

Dental Implications for the Patient with Mental Illness

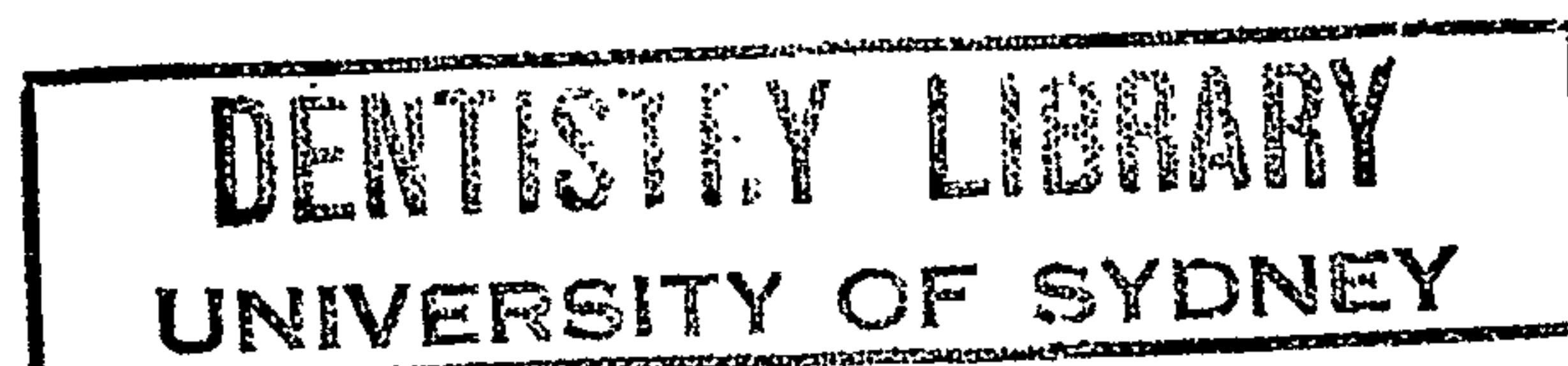
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Summary

Mental illness is prevalent in the modern society. The psychiatric morbidity in general illness has been recognised and has resulted in the growth of consultation-liaison psychiatry. The extent of psychiatric disorders encountered in dentistry is less well documented although the possibility of a hidden psychiatric morbidity in dentistry exists.

The writer has elucidated on the psychiatric morbidity in dentistry and has presented it as a treatise. The aims of this treatise are to give a short account of mental illness (the more common types), discuss the orofacial manifestations of mental illness, review the mental illness of significance to the dentist and examine the side effects of psychotropic medications that can either bring about considerable dental disease or cause significant problems to both the dentist and patient during dental procedures.

Psychogenic facial pain and oral self-mutilation may be presented as a result of mental illness or some deep-rooted psychological factors. Perimyolysis, parotid gland enlargement and hirsutism has been recognised to be the orofacial manifestations of the eating disorders. Bruxism considered to be a parafunctional behaviour with psychogenic overtones may have many deleterious effects on the dentition. Psychological factors need to be considered when constructing full dentures for certain patients.

Certain psychiatric disorders such as dental phobia and hypochondriasis can complicate dental treatment for both the patient and dentist. The dentist has a role in the recognition of child abuse.

Many side-effects of psychotropic medications are of significance to the dentist during dental procedures. The most important ones are that of orthostatic hypotension and excessive sedation. Tardive dyskinesia as a result of antipsychotics will make it difficult for patients to wear full dentures. Xerostomia caused by psychotropic medications can bring about rampant dental caries.

Patients with mental illness have a unique set of factors (as a result of both the nature of the illness and psychotropic medications) that predispose them to a high level of dental disease. There is a need for the psychiatrist and dentist to work together as a team in the management of some of these patients.

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MFY, 11th November, 1991

Dedication

To my father and in memory of my mother.

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List of Abbreviations

BDI	Beck Depression Inventory
CMI	The Cornell Medical Index
DAS	Dental Anxiety Scale
def	Decayed Missing or Filled teeth (in deciduous dentition)
DMF	Decayed Missing or Filled teeth (in permanent dentition)
DBS	Dental Beliefs Survey
DDP	Difficult denture patient
DSM	Diagnostic and Statistical Manual of Mental Disorders
DSM-II	DSM second revision
DSM-III	DSM third revision
DSM-III-R	DSM fourth revision
EPQ	Eysneck Personality Questionnaire
HRC	Help-rejecting complainer
ICD-9	International Classification of Diseases, ninth edition
ICD-10	International Classification of Diseases, tenth edition
MAOI	Monoamine oxidase inhibitor
MHP	Monosymptomatic hypochondriacal psychosis
NAI	Non-accidental injury
NSW	New South Wales
PB	Phantom bite
TD	Tardive dyskinesia
TMJ	Temperomandibular joint

1 Introduction

The incidence of mental illness in the Western world is quite well documented. Approximately 35 million Americans (almost one out of every six) suffer some form of diagnosable mental or psychiatric illness, ranging from a mild neurosis to more serious disturbances, such as schizophrenia or an affective disorder like manic depression (Quock 1985). Surveys of urban communities in the United States have indicated that the six month total prevalence rates for psychiatric disorders is between 11.6% and 13.2% (Myers et al 1984). The lifetime prevalence rates (prevalence during the entire lifetime before interview, irrespective of status at the time of survey) was reported to be as high as 25.2%. In the United States patients with psychiatric disorders occupy approximately 25 % of all hospital beds (Quock 1985).

The Australian Bureau of Statistics published '**Australian Health Survey 1977-78: Chronic Conditions (illness and permanent disabilities)**' which stated that of the population who suffer from some sort of chronic condition, 2.5 % suffer chronically from some sort of mental disorder (this includes behaviour disorders of childhood but not mental retardation or alcoholism).

In the 1983 Australian Health Survey, a study on the illness conditions experienced by the Australian community reported that 3.46 % of the population experience some sort of mental illness during the 2 weeks prior to being interviewed for the study (Australian Bureau of Statistics 1983). Institutionalised persons, overseas visitors on holiday in Australia, students at boarding schools, members of Australian permanent defence forces living on military bases, non-Australian servicemen stationed in Australia and their dependents, diplomatic personnel of overseas governments and non-Australian members of their households, were excluded from this study.

The 'Census of Population and Housing 30 June 1986: Characteristics of In-patients of Health Institutions Australia' stated that 14,630 of the population were in-patients of Psychiatric hospitals or institutions — this is 8.55 % of the 171,140 persons who were counted as in-patients of health institutions (Australian Bureau of Statistics 1986).

In 1987, the Australian Bureau of Statistics presented the following figures for New South Wales:

There were 6,940 "in-patients" in mental health and developmental disability institutions and units.

Approximately 31 % (2,143) were "in-patients" in public psychiatric hospitals.

The most common provisional diagnosis attributed to the "in-patients" in public psychiatric hospitals were schizophrenia-paranoia (20 %) and depression (9 %) (Australian Bureau of Statistics 1987).

The New South Wales Association for Mental Health (1984) published that studies suggest about 20 % of people at any time could be given a psychiatric diagnosis.

The medical profession has recognised the interplay between psychological and organic factors in the causation and presentation of physical illness. Psychological distress (factors) may cause relapse or aggravation of physical symptoms (Scott and Humphreys 1987). Some symptoms may be due entirely to an underlying psychological disorder/psychiatric illness (Evans 1989). This has resulted in the

growth of consultation-liaison psychiatry. Lipowski (1983) put forward the definition of consultation-liaison psychiatry which is widely accepted for its accurate depiction. He proposed that consultation-liaison psychiatry is:

a sub-speciality of psychiatry, one concerned with—

- i) diagnosis, treatment, study and prevention of psychiatric morbidity among physically ill patients and those who somatise, that is, communicate their emotional distress in the form of somatic symptoms and seek medical help for them, and*
- ii) the provision of psychiatric consultation, liaison and teaching of non-psychiatric health workers in all types of health care settings, but especially in the general hospital."*

Consultation occurs when a psychiatrist responds to the request by another health professional to assist with diagnosis and/or management for a patient under the care of this health professional. Liaison refers to an attachment of the psychiatrist over an extended period to a particular non-psychiatric ward or unit for the purposes of case finding, education and consultation (Barrow 1989).

The main reason underlying this type of psychiatric activity is the prevalence of psychiatric disorder in non-psychiatric settings e.g. in the medical wards of general hospitals (Goldberg 1984). Figures as high as 20 % have been indicated for patients of such wards to have a recognisable psychiatric disorder of sufficient proportion either

to account for their admission or to complicate significantly medical, surgical, or other treatment during hospitalisation (Barrow 1989).

There is also a high prevalence of psychiatric illness in general medical practice. In the United Kingdom, Shepherd and co-workers (1984) considered that 25-33 % of general practice patients fit into categories of mental illness and that 14 % of the total number of patients in a practice consult each year with a condition largely or entirely psychiatric. The majority of these patients have a neurotic disorder or a personality disorder. Other earlier workers also quote similar figures. Nielsen and Williams (1980) used the Beck Depression Inventory (BDI) to measure the prevalence of depression in 526 ambulatory patients. They found that 12.2% of this population had mild depression defined by a cutoff score of 13 or more. A review of the medical charts showed that the primary physicians failed to diagnose 50% of these depressed patients. Five hundred and fifty-three consecutive attendees at a general medical practice in London were screened and 20% were identified with a '**conspicuous**' psychiatric disorder (Goldberg and Blackwell 1970). Chancellor and other workers (1977) in an Australian study reviewed patients attending their general practice and found that 17.3 % had emotional or neurotic illness.

Mental (psychiatric) illness is prevalent in our society. It is also recognised that mental illness may play an important and significant role in the presentation of physical symptoms of illness. In general medical practice, it is found that a significant number of patients complained of physical symptoms which are due entirely to mental illness.

The extent of psychological problems or psychiatric illness encountered in the dental practice is less well documented. However, it has been recognised that psychological factors play an important role in the genesis of certain dental problems such as atypical facial pain and temporomandibular joint dysfunction (Moulton 1955, Laskin 1969,

Feinmann and Harris 1984a). Using psychometric measures many researchers have reported high levels of neuroticism, introversion, and depression in some of these dental patients. Hughes and co-workers (1989) conducted a study on 138 attendees of a Psychiatric Pain Clinic at the Glasgow Dental Hospital and School. They found that 91% of these patients had a clinically important psychiatric disorder. These diagnoses were assigned according to the DSM-III (Diagnostic and Statistical Manual of Mental Disorders, third revision) classification devised by the American Psychiatric Association. The possible hidden psychiatric morbidity in dental practice emphasises the importance of, and need for, liaison between psychiatrists and dentists regarding certain dental problems.

It is also imperative that the dental profession is aware of dental problems that can arise for patients receiving certain psychotropic medications. Some of these medications are well known for their anticholinergic effects which can result in hyposalivation and chronic xerostomia (Felder et al 1988). Xerostomia can result in rampant caries. The prevalence of psychiatric morbidity in the dental practice may be unrecognised because patients present with somatic symptoms alone (Hughes et al 1989). Thus, the attitude and awareness of the dentist are often crucial in detection and treatment of the dental problem (Scott and Humphreys 1987). This will not be an easy task as dentists rarely receive any training in psychiatry in undergraduate or postgraduate studies (Feinmann and Harris 1984b).

The writer heads a group of six dentists in the Community Dental Health Unit at the Westmead Hospital Dental Clinical School who often treat patients with mental illness. She needs to know about the nature of the illnesses of these patients in order to devise comprehensive dental treatment plans. She wishes to investigate and evaluate the interplay between dentistry and mental illness/psychiatric disorders. There is a need for a document to enable her (and other dentists in similar situations) to understand the

psychiatric component in dentistry and thus improve management of these patients. As a result she has decided to undertake the preparation of such a document and present it as a treatise.

The aims of this treatise are:

To provide a short background about mental illness.

To discuss the most common orofacial manifestations of mental illness.

To review some mental illness/psychiatric disorders which may be encountered in general dental practice.

To review the side effects of psychotropic medication which can bring about significant dental disease and/or problems.

To review the side effects of psychotropic medication which are of significance to the dentist during dental procedures.

With these aims, the writer wishes to highlight the need to recognise the hidden psychiatric morbidity in dentistry, and promote the concept of the psychiatrist and the dentist working together as a team in the management of certain patients. This concept has also been proposed by others (Blasberg et al 1983, Simmons et al 1986, Hughes et al 1989, Donlon et al 1990).

2 What Is Mental Illness?

An 'illness' of any kind can be defined as the unhealthy condition of the body (The Concise Oxford Dictionary 1984a). The World Health Organization (1965) defined 'health' as,

"a state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity."

There are many other definitions proposed for both illness and health. No single definition has been found to be totally satisfactory and comprehensive (Finlay-Jones 1989, Gelder et al 1989a). The definition of an illness has varied from era to era and from country to country (Finlay-Jones 1989).

Just as 'illness' is a difficult word to define, the term 'mental illness' is as elusive to illustrate. However, in general terms, mental illness is a disorder of the functioning of the mind. The New South Wales Association for Mental Health (1984) explained that:

"Mental illness is a general term which refers to a group of illness affecting the functioning of the mind in the same way that heart disease refers to a group of illness which affect the functioning of the heart."

The concept of mental illness is complicated and usually does not need to be defined for most purposes, but the law requires psychiatrists to diagnose the presence or absence of mental illness (Gelder et al 1989a). This is in relation to mandatory admission to hospital and certain court procedures.

2.1 Classification

As in other branches of medicine, systems of classification are essential in psychiatry (Gelder et al 1989a, Russell 1989). In the clinical practice of psychiatry, there is a great diversity of signs and symptoms. The purpose of classification is to identify groups of patients who share similar clinical features in order to plan the appropriate treatment needed and to predict the course and prognosis of the mental illness. Classification of mental illness will also enable doctors and other health professionals involved, to communicate more effectively and easily about patients. Research can also be conducted with comparable groups of patients.

In general medicine, classification is more straightforward than in mental illness/psychiatric disorders. Most medical problems can be classified on the basis of aetiology (for example, viral or bacterial) and of structural pathology (for example, lobar or bronchopneumonia) (Gelder et al 1989a).

However, mental illness, like some general medical conditions such as migraine, are classified solely on symptoms (Gelder et al 1989a, Russell 1989). A psychiatric diagnosis is usually syndromal as the diagnosis refers to a group of signs and symptoms with a consistent relationship to each other and are indicative of a common course and prognosis. The aetiology and pathogenesis remains obscure.

Many systems of classification exist in psychiatry as no classification presently available is entirely satisfactory. In English speaking countries, the 2 major international classification systems are the ICD-9 and DSM-III-R (Russell 1989).

The DSM-III-R is devised by the American Psychiatric Association. When this classification first appeared in 1952 it was known as the Diagnostic and Statistical

Manual of Mental Disorders (DSM). In an effort to improve its reliability and validity, this classification system has undergone several revisions and were published subsequently as DSM-II and then DSM-III. In 1987, the DSM-III-R was published.

The ICD-9 (International Classification of Disease: Mental Disorders Ninth Revision) has its origins dating back to 1900 in Paris where the First Revision Conference of the International List of Causes of Death was held. However, it was only in 1938 when a section for mental disorders first appeared in the Fifth Revision of this list. Accordingly, many more revisions were made and by 1979, the present ICD-9 was adopted. The ICD-10 is currently in preparation by the World Health Organization in conjunction with psychiatric organisations of many countries.

For the purpose of this treatise, the writer feels that a more thorough explanation of the DSM-III-R and ICD-9 is not relevant. It is important, however, to stress that even though the importance of classification in psychiatric/mental illness is recognised, there are also many strident criticisms voiced. One of the main criticisms has been that there are patients who do not fit neatly into the available categories and the less experienced clinician may strive to fit these patient's symptoms into inappropriate clinical groupings (Gelder et al 1989a, Russell 1989). This criticism is important but this argument can only be applied to improper use of classification. Other criticisms, including that the labelling of patients can attract social stigma, have diminished as particular syndromes have been shown to respond to specific treatments and the prognosis is as predicted. Classification promotes sharing of clinical information for teaching, discussion, and research purposes, as it is a means of organising, assessing and retrieving information for the study of diagnostic entities. Mental disorder cannot be made to be obsolete simply by not giving names to them (Gelder et al 1989a). It is only through public education and awareness that empathy and understanding is

developed towards sufferers of mental disorders to lessen the social stigma that surrounds them.

2.2 A synopsis of a simplified form of classification of mental/psychiatric illness

For the purpose of this treatise, a simplified system of classification based on a medical model of psychiatric illness written by Russell (1989), is used. Russell (1989) felt that this system of classification is sufficient to classify most disorders the clinician is likely to encounter in a general psychiatric setting. The writer feels that a synopsis of this system of classification will be adequate knowledge required by the dentist to begin to recognise and understand the correlation of certain dental problems and mental illness. In Russell's classification there are six main categories:

1. organic brain syndromes,
2. schizophrenia,
3. affective disorders,
4. neuroses,
5. personality disorders, and
6. miscellaneous disorders.

The following information on the six main categories of mental illness is summarised mainly from Russell's (1989) classification unless stated otherwise.

2.2.1 Organic Brain Syndromes

The organic brain syndromes are an exception to the rule that psychiatric diagnoses are made in reference to a group of related symptoms and signs which indicate a common natural history as they represent a pathological process resulting in dysfunction or death of neurones. This physical disturbance of brain functioning is associated with cognitive impairment. There are 2 main types of organic brain syndromes:

1. the acute form, and
2. the chronic form.

2.2.1.1 *The acute form*

The acute form of organic brain syndrome is sudden in onset and is usually of a short duration (often less than a week but can also last for months). Following recovery, it is usually recalled as a bad dream.

This form of organic brain syndrome described as delirium is characterised by disturbed consciousness, perception, orientation, speech, and psychomotor activity. The feeling of fear and suspicion is usually present. Common causes of delirium are:

1. drug intoxication and drug sensitivity and poison,
2. withdrawal states from alcohol, sedatives, anxiolytics,
3. metabolic disturbances, for example, uraemia, hepatic failure,
4. endocrine disturbances, for example, hypoglycaemia, hypothyroidism,
5. nutritional and vitamin deficits,
6. systemic infections such as septicaemia,

7. intracranial infections such as meningitis,
8. anoxia, for example, in respiratory failure,
9. vascular, for example, cerebral haemorrhage,
10. head injury,
11. epilepsy,
12. neoplasms,
13. other intracranial causes, for example, encephalopathy.

(White and Beumont 1989.)

2.2.1.2 The chronic form

The chronic form of organic brain syndrome which involves degeneration of brain tissue is usually irreversible. Better known as dementia, this form of organic brain syndrome is characterised by a deterioration in intellectual performance, memory, judgement, and insight. A personality change together with a disturbance in both work and social relationships and functioning is evident (White and Beumont 1989). The most significant causes of dementia are as follows:

1. degenerative diseases such as senile dementia, Alzheimer's disease,
2. intracranial lesions, for example, cerebral neoplasms,
3. vascular disturbances, for example, multi-infarct dementia,
4. infections, for example, neurosyphilis, Jakob-Creutzfeld's disease,
5. trauma, such as, repeated head injuries,
6. other neurological conditions such as multiple sclerosis,
7. endocrine disorders, for example, Addison's disease,
8. cerebral anoxia,
9. avitaminosis, for example, in thiamine deficiency,

10. metabolic and toxic causes, for example, as in chronic uraemia, chronic hepatic failure,
11. certain drugs, for example, Levodopa.

(White and Beumont 1989.)

It is important to realise that delirium and dementia may coexist (White and Beumont 1989).

