano C, et al. (2017) Emerging criteria for the low-coherence cannot classify category. *Attachment & Human Development* 19(6): 613-634.
- Spiegelberg H (1975) Phenomenology through Vicarious Experience. *Doing Phenomenology: Essays on and in Phenomenology*. Dordrecht: Springer Netherlands, pp.35-53.
- Spitzer RL, First MB and Wakefield JC (2007) Saving PTSD from itself in DSM-V. *Journal of anxiety disorders* 21(2): 233-241.

- Squire LR (1977) ECT and memory loss. *The American Journal of Psychiatry* 134(9): 997-1001.
- Staab R, Campagna J, Ma J, et al. (2022) Rapid refeeding in anorexia nervosa: A dialectic balance. *International Journal of Eating Disorders* 55(5): 653-663.
- Stabenau JR, Creveling CR and Daly J (1970) The "pink spot," 3, 4-dimethoxyphenylethylamine, common tea, and schizophrenia. *American Journal of Psychiatry* 127(5): 611-616.
- Stanghellini G and Aragona M (2016) Phenomenological Psychopathology: Toward a Person-Centered Hermeneutic Approach in the Clinical Encounter. In: Stanghellini G and Aragona M (eds) *An Experiential Approach to Psychopathology: What is it like to Suffer from Mental Disorders?* Cham: Springer International Publishing, pp.1-43.
- Starcevic V (2015) Hypochondriasis: treatment options for a diagnostic quagmire. *Australasian Psychiatry* 23(4): 369-373.
- Stein A, Pearson RM, Goodman SH, et al. (2014) Effects of perinatal mental disorders on the fetus and child. *The Lancet* 384(9956): 1800-1819.
- Stein DJ, Hollander E and Josephson SC (1994) Serotonin reuptake blockers for the treatment of obsessional jealousy. *Journal of Clinical Psychiatry*.
- Steinhausen H-C and Weber S (2009) The outcome of bulimia nervosa: findings from one-quarter century of research. *American Journal of Psychiatry* 166(12): 1331-1341.
- Stephoe A, Deaton A and Stone AA (2015) Subjective wellbeing, health, and ageing. *The Lancet* 385(9968): 640-648.
- Steven T (2011) Early versus late onset obsessive-compulsive disorder: Evidence for distinct subtypes. *Clinical Psychology Review* 31(7): 1083-1100.
- Stevenson J, Brodaty H, Boyce P, et al. (2011a) Personality disorder comorbidity and outcome: Comparison of three age groups. *Australian & New Zealand Journal of Psychiatry* 45(9): 771-779.
- Stevenson J, Brodaty H, Boyce P, et al. (2012) Does age moderate the effect of personality disorder on coping style in psychiatric inpatients? *Journal of Psychiatric Practice* 18(3): 187-198.
- Stevenson J, Dwyer A, Boyce P, et al. (2011b) The effect of age on prevalence, type and diagnosis of personality disorder in psychiatric inpatients. *International Journal of Geriatric Psychiatry* 26(9): 981-987.
- Stevenson J and Meares R (1992) An outcome study of psychotherapy for patients with borderline personality disorder. *American Journal of Psychiatry* 149(3): 358-362.
- Stoffers-Winterling JM, Storebø OJ, Kongerslev MT, et al. (2022) Psychotherapies for borderline personality disorder: a focused systematic review and meta-analysis. *The British Journal of Psychiatry* 221(3): 538-552.

