Chapter 26: Old Age Psychiatry

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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 themself 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., 2018). 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 extra-pyramidal 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 (Dombrovski 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.

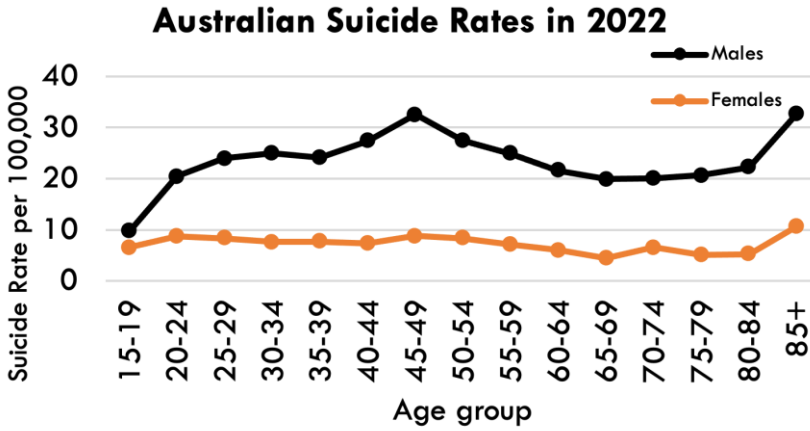

Figure 26.1. Suicide rates in Australia in 2022 (rate per 100,000).

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 themself, 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 Snowdon, 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 (Granholm 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., 2011).

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., 2011). 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., 2011). 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. < 3rd 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**

Cite as:

Peisah, C., Snowdon, J., Stevenson, J., Narchal, S., Wand, A. (2024). Old age psychiatry. In Boyce, P., Harris, A., and Malhi, G.S. (Eds.), *The Sydney textbook of psychiatry* (pp. 383–406). The University of Sydney.

References

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.

Aizenstein HJ, Baskys A, Boldrini M, et al. (2016) Vascular depression consensus report–a critical update. *BMC medicine* 14: 1-16.

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.

American Psychiatric Association (2022) *Diagnostic and statistical manual of mental disorders : DSM-5-TR.* Washington, DC: American Psychiatric Association Publishing.

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.

Beunders AJ, Orhan M and Dols A (2023) Older age bipolar disorder. *Curr Opin Psychiatry* 36(5): 397-404.

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.

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.

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*.

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).

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.

Colijn MA, Nitta BH and Grossberg GT (2015) Psychosis in later life: a review and update. *Harvard review of psychiatry* 23(5): 354-367.

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*.

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, Snowdon J, et al. (2013) Contacts with health professionals before suicide: missed opportunities for prevention? *Comprehensive Psychiatry* 54(7): 1117-1123.

Diefenbach GJ and Goethe J (2006) Clinical interventions for late-life anxious depression. *Clinical Interventions in Aging* 1(1): 41-50.

Dombrovski AY and Mulsant BH (2007) ECT: the preferred treatment for severe depression in late life. *International Psychogeriatrics* 19(1): 10-14.

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.

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.

Fong TG, Tulebaev SR and Inouye SK (2009) Delirium in elderly adults: diagnosis, prevention and treatment. *Nature Reviews Neurology* 5(4): 210-220.

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.

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.

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.

Kalish VB, Gillham JE and Unwin BK (2014) Delirium in older persons: evaluation and management. *American Family Physician* 90(3): 150-158.

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.

Kurrle S, Brodaty H and Hogarth R (2012) *Physical comorbidities of dementia.* Cambridge University Press.

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.

Laver K, Cumming R, Dyer S, et al. (2016) Clinical practice guidelines for dementia in Australia.

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.

Lonergan E, Luxenberg J and Sastre AA (2009) Benzodiazepines for delirium. *Cochrane Database of Systematic Reviews*.(4).

Maglione JE, Thomas SE and Jeste DV (2014) Late-onset schizophrenia: do recent studies support categorizing LOS as a subtype of schizophrenia? *Curr Opin Psychiatry* 27(3): 173.

Malhi GS, Outhred T, Hamilton A, et al. (2018) 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.

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.

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.

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.

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.

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.

Nagendra J and Snowdon J (2020) An Australian study of delusional disorder in late life. *International Psychogeriatrics* 32(4): 453-462.

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.

NSW Heath (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.

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'Neill N and Peisah C (2021) Capacity and the Law. 4 ed.: Australasian Legal Information Institute (AustLII) Communities.

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.

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, Forlenza O and Chiu E (2009) Ethics, capacity, and decision-making in the practice of old age psychiatry: an emerging dialogue. *Curr Opin Psychiatry* 22(6): 519-521.

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.

Reinhardt MM and Cohen CI (2015) Late-life psychosis: diagnosis and treatment. *Current psychiatry reports* 17: 1-13.

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.

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.

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.

Shneidman E (1999) Perturbation and lethality: A psychological approach to assessment and intervention.

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.

Snowdon J, Draper B, Brodaty H, et al. (2010) Prevalence and treatment of late life depression. *Australian and 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.

Steptoe A, Deaton A and Stone AA (2015) Subjective wellbeing, health, and ageing. *The Lancet* 385(9968): 640-648.

Stevenson J, Datyner A, Boyce P, et al. (2011) The effect of age on prevalence, type and diagnosis of personality disorder in psychiatric inpatients. *International journal of geriatric psychiatry* 26(9): 981-987.

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.

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.

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.

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.

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.

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.

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.

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.

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.

Wilson JE, Mart MF, Cunningham C, et al. (2020) Delirium. *Nature Reviews Disease Primers* 6(1): 90.

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.