2.2.2 Schizophrenia

The name 'schizophrenia', comes from the Greek words 'schizos' which means split and 'phrenos' which means mind, is often misunderstood and has lead to the misconception that a person who suffers from schizophrenia has a split or dual personality (Schizophrenia Australia 1986). Schizophrenia is a mental illness where a person's feelings, thoughts, and behaviour, becomes disorganised and do not fit together (The New South Wales Association for Mental Health 1984). There are various forms of schizophrenia. Characteristic features which include delusions, paranoid beliefs, and hallucinations are brought on by thought disorder and disturbances in thought content and perception. Emotional response may be abnormal; feelings expressed may be inappropriate to the circumstances, for example, laughing over something sad (Schizophrenia Fellowship of New Zealand and New South Association for Mental Health 1985). The tendency to withdraw from people, the loss of initiative, energy, and motivation are also symptoms of schizophrenia (Schizophrenia Fellowship of New Zealand and New South Association for Mental Health 1985). The intellect and memory is usually regarded as unchanged and normal but the attention span and ability to perform normal tasks is poor. This illness may result in permanent deterioration of the personality. There is a high risk of suicide due to the tendency for mood fluctuations and changed personality.

This form of psychosis is termed '**functional**'; as yet no organic cause for this group of related conditions has been discovered. Research has indicated that genetic, biochemical, viral, parent and child relationship, and environmental factors, could all be implicated (Jones 1989). The onset of schizophrenia may be acute where the sudden change in behaviour is evident over a few weeks, or slow, where the deterioration of the personality takes place over a long period (Schizophrenia Fellowship of New Zealand and New South Association for Mental Health 1985). The prognosis is better when the illness has a history of acute symptoms precipitated by some sort of psychological stress and the person is a '**well balanced, normal**' individual before this episode of mental illness (Schizophrenia Fellowship of New Zealand and New South Association for Mental Health 1985). Chronic symptoms may precede an acute attack as well as follow it.

Many different studies internationally have shown that schizophrenia has a lifetime prevalence (this means the proportion of persons in the community who have suffered an illness episode, irrespective of whether they are still ill at the time of study) of between 0.5 % and 1 % of the population (Jones 1989). Professor Silvano Arieti, the Professor of Clinical Psychiatry of the New York Medical College made the following quotation about schizophrenia:

"...no war in history has produced so many victims, wounded so many people. No earthquake has exacted so high a toll; no other condition that we know has deprived so many people of the promise of life."

(Schizophrenia Australia 1986).

2.2.3 Affective Disorders

The term 'affective disorders' is used in reference to a group of mental disorders where there is a primary disturbance and change of mood or feeling (affect). The nature of this disturbance is usually depressive. The other extreme where the mood/feeling is elevated excessively leading to elation and mania can also occur. Depression can be a normal emotion and this symptom is usually reactive or situational i.e. this feeling is presumed to be a consequence of certain events in a person's life (Judd and Burrows 1989). Depression has sometimes been referred to as the "*common cold of psychopathology*" (Seligman 1975 cited by Beck et al 1979). Transient depression is a common response to stress and can present as part of some other medical and surgical condition (Petty 1989). Five percent of the adult population in Australia may suffer from a depressive disorder of some sort whilst a smaller number have a manic-depressive illness (The New South Association for Mental Health 1984). Depression is regarded as a form of psychiatric disorder when the emotional changes are persistent and accompanied by psychotic features and the risk of suicide. Psychotic symptoms of hallucinations and delusion, loss of self-esteem, loss of appetite, and the sense of helplessness, are all features of this depressed phase of the disorder. Changes in sleep pattern, overwhelming anxiety and feeling of agitation or retardation are usually evident. Episodes of this depressed mood is known as 'unipolar affective disorder'.

'Bipolar affective disorder' is the term given to episodes of depression which are interspersed with episodes of elation (mania) where the feeling of euphoria is far more intense than the circumstances would warrant, more intense than ordinary happiness (The New South Wales Association for Mental Health 1984). At first, during an episode of mania, a person has a general mood of well being and a feeling of grandeur. There is also increased activity, rapid thinking and output of ideas (Judd and

Burrows 1989). Sleep is often impaired and the manic patient is too busy to stop to eat. As the illness progresses, hyperactivity marked with instability and agitation follows. A mild form of this upswing in mood which is of limited duration is described as hypomania. When the changes are more marked and persistent, lasting weeks leading to a loss of contact with reality, this increase in mental and physical activity is known as mania (Smith et al 1986).

Episodes of this type of mental illness may be mild or severe, may last for a short period or longer, and may be of one type of mood change (unipolar affective disorder) or vary between the two different mood changes (bipolar affective disorder). In between the episodes of mood changes, the person can be free of any symptoms.

The mood swings in affective disorders are believed to be related to biochemical changes (research has implicated an association with abnormalities of neurotransmitters in the brain), genetic predisposition, psychological stress, and changes in biological rhythms (mania and depression occurs at particular times of the year) (Smith et al 1986). Other factors such as sociological factors, life events, childhood environment, and personality may also play a role in the aetiology of affective disorders (Judd and Burrows 1989).

It is important to realise that depression and mania can occur as a response to drug treatment for other medical illness. Drugs such as steroids, thyroid preparations, and Levodopa, may precipitate episodes of mania (Smith et al 1986). Some drugs used to treat hypertension, some tranquillisers, alcohol, and even the contraceptive, can cause depression (Smith et al 1986). Depression can also be concomitant with chronic medical illness (including other psychiatric disorders such as schizophrenia) especially when present in the geriatric population (Judd and Burrows 1989).

Primary affective disorders refer to a past history of mental illness where only mania or depression is present. Secondary affective disorders refer to those patients who have other psychiatric disorders as well as mania or depression (Robin and Guze 1972 cited by Judd and Burrows 1989).

2.2.4 Neuroses (neurotic illnesses)

Generally, neuroses refer to mental illness which is less severe than the psychoses, which are mental illnesses like schizophrenia or depression where the sufferers are usually in some way out of touch with the world (The New South Wales Association for Mental Health 1984). Sufferers of neuroses have symptoms which are closer to normal experience (e.g. anxiety) but are illogical and exaggerated reactions to stress. Neurotic illnesses are characterised by 3 features:

1. There is no organic brain disease present, therefore neuroses are functional disorders.
2. The sufferer is not out of touch with external reality no matter how severe the neurotic illness is (i.e. symptoms of hallucinations and delusions are not present)
3. Neurotic illnesses have a discrete onset rather than a cumulative development of symptoms early on in life.

(Gelder et al 1989b.)

Symptoms of neurotic illnesses are presented as anxiety, depression (less severe in form than in the affective disorders) and disproportionate reactions to stressful factors. The aetiology of neurotic illnesses is not fully understood. However, it is generally believed that neuroses arise when stressful factors in the sufferers lives overwhelm

their ability to cope, irrespective of supportive relationships present. Sufferers usually have an inherited predisposition to neuroses and are of certain personality types (Gelder et al 1989c). Their upbringing and factors relating to family relationships and employment are believed to play a role in the aetiology of neuroses as well.

Numerous forms of neuroses exist; some of the more common forms of neurotic illnesses are as follows:

1. Anxiety states — these are episodes of irrational worry, panic attacks (spells of fear and anxiety which arise spontaneously in situations that most people would not be afraid of), and physical symptoms (such as chest pains, palpitations, dizziness) (Andrews 1989). These episodes are usually related to a past '**traumatic**' or '**upsetting**' event.
2. Phobic states or illogical fears of certain circumstances and surroundings or objects. The most significant phobic state to dental surgeons is that of dental phobia where all dental treatment is avoided, possibly resulting in severe dental disease.
3. Obsessive-compulsive neuroses — these are illnesses where obsessions and rituals preoccupies the sufferers to the exclusion of reasoning and causes intense feelings of guilt and anxiety to the sufferers. Some sufferers have a compulsion about removing '**germs**' from their mouths (McSwiggan 1991). Consequently, they use large quantities of mouth washes and carry out excessive tooth brushing which may result in severe abrasion lesions in the oral cavity.
4. Hypochondria — the sufferers of this neurosis show a morbid preoccupation with their state of health, body processes and functions.

Neuroses are episodic but work and personal relationships could be affected when they become chronic and increase in severity.

2.2.5 Personality Disorders

The word personality is explained by The Concise Oxford Dictionary (1984b) to mean: "*i) personal existence or identity; ii) distinctive personal character; or iii) person especially a celebrity*". However, in Psychiatry, the term personality refers to the individual's qualities that are apparent in an individual's attitudes and ways of behaviour in response to a wide range of situations (Gelder et al 1989d). The personality of an individual with mental illness is of great concern to the psychiatrist as an individual's personality determines how he reacts when ill and makes him more vulnerable to develop emotional disorders when experiencing stressful circumstances.

Personality disorders are hard to define precisely (Parker and Boyce 1989). These disorders are characterised by a behaviour pattern which is psychosocially maladaptive, inflexible, and persistent. The individual's relationship with other people is poor. As a result of the individual's inflexible, and repetitive abnormal personality traits, the individual and those involved with him suffer. There is only a very fine line distinguishing neurotic disorders from personality disorders.

Personality disorders are named according to their similarity to other psychiatric illnesses (e.g. paranoid, schizoid) or with the behaviour traits in the individuals (e.g. dependent, passive-aggressive). As a consequence of difficulties with interpersonal relationships at home or work, or problems with society in general, the individual who has a personality disorder may develop anxiety and depression, too.

The attitudes and behaviour of an individual with personality disorder suggests that an arrested development of personality may have occurred. Psychotherapy can sometimes be effective in modifying the behaviour of individuals with personality disorders, but generally, these are difficult to treat.

2.2.6 Miscellaneous Disorders

This category include habit disorders, psychophysiological disorders, transient situational disturbances, and disorders occurring in childhood and adolescence.

In habit disorders, certain behaviour patterns, accompanied by physical and psychological symptoms, are present. Examples of habit disorders are the sexual disorders, the addictive use of alcohol and drugs, and the eating, disorders. In this group of disorders, the eating disorders of anorexia nervosa and bulimia nervosa (both dieting disorders) are of significance to the dentist. The consequences and dental implications of anorexia and bulimia nervosa will be highlighted in a later chapter of this treatise.

Psychophysiological disorders are conditions in which stress affects organs physically and alter their functions via the autonomic nervous systems. Examples of such disorders are hypertension, migraine, asthma, and peptic ulcers, suffered by some high powered business executives.

Transient situational psychiatric disturbances are situations where the individual's mood, conduct, and behaviour, are brought on directly by stress and resolve when the stressful circumstances are over.

Psychiatric disorders in childhood and adolescence can be similar to those seen affecting adults e.g. schizophrenia, and bipolar affective disorders. Other psychiatric conditions which occur during the stage of development are those such as emotional disorders, behaviour disorders, and abuse disorders. It is felt that these psychiatric disorders may sometimes be related to a manifestation of disturbance in the child's/adolescent's family life or socioeconomic disadvantages (Waters and Brennan 1989).

2.3 Intellectual disability (mental retardation)

There is often a misconception that mental illness is the same as intellectual disability (The New South Association for Mental Health 1984). This is not so. Intellectual disability is also commonly referred to as intellectual handicap or developmental disability. It is usually identified and evident at birth or early childhood. This intellectual impairment causes learning problems and development takes place at a slower rate, resulting in a lower level of self and social functioning (Molony 1989, New South Wales Council for Intellectual Disability 1991). People with intellectual disability can also suffer from mental illness; the more common ones include personality disorders and behaviour disorders like head banging and other self-injurious behaviour (Molony 1989). About 3% of the population of New South Wales have some form of intellectual disability (New South Wales Council for Intellectual Disability 1991).

2.4 Treatment

Many people have one episode of mental illness and never break down again (The New South Wales Association for Mental Health 1984). However, a certain percentage of people do have chronic episodes of this illness. Different types of

treatment are used for the different forms of mental illness. A range of treatments is usually required for any one individual (The New South Wales Association for Mental Health 1984). Current treatments include: psychotherapy, behaviour therapy, medication, electric-convulsive therapy and alternative techniques such as orthomolecular medicine, relaxation and meditation.

2.5 Overview

This chapter on mental illness is by no means sufficient knowledge to prepare the dental practitioner to diagnose and treat psychiatric disorders. As demonstrated in the different categories of mental illnesses, many signs and symptoms of these psychiatric disorders overlap. In fact, many of these different disorders can co-exist, for example, depression and schizophrenia. It needs the expertise of a psychiatrist and other such health care workers to diagnose and treat. The information provided in this chapter is meant to help the dentist plan and carry out comprehensive and appropriate dental procedures, successfully, for an individual who is suffering from some sort of mental illness (and is in need of dental treatment). A deeper understanding of the psychopathology of the different types of mental illness may be needed to help the dentist recognise some symptoms in certain patients. The writer recommends that an up-to-date text book on psychiatric illness should be kept in the dental surgery for this purpose, e.g. *The Textbook of Psychiatry*, edited by Beumont PJV and Hampshire RB, published by Blackwell Scientific Publications, 1989 (or the latest edition).

When certain symptoms of mental illness are detected or recognised by the dentist, timely referral to the appropriate health care personnel (general medical practitioner, psychiatrist, psychotherapist, or social worker) is important. The writer has not attempted to dwell much on the various types of treatment of mental illness as she feels that this is not within the scope of this treatise.

3 Psychogenic Facial Pain

Orofacial manifestations of certain systemic diseases are well documented; for example, the oedematous, bluish-red appearance of the gingivae of a patient with acute myelogenous leukaemia and extra-orally, the presence of cervical lymphadenopathy. Mental illness (certain types) may also affect the orofacial region (Harris and Davies 1980, Jackson 1980).

Moulton, Harris and Davies were among the first workers who believed that the orofacial region can be affected by a wide spectrum of mental illness (Moulton 1955, Moulton 1966, Harris 1975, Harris and Davies 1980). Both Harris and Davies (1980) stated that the association of emotional and mental suffering with symptoms in the mouth could be interpreted in many different ways, on the basis of the complex anatomical, physiological, and developmental aspects of oral function. It has also been proposed by many that the anatomic, physiologic and psychologic characteristics of the orofacial region are more complex than in any other part of the body (Burton 1969, Smith et al 1969). Both the mouth and face have leading roles in self-expression and emotional response of an individual (Moulton 1966, Harris 1975, Harris and Davies 1980). Thus, emotional distress and mental anguish, will understandably evoke a response in the orofacial region. It is this response that predisposes the orofacial region to psychosomatic/psychogenic disorders of which the most significant is pain. Psychogenic pain will be the first orofacial manifestation of mental illness to be discussed in this treatise. This whole chapter deals with psychogenic facial pain. Other orofacial manifestations will be discussed in the following chapters.

3.1 Psychogenic pain in the orofacial region

The term '**pain**' comes from the Latin word '**poena**' which means punishment and is only a linguistic expression for this subjective phenomena (Smith et al 1969). What is pain? Pain has been described as "*suffering or distress of the body or mind when hurt by injury or disease*" (The Concise Oxford Dictionary 1984c). This is essentially an unpleasant experience. Pain is not a simple or straightforward sensation. Like pleasure, it is both a perception and a feeling reaction, but its opposite (Burton 1969, Joy and Barber 1977). Perception of pain is influenced by many factors, some of which are culture, gender, age, present emotional status, and past experience (Harris 1974, Joy and Barber 1977).

Pain is probably the most common symptom encountered in both the medical and dental professions (Scott and Humphreys 1987). Pain in the orofacial region has always been thought to be caused either by local pathology or referred from other nearby structures. The third possible causative factor of pain in the face and mouth may have no organic basis, i.e. it is of psychogenic origin (Moulton 1955, Friedman 1966, Burton 1969, Harris 1974, Harris 1975, Remick et al 1983a). Atypical facial pain affects approximately 10% of patients who present to dentists with chronic orofacial pain complaints (Remick et al 1983a). Mersky estimated that 50 % of psychogenic pain is sited in the head region (Mersky 1984). Other workers have also reported that the head was among the three most common sites of psychological pain complaints (Watson et al 1981). Many possible mechanisms for the production of psychogenic pain has been proposed. Some of the main ones are as follows:

1. Pain caused by sustained muscle contraction (muscle tension), such as that which occurs with emotional distress, is a common mechanism in certain neuroses (anxiety, minor depression, hypochondriasis).

Anxiety leads to tension in the muscle which in turn causes the build-up of locally produced metabolites. This collection of waste-products results in pain. This pain increases the anxiety and thus a vicious circle is set up. A pain pattern as described is common in pain in the shoulder, neck, lower back, and tension headaches.

2. Pain caused by a delusion or hallucination. In psychoses such as schizophrenia or endogenous depression, delusions or hallucinations may give rise to such pain, although this is not common.
3. Pain caused by repression of emotional conflict which subsequently leads to the conversion of this conflict into somatic symptoms. Psychogenic pain of this type (due to conversion hysteria) may be associated with abnormal personalities, anxiety and/or depression.

(Merskey 1984, Dworkin and Burgess 1987, Scott and Humphreys 1987)

Psychogenic pain has some distinguishing features which differentiates it from pain due to an organic cause. Some of the distinguishing features are as follows:

1. The pain distribution does not conform to the anatomical or dermatome distribution of the nervous system.
2. This pain may occur bilaterally and does not have a trigger zone.
3. The pain is continuous for prolonged periods by day and may last for a long time (weeks to years) with little change.
4. The pain is not relieved or only temporarily relieved by analgesics.
5. The pain can be described by the patient to be related to emotional factors and discussion of this problem may bring about an emotional response e.g. crying.

6. There may be a long history of multiple consultation with various health professionals or numerous investigations carried out without any evidence of organic pathology.

(Harris 1974, Mock et al 1985, Scott and Humphreys 1987)

7. The pain is associated with other emotional or psychiatric signs and symptoms such as headaches, neckaches and back pain (Blasberg et al 1983, Donlon et al 1990, McSwiggan 1991).

Before the diagnosis of psychogenic pain can be confirmed, all possible avenues to look for an organic cause of the pain must be exhausted. Although psychogenic pain does not have an organic basis, it is not a **'figment of the patient's imagination'** (Friedman 1966, Harris 1974). The suffering experienced is real, a source of anxiety and a reason for complain by the patient.

Psychogenic pain in the orofacial region has been subdivided into a number of different disorders:

1. Facial arthromyalgia
2. Atypical facial pain
3. Oral dysaesthesia
4. Atypical odontalgia.

(Harris and Davies 1980)

3.1.1 Facial Arthromyalgia

Facial arthromyalgia is now more widely known as temporomandibular joint (TMJ) dysfunction syndrome (Greene et al 1969, Speculand et al 1983, Grzesiak 1991). Other names given to this phenomenon include myofascial pain dysfunction syndrome, mandibular dysfunction syndrome, arthrosis of the TMJ, and Costen's syndrome (Lupton 1969, Thomson 1971, Goodman 1976, Greene and Laskin 1974). In facial arthromyalgia, there is seldom evidence of organic pathology present.

Facial arthromyalgia affects the TMJ and the associated musculature (Fine 1971, Thomson 1971, Brooke 1977, Feinmann 1983, Grzesiak 1991). A prolonged dull ache (with acute exacerbations at times) in the joint and masticatory muscles in the preauricular area is usually presented (Lupton 1969, Brooke et al 1977, Harris and Davies 1980, Feinmann 1983, Feinmann and Harris 1984a). This pain may involve the side of the head and neck where the affected joint is located. Other symptoms such as clicking, sticking, stiffness, and trismus of the TMJ (which seem to indicate disturbed joint function) may also be found although no organic cause is evident. If there is some minor evidence of organic pathology, this is not commensurate with the degree of pain felt. Facial arthromyalgia may affect either of the joints or even both, in some instances. The muscle tenderness and joint symptoms in this region may sometimes be accompanied by aural symptoms such as tinnitus and the feeling of fullness of the ear (Harris and Davies 1980, Feinmann and Harris 1984a).