- Storey JE, Rowland JT, Conforti DA, et al. (2004) The Rowland universal dementia assessment scale (RUDAS): a multicultural cognitive assessment scale. *International Psychogeriatrics* 16(1): 13-31.
- Stovall-McClough KC and Dozier M (2016) Attachment states of mind and psychopathology in adulthood. *Handbook of attachment: Theory, research, and clinical applications*. 715-738.
- Stunkard AJ (1959) Eating patterns and obesity. *Psychiatric Quarterly* 33(2): 284-295.
- Stunkard AJ, Grace WJ and Wolff HG (1955) The night-eating syndrome. A pattern of food intake among certain obese patients. *American Journal of Medicine* 19: 78-86.
- Suppes T, Webb A, Carmody T, et al. (1996) Is postictal electrical silence a predictor of response to electroconvulsive therapy? *Journal of Affective Disorders* 41(1): 55-58.
- Swayden KJ, Anderson KK, Connelly LM, et al. (2012) Effect of sitting vs. standing on perception of provider time at bedside: a pilot study. *Patient Education and Counseling* 86(2): 166-171.
- Taipale H, Tanskanen A, Mehtälä J, et al. (2020) 20-year follow-up study of physical morbidity and mortality in relationship to antipsychotic treatment in a nationwide cohort of 62,250 patients with schizophrenia (FIN20). *World Psychiatry* 19(1): 61-68.
- Takano H, Motohashi N, Uema T, et al. (2007) Changes in regional cerebral blood flow during acute electroconvulsive therapy in patients with depression: positron emission tomographic study. *The British Journal of Psychiatry* 190(1): 63-68.
- Taylor DJ, Wilkerson AK, Pruiksma KE, et al. (2018) Reliability of the structured clinical interview for DSM-5 sleep disorders module. *Journal of Clinical Sleep Medicine* 14(3): 459-464.
- Taylor DM, Young AH and Barnes TRE (2021) *The Maudsley Prescribing Guidelines in Psychiatry*. Wiley-Blackwell.
- Taylor MA and Fink M (2003) Catatonia in Psychiatric Classification: A Home of Its Own. *American Journal of Psychiatry* 160(7): 1233-1241.
- Taylor S, Jang KL and Asmundson GJ (2010) Etiology of obsessions and compulsions: A behavioral-genetic analysis. *Journal of Abnormal Psychology* 119(4): 672-682.
- Teasdale SB, Ward PB, Rosenbaum S, et al. (2017) Solving a weighty problem: Systematic review and meta-analysis of nutrition interventions in severe mental illness. *British Journal of Psychiatry* 210(2): 110-118.
- Teesson M, Slade T and Mills K (2009) Comorbidity in Australia: findings of the 2007 national survey of mental health and wellbeing. *Australian & New Zealand Journal of Psychiatry* 43(7): 606-614.

- Teicher MH, Samson JA, Anderson CM, et al. (2016) The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience* 17(10): 652-666.
- Tennant CC (2002) A student mental health and welfare program in a medical faculty. *The Medical Journal of Australia* 177(1): S9.
- Testad I, Corbett A, Aarsland D, et al. (2014) The value of personalized psychosocial interventions to address behavioral and psychological symptoms in people with dementia living in care home settings: a systematic review. *International Psychogeriatrics* 26(7): 1083-1098.
- Tharyan P and Adams CE (2009) Electroconvulsive therapy for schizophrenia. *Cochrane Database of Systematic Reviews* 4.
- The Australian Research Council and Universities Australia (Updated 2018) National Statement on Ethical Conduct in Human Research 2007. Commonwealth of Australia, Canberra: The National Health and Medical Research Council.
- The National Eating Disorders Collaboration (2010a) Eating disorders prevention, treatment & management: An evidence review. Sydney: NEDC.
- The National Eating Disorders Collaboration (2010b) Eating disorders: The way forward: An Australian national framework. Sydney: NEDC.
- The National Eating Disorders Collaboration (2012) An integrated response to complexity – National eating disorders framework 2012. Sydney: NEDC.
- The National Institute of Mental Health (NIMH) (2013) *Research Domain Criteria (RDoC)*. Available at:
<https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc/index.shtml>.
- The UK ECT Review Group (2003) Efficacy and safety of electroconvulsive therapy in depressive disorders: a systematic review and meta-analysis. *Lancet* 361: 799-808.
- The World Medical Association (2013) World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. World Medical Association.
- Thomas E and Stein DJ (2017) Novel pharmacological treatment strategies for posttraumatic stress disorder. *Expert Review of Clinical Pharmacology* 10(2): 167-177.
- Tor P-C, Bautovich A, Wang M-J, et al. (2015) A systematic review and meta-analysis of brief versus ultrabrief right unilateral electroconvulsive therapy for depression. *The Journal of Clinical Psychiatry* 76(9): 20683.
- Torgersen S (2009) The nature (and nurture) of personality disorders. *Scandinavian Journal of Psychology* 50(6): 624-632.
- Trevarthen C (2018) Conversations with a two-month-old 1. *Parent-infant Psychodynamics*. Routledge, pp.25-34.

- Trevethan R (2017) Sensitivity, specificity, and predictive values: foundations, pliabilitys, and pitfalls in research and practice. *Frontiers in Public Health* 5: 308890.
- Trubetskoy V, Pardiñas AF, Qi T, et al. (2022) Mapping genomic loci implicates genes and synaptic biology in schizophrenia. *Nature* 604(7906): 502-508.
- True WR, Rice J, Eisen SA, et al. (1993) A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Archives of General Psychiatry* 50(4): 257-264.
- Tyrer P and Tyrer H (2015) Nidotherapy: The expansion of environmental treatment. *Australian & New Zealand Journal of Psychiatry* 49(10): 934.
- United States Census Bureau (2016) Available at: <http://www.census.gov/popest/> (accessed 30th March).
- Uphoff E, Ekers D, Robertson L, et al. (2020) Behavioural activation therapy for depression in adults. *Cochrane Database of Systematic Reviews*.(7).
- Urfer-Parnas A (2019) Eugène Minkowski. Oxford University Press.
- Vaillant GE (2012) *Triumphs of experience*. Harvard University Press.
- Vaillant GE and Milofsky E (1980) Natural history of male psychological health: IX. Empirical evidence for Erikson's model of the life cycle. *The American Journal of Psychiatry* 137(11): 1348-1359.
- Vaillant GE and Vaillant CO (1990) Natural history of male psychological health, XII: a 45-year study of predictors of successful aging at age 65. *American Journal of Psychiatry* 147(1): 31-37.
- van Buitenen N, van den Berg CJ, Meijers J, et al. (2020) The prevalence of mental disorders and patterns of comorbidity within a large sample of mentally ill prisoners: A network analysis. *European Psychiatry* 63(1): e63.
- Van der Kolk B (2014) The body keeps the score: Brain, mind, and body in the healing of trauma. *New York*.
- van der Velden AM, Scholl J, Elmholt E-M, et al. (2023) Mindfulness training changes brain dynamics during depressive rumination: A randomized controlled trial. *Biological Psychiatry* 93(3): 233-242.
- van Diermen L, van den Amele S, Kamperman AM, et al. (2018) Prediction of electroconvulsive therapy response and remission in major depression: meta-analysis. *The British Journal of Psychiatry* 212(2): 71-80.
- Van Orden KA, Wiktorsson S, Duberstein P, et al. (2015) Reasons for attempted suicide in later life. *The American Journal of Geriatric Psychiatry* 23(5): 536-544.
- Vandenbroucke JP, Elm Ev, Altman DG, et al. (2007) Strengthening the Reporting of Observational Studies in Epidemiology (STROBE): explanation and elaboration. *Annals of Internal Medicine* 147(8): W-163-W-194.
- Vasileva M, Graf RK, Reinelt T, et al. (2021) Research review: A meta-analysis of the international prevalence and comorbidity of mental disorders in

- children between 1 and 7 years. *Journal of Child Psychology and Psychiatry* 62(4): 372-381.
- Veale D (2001) Cognitive-behavioural therapy for body dysmorphic disorder. *Advances in Psychiatric Treatment* 7: 125-132.
- Videler AC, van Alphen SP, van Royen RJ, et al. (2018) Schema therapy for personality disorders in older adults: a multiple-baseline study. *Aging & Mental Health* 22(6): 738-747.
- Vita A, Barlati S, Ceraso A, et al. (2021) Effectiveness, Core Elements, and Moderators of Response of Cognitive Remediation for Schizophrenia: A Systematic Review and Meta-analysis of Randomized Clinical Trials. *JAMA Psychiatry* 78(8): 848-858.
- Vöhringer PA and Perlis RH (2016) Discriminating Between Bipolar Disorder and Major Depressive Disorder. *Psychiatric Clinics* 39(1): 1-10.
- Volkert J, Gablonski T-C and Rabung S (2018) Prevalence of personality disorders in the general adult population in Western countries: systematic review and meta-analysis. *The British Journal of Psychiatry* 213(6): 709-715.
- Vos T, Lim SS, Abbafati C, et al. (2020) Global burden of 369 diseases and injuries in 204 countries and territories, 1990:2013;2019: a systematic analysis for the Global Burden of Disease Study 2019. *The Lancet* 396(10258): 1204-1222.
- Wade DT and Halligan PW (2004) Do biomedical models of illness make for good healthcare systems? *BMJ* 329(7479): 1398-1401.
- Wade TD, Bergin JL, Tiggemann M, et al. (2006) Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *Australian & New Zealand Journal of Psychiatry* 40(2): 121-128.
- Wald I, Lubin G, Holoshitz Y, et al. (2011) Battlefield-like stress following simulated combat and suppression of attention bias to threat. *Psychological Medicine* 41(4): 699-707.
- Walton MM (2015) Sexual equality, discrimination and harassment in medicine: it's time to act. *Medical Journal of Australia* 203(4): 167-169.
- Wand AP, Peisah C, Draper B, et al. (2018) Why do the very old self-harm? A qualitative study. *The American Journal of Geriatric Psychiatry* 26(8): 862-871.
- Wand APF, Draper B, Brodaty H, et al. (2019) Self-harm in the very old one year later: has anything changed? *International Psychogeriatrics* 31(11): 1559-1568.
- Watson HJ, Yilmaz Z, Thornton LM, et al. (2019) Genome-wide association study identifies eight risk loci and implicates metabo-psychiatric origins for anorexia nervosa. *Nature Genetics* 51(8): 1207-1214.
- Watson JB and Rayner R (1920) Conditioned emotional reactions. *Journal of Experimental Psychology* 3(1): 1.