Facial arthromyalgia has been associated with a wide range of factors such as bruxism, loss of teeth resulting in decrease in vertical dimension, malocclusion, and arthritis of the joint (Moulton 1955, Laskin 1969, Solberg et al 1972, Goodman 1976, Brooke et al 1977, Attanasio 1991). Investigations including extensive radiographic studies seldom show evidence of organic disease when this pain is ascribed to anxiety or

agitation associated with depression. This pain disorder is found to be more frequent in females and the highest occurrence is among the younger, premenopausal age group (Feinmann 1983, Scott and Humphreys 1987).

In the 1960's, many studies were carried out to delineate the personality characteristics of these patients. McCall and his associates (1961) administered the Minnesota Multiphasic Personality Inventory to 3 distinct groups of army personnel; 70 patients with TMJ dysfunction, 70 subjects who were healthy and with no need of dental treatment, and 70 patients who were about to undergo oral surgery. They found that, as a group, the patients with TMJ dysfunction were easily identifiable from the other two groups in that anxiety is the major factor reflected in their response to questions in this standard personality inventory. These patients described themselves being easily angered, restless, worried and indecisive, more nervous than most people, suspicious, and fighting their hardest battle with themselves. Lupton (1969) published his investigations into the personality characteristics, responsiveness to pain, and muscular activity under stressful and non-stressful situations of 200 patients with TMJ dysfunction carried out at the TMJ Research Centre of the University of Illinois College of Dentistry. From this sample a significant number of these patients demonstrated psychological factors linked to their TMJ dysfunction of non-organic origin. Treatment of these psychological factors also brought relief for the TMJ symptoms.

Gross and Vacchiano (1973) designed an investigation to clarify the personality characteristics of 56 patients with TMJ dysfunction. They found that compared with a similar control group, the patients with TMJ dysfunction had low ego strength, were highly emotional, easily annoyed, and were generally dissatisfied with the world and their life circumstances. Thomson (1971) carried out a study on 100 patients with TMJ pain and on 100 unselected patients or members of staff without TMJ pain (as a

control group) at the London Eastman Dental Hospital. He found that in 61 % of the patients with TMJ pain some sort of emotional upset was a feature.

Fine (1971) studied 2 groups of patients, a group of 50 patients with TMJ syndrome of non-organic origin (facial arthromyalgia) and another group of 50 patients with facial pain due to a variety of dental causes, to investigate the possible significance of psychological factors related to this syndrome. Fine's comparison of these two groups elicited that 76 % of the group with the TMJ syndrome had psychiatric symptoms while only 20 % of the control group had such a diagnosis. The psychiatric diagnosis was predominantly either anxiety and/or depression. In 52 % of cases, symptoms of anxiety or depression associated with bereavement, marital problems, or living with a chronically ill relative, had pre-empted the onset of pain by at least 6 months.

Speculand and his co-workers (1984) compared the illness behaviour of 100 patients with TMJ dysfunction and 100 asymptomatic patients as controls. The 62 item version of the Illness Behaviour Questionnaire of Pilowski and Spence was used. It was found that the TMJ dysfunction patients had significantly higher scores on measures of disease conviction and anxiety or depression. These patients were also less likely to deny the existence of problems in their lives than the control group.

Anxiety, depression, emotional upset, the use of denial (to block out the existence of problems), and repression of feelings seem to be factors implicated in TMJ dysfunction of non-organic origin (facial arthromyalgia). These patients are often unaware of their state of high arousal and focus on one symptom of anxiety which may be pain in the TMJ (McSwiggan 1991). Other workers have also identified stress as one of the significant psychological factors (Laskin 1969, Reading and Raw 1976, Brooke et al 1977, Speculand et al 1984, Dettmar and L'Estrange 1987). Stress brings

about increased muscle tone in the masticatory muscles. This muscular hyperactivity may in turn produce fatigue and pain.

Over the years, a large number of studies have been carried out to investigate the relationship between non-organic TMJ dysfunction (facial arthromyalgia) and psychological factors as it is considered the most common type of psychogenic pain in the orofacial region (Harris and Davies 1980, Feinmann et al 1984).

3.1.2 Atypical Facial Pain

Atypical facial pain also known as atypical neuralgia (Harris and Davies 1980, Mock et al 1985) has been described as a poorly localised, diffuse pain felt in the facial bones or alveoli of the jaws and/or deep in the soft tissues (Lascelles 1966, Feinmann 1983, Mock et al 1985). The quality of pain may vary from a dull ache to a severe throbbing sensation (Harris and Davies 1980, Feinmann 1983). Atypical facial pain occurs in areas supplied by the second and third cervical or fifth and ninth cranial nerves (Lascelles 1966). However, the distribution of the pain is not anatomical in nature and the pain may cross over to the opposite side (Lascelles 1966, Harris and Davies 1980, Mock et al 1985). No organic cause can be elicited for this pain (Harris and Davies 1980, Feinmann 1983, Mock et al 1985). This disorder occurs more frequently in females than males and the highest incidence is among post-menopausal women (Smith et al 1969, Feinmann 1983, Remick et al 1983a, Mock et al 1985). Atypical facial pain lasts for six months or longer (Smith et al 1969, Mock et al 1985). Two variants of this disorder, atypical odontalgia and oral dysaesthesia, will be discussed separately further on.

It has long been recognised that atypical facial pain (of non-organic cause) is closely related to the presence of a psychological disorder although there is disagreement regarding the type of the psychological disorder involved.

Webb and Lascelles (1962) obtained significant results when using antidepressant drugs on 31 patients suffering from facial pain and depression. The degree of relief of the patient's facial pain was accompanied with the loss of their depressive symptoms. In 1966, Lascelles carried out a detailed general medical and psychiatric study on 93 patients suffering from prolonged facial pain. He revealed the presence of '**atypical depressive symptoms**' in the majority of patients in his study. The symptoms of the '**atypical**' depression were intense fatigue, agitation, and sleep disorders, whilst the classical features of depressive illness (melancholia, retardation, weight loss, and self reproach) were absent. In this double-blind controlled trial, the efficacy of phenelzine (a monoamine oxidase inhibitor) was demonstrated, as the improvement of the facial pain coincided with the improvement in depression.

Smith and his co-workers (1969) conducted a psychiatric study of 32 patients having '**atypical facial pain**', who were seen at the Mayo Clinic over an 8 month period. Information obtained from various interviews and results of the Minnesota Multiphase Personality Inventories (completed by each patient) demonstrated that, as a group, they were perfectionistic, driving, success-oriented, hypochondriacal, depressed individuals with difficulty in interpersonal relationships. Smith and his co-workers felt that the above personality characteristics of the patients brought about resentment and anger which were repressed. They also concurred that this repression in turn produced both depression and facial pain which expresses the patients' distress.

A group of Canadian psychiatrists, Remick and others (1983a) found that 68% (46) of their patients with atypical facial pain had a specific psychiatric disorder, according to

the Diagnostic and Statistical Manual of Mental Disorders III criteria. The various types of psychiatric disorders found included somato form disorders, personality disorders, schizophrenia, and atypical psychosis.

A retrospective study of atypical facial pain was carried out by Mock and his co-workers (1985) at the Mount Sinai Hospital of the University of Toronto. Thirty-four patients, 76.5 % female and 23.5 % male, were in this group. Mock and his co-workers felt that psychological factors played a dominant role in most cases although they could not demonstrate any definable psychiatric illness.

Many interesting case reports regarding the underlying psychiatric disorder related to atypical facial pain has been published. One of them was presented by Oldham (1974), where a patient suffering from the Von Munchausen syndrome (this condition consists of the imitation, production and encouragement of illness consciously, to deceive clinicians and is usually associated with a severe personality disorder) feigned atypical facial pain which was assiduously investigated. Another such interesting case report was presented by Delaney (1976). In his paper, Delaney presented three cases where the atypical facial pain was the major presenting complaint underlying a severe emotional conflict. He suggested that the facial pain may have served as a defence against the emergence of psychosis. Treatment with antipsychotic drugs and psychotherapy resulted in the psychotic symptoms disappearing.

The most significant, single feature found among the literature on atypical facial pain is that a large number of patients with atypical facial pain had various, usually multiple procedures performed in an attempt to relieve the pain (Remick et al 1983a, Remick et al 1983b, Mock et al 1985). A large number of invasive procedures such as dental extraction and sinus cavity explorations which had been unnecessarily carried out not only fail to alleviate the pain but seemed to aggravate the symptoms causing more

distress and pain. In most of these patients, the symptoms noticeably decreased when active treatment was stopped (Mock et al 1985). It has been suggested by these workers that dental or surgical procedures tend to precipitate, perpetuate, or exacerbate atypical facial pain. They felt that when a patient with atypical facial pain is presented, no invasive procedures should be carried out unless overwhelming evidence indicates so. This concept had been proposed as early as 1924 by Frazier and Russel who first described this pain (Frazier and Russell 1924 cited by Mock et al 1985). In view of the overwhelming evidence which indicates that some sort of underlying psychological problem seems to play a significant role in atypical facial pain, the writer feels it is mandatory that a psychological evaluation be carried out in the early stages of the investigation.

3.1.3 Oral Dysaesthesia

Oral dysaesthesia is better known as the burning mouth syndrome (Lamey and Lewis 1989). Other names for it which include glossopyrosis (meaning burning tongue), glossodynia (meaning painful tongue) and hot tongue syndrome aptly describes the most common site of occurrence in this pain disorder (Zucker 1972, Harris 1974, Browning et al 1987, Van der Ploeg et al 1987, Zilli et al 1989). In oral dysaesthesia, there may also be a disturbance of oral sensation and taste present with the burning sensation in the mouth (Feinmann 1983, Main and Basker 1983, Feinmann and Harris 1984a, Grushka 1987). The site of burning is variable but it affects the anterior 2/3's of the tongue most often, followed by the palate (upper denture bearing tissues), lip, and the lower denture bearing area (Main and Basker 1983, Van der Ploeg et al 1987, Lamey and Lewis 1989). The occurrence of oral dysaesthesia in sites other than the tongue have probably brought about other names such as 'stomatopyrosis' and 'stomatodynia' (Lamey and Lewis 1989). There may be more than one site involved and the burning sensation is nearly always felt bilaterally (Van der Ploeg et al

1987, Lamey and Lewis 1989). The appearance of the oral tissue is usually normal (Lamey and Lewis 1989, Grushka and Sessle 1991). The presence of an unpleasant metallic taste (Grushka 1987) and a dry mouth may sometimes be reported (Main and Basker 1983, Grushka 1987, Lamey and Lewis 1989). This burning sensation may be present on waking and persists throughout the day, begins on waking, and worsens as the day progresses, or may be absent on certain days (Lamey and Lewis 1989).

Oral dysaesthesia occurs much more frequently in women than in men (Harris 1974, Main and Basker 1983, Browning et al 1987, Van der Ploeg et al 1987). Workers such as Lamey and Lewis (1989) have reported a ratio as high as 7:1. The average age of the patient is more than 50 years old although patients of 28 years of age have also been reported (Lamey and Lewis 1989).

Just as for facial arthromyalgia, oral dysaesthesia has been attributed to various causative factors. Detailed investigations including a thorough clinical examination, complete review of past and present medical history (including drug history), laboratory culture for fungi (*Candida albicans*), complete blood count (for assays of vitamins B₂, B₆, B₁₂ and iron), non fasting blood glucose determination, saliva analysis, and even biopsies have shed some light on this topic (Main and Basker 1983, Zegarelli 1984, Lamey and Allan 1986, Browning et al 1987, Lamey and Lewis 1989). Undue stress (or overloading) on the denture bearing tissues by badly constructed dentures, leakage of residual monomer from denture material, vitamin B complex deficiency, candida infections, xerostomia, and diabetes, have been proven to be the aetiological factors in many cases of oral dysaesthesia (Basker et al 1978, Main et al 1983, Lamey and Allam 1986, Browning et al 1987, Lamey and Lewis 1989, Grushka and Sessle 1991). However, in many, no oral or other organic cause can be identified and even when the known aetiological factors mentioned above have been investigated and corrected, these cases of oral dysaesthesia are not improved.

Psychological factors are also considered to play an important role in the aetiology of oral dysaesthesia. Zucker (1972) was of the opinion that the differential diagnoses of psychogenic tongue symptoms included the anxiety state, hypochondriacal reactions, conversion reaction and masochistic reaction. Harris (1974) believes that oral dysaesthesia without an organic cause is a common condition in cancerphobic patients and those suffering from exogenous depression. Lowental and Pisanti (1978) concluded from their study of 44 patients that the most common causes of this syndrome were depression and hypochondriasis.

Zegarelli (1984) analysed 57 patients with a chief complaint of burning mouth syndrome over a period of one and a half years. This analysis included a thorough medical history including medication history, clinical examination, and appropriate haematological and microbiological tests when indicated. Twenty-one patients (37 %) were found to have psychogenic factors implicated as the aetiological factor in their condition. Psychogenesis was also a contributing factor in 6 additional cases (11 %). Van der Ploeg and his co-workers (1987) carried out a psychological questionnaire survey of 184 patients who had burning mouth syndrome. Seventy-two of the patients had been referred to the oral surgery department of the Free University of Amsterdam whilst the rest of the patients were members of a group called Association of Burning Mouth Syndrome. In this Dutch study, 4 psychological tests which measured state and trait anxiety, depression, bodily and somatic response to stress, neurotic lability, neurosomatic lability and introversion-extroversion were carried out. Van der Ploeg and his co-workers concluded that psychological aspects such as anxiety, depression, and neurotic tendencies are of significance in a large number of patients with oral dysaesthesia.

In another study of systematic psychiatric interviews, Browning and his co-workers (1987) in England found that 44 % out of 25 patients with burning mouth syndrome

had a psychiatric disorder as compared with 16 % of a control group of 25 dental patients with organic disease. They felt that anxiety mixed with depressive symptoms and a complex social history forms part of the psychiatric disorder in these patients. Results from studies (1989) into the effectiveness of the Hospital Anxiety and Depression scale (Lamey and Lamb 1989) and the General Health Questionnaire (28 item version) together with the Irritability, Depression and Anxiety scale (Zilli et al 1989) to assess and screen for psychiatric illness in patients with oral dysaesthesia (without any organic disease) seem to implicate anxiety and depression as some of the main aetiological factors.

The high incidence of depression found among patients with oral dysaesthesia suggests that xerostomia might be one of the causative factors as there is usually a decrease of salivary flow in depression (McSwiggan 1991). It has been found that in some patients with oral dysaesthesia, the burning pain is relieved by eating and drinking (Harris 1974, Feinmann 1983, Feinmann and Harris 1984a). This is unlike pain caused by organic pathology as drinking and eating will make this pain worse. This distinction may be quite crucial in making the diagnosis of oral dysaesthesia linked to some sort of psychiatric disorder.

3.1.4 Atypical Odontalgia

Atypical odontalgia which is toothache or tooth pain not associated with any lesion was earlier described by Harris in 1974 as 'idiopathic periodontalgia'. It is now believed to be a variant of atypical facial pain (Rees and Harris 1979, Brooke 1980, Feinmann 1983, Feinmann and Harris 1984a). As for the other presentations of psychogenic facial pain, there is no clinical or radiological evidence of disease in the teeth and related periodontal membrane.

The pain felt in atypical odontalgia is described as severe, throbbing, and continuous in nature. The pain may be localised and start in one quadrant but can also become widespread involving the opposite side (Rees and Harris 1979). Some patients have complained of pain which is present in all quadrants at the same time. The pain is usually exacerbated by any thermal or tactile stimuli (Rees and Harris 1979, Feinmann 1983).

The history of this pain may vary from a few months to as long as 20 years (Rees and Harris 1979). Atypical odontalgia resembles most of the signs and symptoms of pulpal or periodontal pathology. As a result, the offending tooth usually receives endodontic treatment or is extracted (Rees and Harris 1979, Brooke 1980). The relief of the pain is only transient and the recurring pain is described as greater in intensity. Sometimes all teeth in a certain quadrant of the mouth may be extracted without permanent pain relief. In spite of the edentulousness, atypical odontalgia can persist and sometimes may result in extensive investigations of the surrounding structures such as the antrum, nose, salivary glands, and even the trigeminal nerve (Rees and Harris 1979).

Most of the sufferers of atypical odontalgia are female (Rees and Harris 1979, Brooke 1980). Bruxism, migraine, tension headaches, and other such conditions related to emotional stress, are usually present among those who suffer from atypical odontalgia.

Harris (1974) who first referred to 'atypical odontalgia' as 'idiopathic periodontalgia' suggested that the condition may have been precipitated by a dental procedure such as an extraction or placement of a bridge. He believed that emotional problems play a significant role in many patients presenting with atypical odontalgia. He also found that many of these patients suffer from recurrent migraine. Harris also believed that the condition is a state of hyperalgesia of periodontal pain receptors and the association with migraine suggested a vascular disturbance.

Later, Rees and Harris (1979) postulated that this condition is a symptom of the underlying psychiatric disorder. They believed depression and other personality disorders to be responsible. They also stressed that since depression can undergo remissions and exacerbation, the atypical odontalgia may follow a similar pattern. Associated with this underlying disorder of affect is the temporary or permanent biochemical defect producing vascular changes. The vasodilation in the blood supply of the affected tissues occurs as a result of the deficiency of catecholamines, in particular noradrenaline. Rees and Harris also found that many of the group of patients they studied, seem to demonstrate marked hyperaemia of the gingiva or oral mucosa. They felt that this is evidence in support of their hypothesis that the vascular changes in the teeth and surrounding periodontal membrane give rise to the condition of atypical odontalgia, which is seen as a localised form of the more diffuse atypical facial pain or neuralgia. Rees and Harris (1979) studied 44 cases of atypical odontalgia and 75 % of the patients responded well to antidepressant drug therapy. These results were superior to that of Brooke.

Brooke (1980) carried out a study on 22 cases of atypical odontalgia. He had a success rate of 50 % with antidepressant and tranquilliser drug therapy. His findings were, however, similar to that of Rees and Harris in that a significant number of the patients had a past or present history of depression and that many also suffered from stress related conditions such as bruxism, migraine, or tension headaches. All the patients in Brooke's study were female whilst in the study conducted by Rees and Harris, 82 % were female. Both the studies indicated that the more invasive dental treatment was carried out on the offending teeth, the more severe and complicated the history of the atypical odontalgia presented. Brooke felt that the higher rate of success achieved by Rees and Harris with the antidepressant therapy was probably due to the longer duration for which this therapy was carried out. In both studies, it is indicated that adults of all ages were vulnerable.

Marbach (1978a) reported on the phenomenon of phantom tooth pain. He described phantom tooth pain as *"persistent pain in teeth whereby neither endodontic therapy, apicetomy nor extraction of the offending teeth render the region free of pain"*. He studied 25 cases of persistent pain in teeth which had received endodontic treatment and in some cases, pain felt in the edentulous region where teeth had been extracted to alleviate the pain. Marbach felt that this pain is akin to that of the better known phenomenon, '**phantom limb pain**'. He based his hypothesis on that of Melzack's who proposed a '**central biasing mechanism**' for pain which are consistent with the gate-control theory of pain. Marbach proposed the hypothesis that two central nervous system mechanisms i.e. the central biasing and pattern generating, explains '**phantom tooth pain**' the best at this time. The implications of this hypothesis to dental procedures are as follows: In some people, extirpation of the pulp or extraction of the tooth may result in a loss of sensory input in the trigeminal system. This results in decrease of tonic inhibition which subsequently produces sustained neural activity. Long term pain from the area of the affected tooth may then spread to other regions of the face. Marbach formulated that treatment should then be directed towards reducing the sensory input.

Of all the various forms of presentation of psychogenic facial pain, atypical odontalgia seems to be the least reported and studied. Harris, Rees and Brooke proposed that there is an element of psychopathology involved whilst Marbach ascribed atypical odontalgia to an aetiology similar to that of '**phantom limb pain**'. Although there are differing views about the aetiological factors involved, all these writers feel emphatically that invasive dental procedures should be avoided in cases of atypical odontalgia or '**phantom tooth pain**' (Harris 1974, Marbach 1978a, Rees and Harris 1979, Brooke 1980).

3.2 Overview

At this moment, the most current study regarding orofacial psychogenic pain was carried out by Feinmann and Harris (1984a and 1984b). The study was based in the oral surgery departments of the Eastman Dental Hospital and King's College Hospital, London, and began in October 1979 with the results published in 1984.

The study was carried out on 93 subjects (50 with a diagnosis of facial arthromyalgia and 43 with a diagnosis of atypical facial pain including its two variants, oral dysaesthesia and atypical odontalgia). The patients received dental and medical examinations (including radiological and blood tests) to eliminate the possibility of an organic lesion. The patients were also assessed with three standardised psychiatric questionnaires (The Clinical Interview Schedule, The Montgomery-Asberg Depression Rating Scale and The Eysenck Personality Questionnaire). Demographic and social details in addition to major life events prior to the development of the pain were recorded. A double-blind controlled clinical trial was also carried out on these patients to assess the efficacy of dothiepin (an antidepressant) against a placebo and a soft bite-guard in the treatment of psychogenic facial pain. From the first part of the study, Feinmann and Harris (1984a) found that the pain disorders appeared to be associated with adverse life events and long term problems. Of these patients, 35% were classified as depressive neuroses and 20 % received a diagnosis of non-depressive neurosis. The second part of the study came to the conclusion that 80 % of these patients with psychogenic pain responded to the antidepressant therapy.

From various studies and tests carried out on patients with psychogenic facial pain over the years, the exact nature of the psychiatric disorder underlying this pain syndrome is still not clear although depression and atypical depression seem to be the most common diagnosis. Patients with schizophrenia have also been known to report

pain in the head which is related to specific delusion or hallucinatory processes (Watson et al 1981). It is also suspected that there is a relationship between certain personality traits, emotional stress, and the resultant presentation of psychogenic facial pain. A large number of patients with psychogenic facial pain also complain of other chronic medical problems such as low back pain and tension headaches (Moulton 1955, Berry 1969, Smith et al 1969, Harris 1974, Blasberg et al 1983, Donlon et al 1990). Berry (1969) carried out a study to determine the incidence of chronic minor illness such as migraine, back, neck and shoulder pain, pruritic skin diseases, in 100 patients with facial arthromyalgia. He found that these symptoms were ten times higher in patients with facial arthromyalgia than the general population. As early as the 1950's, Moulton noted that most patients with atypical facial pain also complained of these chronic minor illnesses (Moulton 1955). This occurrence of psychogenic facial pain disorders together with other minor medical illness related to stress imply that facial pain may be part of a person's bodily response to stress. Patients with chronic pain elsewhere have also been found to be suffering from depression and/or anxiety (Ranga Rama Krishnan et al 1985a, Ranga Rama Krishnan et al 1985b, Merskey et al 1987). There is a dilemma regarding 'cause or effect' - does the pain cause depression, or is it a result of depression? (McSwiggan 1991.)

The results of Feinmann and Harris (1984b) can be compared favourably with those of Lascelles. Lascelles (1969) had a 73 % improvement when he used the antidepressant drug phenelzine and chlordiazepoxide hydrochloride to treat 93 patients with psychogenic facial pain. He reported the greatest success in those patients who were found to be suffering from a depressive disorder. The success of the antidepressants in relieving pain in the absence of depressive symptoms may be due to the drug's vasodilatory and muscle relaxant properties or placebo effect.

The literature suggests that patients with psychogenic facial pain have undergone a large number of unnecessary invasive procedures ranging from dental extractions and root canal therapies to sinus explorations (Marbach 1978a, Rees and Harris 1979, Brooke 1980, Remick et al 1983b, Mock et al 1985). In a study by Remick and co-workers (1983b) on the ineffective dental and surgical treatment carried out on patients with atypical facial pain, 21 out of 58 patients had 65 dental and surgical treatments with only 1 patient showing less pain as a result of the treatment. Sixty-nine percent of these patients suffered from a psychiatric illness and they received more ineffective treatments than those patients in this study who were later found to have a specific medical or dental cause for their atypical facial pain. In a later study by Mock and his co-workers (1985) on 34 cases of atypical facial pain who were selected randomly, 73.5 % of these patients had some form of treatment in an attempt to relieve the pain. Half of the treatment consisted of extraction or endodontic treatment. In all these patients who received treatment, the pain symptom either persisted or were exacerbated after treatment. The results from these 2 recent studies and many earlier ones suggest that in patients with psychogenic facial pain, no active dental or surgical treatment should be undertaken unless there are definitive indications. The treatment may not only be ineffective and unnecessary; it has been shown in many cases that the dental or surgical procedures may initiate, perpetuate, or worsen the psychogenic facial pain. This idea was proposed as early as 1924 by Frazier and Russell (cited by Mock et al 1985). When investigating and treating facial pain with no discernible organic cause, a psychiatric assessment may prove to be more productive and suitable, rather than initiating dental and surgical procedures.

It is impossible to distinguish the intensity and character of psychogenic facial pain in the face due to a organic cause from that caused by psychogenic factors. However, it will be wrong to regard psychogenic facial pain as less distressing and false. In retrospect, psychogenic facial pain may be seen as an appeal for help.

4 Dental and Orofacial Manifestations of the Eating Disorders (Anorexia Nervosa and Bulimia Nervosa)

Psychogenic facial pain, discussed in the previous chapter, is a symptom of an underlying mental illness such as depression or psychological stress/disturbance. This orofacial manifestation is felt and described by the patient. The pain is real to the patient although the signs for an organic cause are not apparent to the clinician consulted. This chapter will deal with eating disorders which demonstrate dental and orofacial changes discernible to the clinician.

Anorexia nervosa and bulimia nervosa are both eating disorders where the common factor is an abnormal, intense pre-occupation with food consumption and body weight control (Herzog and Copeland 1985, Roberts and Li 1987). They are also sometimes referred to as '**dieting disorders**' (Touyz and Beumont 1989). Both these disorders are thought to be associated with complex psychological, physiological and sociological factors (Huon and Brown 1984, Wolcott et al 1984, Touyz and Beumont 1989). They are on the increase in Western cultures where '**thinness**' is deemed to be desirable and fashionable (Casper et al 1980, Milosevic and Slade 1989, Touyz and Beumont 1989).

4.1 Anorexia nervosa

Anorexia nervosa may be a misleading name for the disorder. The term '**anorexia**' means '**loss of appetite**' but in anorexia nervosa, there is no real appetite loss until the later stages of the illness (Hasler 1982, Stege et al 1982). This illness is characterised by the intense fear of gaining weight and the practice of self-imposed

starvation in the fanatical pursuit of 'thinness' (Hasler 1982, Touyz and Beumont 1989). People suffering from anorexia nervosa refuse to maintain normal body weight and are characterised by a weight loss of at least 25% of original body weight (Abrams and Ruff 1986, Touyz and Beumont 1989). They may have a distorted perception of their own body image, obsessional traits, neuroendocrine abnormalities, and also suffer from depression (Crisp et al 1976, Brady 1980, Touyz and Beumont 1989). Anorexia nervosa develops from puberty through to the 40's but the most common time of onset is during the teenage years of 14 to 17 (Brady 1980, Wolcott 1984). It occurs in females approximately 20 times more often than in males (Beumont 1972).

The systemic consequences of anorexia nervosa include hypothermia, dehydration and electrolyte imbalance, dermatological problems, amenorrhoea in females, hypotension and/or bradycardia. In untreated cases of anorexia nervosa, death can result (Halmi 1974, Brady 1980, Touyz and Beumont 1989). This could be the result of either severe metabolic breakdown (and malnutrition) or suicide (Andrews 1982, Sohlberg 1990). The mortality rate has been reported to be as high as 21% (Stege et al 1982).

4.2 Bulimia nervosa

Bulimia in Greek means 'ox hunger' or 'voracious appetite' (Casper et al 1980, Wolcott et al 1984). Bulimia nervosa is characterised by a gluttonous appetite resulting in the ingestion of very large quantities of food. These recurrent episodes of 'bingeing' are followed by vomiting or other compensatory behaviour to get rid of the excess food ingested (Andrews 1982, Huon and Brown 1984, Wolcott et al 1984, Touyz and Beumont 1989). These forms of compensatory behaviour include self-induced vomiting, fasting, use of laxatives, and excessive exercising (Hellstrom 1974, Milosevic and Slade 1989, Touyz and Beumont 1989). Patients with bulimia nervosa maintain a normal, or near to normal, bodyweight although they, too, have an

obsession with food consumption and weight control (Andrews 1982, Abrams and Ruff 1986, Robert and Li 1987). Bulimia nervosa is found in both sexes but the cases reported are predominantly females. Bulimia nervosa usually starts in adolescence (Wolcott et al 1984, Abrams and Ruff 1986). The practice of restricted eating, bingeing followed by the various compensatory behaviours usually result in the feeling of guilt, anxiety, depression, and self-disgust, as the bulimics feel totally out of control (Casper et al 1980, Wolcott et al 1984, Touyz and Beumont 1989). Casper and his associates (1980) studied the eating habits of 105 hospitalised female patients (with the diagnosis of anorexia nervosa) and found that 47 % of them periodically resorted to bulimia. It is estimated that 20 % to 50 % of patients with anorexia nervosa also suffer from bulimia (Casper et al 1980, Roberts and Li 1987, Touyz and Beumont 1989). These two eating disorders may coexist or present as separate illness entities.

The systemic consequences of bulimia nervosa include electrolyte imbalance, alkalosis, hypochloremia, steatorrhoea, oedema, kidney disorders, acute dilation of the stomach, and cardiac arrhythmia (Wolcott et al 1984, Brady 1985, Touyz and Beumont 1989).

4.3 The dental and orofacial manifestations

Bulimia nervosa and anorexia with bulimia present dental and orofacial changes. These disturbances have not been recognised until recently and are only evident later on in the illness, after systemic consequences are apparent (Brady 1980). Although the dental and orofacial sequela do not result in the death of the patient, some of these changes may result in irreversible damage (Hellstrom 1974, Stege et al 1982). The dental and orofacial changes that may result are as follows:

4.3.1 Perimylolysis (Dental erosion)

This term, perimylolysis, has been used to describe the well acknowledged destruction of teeth structure due to persistent vomiting (Allan 1969, Hurst et al 1977, Andrews 1982, Negus and Todd 1986, Jensen et al 1987). Hellstrom (1974, 1977) who conducted several extensive studies on oral complications in anorexia nervosa (both with and without bulimia) defined perimylolysis as *"a loss of enamel and dentine on the lingual surfaces of the teeth as a result of chemical and mechanical effects caused mainly by regurgitation of gastric contents and activated by the movements of the tongue"*. In the severe chronic cases of bulimia nervosa and anorexia nervosa with bulimia, it has been reported that all surfaces of the teeth may be affected by the acid erosion (White et al 1978, Simmons et al 1986).

The erosion patterns that develop in bulimia nervosa and anorexia nervosa with bulimia are similar in all reported cases (Allan 1969, Hurst et al 1977, White et al 1978, Brady 1980, Andrews 1982, Kleier et al 1984, Wolcott et al 1984, Negus and Todd 1986, Simmons 1986, Roberts and Li 1987, Milosevic and Slade 1989, Hellstrom 1990). In perimylolysis, the enamel loss from the teeth can vary from minor changes in the lingual surfaces of anterior teeth to extensive buccal and lingual enamel loss in anterior teeth and moderate destruction of buccal and lingual enamel of posterior teeth. The mandibular teeth are sometimes less affected. Dentine may thus be exposed and damaged. The teeth may be sensitive to thermal changes and to chewing or brushing. Generally, these surfaces of the teeth are smooth and 'shiny' in appearance. The margins of the defect are rounded in contrast to defects caused by abrasion which are sharp and have a notched appearance. Stains are usually absent (Stegé et al 1982, Simmons et al 1986). Restorations present in these teeth are usually unaffected and 'stand out' or seem to protrude above the teeth substance around it. In severe cases of perimylolysis where occlusal erosion of posterior teeth occurred,

vertical dimension may be decreased. Anterior open-bites may result when excessive enamel is lost from the anterior teeth. It is also felt that parafunctional habits such as bruxism, teeth clenching, and abnormal swallowing habits may further contribute to tooth substance loss in these patients (Brady 1980, Stege et al 1982). Severe psychological problems which can bring about such parafunctional habits will be reviewed in a later chapter.

Simmons and co-workers (1986) examined 66 patients with bulimia and found that 38 % demonstrated evidence of significant enamel erosion caused by chronic vomiting. The prevalence of erosion in those who have been vomiting for four years or less was significantly lower than the prevalence of erosion in those who had been vomiting for more than four years. Numerous observations were made by researchers regarding the degree of perimylolysis in these patients (Brady 1980, Stege et al 1982, Kleier et al 1984, Wolcott et al 1984, Simmons et al 1986, Jensen et al 1987). It is felt that the degree of tooth substance lost depends on the number of times the self-induced vomiting is carried out and the dental health awareness and practices of the patient. Someone who carries out fluoride mouth rinses will have less teeth damage than someone who allows the acidic gastric contents to collect around the teeth, in the buccal spaces between the cheeks and dentition, in the lingual spaces, and on the tongue surface. A rinse with an alkaline solution of sodium bicarbonate or a slightly alkaline mineral water to neutralise the acid has also been recommended. These mouthrinses are preferable to immediate tooth brushing as the enamel (damaged by the acid) is susceptible to the physical action of toothbrushing (Simmons et al 1986, Jensen et al 1987). Excessive eating or a diet consisting of citrus fruit juices or sweetened soft drinks is seen as a contributory factor in the degree of tooth enamel loss.

The 'binge and purge' episodes in patients with bulimia nervosa or anorexia nervosa with bulimia are carried out surreptitiously as there are feelings of guilt and lessened self-esteem associated with these eating disorders. However, the erosion of the dentition gives away the secret. The writer considers perimyolysis to be one of the worst sequelae of these eating disorders. The damage done to the dentition is irreversible.

4.3.2 Dental Caries and Gingival Inflammation

There are many conflicting reports regarding the caries incidence among patients with these two eating disorders. Some workers (Hellstrom 1974, 1977, 1990, Hurst et al 1977, Brady 1980) have reported increased caries incidence whilst others (Simmons et al 1986, Roberts and Li 1987, Milosevic and Slade 1989) have reported an unaffected caries incidence. This disparity has been noted by these workers involved and many possible explanations have been put forward. Individual oral hygiene and dental health practices like the use of fluoride mouthwashes and regular visits to the dentist are factors to be considered. The cariogenicity of one's diet would influence caries incidence. A diet rich in carbohydrates, excessive consumption of citrus fruits, the large intake of fruit juices and sugary soft drinks favoured by these patients as a result of dehydration, thirst, and binge eating, can have deleterious effects (Hurst et al 1977). Certain medications prescribed could also contribute to an increase in caries incidence. Some antidepressants that can decrease salivary flow (already low due to dehydration) are prescribed to alleviate depressive symptoms which may accompany these two eating disorders will increase the incidence of caries. Other factors, which influence caries experience in general, such as genetic predisposition, and fluoride exposure during the important tooth forming years must also play a role (Roberts and Li 1987). Alteration in salivary composition due to electrolyte imbalance may lower the buffering and remineralisation capacity of saliva (Hellstrom 1974, 1990, Hurst et al

1977, Brady 1980). The lack of, or lowered buffering capacity of saliva, may render teeth more susceptible to acid. However, Milosevic and Slade(1989) have recently found normal buffering capacity among these patients.

Different studies on the incidence of gingival inflammation (periodontal disease) among these patients have also presented different results. Reports of increased incidence of periodontal disease (Hurst 1977, Brady 1980, Hellstrom 1990), as well as an unaffected periodontal health have been reported by many others. Some of the factors affecting caries incidence among patients of these eating disorders may have a similar influence on periodontal health. Wolcott and his co-workers (1984) felt that patients with good oral hygiene and lower incidence of caries usually have a good and unaffected periodontal condition. Some workers like Roberts and Li (1987) believe that patients who suffer from anorexia nervosa frequently are also depressed and have low distorted self-image. These patients are usually not interested in their oral health or motivated about dental hygiene. They go on to propose that patients with bulimia nervosa are not likely to have such a derogatory perception of themselves and are in fact bright, well-educated individuals who are concerned about the effects of their secretive episodes of '**purge and binge**' on their dentition. As a result, these patients are likely to practice good oral hygiene and their periodontal health will be unaffected by the vomiting episodes. A rare case of a lingual abscess due to a grossly carious lower molar was reported in a patient with anorexia nervosa. (Keith and Flint 1989).

The incidence of dental caries and periodontal disease in patients with bulimia nervosa and anorexia nervosa with bulimia is very much governed by the level of oral hygiene and cariogenicity of the diet when all other factors are equal.

4.3.3 Oral Mucous Membrane Lesions

Oral mucosal (even pharyngeal mucosa at times) erythema as a consequence of chronic irritation by gastric contents has been reported (Abrams and Ruff 1986). Dry, red, and cracking lips, have also been noticed in bulimic patients. This could be due to local irritation of the gastric contents or a result of systemic factors such as vitamin C deficiency due to improper diet and altered metabolism (Abrams and Ruff 1986).

4.3.4 Parotid Gland Enlargement

Enlargement of the salivary glands, the parotid glands in particular, has been reported in patients with bulimia nervosa and anorexia nervosa with bulimia (Lavender 1969, Bernard and Shearn 1974, Dawson and Jones 1977, Levin et al 1980, Hasler 1982, Wolcott 1984, Brady 1985, Abrams and Ruff 1986, Touyz and Beumont 1989). This enlargement is usually painless and may occur unilaterally or bilaterally. The swelling may be so slight that the patient is unaware of it, or it may be so extensive that the shape of the face is distorted, resulting in great concern for the patient.

The association of this benign parotid enlargement in these two eating disorders has been reported in individual case reports before it was recognised as one of the orofacial sequela of the **'binge and purge'** syndrome. One of the first case reports was by Lavender (1969) who described his observations in a letter in the **'Lancet'**. A case report of a 19 year old woman with a two year history of parotid and submaxillary gland enlargement linked with malnutrition was published by Bernard and Shearn (1974). This malnutrition was due to surreptitious self-induced vomiting. Later on, Dawson and Jones (1977) published a case report of a Ugandan woman who was suffering from malnutrition and vomiting-induced hypokalaemic alkalosis. This patient

had obvious bilateral, smooth parotid swellings which gradually regressed following correction of her malnutrition.

In the 1980's, this parotid swelling was quite extensively observed by workers studying the dental implications of bulimia nervosa and anorexia nervosa with bulimia. Levin and his co-workers (1980) reported about 7 women with bulimia nervosa who had benign bilateral painless parotid enlargement. This swelling was generally intermittent and usually developed two to six days after a binge overeating episode ceased. Several of them had mild hypokalaemic alkalosis and a moderate elevation in serum amylase levels. Hasler (1982) presented a case reported to support his proposal that parotid gland enlargement should be considered as an additional orofacial finding of anorexia nervosa with bulimia. Brady (1985) reported that enlargement of the parotid occurs in about 10 % of the patients with bulimia who attend the Eating Disorder Service at the University of Pennsylvania. At that time, 15 to 20 new cases were seen each month. Brady reported that the parotid enlargement seem to occur more often in those patients who binge and vomit one or more times every day. About 50 % of these patients with parotid enlargement also show elevated serum amylase levels.