- Weathers FW, Bovin MJ, Lee DJ, et al. (2018) The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5): Development and initial psychometric evaluation in military veterans. *Psychological Assessment* 30(3): 383.
- Weinberger DR, Glick ID and Klein DF (2015) Whither Research Domain Criteria (RDoC)? The Good, the Bad, and the Ugly. *JAMA Psychiatry* 72(12): 1161-1162.
- Weissman MM, Bland RC, Canino GJ, et al. (1994) The cross-national epidemiology of obsessive-compulsive disorder. *Journal of Clinical Psychiatry* 55: 5-10.
- Wells A (2005) The metacognitive model of GAD: Assessment of meta-worry and relationship with DSM-IV generalized anxiety disorder. *Cognitive Therapy and Research* 29: 107-121.
- Wesseloo R, Kamperman AM, Munk-Olsen T, et al. (2016) Risk of Postpartum Relapse in Bipolar Disorder and Postpartum Psychosis: A Systematic Review and Meta-Analysis. *American Journal of Psychiatry* 173(2): 117-127.
- West M and Coia D (2019) Caring for doctors, caring for patients. *General Medical Council*.
- Westbury JL, Gee P, Ling T, et al. (2018) RedUSe: reducing antipsychotic and benzodiazepine prescribing in residential aged care facilities. *Medical Journal of Australia* 208(9): 398-403.
- Westerman T (2021) Culture-bound syndromes in Aboriginal Australian populations. *Clinical Psychologist* 25(1): 19-35.
- Wetherell JL, Petkus AJ, McChesney K, et al. (2009) Older adults are less accurate than younger adults at identifying symptoms of anxiety and depression. *The Journal of Nervous and Mental Disease* 197(8): 623-626.
- Whiteford HA, Degenhardt L, Rehm J, et al. (2013) Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. *The Lancet* 382(9904): 1575-1586.
- Wijeratne C and Draper B (2011) Reformulation of current recommendations for target serum lithium concentration according to clinical indication, age and physical comorbidity. *Australian & New Zealand Journal of Psychiatry* 45(12): 1026-1032.
- Wikman A, Molloy GJ, Randall G, et al. (2011) Cognitive predictors of posttraumatic stress symptoms six months following acute coronary syndrome. *Psychology & Health* 26(8): 974-988.
- Wilkins G, Andrews G, Bell C, et al. (2019) Managing anxiety disorders in adults. *Medicine Today* 20(12): 12-22.
- Williams NR and Okun MS (2013) Deep brain stimulation (DBS) at the interface of neurology and psychiatry. *The Journal of Clinical Investigation* 123(11): 4546-4556.
- Wilson JE, Mart MF, Cunningham C, et al. (2020) Delirium. *Nature Reviews Disease Primers* 6(1): 90.

- Winnicott DW (1986) The theory of the parent-infant relationship. *Essential Papers on Object Relations*. 233-253.
- Winston AP (2020) Eating disorders and diabetes. *Current Diabetes Reports* 20: 1-6.
- World Health Organisation (2012) *Global burden of mental disorders and the need for a comprehensive, coordinated response from health and social sectors at the country level: report by the Secretariat*. Available at:
<https://iris.who.int/handle/10665/78898>.
- World Health Organisation and the United Nations (2023) *Mental health, human rights and legislation: guidance and practice*. Geneva: World Health Organisation and the United Nations (represented by Office of the High Commissioner for Human Rights).
- World Health Organization (2011) Global burden of mental disorders and the need for a comprehensive, coordinated response from health and social sectors at the country level: report by the Secretariat. Geneva: World Health Organization Retrieved from
<https://iris.who.int/handle/10665/78898>
- World Health Organization (2019) *International statistical classification of diseases and related health problems (11th Revision)*. Geneva: World Health Organisation.
- Wragg RE and Jeste DV (1989) Overview of depression and psychosis in Alzheimer's disease. *The American Journal of Psychiatry*.
- Wu H and Hilmer S (2015) How to treat: Delirium. *Australian Doctor*.
- Xia J, Merinder LB and Belgamwar MR (2011) Psychoeducation for Schizophrenia. *Cochrane Database of Systematic Reviews*. (6).
- Yehuda R, Southwick SM, Nussbaum G, et al. (1990) Low urinary cortisol excretion in patients with posttraumatic stress disorder. *The Journal of Nervous and Mental Disease* 178(6): 366-369.
- Yehuda RE and McFarlane AC (1997) *Psychobiology of posttraumatic stress disorder*. New York Academy of Sciences.
- Young-Southward G, Jackson A and Dunan J (2020) Group CBT for mild to moderate depression and anxiety: an evaluation of patient satisfaction within a primary care mental health team. *the Cognitive Behaviour Therapist* 13: e8.
- Young JT, Bellgrove MA and Arunogiri S (2021) Assessment of attention-deficit hyperactivity disorder in people with substance use disorder: Another case of what gets measured gets done. *Australian & New Zealand Journal of Psychiatry* 55(8): 744-746.
- Yule AM and Kelly JF (2019) Integrating treatment for co-occurring mental health conditions. *Alcohol Research: Current Reviews* 40(1).
- Yunxi Z, Yifan C, Xiaoying S, et al. (2024) Global, regional and national burdens of bipolar disorders in adolescents and young adults: a trend analysis from 1990 to 2019. *General Psychiatry* 37(1): e101255.