In spite of the numerous observations regarding parotid gland enlargement associated with bulimia, the pathophysiological causes of this swelling have not been established. All the workers who observed the swellings have put forward different theories regarding the mechanisms of parotid swellings in these patients. Brady (1985) feels that the fervent repeated stimulation of the salivary glands from repeated episodes of binge eating resulting in '**work hypertrophy**' of the salivary gland tissues is the most likely cause. The elevated levels of salivary amylase observed in Levin's study had similarly been observed in several women who ingested large amounts of starch daily and also had parotid gland swellings (Silverman and Perkins 1966, Levin 1980). Foods rich in carbohydrates might be favoured by anorexic and bulimic patients when

they binge eat. Reports regarding swelling of the parotid gland being associated with repeated secretive self-induced vomiting have suggested that the swelling of the gland could be due to the irritating effects of the vomitus on the opening and lining of the salivary ducts (Brady 1985). Suggestions of the immune system being involved have been made – indirect humoral effect on the salivary glands caused by the sudden stimulation of the pancreas in a person who is fasting and then begins binge eating (Dreiling et al 1978, Kakizaki et al 1978, Levin et al 1980). This indirect humoral effect on the salivary glands is probably responsible for the development of the parotid enlargement. Swelling of the parotid gland (termed '**nutritional mumps**') has been reported in communities where starvation and malnutrition are widespread (Katsilambros 1961, Batsakis and McWhirter 1972). Starvation and malnutrition is common among anorexic and bulimic patients.

Even though the mechanism of the parotid gland swelling in patients with bulimia nervosa and anorexia nervosa with bulimia is not clear, the writer feels that undoubtedly this association has been established and given due recognition as one of the symptoms of these eating disorders.

4.3.5 Hirsutism and Other Minor Symptoms

Hirsutism sometimes also referred to as '**lanugo**', is the excessive growth of fine downy hair. Many workers who studied patients with anorexia nervosa have noticed an unusual growth of fine downy hair particularly over the face, neck, and limbs (Brady 1980, Hasler 1982, Touyz and Beumont 1989). One of the most extensive studies on clinical features of anorexia nervosa was carried out by Halmi (1974). Of the 94 patients he studied, 18 % had hirsutism.

Dry scaly skin has also been recognised as one of the numerous physical manifestations of anorexia nervosa (Brady 1980, Touyz and Beumont 1989).

Chronic hoarseness of the voice due to the irritation of the throat by repeated assaults of acidic vomitus has also been observed by some workers (Brady 1985, Scully and Cawson 1987, Touyz and Beumont 1989).

Hirsutism of the facial region especially with a dry scaly skin should be recognised as one of the possible orofacial manifestations of anorexia nervosa.

4.4 Overview

'Anorexia nervosa' was recognised as early as in 1694 when it was first described in the medical literature by Morton (cited by Loudon 1980). In recent years, this eating disorder has been noted to be on the increase. In 1976, Crisp and his colleagues reported that one out of 100 schoolgirls over the age of 16 years attending private schools in London was probably affected.

'Bulimia nervosa' has now been recognised as a different entity from anorexia nervosa although the symptom of bulimia is common amongst many of those who suffer from anorexia nervosa. Huon and Brown (1984) reviewed the literature available between 1979 and 1983 on bulimia and reported that this behaviour is now widespread among the obese, those with anorexia nervosa, and those of normal weight.

The most likely orofacial and dental sequela of these two eating disorders has been recognised to be perimylolysis, parotid gland enlargement, possible dental caries

increase, and deterioration of the periodontal health depending on the individual's oral hygiene and dental awareness.

A review of the dental literature has indicated that unless the primary psychiatric disorder is being managed and the episodes of '**binge and purge**' are under control, dental treatment for the perimyolysis should only be palliative (Brady 1980, Stege et al 1982, Kleier et al 1984, Wolcott 1984). Definite restorative procedures should only be undertaken when the patient is on the road to recovery whilst undergoing therapy for the eating disorder. Dental hygiene education will be useful for these patients. The possible dental sequelae of the eating disorders might act as a deterrent to their behaviour. Due to the surreptitious nature of these eating disorders, the dental surgeon might have a role to play in diagnosis when presented with some of the dental and orofacial manifestation during a dental examination. Like many workers (Brady 1980, Simmons et al 1986), the writer feels that there is the need for psychiatric and dental liaison in the treatment of some patients with these eating disorders.

5 Oral Self-mutilation

Among certain groups of the population, self-mutilation seems to be relatively common. Some of these self-inflicted injuries may be motivated and sustained by the secondary gains received either directly or indirectly after the episode of factitious injury (Shira et al 1959, Altom and Di Angelis, 1989). Such self-inflicted injuries may be frequent among prison inmates who may want distraction from boredom and allocated duties in the institution (Claghorn and Beto 1967 cited by Ayer and Levin 1974).

In particular cultures such as those among certain tribes in Africa, non-therapeutic extractions and mutilation of the dentition are performed for a variety of social, religious, and cultural reasons. (Halestrap 1971, Gould et al 1984). Self-mutilation has also been frequently reported among certain types of developmental disabilities such as severe intellectual disability, Lesch-Nyhan syndrome, and Cornelia de Lange syndrome (Dura et al 1988, Jankovic 1988), and those under psychiatric care. (Ayer and Levin 1974, Sneddon and Sneddon 1975, Yesavage 1983a, 1983b, Altom and Di Angelis 1989, Thomas 1989). A review of the literature seem to indicate that some patients with certain psychiatric illnesses have been known to carry out many types and degrees of self-mutilating acts ranging from something minor like scratching to more dramatic and horrifying behaviour such as enucleation of the eyes, castration and glossectomy (Tenzer and Orozco, 1970, Sneddon and Sneddon 1975, MacLean and Robertson, 1976, Simpson 1976, Shore et al 1978, Pattison and Kahan 1983, Svirsky and Sawyer, 1987).

In this section of the treatise, oral self-mutilation which is associated with some sort of psychological or psychiatric illness, will be reviewed.

5.1 Classification of oral self-mutilation

Ayer and Levin (1974) classified self-injurious behaviour into 2 broad categories based on the possible aetiologic factors – organic or functional. Oral self-mutilation which is a form of self-injurious behaviour can be described as either organic oral self-mutilation or functional oral self-mutilation.

Organic oral self-mutilation refers to the group of self-injurious behaviour committed by the person unknowingly, involuntarily, and in a compelling manner (Ayer and Levin 1974). This type of oral self-mutilation is characteristic of hereditary disorders with biochemical and enzymatic deficiencies or intellectual disability. The most renowned of such a disorder is the Lesch-Nyhan syndrome which is a sex-linked recessive biochemical disorder caused by a deficiency of the enzyme hypoxanthine-guanine phosphoribosyl-transferase that is involved in purine metabolism. These patients behave aggressively and carry out unusual self-injurious behaviour even before reaching the age of two (Lesch and Nyhan 1964, La Banc and Epker 1981). Shear and co-workers (1971) reported on 2 children with the de Lange syndrome who bit their lips and fingers constantly, causing extensive scarring and destruction. DenBesten and McIver (1984) presented a case where an 18 month old male child, severely intellectually disabled due to toxoplasmosis mutilated his lips, and tongue.

Functional oral self-mutilation refers to the group of self-injurious behaviour which is carried out with intent and irrationally. Ayer and Levin (1974) felt this sort of self-destructive behaviour could be motivated by, and sustained for, secondary gains. They also observed that identification and recognition of certain environmental factors which initiate and reinforce the episodes of self-injurious behaviour would help to control these episodes. Altom and DiAngelis (1989) includes self-mutilation during a

psychotic episode under this category. They feel that oral self-mutilation resulting from a functional cause represents a far greater diagnostic challenge to practitioners.

At present, Ayer and Levin (1974) feel that current ideas in psychotherapy and modern psychiatry ascribes this type of behaviour to the concept of a functional cause. In the dental literature, oral self-mutilation has also been referred to under the common names of oral factitious injuries or self-inflicted gingival injuries regardless of whether the injury involves solely gingival tissues or only the teeth have been damaged, or both (Hasler and Schultz 1968, Blanton et al 1977).

Self-mutilation of oral tissues is sometimes referred to as gingivitis artefacta or factitial gingivitis (Stewart and Kernohan 1972, Stewart 1976). Stewart (1976) suggested that self-inflicted injuries to the oral tissue might be divided into two groups – gingivitis artefacta minor and gingivitis artefacta major. As the names suggested, gingivitis artefacta minor refers to a less severe type of gingival injury. This injury is usually self-inflicted by a fingernail habit provoked by a pre-existing foci of irritation. As soon as the trigger area is identified and treated, or when the offender (patient) made aware of the injurious habit, the habit will eventually stop. In contrast, lesions in gingivitis artefacta major are characteristically more severe and widespread. Gingivitis artefacta major injuries may be a physical manifestation of an emotional or psychological disorder.

Stewart and Kernohan (1972) had also earlier categorised self-inflicted gingival injuries into three types. They are:

1. Type A.

Injuries superimposed upon a pre-existing lesion (or irritation) where the patient continues to damage the site.

2. Type B.

Injuries secondary to an established habit such as fingernail biting or finger sucking.

3. Type C.

Injuries of unknown and/or complex aetiology which are usually based on some emotional disturbance.

When Stewart (1976) introduced the division of oral self-mutilation into two broader categories, Type C represents gingivitis artefacta major.

Perhaps the earliest classification was introduced by Sorrin in 1935 (cited by Blanton et al 1977). He classified destructive oral habits, some of which can result in factitious injuries, into neuroses (such as lip and cheek biting, fingernail biting, and toothpick wedging between the teeth), occupational habits (such as holding of nails in the mouth which is a practice common amongst upholsterers and carpenters, pressure while playing certain musical instruments), and miscellaneous habits (such as pipe or cigarette smoking, thumb sucking, mouth breathing, and incorrect methods of tooth brushing).

In this treatise, the types of oral self-mutilation reported will not be classified into either organic or functional aetiology. The writer feels that, since at the moment there is no concrete proof of ascribing an organic basis to some of these self-injurious behaviour, it is best to wait till the hypothesis is proven. The writer also feels that the division of these case reports regarding oral self-mutilation into those involving teeth from those involving soft tissues, allows her to better review and understand the case reports. The cases of oral self-mutilation described in this chapter will be divided into two main categories:

1. self-mutilation of teeth.
2. self-mutilation of the soft tissues i.e. the gingivae and tongue.

The writer however, recognises that some cases of oral self-mutilation cannot fit into either category. An example of such a case is: a male patient with a history of fractured mandible, and various other lesions involving the mouth and facial regions was later found to be suffering from the Von Munchausen syndrome and the injuries were self-inflicted (Thomas 1989).

5.2 Self-mutilation of teeth

A review of the literature indicates that one of the earliest documented cases of self-mutilation of teeth i.e. non-therapeutic extraction of one's own teeth was presented in 1958 (Meyer 1958). Meyer described the case of a man who systematically extracted nearly every tooth in his mouth during the year following the accidental drowning of a younger brother. Meyer concluded that this act of self-mutilation signified an identity between the dead brother and the teeth. He felt that the extractions carried out during a period of mourning and '**mental imbalance**' fulfilled a psychological need of the patient to rid himself of these physical parts that reminded him of his brother. The dead brother had ceased to play a role in the conception of his own self-image, and as a result he had to extract all his teeth.

Plesset (1959) reported the auto-extraction of 2 lower central incisors by a nine year old girl. She had a previous history of having worked her maxillary deciduous cuspids loose from her mouth. Other observations of this girl included lacerations on the lips and tongue. No explanation for this behaviour was revealed. She was referred to a mental health clinic for investigation and further evaluation of this self-mutilating behaviour.

Gelbier (1963) also reported a case involving a young child – an 11 year old boy who extracted his lower central incisors. This child was described by his mother as being nervous, restless, and fidgety, but it is uncertain whether the self-extractions were carried out due to some emotional problem (the nervous habit of playing with his teeth), or partially caused by a traumatic occlusion.

Goldstein and Dragon (1967) reported the case of a psychotic adolescent who expressed his hostility towards his mother by extracting his upper right central incisor. Six months later, he fractured his right mandible in the process of extracting his lower right canine. Goldstein and Dragon feel that psychotic children often resort to self-mutilation to direct aggression towards themselves. This anti-social behaviour is probably an expression of inner frustration and hostility towards other people.

A case of self-mutilation of teeth involving an adult was presented by Blanton and co-workers (1977). It involved a 61 year old man who placed a pocket knife between his teeth to produce a wedging force to fracture tooth enamel. Subsequently, this knife was also used to smooth the toughened rough fractured enamel surface. This damaging oral habit was performed whenever he was agitated over some disagreement with his wife.

The case report of a 29 year old single man with an 11 year history of schizophrenia who extracted his upper maxillary canine teeth and damaged an adjacent tooth with pliers was presented by Walter-Ryan and Shiriff (1985). This act of self-mutilation was carried out during an exacerbation of psychosis and the patient reported that this act decreased his tension and was painless. Several days before the autoextraction, the patient told his parents that his eye was hanging out of his head. Walter-Ryan and Shiriff felt that this was a form of change in body image and was an indication of the impending self-mutilative act.

Woody and Eisenhauer (1986) described their experience with a 12 year old boy with severe Gilles de la Tourette's syndrome who extracted 3 of his upper central incisors in spite of the presence of a mouth guard. The Gilles de la Tourette's syndrome is a complex neuropsychiatric disorder with many clinical manifestations, some of which are involuntary movements such as vulgar gestures (copraxia) and explosive involuntary utterances of inarticulate noises (barks, yelps, grunts, coughs). This syndrome is also associated with impulsive and obsessive-compulsive conditions (Lowe 1986, Jankovic 1988). This oral self-mutilation in the patient illustrates a severe compulsive behaviour.

The most recent case in the dental literature regarding autoextraction was reported by Altom and DiAngelis (1989). They presented a case report of a 27 year old man who attempted to extract his own teeth with a pair of pliers during a psychotic episode. In doing so, he managed to amputate 12 of his posterior teeth at levels varying from the gingival margin to below the crest of the alveolar bone. This man had several previous psychiatric hospitalisations for other episodes of unusual acts including one of a self-inflicted stab wound to the abdomen. All these bizarre incidents seem to be related to the patient being in a '**hyper-religious state**' in which he received orders from '**God**' to carry out these acts in an attempt to get rid of all his worldly possessions.

The 8 case reports of oral self-mutilation by autoextractions (extraction of one's own teeth) indicated that there seem to be an absence of pain during this self-mutilating behaviour. There also seemed to be a distortion of reality as almost all the patients involved in these 8 case reports felt that the oral self-mutilation served some purpose. This form of self-mutilation can be compared with the self-mutilative act of chronic wrist slashing to reduce unbearable tension levels (McSwiggan 1991). Habit mannerisms such as removing eye-lashes and scratching are also usually present.

5.3 Self-mutilation of oral soft tissue

Self-inflicted gingival injuries can range from very minor surface abrasion of toothbrush trauma (not uncommon among patients who have just been given toothbrushing instructions) to very serious injuries repeatedly inflicted on the gingival tissues despite evidence of obvious damage produced. The latter type of self-mutilation of gingival tissues is usually associated with some emotional disturbances (Stewart 1976, Groves, 1979, Pattison, 1983).

As early as 1959, Shira reported on a case of a soldier who presented with multiple factitious lesions in the oral cavity in order to retire from the army with a pension (Shira et al 1959). One of the other early case reports regarding oral mutilation of gingival tissue is about a 14 year old girl who picked at the gingivae from the buccal aspect of second maxillary molar to the canine (in both the left and right quadrants) with her fingernails, toothpicks, and even with a kitchen knife on certain occasions (Golden and Chosach 1964). This self-mutilation was seen as an oral manifestation of a psychological problem. The psychiatric assessment on this girl concluded that her emotional development, which was not commensurate with her physiological and chronological age, resulted in her having an exaggerated dependency on adults.

Other cases reported also seem to involve relatively young children. Hasler and Schultz (1968) reported a case regarding a 10 year old boy who had gingival ulcerations and recession around the posterior region of the upper teeth. The condition persisted for about 10 months during which an exhaustive investigation took place (complete blood counts, urine analysis, serology, etc.) before a definitive diagnosis of self-inflicted injury by means of 'scratching' the gingiva was made. The child was unhappy at school, felt that he was receiving unduly harsh treatment from his teacher and that his academic performance was poor. The boy had emotional problems and

received gratification from the attention received during the various consultations with different medical professionals. Three other cases of gingival mutilation in children which arose as a result of stress and a sense of deprivation were also reported (Hoffman and Baer 1968). Just as in the earlier cases, these children wanted the attention given when the oral lesions appeared. Children as young as 4 years of age have also been known to carry out self-inflicted gingival injuries. Vogan (1969) described the case of a 4 year old boy who had labial gingival recession of the upper anterior deciduous teeth. This was caused by a thumb sucking habit. The lesion healed when the thumb sucking habit was replaced by that of sticking his tongue out. The psychological basis of this was not elucidated in the case report.

Stewart and Kernohan (1972) reviewed 27 cases of self inflicted gingival injuries (5 of these cases were actually cases of self-extraction of teeth) which included 7 of their own case reports on this subject. They felt that most of these lesions were due to some readily identifiable cause although they manifested deep emotional problems which may be difficult to identify. Stewart and Kernohan compared self-inflicted gingival injuries with those inflicted on the skin (also known as **'feigned eruptions, dermatitis artefacta or factitial dermatitis'**). **'Dermatitis artefacta'** is a well recognised clinical entity in medicine (Fabisch 1980). They felt that many clinical features of self-inflicted injuries to the skin are actually just as applicable and descriptive of the lesions of self-inflicted gingival injuries. These common clinical features are:

1. The lesions do not correspond to those of any known disease.
2. The lesions usually have an unusual configuration with a sharp outline on an otherwise normal background.

3. The lesions are grouped in a peculiar fashion and distributed in areas that can be easily reached by the patient's hand (in the case of self-inflicted gingival injuries) or mouth.
4. The lesions may occur singly but more often they are multiple.

These observations are supported by Svirsky and Sawyer (1987) who reported a case of dermatitis artefacta on the right side of the face of a 33 year old man. Dermatitis artefacta has been studied; psychological investigations have suggested an inward looking, self centred personality while psychiatric investigations pointed to a background of emotional disturbances during the early formative years (Fabisch 1980).

Stewart and Kernohan (1972), however, felt the major difference between self-inflicted gingival injuries and that of the skin is the age of the patients. From their review of the 27 cases, gingivitis artefacta seems to be more common in children whereas dermatitis artefacta is seen in teenagers and adults. This observation is also supported by Pattison (1983) who reviewed 49 cases of self-inflicted gingival injury (some of which involved self-extraction or mutilation of the teeth), and Sneddon and Sneddon (1975) who conducted a follow-up study of 43 cases of self-inflicted skin lesions. Pattison (1983) reported that 78 % of the cases he reviewed were children 12 years of age or younger, 66 % were females and most of the gingival injuries were produced by the patients own finger. A variety of other objects have also been used to produce these lesions, some of which include knives, strands of hair, toothpicks, a stick of cane and baby pacifiers. In the study by Sneddon and Sneddon (1975), 19 out of the 43 patients were in their teens, one was 9 years of age and the rest were adults when the skin lesions were first seen.

Ayer and Levin (1974) who proposed the theory of categorising self-injurious behaviour according to an organic or functional etiology also presented a case about an eight year old girl who caused a buccal gingival defect adjacent to a mandibular incisor by scratching that area. The girl complained that the gum often 'itched' and that resulted in the scratching. Other authors (Golden and Chosach 1964, Hasler and Schultz 1968, Groves 1979) also feel that when no discernible cause is found for these self-inflicted gingival injuries, they represent a manifestation of some sort of psychological or emotional disorder.

Blanton and co-workers (1977) presented four case reports of self-injurious behaviour involving the oral tissues which were a result of emotional problems (Blanton et al 1977). Two of these case reports involved adults aged 61 and 35 years. The other two case reports were about a five year old boy and another who was nearly six years old.

A case of 40 year old woman who self-mutilated her mouth and skin by mechanical and chemical means was also reported (Barrett and Buckley 1988). Psychiatric evaluations revealed severe underlying depression, melancholia, and feelings of inferiority.

The cases reviewed in this section so far involved only self-inflicted injurious lesions of the gingivae or skin. Horrific cases of self-injurious behaviour where the tongue was involved have also been reported. A 29 year old woman self mutilated her tongue by biting it repeatedly (Slawson and Davidson 1964). It was felt that she had a depressive reaction with conversion features and hysteria. A 46 year old woman (who had been hospitalised previously for schizophrenic reactions of the catatonic type) had cut off her own tongue because she had "*received a message from God*" to do so. (Tenzer and Orozco 1970).

The third case involved a 16 year old boy with a diagnosis of Gilles de la Tourette's syndrome who had a compulsive habit of biting his tongue. (Lowe 1986). The right lateral tip of the tongue was missing due to repeated biting. Even though he felt the pain, he felt that the biting relieved the tension. The tongue biting can be regarded as an obsessive-compulsive behaviour.

Self mutilation of the oral soft tissues seems to present a situation as bizarre as that presented by that of self-mutilation of the teeth.

5.4 Overview

Numerous cases of oral self-mutilation involving either soft tissues or teeth (and even both in certain reports) have been reviewed. Many of these offenders (patients) are young children (Hoffman and Baer 1968, Stewart and Kernohan 1972, Pattison 1983, Svirksy and Sawyer 1987). When the destructive oral habit or behaviour (which is causing the damage) is pointed out, many of these habits stopped or were corrected. However, in numerous cases, this was not possible. These cases probably represent some deep underlying emotional, psychological, or psychiatric disorder.

The origin of this self-injurious behaviour may be the need for attention or sympathy and even personal gratification. In many instances, the oral self-mutilative act is carried out during a psychotic episode, common amongst schizophrenic or depressed patients. Indirect hostility has been shown to correlate with self destructive behaviour in both schizophrenic and depressed patients (Yesavage 1983a, 1983b). Oral self-mutilation, a form of self destructive behaviour could perhaps be viewed as a form of indirect hostility. Oral self-mutilative behaviour may also represent either some uncontrollable obsessive-compulsive behaviour as in Tourette's syndrome or a clinical presentation of

the Von Munchausen syndrome. Oral self-mutilation may be a form of self-stimulation to obtain relief from boredom (McSwiggan 1991).

Ayer and Levin (1974) who reviewed the psychiatric and psychological aspect of oral self-mutilating behaviour believe the theory of learning by paradigm (example) which emphasises on conditioning and deconditioning might be successful in stopping/correcting self-injurious behaviour. Psychotherapy has also been proposed to be able to limit episodes of self-injurious behaviour.

Whatever the psychological, emotional, or psychiatric condition that may be the reason behind the oral self-mutilative behaviour, it is important to recognise that extensive and permanent damage can result. These lesions can prove to be an enigma to the unsuspecting dentist who is trained to recognise and treat organic diseases. Numerous fruitless and needless investigations may be carried out and the dentist may still not be any nearer to a solution or diagnosis. As in many other medical conditions where an emotional or psychiatric problem may be the origin, the diagnosis comes about only by exclusion of other organic causes.

The writer feels that oral self-injurious behaviour poses to the dentist not only diagnostic problems but also the treatment of the resulting lesions. It would be prudent for a competent dentist to seek the help of psychiatric professionals when it is suspected that some lesions in the mouth may be caused by oral self-mutilation. This oral self-mutilating behaviour may be a manifestation of an emotional, psychological, or psychiatric problem and may be an attempt by the patient at non-verbal communication to appeal for help.

6 Bruxism, and Psychological Considerations in Prosthetic Dentistry

6.1 Bruxism

Bruxism is the term used to define *"the grinding, clenching or pressing of the teeth at times other than mastication of food or swallowing"* (Nadler 1957, Ramfjord 1961, Arnold 1981, Scott and Humphreys 1987). It is thus regarded as a parafunctional behaviour. It may be a daytime phenomenon and is referred to as diurnal bruxism. When it occurs at night time while a person is asleep, it is referred to as nocturnal bruxism. This non functional occlusion of teeth may occur either unconsciously or subconsciously (Nadler 1957, Reding et al 1966, Arnold 1981).

6.1.1 Prevalence of Bruxism

The prevalence of bruxism has been reported in both children and adults. Using a questionnaire survey among two groups of students – one group of 2,290 students between the age of 16 and 36 years and another group of 1,157 students between the age of 3 and 17 years, Reding and his co-workers (1966) concluded that bruxism was a common enough occurrence to warrant its inclusion among public health problems. They also found no statistical differences between the prevalence of bruxism in males and females. 15% of the students aged between 3 and 17 years reported a history of bruxism.

Another extensive study was conducted in Poland on 4,929 subjects (Wigdorowicz-Makowerowa et al 1979). This population consisted of 2,100 school children (10 to 15 years of age), 429 medical students, 400 military students, 1000 soldiers (20 to 23

years of age), and 1000 middle aged soldiers (39 to 45 years of age). A high incidence of bruxism was reported in each group except among the schoolchildren where the incidence was found to be 3.9%. In the adult population studied bruxism occurred in over 21% of cases.

By means of a questionnaire to 1,052 University students whose average age was 19 years, Glaros (1981) surveyed the incidence of diurnal and nocturnal bruxism. Glaros subdivided the bruxists into exclusively diurnal, exclusively nocturnal and both diurnal and nocturnal groups. About 31% of this population were found to be bruxists and diurnal bruxism occurs more often than nocturnal bruxism.

Results from other studies also indicate that bruxism is a relatively common phenomenon (Seligman et al 1988, Ekfeldt et al 1990).

6.1.2 Aetiology

Many investigators feel that multiple factors may be involved regarding the aetiology of bruxism (Nadler 1957, Arnold 1981, Scott and Humphreys 1987, Cash 1988, Faulkner 1990, Attanasio 1991). These factors include the psychogenic component, occlusal discrepancies, central nervous system involvement, genetics, systemic factors or even allergies. The writer feels that although it is not appropriate to classify bruxism as an oral manifestation of mental illness, the possible psychological factors in the aetiology of bruxism necessitates this dental phenomenon to be investigated in this treatise.

6.1.3 Psychogenic Component of Bruxism

The Cornell Medical Index was administered to 50 female patients at the College of Dentistry, New York University, and it indicated a definite correlation between bruxism and the anxiety state (Thaller 1960). Walsh (1965) suggested that bruxism is a manifestation of anxiety and reported on 2 cases of bruxism related to schizophrenia and paranoid psychosis.

Molin and Levi (1966) conducted a personality investigation of 103 bruxists and matched non-bruxist subjects of the population. It was demonstrated that the bruxist group exhibited more neurotic (anxiety and depressive) traits than did the control subjects. The bruxist group also reported an inclination to react with muscle tensions in other body regions besides the jaw region. Another study using the Cornell Medical Index and the Rosenzweig Picture Frustration study on 86 patients also showed a significantly higher anxiety trait among bruxists than non-bruxists (Thaller et al 1967). The bruxist patients were found characteristically to use hostility turned inward in order to relieve frustration and their bruxist habit may be a means of relieving frustration. The non-bruxist patients tended to manifest their frustration through punitive actions directly.

One hundred and ninety-two school children (12 years of age) in Umeå were involved in a study to evaluate a possible connection between bruxism and emotional disturbances (Lindqvist 1972). The results showed that stress symptoms and nervous disorders were more prevalent in children with bruxism. Bruxism was diagnosed by the presence of atypical wear facets in the dentition of these children.

It has been indicated that emotional or nervous tension, pain or discomfort, together with occlusal interferences, can increase the tonic activity in the jaw muscles leading to

non functional gnashing and clenching of the dentition (Graf 1969). Arnold (1981) felt that manifestation of psychological stress is focused around the oral cavity as it is intimately tied to emotions such as satisfaction, frustration, anxiety, and anger. These emotional associations are made early in life since the mouth is the infant's way of receiving food and the earliest way of exploring the environment. Like Arnold, Mikami (1977) also applied Freud's psychoanalytical theory to give a psychogenic basis to the aetiology of bruxism.

6.1.4 Other Possible Factors Involved in Bruxism

Frisch and his co-workers (1960) did not establish a relationship between bruxism and aggression. Ramfjord (1961) concluded from his clinical and electromyographic study of 34 subjects that the most common cause of bruxism was a discrepancy between centric relation and centric occlusion. He eliminated the bruxism in all 34 patients with occlusal adjustments.

Bruxism during sleep was found to occur most frequently during periods of rapid eye movement which are associated with dreaming (Reding et al 1964, Robinson et al 1969). Rugh and his co-workers (1984) demonstrated that there is no relationship between nocturnal bruxism and experimental occlusal discrepancies. In his study on sleep patterns and habits, Colquitt (1987) concluded that certain sleeping positions apply lateral force to the mandible contributing to the incidence and severity of nocturnal bruxism. Use or abuse of psychotropic drugs, appetite suppressants and the amphetamines may all play an aetiological role in bruxism (Scott and Humphreys 1987).

Bruxism was found to be common among children with cerebral palsy (Rosenbaum et al 1966, Lange et al 1983). It is also frequently a problem in people with severe and profound levels of mental retardation (Dura et al 1988).

The possibility of a combined aetiology for bruxism has been suggested due to the different results obtained from various studies over the years. The involvement of the psychogenic element cannot be discounted and, as suggested by Mikami (1977), management of psychic tension and neuromuscular habits may play a role in the treatment of bruxism.

6.1.5 The Significance of Bruxism to Dentistry

The presence of bruxism can affect the oral cavity in many ways and can have many deleterious effects. The force generated whilst bruxing can be considerable (Arnold 1981) and the resulting trauma can cause a range of toothwear from mild to severe (Gecker and Weil 1963, Nadler 1966, Zeldow 1976, Glaros and Rao 1977). The toothwear is seen as facets and can be localised or present throughout the dentition. When the attrition is excessive and generalised, a loss of facial vertical dimension or mandibular overclosure can result (Reding et al 1966).

Other dental problems that may arise from bruxism include increased tooth mobility, damaged periodontal ligament and periodontitis, hypercementosis, fractured teeth and even pulpitis (Nadler 1957, Zeldow 1976, Glaros and Rao 1977, Arnold 1981, Pavone 1985, Attanasio 1991). Painful and tender masticatory muscles or severe hypertrophy of these muscles may sometimes present (Walsh 1965, Nadler 1966, Arnold 1981). Resorption of alveolar bone and resorption possibly in the temporomandibular joint has been attributed to bruxism (Nadler 1966, Arnold 1981,

Attanasio 1991). Non dentally related problems like preauricular and head pain caused by bruxism has also been reported (Woodrow 1964).

As demonstrated by the list of dental problems that can arise due to bruxism, it is important that bruxism is detected early and treated. The writer feels that bruxism due to undue stress or anxiety or some other psychological factor may be differentiated from that which is due to a mixed aetiology. Consultation with the appropriate mental health professional might be needed.

6.2 The psychological considerations of prosthetic dentistry

As mentioned in Chapter 3, the complex anatomical, physiological and developmental aspects of oral function can result in orofacial manifestations of mental illness. On the same basis, dental treatment may also have profound psychological implications. One of these possible psychological implications can result in dental phobia which will be discussed in the next chapter. On a similar note, dental treatment will have psychological implications. The psychological implications in prosthetic dentistry is one of the most widely studied aspects within the practice of dentistry. The writer has therefore reviewed the literature in this important area. Another reason for reviewing only the psychological implications of prosthetic dentistry is that depression and anxiety (common symptoms of various mental illnesses) has a considerable role to play in the success of prosthetic treatment. Included in this section of the treatise is also the psychological implications of tooth loss/extraction which interacts with or influences the psychological implications of prosthetic dentistry.

Some patients may be unable to adapt to full dentures at all in spite of high technical quality in their dentures. There are a number of factors responsible for this —

anatomical, physiological and psychological (Koper 1964, Smith 1976, Friedman et al 1987). The psychological factors will be reviewed in this section of the treatise.

One of the psychological factors that influences a patient's maladaptive behaviour towards full dentures is the patient's attitude towards the loss of teeth. One of the greatest fears the human being faces is the that of mutilation and certain patients see the loss of teeth as a threat of mutilation (Sosnow 1962). Loss of teeth can also be viewed as damage to one's body image (Pilling 1973, Friedman et al 1987). This loss and disfigurement to the body is seen to be a severe blow to the person's sense of wholeness and self-esteem. However, most significantly, the loss of teeth means the advancement in age (Swoope 1972). Other significant symbols of tooth loss include loss of femininity, loss of virility, loss of attractiveness and vitality, and body degeneration (Swoope 1972, Friedman et al 1988a). It is not surprising that some patients view the loss of teeth with great trepidation.

The symbolic significance of loss of teeth will influence the patient's attitude towards full dentures. To some patients, dentures are not just dentures; there may be a variety of meanings to the recipients (Plainfield 1962, Swoope 1972, Friedman et al 1987). The meaning attached to dentures by some patients is tied together with that of tooth loss and is similarly seen to represent or be associated with growing old, loss of virility and vitality, changes in appearance, and a general decrease in their worth as individuals. The negative emotional significance of tooth loss and dentures will cause certain patients to respond with fear and anxiety (Swoope 1972, Friedman et al 1987). The emotional fight against losing teeth can carry on even until the dentures are completed, resulting in psychologically unacceptable dentures. In contrast, patients who accept the full dentures as a retention of their youth will be able to accept them satisfactorily (Plainfield 1962).

The significance of tooth loss and dentures to certain patients are influenced by parental attitudes and their current life circumstances (Friedman et al 1988a). The attitudes of parents towards body values and ideas (either positive or negative) are usually assumed and learnt by their children. One's ability to cope with the loss of teeth can be impaired if the situation of present life circumstances is stressful and emotionally taxing. The symbolic meaning of teeth (just as the symbolic meaning of tooth loss and dentures) can help influence a patient's adaptive response to dentures.

There are other emotional factors which need to be considered for the success of full denture treatment. High morale and good self-image is related to greater flexibility in adapting to full dentures and ensuing satisfaction (Silverman et al 1976). Chamberlain and co-worker (1985) used the Beck Depression Inventory to measure the prevalence of depression among 120 patients who sought full denture treatment at The University of Michigan School of Dentistry. About 11% of these patients were found to have depressive symptoms. Depressive symptoms are usually accompanied by negative self-feelings and distorted body image, and these may bring about difficulty in coping with denture wearing effectively (Beck et al 1979, Rich and Kabcenel 1987). The common presence of depressive symptoms among the age group that wear full dentures may need to be another psychological consideration in complete denture prosthodontics. Pitts (1985) has identified a similarity between the difficult denture patient (DDP) and the help-rejecting complainer (HRC). DDPs are patients with abnormal and uncommon denture problems whilst HRCs describe patients who seek help from psychotherapists and yet reject both the treatment and attempts of the therapist to help them. Pitts hypothesised that the HRC and DDP are working through the same mechanism. Some of the characteristics of this mechanism include the need for both the DDP and HRC to seek treatment from someone that they will ultimately reject. They have the self-fulfilling prophecy of failure of the treatment they seek.

It can be said that anxiety, fear, and depression, are among the many emotional/psychological factors that can influence a patient's ability to adapt to dentures. Friedman and his co-workers(1987) have identified three types of maladaptive responses (as a result of fear, anxiety, and depression) to full dentures. In maladaptive Class I response, the patient adapts to the dentures physically but not psychologically. In maladaptive Class II response, the patient is unable to adapt to dentures physically or psychologically. He becomes a difficult denture patient who complains frequently and requires a protracted amount of attention. The patient with maladaptive Class III response cannot and does not wear the denture. This patient is emotionally overwhelmed by the experience and becomes depressed and withdraws from society.

As illustrated above, it is of great importance that an assessment of the psychological factors that governs the patient's attitudes to tooth loss and acceptance of dentures becomes part and parcel of the dentist's responsibility for certain patients. The incidence of depression or depressive symptoms and other mental illness among the geriatric population has been recognised to be substantial (Butler 1975, Levenson 1983, Schmahl 1984, Whittle 1987). The possibility that psychological factors may complicate prosthodontic treatment for these geriatric patients has to be recognised.

The Cornell Medical Index (CMI) has been recommended for use as a prognostic aid for full denture patients (Bolender 1969). Bolender and his co-workers established a definite correlation between increase in total CMI score above 25 and decrease in patient satisfaction. Many researchers in this field of study have stressed the importance of a perceptive dentist (who recognises the patient's needs and feelings) and a good dentist-patient relationship in reducing the part that psychological factors play in regards to patient's adaptability to full dentures and ensuing satisfaction

(Plainfield 1962, Koper 1964, Swoope 1972, Swoope 1973, Silverman et al 1976, Smith 1976, Pitts 1985, Friedman 1988b).

6.3 Overview

Many factors seem to be implicated in bruxism. To date, no one factor has been shown conclusively to be responsible for this relatively common phenomenon. The writer feels that bruxism can perhaps be viewed as a parafunctional behaviour that may have psychogenic overtones.

Patients dissatisfaction with dentures or their inability to adapt to them may not be a result of poor technical quality of the dentures or lack of adequate denture supporting tissues. It is important to understand that psychological factors may be responsible for the maladaptive response towards full dentures.

In certain people emotional factors such as anxiety, fear, and depression seem to play a significant role in the propagation of bruxism and can affect their ability to adapt to full dentures. In dentistry, psychological/emotional factors such as stress have also been implicated in other oral lesions like aphthous ulcers and acute necrotising ulcerative gingivitis (Harris 1975, Harris and Davies 1980, Poporad and Kaye 1983). In order for dental treatment to be carried out successfully, it is necessary for the dentist to realise that psychological factors may play a significant role.

7 Psychiatric Disorders with Special Relevance to Dentistry

The previous chapters on orofacial manifestations of mental illness have dealt with specific dental problems as a result of certain psychiatric disorders. In this chapter, issues that are dealt with are psychiatric disorders that are relevant to dentistry even though a specific orofacial manifestation may not be present.

7.1 Anxiety

Anxiety is a common symptom and, to a certain degree, is normal, and can have a protective effect (Sandlin 1987, Andrews 1989). In 1908, Yerkes and Dodson described a relationship between anxiety and performance whereby performance at skilled tasks is enhanced as anxiety is increased above a baseline of relaxation but a rapid deterioration in performance occurs when anxiety becomes severe (Andrews 1989). This relationship is well illustrated by Figure 1.

It is important to be aware of both the facilitating and debilitating effects of anxiety (Sandlin 1987, Andrews 1989). It is the debilitating effects of anxiety regarding dental treatment and procedures that is implied by the writer of this treatise under this section on dental anxiety/phobia.

7.1.1 Dental Phobia

Since its origins, dentistry has been associated with anxiety and fear more than any other branch of health care (Borland 1962, Kleinknecht et al 1973, De Martino 1987). With the introduction of anaesthetics, pain induced by certain dental procedures which

resulted in the anxiety and fear felt by patients regarding dental treatment is under better control but nevertheless, a proportion of the population throughout the world still has a high level of fear and anxiety towards dental treatment (Freeman 1985, De Martino 1987, Scott and Humphreys 1987).