- Zhang K, Hu X, Ma L, et al. (2023) The efficacy of schema therapy for personality disorders: a systematic review and meta-analysis. *Nordic Journal of Psychiatry* 77(7): 641-650.
- Zhu Y, Li C, Huhn M, et al. (2017) How well do patients with a first episode of schizophrenia respond to antipsychotics: A systematic review and meta-analysis. *European Neuropsychopharmacology* 27(9): 835-844.
- Zimmerman M, Galione JN, Chelminski I, et al. (2012) Does the diagnosis of multiple Axis II disorders have clinical significance? *Annals of Clinical Psychiatry* 24(3): 195-201.
- Zipfel S, Giel KE, Bulik CM, et al. (2015) Anorexia nervosa: aetiology, assessment, and treatment. *The Lancet Psychiatry* 2(12): 1099-1111.
- Zipfel S, Wild B, Groß G, et al. (2014) Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): randomised controlled trial. *The Lancet* 383(9912): 127-137.
- Zohar J, Juven-Wetzler A, Sonnino R, et al. (2011a) New insights into secondary prevention in post-traumatic stress disorder. *Dialogues in Clinical Neuroscience* 13(3): 301-309.
- Zohar J, Yahalom H, Kozlovsky N, et al. (2011b) High dose hydrocortisone immediately after trauma may alter the trajectory of PTSD: interplay between clinical and animal studies. *European Neuropsychopharmacology* 21(11): 796-809.

Index

- ACE model, 27, 29, 227, 228
- Activity, Cognition and Emotion (ACE) model, 204
- acute stress disorder, 265
- adaptation, 10, 36, 299, 345, 380, 397, 513, 523, 526, 527
- ADHD, 335, 355, 357, 367, 370, 371, 372, 373, 374, 381, 440, 539, 540
- aetiology, 28, 87, 97, 126, 179, 181, 185, 192, 196, 201, 206, 223, 243, 246, 250, 258, 261, 348, 353, 377, 378, 379, 398, 431, 457
- affective instability, 230, 291
- agoraphobia, 240, 244, 245, 246, 248, 249, 253, 481
- alcohol use disorder, 440, 447
- Alzheimer's disease, 59, 60, 61, 62, 66, 67, 161, 162, 401, 402
- anorexia nervosa, 272, 273, 274, 276, 277, 278, 279, 280, 282, 283, 380, 415, 433, 452
- antidepressants, 27, 202, 210, 211, 212, 213, 214, 215, 235, 252, 253, 256, 260, 335, 344, 369, 381, 387, 388, 435, 457, 531, 535, 536, 537, 543, 551
- antipsychotic medications, 181, 185, 193, 370, 381, 403, 532, 533
- antipsychotics, 28, 46, 114, 194, 235, 330, 350, 387, 389, 393, 394, 399, 400, 403, 412, 418, 435, 452, 531, 532, 533, 535, 539, 542
- anxious ambivalent attachment, 171
- anxious avoidant attachment, 171
- apraxia, 68
- attachment theory, 8, 172, 174
- auditory hallucinations, 108, 187, 370, 393, 410, 551
- Australian Aboriginal and Torres Strait Islander, 15
- Australian Health Practitioner Regulation Agency, 11, 16
- avoidance behaviour, 472, 477, 503, 504, 505
- avoidant/restrictive food intake disorder, 272
- behavioural activation, 472, 474, 479, 485, 487, 489, 490, 491, 492, 493
- behavioural experiments, 502, 503, 505
- benzodiazepines, 193, 252, 253, 271, 300, 393, 398, 400, 410, 412, 417, 420, 531, 537
- binge eating disorder, 272, 273, 282, 283, 452, 539
- biomedical model, 25, 72, 179, 329
- biopsychosocial, 26, 71, 74, 168, 172, 192, 251, 342, 359, 383, 391, 508, 512, 540
- blood-injection-injury phobia, 248, 249
- Body dysmorphic disorder, 256, 261
- borderline personality disorder, 288, 289, 290, 291, 293, 295, 297, 298, 299, 300, 301, 302, 304, 396, 414, 415, 528, 529
- boundaries, 4, 22, 24, 27, 102, 137, 294, 297, 378, 516, 522
- bulimia nervosa, 272, 273, 274, 279, 280, 282, 283, 452
- burnout, 12, 13, 15, 20
- cardiovascular disease, 198, 243, 304, 312, 450, 452, 454
- case-cohort study, 144