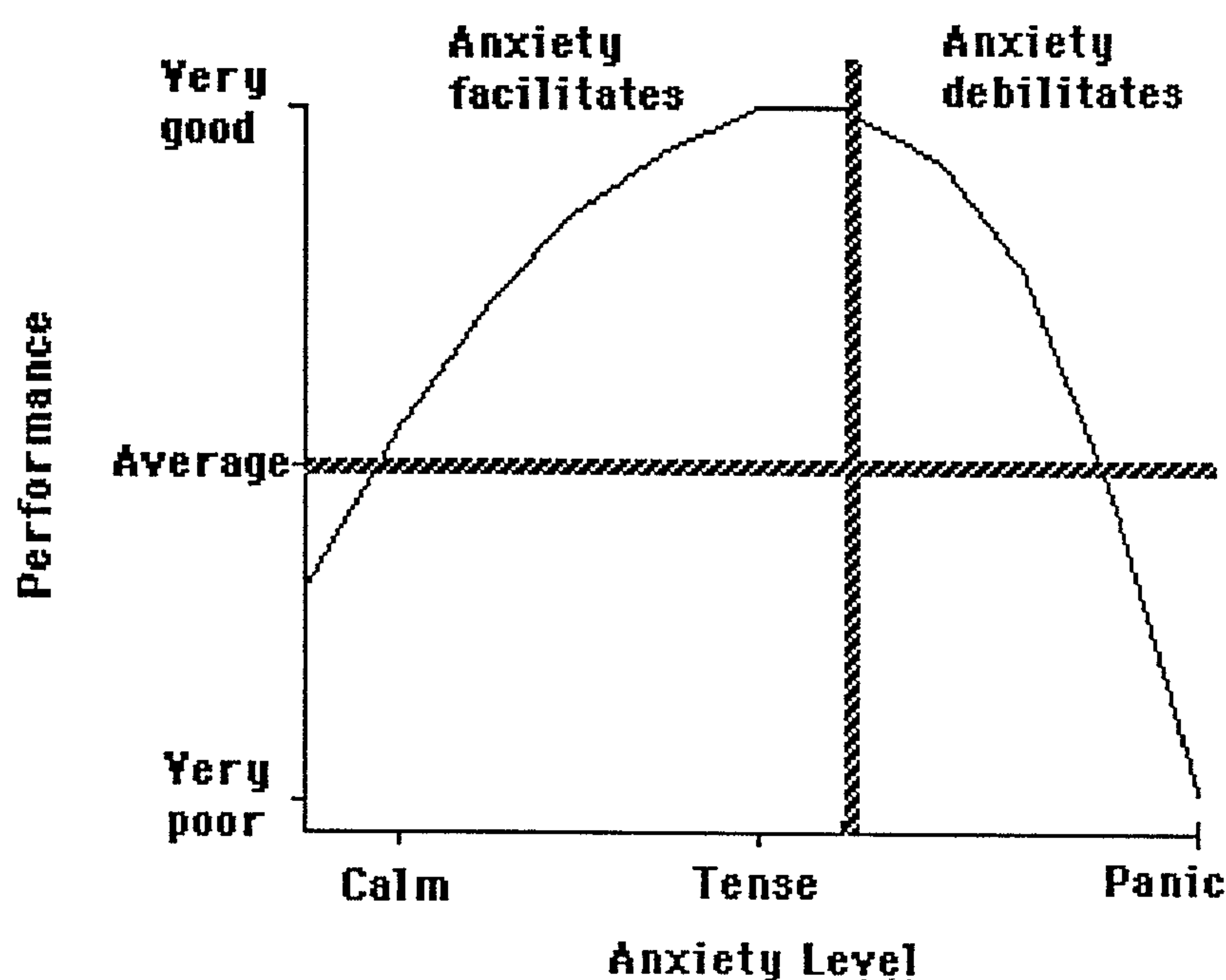


Figure 1. The Yerkes Dodson curve
Relationship between performance and the anxiety level
(Adapted from Andrews 1989)

This fear and anxiety regarding dental treatment has been referred to in the literature as dental fear, dental anxiety, and dental phobia or odontophobia (Borland 1962, Lauth 1971, Hall and Edmondson 1983, Freeman 1985, Schuurs et al 1985, Smith et al 1987). For uniformity, the writer will refer to this inordinate fear of dental treatment as dental phobia. Dental phobia can become so severe that all dental treatment is avoided and serious consequences to the dental health can result (Gelder et al 1989e). As with other types of phobia, this fear of dental procedures is irrational and so persistent that these people only seek dental care when in great pain and demand sedation or a general

anaesthesia for all dental procedures (Hall and Edmondson 1983, De Martino 1987, Lindsay et al 1987, Smith et al 1987).

7.1.1.1 Prevalence of dental phobia

Unsworth (1984) reported that in the Adult Dental Health Survey in England, 41% of adults admitted that they put off going to the dentist because they were scared. Between 73% and 79% of a population of 609 undergraduate students at Kent State University reported some level of dental fear (Scott and Hirschman 1982). Eight to fifteen percent of these subjects were highly anxious about dentistry. In a recent epidemiological study carried out in a large United States city (Seattle), it was reported that 20% of this population are "*somewhat afraid, very afraid or terrified*" about dental treatment (Milgrom et al 1988). In another recent study, in Japan, 6 to 14% of University students were "*very afraid*" or "*terrified*" of the dentist (Domoto et al 1988). In Singapore, the prevalence rate for high dental fear among young adults has been reported to be between 78 and 208 fearful young adults per 1000 of the population (Teo et al 1990). In Singaporean children, it was found that 177 children per 1000 population were fearful of the dentist (Chellapah et al 1990).

As seen from the above figures, there is a degree of fear of dental treatment among most people and a smaller population actually suffer levels of anxiety and fear high enough to be classified as phobia. This fear of dental treatment is significant to all dentists as on the average it takes 20% more time to treat anxious patients and dentists tend to work below their best when patients' anxiety is a prominent factor (Unsworth 1984). Surveys have shown that fear and anxiety related behaviour is a difficult aspect of patient management (Borland 1962, O'Shea et al 1984, Corah et al 1985b, Milgrom et al 1988, Smith et al 1990). From the dentists point of view, these anxious and

fearful patients are not only difficult to manage but can cause a significant amount of stress for the dentist and staff.

7.1.1.2 Factors associated with dental phobia

Fear of dental treatment may be developed by direct exposure and experience (this is known as '**aversive conditioning**'). It could also be acquired by observing pain and fear reactions in others exposed to the aversive stimuli without actually experiencing the direct trauma; this is known as '**vicarious learning**' or '**modelling**' (Hall and Edmondson 1983, Rankin and Harris 1984).

When the fear and anxiety which arises are out of proportion to the demands of the situation and cannot be reasoned away, or rationalised and leads to avoidance of the fearful situation, a phobia is said to be present. This phobia is beyond voluntary control. In reaction to a phobic situation or circumstance, a defence mechanism will be set up where the individual will protect himself by avoiding the stimulus or the circumstances that elicit it (Sandlin 1987, Andrews 1989, Gelder et al 1989f).

Fear of dental treatment or procedures, whether to the extent of a phobia or not, is distressing for both the dentist and patient. Over the years, many studies have been carried out in an attempt to understand this sort of behaviour and find means to overcome this agonising situation. From these numerous studies, dental fear or phobia has been found to have a multifactorial aetiology.

Women patients have been found to have higher scores for dental phobia and also consider themselves to be more fearful of dental treatment when compared with men (Lautch 1971, Kleinknecht et al 1973, Corah et al 1978, Wardle 1982, Schuurs et al 1985, Smith et al 1987). Many tests or questionnaires have been designed to measure

the level of anxiety towards dental procedures. Two of them are the Dental Beliefs Survey (DBS) developed by Getz and co-workers and the Dental Anxiety Scale (DAS) by Corah (Kunzelmann and Dünninger 1990). The writer has not personally had any experience with application of these of questionnaires.

A very large number of dental phobic patients had past traumatic dental experiences (Lautch 1971, Kleinknecht et al 1973, Hall and Edmondson 1983, Berggren and Meynert 1984, Smith et al 1987). From various studies, it is obvious that past traumatic dental experiences leave deep-rooted fear in the patient. When these events occur in childhood, the memory lingers on into adulthood and may be unintentionally passed on to the next generation. Children dental phobics usually have either a parent or an immediate member of the family who also has this fear. Peer group influences are also significant in the propagation of dental phobia in children (Lautch 1971, Kleinknecht et al 1973, Berggren and Meynert 1984). This relationship of dental fear in the family is especially noted between mother and daughter.

According to some researchers unpleasant dental events produces negative affective changes which propagates the development of this dental anxiety (Brown and Smith 1979, Freeman 1985). This is known as a primary effect and is significant especially in children.

Perhaps the most significant aspect of traumatic past dental experience is the impression left in the patient's mind. This impression will influence the fear of the unknown. A combination of the initial fear experienced during the traumatic event and the fear of the unknown profoundly influences the development of dental phobia. This is known as a secondary effect of a past traumatic dental experience (Freeman 1985). This probably explains why there is a clear association between past and expected pain to certain dental procedures than between past and experienced pain, among dental

phobics (Wardle 1982, Kent 1985, Kent and Warren 1985, Kent 1990). The level of pain anticipated before a certain dental procedure is higher than that actually experienced during the subsequent procedure.

As mentioned earlier, family and peer groups influences play a considerable part in increasing the level of dental anxiety. This means of acquiring dental information about unpleasant dental experiences resulting in dental phobia is through vicarious learning or modelling (Brown and Smith 1979, Hall and Edmondson 1983, Berggren and Meynert 1984, Rankin and Harris 1984).

Perhaps one of the most interesting factors associated with dental anxiety or phobia is the personality of the patient. The personalities of phobic patients may in general be described as timid, shy, dependent, and immature (Lautch 1971, Hall and Edmondson 1983). Dental phobic patients may be anxious by nature in a wide range of circumstances. It was found that some dentally anxious patients score identically on the Eysneck Personality Questionnaire (EPQ) as anxiety neurotics (Lautch 1971). Others have found either the presence of a higher level of neuroticism or psychosomatic symptoms such as tension headache and stomach ache among dental phobics (Hall and Edmondson 1983, Berggren and Meynert 1984). Some of these patients had received past treatment for psychiatric conditions or were still being treated (Berggren and Meynert 1984).

The mouth has a very high cortical representation (McSwiggan 1991). It is the baby's most important area for assessing the world, and getting pleasure (eating). The psychological importance of the mouth is established at this early age. The psychological significance of the mouth has a part to play in the development of dental anxiety among some dental phobics (Borland 1962). These individuals usually have a distorted emotional concept of the mouth.

Other researchers have found a lower level level of education and those belonging to a lower socioeconomic class to be associated with higher level of dental anxiety among the dental phobics (Berggren and Meynert 1984, Schuurs et al 1985). Dental phobia has also been found to be prevalent among edentulous patients (Schuurs et al 1985, Vervoorn et al 1989, Stouthard and Hoogstraten 1990). In a comparison study of dental anxiety between dentate and edentulous subjects, the proportion of anxious subjects is lower in the edentulous group, yet the proportion of phobic subjects is more than twice that in the dentate group.

Patient-dentist relationship may play a significant role for patients with dental phobia. In their study of 322 Western Washington State College students regarding fear of dentistry, Kleinknecht and his co-workers (1973) found that the perception of the dentist as a person was important in determining the patient's attitude towards dentistry. Subjects in this study who were positive about dentistry reported that they liked their dentist whereas those who reported adverse reactions to dentistry had a personal dislike of their dentists. From their investigation regarding causes, symptoms, and consequences of dental fear (among 160 adults who suffered from dental fear and avoidance of treatment), Berggren and Meynert (1984) felt that the interaction between dentists and their patients plays an important role. They found that the most desired qualities in dentists were being understanding, ability to avoid inflicting pain, and professional behaviour. In many other studies on treatment of dental phobia, characteristics of the dentist prescribed to be an important factor in alleviating fear include (besides those mentioned above) being tolerant, sympathetic, and showing a genuine interest in helping the patient overcome their fear of dentistry (Borland 1962, Hall and Edmondson 1983, Scott and Humphreys 1987, Smith et al 1987). In recent studies on dental fear, data showed that patients who suffered from dental fear had more negative beliefs about the dentist and were less satisfied in general regarding their treatment (Corah et al 1985a, Kunzelmann and Dünninger 1990).

It is quite obvious to the writer of this treatise that many factors play a role in the aetiology of fear of dentistry or dental phobia. It is of utmost importance for the dentist to be aware of the circumstances which surround dental phobia and perhaps to be able to make the differentiation between the dental phobia brought about by past traumatic dental experiences or as a result of '**vicarious learning**' from family or peers. In some patients the fear of dentistry may be only one among many fears that they may have. In any of these situations the dentist will need help from other health professionals to manage this fear.

Patients who have dental phobia are usually fearful of a number of definite events or procedures in the delivery of dental treatment (Gale and Ayer 1969, Kleinknecht et al 1973, Scott and Hirschman 1982, Berggren and Meynert 1984, Corah et al 1985b, Freeman 1985, De Martino 1987, Domoto et al 1988, Milgrom et al 1988). The sight of the anaesthetic needle and subsequent injection, the sight and sound of the drill, extraction of a tooth, and the anticipation of treatment and pain, were regarded as the events that dental phobics were most fearful about. Being told that they had bad teeth or being laughed at by the dentist can induce some degree of dental anxiety too.

Patients who have this fear of dentistry usually do not seek treatment until they have no choice i.e. when the pain becomes uncontrollable. These patients will put off their appointments (cancellations or not showing up) or are late for their appointments (Borland 1962, Gale and Ayer 1969, Berggren and Meynert 1984, Lindsay et al 1987, Smith et al 1987, Milgrom et al 1988, Smith et al 1990). Many avoid dental treatment to such an extent that poor oral health is the unfortunate result. Due to the complex nature relating to the aetiology of dental phobia, management of this requires behaviour therapy or modification, pharmacological pain control, and most important of all, a dentist who has a genuine interest in treating these patients.

7.2 Hypochondriasis

Hypochondriasis is the condition where the individual exhibits a morbid preoccupation with his health or bodily functions (Kenyon 1976, Meares 1989). This excessive concern with one's health is a form of abnormal illness behaviour (Pilowsky 1978). Patients with hypochondriasis present with a firm belief that they have some specific illness and may present with pain (Bianchi 1973, Scott and Humphreys 1987). This belief of having a serious disease is based on the individual's interpretation of physical signs or sensations and persists despite medical reassurances (Gelder et al 1989g, Meares 1989).

Hypochondriacal beliefs are ubiquitous in the elderly (Henderson and Rosenman 1989) and found more commonly among the lower socioeconomic status groups (Pilowsky 1978). Bianchi (1973) and Pilowsky (1978) are in agreement that individuals with hypochondriacal beliefs are from large families with a history of illness and operations.

Kenyon (1964) feels that the commonest region for hypochondriacal symptoms is the head and neck. Scott and Humphreys (1987) supports this notion and feel that this is a psychiatric disorder of special relevance to dentistry. Hypochondriasis has been defined by the DSM-III-R as being a primary disorder (cited by Mears 1989, Gelder et al 1989g) whilst some researchers like Kenyon (1964) and Pilowsky (1970) disagree. Hypochondriasis as a primary condition unassociated with any other signs of mental illness, has a poor prognosis and suicide is a risk amongst these patients (Bebbington 1976, Scott and Humphreys 1987). Kenyon (1964) and Pilowsky (1970) found this hypochondriacal phenomenon to present with other psychiatric illness such as schizophrenia, depression, or personality disorders.

The writer has not come across any mention of dental health being the centre of a hypochondriacal belief in the dental literature except from Scott and Humphreys (1987) who raised the possibility of this event. The writer however feels that it is essential that the dental surgeon who treats patients with mental illness is aware of this possibility.

7.3 Dysmorphobia and monosymptomatic hypochondriacal psychosis (MHP)

Dysmorphobia and MHP are two closely allied disorders (Leader 1978, Scott and Humphreys 1987). The writer feels that these two psychiatric conditions are similar to hypochondriasis. Patients with MHP can be successfully treated with pimozide whilst this drug does not seem to help patients with dysmorphobia (Reilly and Beard 1976, Leader 1978, Mack 1985).

7.3.1 Dysmorphobia

In dysmorphobia, the individual has an excessive concern or fear of being deformed and the unsightly appearance is usually fixed to a single feature (Hay 1970). To other people, the appearance is normal or there is very minor abnormality. This concern is disproportionate to any trivial blemish which may be present. The common dissatisfactions and complaints are the nose, ears, mouth, breasts, buttocks, and penis, but any part of the body may also be involved (Hay 1970, Gelder et al 1989h).

Hay (1970) found that out of his 17 dysmorphic patients, 11 had severe personality disorder, 5 had schizophrenia and 1 a depressive illness. Andreasen and Bardach (1977) found dysmorphobia to be a symptom among patients with personality disorders, whilst Connolly and Gipson (1978) reported it to be an early symptom

associated with schizophrenia. In individuals with psychosis this preoccupation is usually delusional whilst in those with personality problems, it is usually an overvalued idea (McKenna 1984, Gelder et al 1989h). Individuals with dysmorphobia blame all their difficulties in life on this supposed deformity. In dentistry, a patient with dysmorphobia will have the delusion that some part of his face or jaw is deformed (Scott and Humphreys 1987).

7.3.2 Monosymptomatic Hypochondriacal Psychosis (MHP)

MHP is a rare condition in which the individual holds a specific, well localised delusion concerning some part of the body (Leader 1978, Mack 1985, Scott and Humphreys 1987). This false belief is not amenable to reason. Examples of this delusion are that the tooth is infected with worms or the skin is full of spiders (Mack 1985). This delusion is separate from other mental functions which are unimpaired. Even though thought processes and personality remains unaffected, the individual's life style or habits are modified (Munro 1980).

Individuals with MHP express delusions that they smell or are infected (Leader 1978) and will seek medical treatment which they genuinely think necessary. As this treatment is often not possible, the clinicians consulted are accused of incompetence and the need for psychiatric help is usually refused (Mack 1985). As mentioned earlier, MHP responds well to treatment with pimozide (Riding and Munro 1975, Reilly and Beard 1976, Leader 1978, Mack 1985). However, McSwiggan (1991) feels that this drug is unlikely to be specific as it is just another antipsychotic.

In the dental literature, Mack (1985) reported on a patient who held the delusion that there was an unceasing growth of hair on his dentures. This is possibly the first reported case where the delusion is fixed upon a prosthesis rather than to a natural part

of the body. This patient was diagnosed to have MHP and was successfully treated with pimozide after 8 weeks of the drug therapy. The other interesting fact in this report is that the wife of the patient was in complete agreement with the husband's history. Such corroboration is termed '**folie a' deux**', also known as '**induced psychosis**', a delusion which develops in a person as a result of a close relationship with another person who already has an established delusion (Scott and Humphreys 1987, Gelder et al 1989i). In such instances, both individuals need to consult a psychiatrist.

7.3.3 The '**Phantom Bite**' Syndrome

'**Phantom bite**' (PB) is a term used to describe a single hypochondriacal false belief that an individual has regarding his abnormal dental occlusion (Marbach 1978b). This delusion is chronic and patients with PB have a superficial knowledge about dental treatment (including anatomy and physiology). Marbach (1978b) states that this quest for the perfect bite results in repeated treatment failures which the patient seems to be unable to understand or learn from and he will go on year after year from one dentist to another. Marbach and his co-workers (1983) state that PB can be classified under either MHP or dysmorphobia.

7.4 Child abuse

Child abuse refers to the non-accidental injury of children (Humphreys and Scott 1987). The writer has included this topic under this section on '**Psychiatric disorders with special relevance to dentistry**' because dentists may play an important role (and may be in a special position, as will be shown later) in identifying the psychiatric disorder.

Non-accidental injury (NAI) of children, child abuse and the '**battered child syndrome**' are used synonymously to describe children with numerous unexplained bruises, fractures, and head injuries (Kempe et al 1962). NAI is inflicted by the caretaker of the children and are usually the parents. In the United Kingdom, it was established that at least 0.5% of children under three years of age will suffer serious injury as a result of NAI (Scott 1977). Approximately 1% of children are abused or neglected each year in the United States of America (Schmitt 1986a).

NAI in children may vary from minimal bruising to severe brain injury or death (Tate 1972, Robertson 1982, Sims 1985). Schmitt (1986a) has identified 10 types of child abuse from a report on the National Study (in the United States of America) of the incidence and severity of child abuse and neglect between 1 May, 1979 and 30 April, 1980. They are:

1. physical abuse,
2. sexual abuse,
3. failure to thrive due to nutritional neglect,
4. intentional drugging or poisoning,
5. Munchausen syndrome by proxy,
6. health (medical) care neglect,
7. dental neglect,
8. safety neglect,
9. emotional abuse and neglect, and
10. physical neglect.