- catatonia, 90, 114, 116, 189, 388, 413, 538
- catatonic, 46, 116, 119, 346, 347, 348, 545, 550
- Child and Adolescent Mental Health Service, 359
- chronic pain, 43, 413, 450, 452, 455, 456
- classical conditioning, 475, 476
- clinical epidemiology, 141, 144
- Cluster B personality disorders, 290
- Cluster C personality disorders, 289, 298
- cognitive behaviour therapy, 9, 252, 269, 298, 529
- cognitive behavioural therapy, 8, 209, 210, 260, 305, 358, 386, 397, 435
- cognitive challenging, 463
- cognitive impairment, 39, 56, 59, 61, 70, 229, 276, 293, 335, 386, 392, 395, 396, 397, 398, 439, 553
- cognitive processing, 171
- cognitive testing, 39, 47, 69, 111, 393
- cognitive therapy, 254, 298, 472, 492, 493, 494, 495, 496, 497, 501
- community treatment order, 83, 84, 541
- comorbidity, 8, 9, 77, 265, 292, 387, 396, 434, 440, 441, 444, 450, 451, 452, 453, 455, 457
- compensatory behaviours, 275, 276, 280, 281
- confidence interval, 163
- control group, 13, 144, 156
- conversion disorder, 52, 123, 427
- countertransference, 170, 174, 176, 297, 414, 523, 525, 526, 527
- cultural competence, 323, 331
- decision-making capacity, 46, 82, 83, 88
- delirium, 56, 69, 70, 90, 107, 112, 347, 358, 386, 387, 392, 394, 397, 398, 399, 400, 416, 417, 418, 550
- delusions, 24, 42, 45, 53, 54, 55, 79, 100, 104, 105, 106, 121, 122, 132, 179, 187, 188, 189, 196, 295, 346, 348, 389, 393, 398, 402, 413, 425, 435, 436
- dementia, 51, 56, 60, 61, 62, 66, 67, 112, 133, 150, 188, 222, 261, 304, 328, 386, 387, 392, 393, 394, 396, 397, 398, 400, 401, 402, 403, 404, 410, 416, 417, 484, 493
- dependence, 17, 168, 173, 217, 252, 253, 293, 300, 301, 336, 443, 447, 537
- depersonalisation, 111, 526
- derealisation, 111
- developmental milestones, 353
- diagnostic criteria, 8, 133, 136, 148, 272, 274, 275, 355, 370, 371, 395, 440, 441
- diagnostic test, 161
- dialectical behaviour therapy, 297, 305, 381, 397
- dopamine, 118, 181, 185, 348, 372, 447, 531, 532, 533, 535, 542, 548
- DSM-5-TR, 22, 27, 200, 224, 225, 226, 240, 264, 275, 313, 324, 326, 337, 341, 346, 353, 400, 428, 429, 430, 434, 436, 438, 460
- dysphoria, 95, 297, 304, 389, 396
- eating disorders, 8, 272, 276, 277, 281, 282, 291, 296, 366, 415, 433, 452, 454, 494
- effect size, 163, 185, 468, 546, 551

- electroconvulsive therapy, 42, 215, 413, 531
- Electroconvulsive therapy, 236, 344, 545
- emotional dysregulation, 273, 288, 294, 296
- emotional regulation, 278, 298
- epilepsy, 43, 244, 422, 429, 538, 539
- euthymia, 95, 221, 389
- Excoriation disorder, 256, 262
- executive functions, 58, 60, 64
- experimental studies, 145
- exposure and response prevention, 260, 435
- Externalising problems, 355
- extinction, 267, 268, 269, 270, 455, 479
- family therapy, 196, 360, 380
- fear conditioning, 266, 267, 268
- fight-flight response, 519
- formulation, 5, 7, 32, 71, 72, 73, 80, 85, 172, 215, 218, 374, 413, 414, 469, 512, 513, 514, 515, 540
- frontal lobe dysfunction, 64, 65
- functional impairment, 24, 115, 200, 201, 204, 226, 242, 245, 248, 249, 258, 259, 305, 353, 370, 393, 397, 398
- generalised anxiety disorder, 240, 259, 345, 443
- genetic factors, 201, 223, 338
- genetic predisposition, 71, 243, 246, 248, 250, 304, 348, 395
- genogram, 360, 361
- habituation, 481
- hallucinations, 24, 42, 45, 47, 55, 79, 104, 105, 107, 108, 110, 111, 121, 122, 179, 187, 196, 295, 326, 327, 348, 350, 364, 393, 394, 398, 402, 410, 417, 418
- hermeneutics, 134, 135
- Hoarding disorder, 256, 261
- holistic care, 444
- HPA axis, 202, 268, 271, 341
- hypnotics, 537, 538
- hypomanic, 205
- hypothesis testing, 163
- ICD-11, 27, 28, 29, 133, 224, 288, 294, 295, 324, 353
- illusions, 55, 107, 108, 417, 418
- impulse control, 261, 367
- informed consent, 164, 216, 399
- insight, 27, 33, 45, 46, 47, 57, 77, 82, 86, 88, 89, 112, 121, 189, 221, 239, 257, 259, 279, 348, 391, 453
- insomnia, 75, 205, 250, 264, 369, 460, 462, 463, 464, 465, 493, 537, 538
- intergenerational trauma, 319, 320
- Internalising problems, 355
- intrusive thoughts, 54, 104
- involuntary treatment, 7, 81, 82, 83, 216, 329, 414, 549
- John Bowlby, 167
- Karl Jaspers, 86, 87, 130
- Kurt Schneider, 133
- Lewy body dementia, 60, 67
- LGBTIQ+, 14
- lithium, 42, 215, 218, 222, 234, 235, 236, 237, 330, 347, 348, 349, 388, 392, 412, 457, 531, 532, 539
- longitudinal study, 144, 147, 156
- Ludwig Binswanger, 132
- major depressive disorder, 142, 143, 200, 201, 203, 204, 205, 209, 211, 250, 271, 337, 338, 340, 353, 384, 410, 440, 443, 538

- mania, 26, 90, 94, 95, 97, 136, 188, 221, 222, 223, 224, 225, 226, 227, 229, 230, 231, 232, 233, 234, 236, 370, 382, 388, 389, 392, 394, 398, 408, 410, 413, 548
- manic episode, 29, 205, 224, 545, 546
- manic-depressive illness, 222, 538
- medical students, 4, 5, 11, 12, 13, 14, 16, 19, 20, 32, 33, 36
- Mental Health Act, 7, 81, 82, 83, 216, 348, 541, 549
- Mental Health Review Tribunal, 83
- mental state examination, 7, 39, 49, 50, 56, 79, 85, 87, 88, 130, 190, 364, 393
- meta-analysis, 149, 159, 399, 440, 451, 468, 492, 542
- Mild Cognitive Impairment, 60, 61
- Mini-Mental State Examination, 59
- mixed mood states, 26, 27
- monoamine oxidase inhibitors, 202, 253, 535
- Montreal Cognitive Assessment, 56, 59, 60, 401
- mood stabilising agents, 235
- multidisciplinary team, 196, 199, 281
- multiple sclerosis, 60, 422, 432
- Mutual Maintenance Model, 455
- Narcissistic personality disorder, 288
- narcolepsy, 116, 460, 539
- negative symptoms, 51, 188, 189, 190, 551
- negative thoughts, 54, 264, 495, 501, 502, 503, 505
- neurodegenerative disease, 383
- neurodegenerative disorders, 58, 59, 63, 66, 67
- neurodevelopmental disorders, 22, 185, 358, 381, 539
- nosology, 126, 133, 138, 181, 222
- obesity, 149, 330, 452, 454, 456, 462, 468
- observational studies, 156, 160
- obsessional jealousy, 262
- obsessive-compulsive disorder, 240, 273, 283, 494
- Olfactory reference syndrome, 256, 263
- operant conditioning, 475, 477, 479, 484, 490
- orientation, 35, 56, 59, 61, 62, 63, 67, 111, 296, 365, 366, 401, 417, 468, 469, 515
- panic attack, 241, 242, 248, 494, 538
- panic disorder, 121, 240, 242, 243, 245, 253, 254, 268, 345, 452, 503
- Parkinson's disease, 67, 90, 116, 117, 118, 120, 393
- perceptual disturbances, 55, 56
- perinatal, 9, 176, 333, 334, 337, 339, 340, 341, 342, 343, 344, 345, 352
- perinatal period, 9, 333, 334, 337, 339, 343, 344, 345
- perpetuating factors, 76, 78, 383
- pharmacotherapy, 198, 207, 215, 233, 234, 252, 253, 270, 369, 370, 374, 387, 399, 432, 433, 474, 546
- phenomenology, 7, 85, 87, 88, 89, 100, 107, 127, 128, 131, 134, 136, 137, 140, 204, 364, 438
- Pica, 272, 283
- positive symptoms, 158, 186, 187, 190, 350, 393, 551
- precipitating factors, 75
- predisposing factors, 73, 74, 75, 79

- primary care, 7, 9, 202, 208, 242, 288, 384, 509
- professional identity, 11, 18
- protective factors, 72, 77, 78, 141, 379
- psychiatric assessment, 49, 135, 276, 323, 393, 414, 444
- psychodynamic, 8, 9, 209, 220, 252, 282, 297, 299, 475, 521, 522, 523, 526, 528, 529
- psychoeducation, 196, 207, 209, 217, 232, 233, 253, 299, 300, 342, 351, 359, 397, 462, 463, 465, 472, 515, 519
- psychomotor agitation, 50, 52, 116, 224
- psychomotor retardation, 90, 94
- psychosocial factors, 181, 204, 383, 453
- psychotropics, 383, 399, 403
- qualitative, 130, 144, 154, 159
- quantitative, 130, 154, 155, 159
- randomised controlled trial, 157, 299, 492
- refeeding syndrome, 279
- reinforcement, 298, 368, 376, 377, 472, 477, 478, 487, 488, 489, 490
- relapse, 79, 186, 193, 199, 252, 253, 270, 343, 346, 348, 349, 369, 370, 435, 440, 446, 448, 472, 493, 506, 548
- reliability, 27, 139, 155, 160, 401, 523
- rumination, 272, 472, 488, 492
- rumination disorder, 272
- schema therapy, 176, 397
- schemas, 298
- schizoaffective disorder, 27, 28, 184
- screening tools, 58, 59, 62, 67, 70, 340, 341, 385, 398
- sedation, 193, 212, 213, 387, 389, 394, 400, 411, 417, 454, 493
- sedatives, 120, 537, 538
- selective serotonin reuptake inhibitor, 260
- serotonin and noradrenaline reuptake inhibitors, 211, 253
- serotonin hypothesis, 202
- sick role, 173, 424, 425, 426, 431, 436
- sleep apnoea, 460, 462
- sleep diary, 462, 465
- sleep hygiene, 9, 75, 77, 78, 209, 231, 342, 461, 462, 463, 464, 465, 517
- sleep restriction, 463, 464
- sleep-wake disorder, 460
- social anxiety disorder, 189, 240, 246, 247, 248, 253, 254, 345, 434, 479
- specific phobia, 240, 245, 248, 253
- statistical significance, 149, 163, 164
- stepped care, 444, 530
- stimulants, 230, 372, 440, 539
- stimulus control, 463, 464
- stress diathesis model, 202, 295
- stress response, 139, 168, 174, 265, 267, 337, 519
- stress-diathesis model, 337
- Structured Problem Solving, 467, 468, 469
- substance use disorder, 43, 81, 247, 265, 353, 439, 440, 441, 443, 444
- suicidal ideation, 15, 40, 49, 53, 88, 205, 291, 367, 468, 518
- systematic review, 12, 155, 158, 159, 176, 451
- tardive dyskinesia, 50, 189, 193, 197
- therapeutic alliance, 172, 173, 176, 370

- therapeutic relationship, 5, 32, 78,
231, 281, 449, 511, 515, 521,
522, 524, 525, 526, 528
- thought monitoring, 496
- tolerance, 212, 298, 443, 522, 526
- Transcranial magnetic stimulation,
551
- transference, 170, 174, 176, 293,
297, 299, 414, 523, 524, 525,
526, 527
- treatment resistant, 385, 388, 521,
539, 546
- tremor, 91, 116, 119, 120, 197,
213, 250, 428, 538
- trichotillomania, 261, 262
- tricyclic antidepressants, 213
- validity, 27, 139, 142, 147, 151,
160, 181, 204, 288, 341, 353,
355, 395, 401, 499, 502, 505
- visual processing, 67, 434
- withdrawal, 55, 90, 102, 106, 120,
122, 134, 171, 184, 186, 187,
190, 252, 293, 295, 345, 358,
386, 393, 400, 408, 409, 418,
419, 443, 447, 472, 537, 538
- working memory, 58, 59, 61, 63