Physical abuse is the most prevalent and is responsible for more cases of NAI in children than other types of abuse or neglect.

Robertson and his colleagues (1982) carried out a comparison study, on the prevalence and site of recent injury of any type, between 400 normal children and 84 children of similar age where NAI was proven or suspected. They found that 60% of the proven or suspected NAIs involved the head and the face of children of all ages. In an earlier study where medical records of 260 cases of child abuse admitted to The Children's Hospital in Boston between 1970 and 1975 were reviewed, it was found that 65% of the abused children received injuries to the head/face and intra-oral region (Becker et al 1978). Of the 386 injuries sustained by these 260 children, 33% were to the head, 61% the face (contusions, ecchymoses, abrasions, lacerations, fractures, burns and bites), and 6% the intra-oral structures.

Needleman reviewed numerous reports on the types of injuries sustained by the physically abused child and concluded that trauma to the head and associated areas occur in approximately 50% of the cases (Needleman 1986). He also stated that soft tissue injury (most frequently bruises) is the most common injury inflicted to the head and face and is the single most frequent injury sustained in child abuse. Injuries to the upper lip and maxillary labial frenum is a common oral injury which may be an identifying characteristic in NAI children (Tate 1972, Needleman 1986). This lesion may present where either a spoon or a bottle has been forced into the child's mouth or as the result of a blow to the mouth in an effort to silence a crying child. Bite wounds have also been identified in cases of NAI to children (Wagner 1986).

The head and/or facial areas are exposed and accessible; as the head is often considered to represent the whole being or 'self', it is not surprising that NAI in children often involve these areas (Sopher 1977, Needleman 1986). Bruises and injuries of varying age on other parts of the body are usually another common finding associated with orofacial trauma due to NAI (Tate 1972, Sims 1985, Kittle et al 1986). Brain damage

due to assaults by their parents has been diagnosed among many (between 2 and 4 percent) institutionalised children (Scott 1977).

The common occurrence of child abuse injuries to the head and oral cavity indicates that the dentist has a unique opportunity to identify and report suspicious cases in order to lessen the high mortality rate (Tate 1972, Sopher 1977, Becker et al 1978, Kittle et al 1986). The writer supports this statement wholeheartedly but like all other professionals is worried about implicating or suspecting innocent parents or caretakers thereby causing undue distress.

Researchers in the field of child abuse have presented many guidelines to help the inexperienced professional to recognise NAI in children. The common characteristics in the findings of many studies and investigations on child abuse have been identified as:

1. delayed reporting/presentation of injuries for medical help,
2. vague, inconsistent or unlikely explanations given for the cause of injury,
3. presence of multiple injuries in varying stages of healing,
4. caretakers or parents who are irritable and uncooperative to appropriate questioning

(Tate 1972, Scott 1977, Kittle et al 1986, Needleman 1986, Schmitt 1986b).

The family social history is often significant. In a controlled study of 214 parents of battered babies, it was shown that they were young and predominantly of lower social class (Smith et al 1973). Among the mothers, 76% had an abnormal personality and 48% were neurotic. Among the fathers, 64% had an abnormal personality, more than

half being psychopaths. 11% of the mothers and 29% of the fathers had criminal records. It was also found that siblings of these abused children are also likely to be assaulted. Premature parenthood seems to be a significant finding in cases of child abuse (Sims 1985, Sobel 1986). The abused child often shows signs of psychological disturbances. Abused children may appear overly vigilant or display a '**frozen watchfulness**', staring constantly and scanning the environment for danger while their faces are immobile at the same time (Scott 1977, Kittle et al 1986). These children seldom smile and do not usually make eye contact. Other indications of child abuse may also include the general lack of cleanliness of the child, small stature and size with respect to age, and evidence of malnutrition, and physical signs such as the child's gait or the presence of a limp when moving.

Robertson and his colleagues (1982) indicated that the prevalence and site of injuries may vary with age and provide useful guidelines as to what types of injuries are compatible with normal activity and a normal level of care in young children. For example, whilst minor bruising of the head and face is not uncommon in young children, such injuries are rare in children of less than 9 months (as they cannot crawl or walk yet) or more than 3 years (the children would have developed better coordination and protective skills). Robertson's statement is also supported by Schmitt (1986b). A point of interest to dentists is that a study on abused and neglected children by Badger (1986) displayed no significant differences in def/DMF rates from the national averages in the United States. There does not seem to be any increase in caries incidence among these children.

7.5 Overview

Dental phobia can result in a considerable degree of dental disease for the sufferers as they will avoid dental treatment until they have no choice (i.e. severe pain or dentition

infected). To treat these patients is stressful for the dentist and his staff. The dentist needs to manage this debilitating condition before he can carry out dental treatment effectively.

It is important for a dentist to be aware that psychiatric disorders such as hypochondriasis, dysmorphobia and monosymptomatic hypochondriacal psychosis (which causes the patient to have a distorted self body image), can result in the presentation of unusual and rather puzzling dental conditions. Appropriate mental health professionals will have to be approached to help manage this problem.

Involvement of the head and face in definite or suspected cases of non-accidental injury in children has been found to be high; the writer feels that the dentist and his staff are in a strategic position to identify child abuse. Where orofacial trauma occurs in child abuse, the dentist may be the first person approached by the caretakers or parents for help. In cases of child abuse, both the abused child and the parents or caretakers who are the culprits need urgent help. The dentist who recognises and identifies this has a responsibility to report his findings to the appropriate authorities.

8 Side Effects of Psychotropic Drugs with Dental Implications

Psychotropic or psychotherapeutic drugs are the medications used to treat the various mental illnesses. These drugs are not curative but are used to treat the symptoms of the mental illness and ameliorate its course (Hollister 1980). Psychotropic drugs are classified according to their pharmacological action, hence the main classes: antipsychotic drugs, antidepressant drugs, mood-stabilising drugs, and anti-anxiety and hypnotic compounds (Gelder et al 1989j, The Psychotropic Guidelines Sub-Committee Victorian Drug Usage Advisory Committee 1989a). Not all the drugs used in the treatment of mental illness fit perfectly into this classification as some of these drugs are used to treat more than one group of symptoms. A notable example is lithium carbonate (Gelder et al 1989j, The Psychotropic Guidelines Sub-Committee Victorian Drug Usage Advisory Committee 1989b). It has a moderate antidepressant effect and can control the symptoms of mania. However, it is used mainly to prevent recurrences of depressive and manic symptoms.

As with any medication, psychotropic drugs have side effects. Of these undesirable side effects, the ones with the most significance to dentists are tardive dyskinesia and xerostomia. These 2 occurrences will be discussed separately and in more detail in the next two chapters. Other adverse effects of psychotropic drugs of concern to the dentist will be grouped according to their action on specific organ systems.

8.1 Adverse effects on the cardiovascular system

The cardiovascular system is affected by the antipsychotics, antidepressants and lithium. Antipsychotic medications can cause tachycardia, fluctuations in blood

pressure, and orthostatic (postural) hypotension (Hollister 1980, Quock 1985, Friedlander and Brill 1986a, Harris 1988a). Cardiac effects such as palpitations, arrhythmia, tachycardia and orthostatic hypotension may also be caused by tricyclic antidepressants (Beck et al 1979, Hollister 1981, Quock 1985, Harris 1988b). Orthostatic hypotension is also a troublesome side effect of the monoamine oxidase inhibitor antidepressant. Reversible electrocardiogram changes in the T-wave can also occur in patients on lithium (Beck et al 1979, Gelder et al 1989j, Johnson 1989).

Of all the side effects on the cardiovascular system caused by the psychotropic drugs, orthostatic hypotension would seem to be the one that can affect the dentist most. Antipsychotics and tricyclic antidepressants block alpha-adrenergic receptors resulting in a sympatholytic effect in peripheral vasculature. The monoamine oxidase inhibitor antidepressants may mediate the orthostatic hypotension side effects centrally, involving reduction of the peripheral sympathetic outflow (Quock 1985). Dental procedures are usually delivered to patients who are in a reclining position. After dental treatment (especially a lengthy procedure), the impairment of sympathetically mediated vascular reflexes (caused by psychotropics) predisposes the patient to uncompensated, considerable venous pooling when getting up quickly to leave the dental chair. This sudden reduction in cerebral blood flow can result in dizziness and syncope. This unpleasant episode can be prevented if the dentist is aware of this adverse effect on patients who are taking this type of medication and therefore carries out certain precautions. The patient should be warned of this postural hypotension and possible fainting. The patient should be allowed to sit upright in the chair and rest for a few minutes before getting out of the chair. The patient should then be helped out of the chair slowly (Beck et al 1979, Quock 1985). The elderly are more vulnerable to fainting due to orthostatic hypotension (induced by psychotropics) because of potential compromise to a cerebral circulation which is already impaired due to age changes (Blackwell 1981a). A fainting episode due to orthostatic hypotension is unpleasant for

both the patient and dentist. This may cause undue alarm in the patient and reduces the confidence level of the patient in the clinician's ability.

8.2 Adverse effects on the central nervous system

Psychotropic medications can affect the central nervous system in a few ways. One of the most prominent adverse reactions to both antipsychotic drugs and tricyclic antidepressants is sedation (excessive drowsiness). The precise mechanism for this side effect is probably nonspecific (Quock 1985). Therapeutic dosages of these medications will produce varying degrees of drowsiness and sedative effects. The sedative effects tend to diminish with prolonged use as tolerance to this effect of these drugs may develop (Gelder et al 1989j, Johnson 1989). This sedative effect can interfere with a patient's routine and the writer feels that this will affect the patient's interest in oral health and ability to carry out the daily oral hygiene measures required.

Both antipsychotics and tricyclic antidepressants have been known to lower seizure threshold in patients with epilepsy (Dallos and Heatherfield 1969, Toone and Fenton 1977, Remick and Fine 1979, Blackwell 1981a, Quock 1985, Johnson 1989). In epileptic patients where convulsive threshold is already altered, psychotropics could cause an epileptic seizure. Amitriptyline (an antidepressant) and chlorpromazine (an antipsychotic) are the most commonly implicated psychotropic drugs to have this adverse effect (Remick and Fine 1979, Blackwell 1981a). However, this is a rare occurrence and these seizures occur only with large doses of psychotropic drugs, and in those patients who have a history of seizures or who are predisposed to have seizures (Toone and Fenton 1977, Edwards 1979, Blackwell 1981a). This predisposition may be due to some sort of brain injury or to age .

Seizures induced this way will prove to be another unnecessary hazard to both the dental surgeon and patient if it occurs in the dental surgery. The writer feels that although the dentist is not in the position to be able to control or prevent it, it is vital that the clinician is aware of the possibility of such an event and is prepared for it.

Tardive dyskinesia and other movement disorders associated with antipsychotics and certain antidepressants also have a bearing on dentistry. These movement disorders, tardive dyskinesia in particular, will be discussed in greater detail in a later chapter.

8.3 Adverse effect on the haematological system

Antipsychotics and tricyclic antidepressants (in particular mianserin) are also known to depress the haemopoietic system resulting in granulocytosis (a medical condition characterised by marked diminution in the number of polymorphonuclear leucocytes) (Friedlander and Brill 1986a, Friedlander and Brill 1986b, Gelder et al 1989j). The writer feels that this occurrence may interfere with healing in the event of surgical dental procedures.

Lithium, on the other hand, has been sometimes found to cause benign, reversible neutrophil leucocytosis with an absolute increase in neutrophil mass and production (Johnson 1989, Gelder et al 1989j).

8.4 Other unwanted effects of psychotropic drugs

Xerostomia is another adverse effect of psychotropic drugs which will be discussed in the next chapter. Other anticholinergic side effects of psychotropic drugs such as urinary retention, constipation, and blurred vision, may cause the patient considerable general discomfort (Johnson 1989). The writer feels that, indirectly, the dentist may be

affected because this patient may prove to be difficult to treat as a result of the general discomfort.

Certain medications in the different classes of psychotropic drugs have specific, unique side effects which may be of significance to the dentist. Amitriptyline, a commonly prescribed tricyclic antidepressant, has been reported to cause a craving for carbohydrates with an associated weight gain (Arenillas 1964, Paykel et al 1973, Smith et al 1986). The writer feels that this craving for carbohydrates may result in an increase of dental caries if xerostomia is also present and preventive measures such as daily oral hygiene procedures and the use of fluoride, are not carried out. Others have reported an increase in appetite rather than just a craving for carbohydrates (McSwiggan 1991).

Patients on lithium have also complained of a metallic and unpleasant taste in their mouth (Scully and Cawson 1987, Gelder et al 1989j). Non specific stomatitis (in the oral mucosā) due to lithium therapy has also been reported (Muniz and Berghman 1978, Bar Nathan et al 1985).

Other adverse effects of long term lithium therapy may include impaired renal function (due to kidney damage) and development of hypothyroidism (due to interference with thyroid production) (Gelder et al 1989j, Johnson 1989). The writer feels that these medical conditions may again affect the delivery of dental treatment indirectly. In hypothyroidism, surgical procedures or infection could bring on a hypothyroid coma while impaired renal function is associated with bleeding problems and increased susceptibility to infection (Little and Falace 1988a).

8.5 Psychotropic drug interactions

In clinical practice, drug interactions are well recognised. Psychotropic drugs are susceptible to a wide range of interactions with other drugs (Hollister 1980, Gelder et al 1989j, Johnson 1989). The potential drug interactions of concern to the dentist will be discussed in this section.

Central nervous system depressants such as barbiturates, benzodiazepines, and narcotic analgesics, can have the depressant (synergistic) effect enhanced by antipsychotics and tricyclic antidepressants (Hollister 1980, Friedlander and Brill 1986a, Little and Falace 1988b, Gelder et al 1989j, Johnson 1989). Severe respiratory depression may be precipitated and at the least, extreme drowsiness or sleepiness may occur. The dentist will have to be aware of this drug interaction when prescribing barbiturates, benzodiazepines or other sedatives to alleviate anxiety in these patients (already on psychotropic drugs) before any dental procedures. When prescribing narcotic analgesics for pain, this possible drug interaction with psychotropic drugs will have to be kept in mind. Another dangerous drug interaction that can occur when a patient on long term monoamine oxidase inhibitor antidepressant therapy who is prescribed meperidine (pethidine) for pain relief is that of hyperpyrexia (Shee 1960, Hollister 1980). In these patients, hyperpyrexia can produce delirious behaviour and convulsions which may be fatal.

The use of a local anaesthetic containing a sympathomimetic amine vasoconstrictor in patients on psychotropic drugs has been discussed extensively (Bookes et al 1972, Jastak and Yagiela 1983, Quock 1985, Humphreys and Scott 1987, Little and Falace 1988b, Cawson and Spector 1989, Gelder et al 1989j). From these various discussions, two distinct thoughts have evolved. Some believe that the tricyclic antidepressants, monoamine oxidase inhibitors, and certain antipsychotics, will

increase the pressor effects of the sympathomimetic amines (noradrenaline, adrenaline and phenylephrine) in local anaesthetics resulting in a severe hypertensive crisis and subsequent cerebrovascular accident. The other group believe that this is clinically irrelevant if the dosage is limited (as in the amounts of local anaesthetic normally used) and an intravascular injection has been avoided. Many dentist err on the side of caution in their clinical procedures and will use a local anaesthetic without a sympathomimetic amine as a vasoconstrictor when treating a patient on psychotropic drugs (Humphreys and Scott 1987). The writer feels that the choice of use of local anaesthetic with any particular vasoconstrictor is entirely that of the dentist's; she regularly uses a local anaesthetic without a sympathomimetic amine as a vasoconstrictor when treating these patients.

Psychotropic drug interactions with many other drugs are well documented and clinically important. However, not all of these interactions will be encountered by the dentist or are of significance to the delivery of dental treatment. The dentist is not responsible for prescribing those drugs in Australia and so will not be discussed here.

8.6 Overview

In psychiatry, the treatment of mental illness involves not only pharmacotherapy but may also require psychoanalysis, psychotherapy, and behaviour cognitive therapy. A small but select few do benefit from electroconvulsive therapy or psychosurgery. The writer feels that the treatment of mental illness is not within the parameters of this treatise but she understands that pharmacotherapy, alone, might not be sufficient. Psychotropic medications do not cure mental illness but are used to influence psychological and mental processes, and to control certain behaviours present in mental illnesses. It has been proposed that the integration of pharmacotherapy and

psychotherapy will aid patients to achieve the dual goals of symptom alleviation and enrichment of interpersonal experience (Goldhamer 1983).

Pharmacotherapy not only produces desired effects, but also has many adverse actions. It is very important that the dentist is aware of the unwanted side effects of psychotropic drugs on the various body organ systems in order to prevent a crisis in the dental surgery. The patient on psychotropic medication will not be unduly placed in any potentially dangerous situation if the dentist is knowledgeable regarding the interaction of psychotropic drugs with other drugs that may be used in dentistry.

9 Tardive Dyskinesia

Tardive dyskinesia (TD) was first briefly reported by Shonecker in 1957 and later described in detail and with more accuracy – this is the modern description, by Sigwald in 1959 (cited by Fahn 1984). TD is now recognised as a major and significant iatrogenic disorder (Paulson 1975, Jeste and Wyatt 1981). This distinct clinical entity refers to that of a persistent movement disorder which is an undesirable side effect of prolonged use of drugs that block dopamine receptors (Crane 1973, Paulson 1975, Fahn 1984, Harris 1988a, Jones 1989, The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c).

9.1 What is tardive dyskinesia

Tardive dyskinesia (TD) a movement disorder (The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c), is a clinical representation of pharmacological interference with the extrapyramidal system (Carruthers 1971, Turek et al 1972, Kane and Smith 1982, Harris 1988b). The abnormal movements are involuntary or semi-voluntary (Paulson 1975, Fahn 1984, Bassett et al 1986), and can affect the limbs, trunk, neck and most significant of all (to dentists) the mouth. These movements can be controlled briefly by the patient when he is made consciously aware of them. However, anxiety increases the abnormal movements (The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c).

9.2 Drugs which cause tardive dyskinesia

The most common type of drug which blocks the uptake of dopamine at dopamine receptors are antipsychotics. In the literature, antipsychotics and neuroleptics are used synonymously although this is not entirely accurate. The term neuroleptic indicates any drug that can induce parkinsonism as a toxic adverse effect (Fahn 1984). The first neuroleptic discovered was reserpine which depletes dopamine stores in tissues. Antipsychotic drugs are also neuroleptics but the mechanism of action is that of blocking dopamine receptors rather than depleting the dopamine (Carlsson 1978). Although reserpine and antipsychotics are both neuroleptics, reserpine does not cause TD (Fahn 1984). This writer feels that it is more appropriate to adopt the phrase **'certain neuroleptics induce TD'** rather than **'neuroleptics induce TD'**. The antipsychotic medication associated with TD are the phenothiazines (e.g. fluphenazine and trifluoperazine), the butyrophenones (e.g. haloperidol and doperidol), the thiozanthenes and the diphenylbutylpiperidines (e.g. pimozide) (Jones 1989, The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c).

Other drugs shown to induce TD include certain tricyclic antidepressants (Fann, et al 1976, Jeste et al 1979, The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c). Certain antiemetics such as prochlorperazine (Stemetil) or metaclopramide (Maxolon) have also been implicated in the development of TD (Bassett et al 1986). Large doses of lithium, anticholinergics and antihistamines can cause TD (The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c). Cases of severe TD have been reported in patients when a phenothiazine or butyrophenone is used in conjunction with lithium (McSwiggan 1991).

9.3 Clinical features of tardive dyskinesia affecting the orofacial region

In the orofacial region, TD manifests as a repetitive pattern of chewing movements of the facial and masticatory muscles, occasionally with smacking open of the mouth, protrusion of the tongue (known as fly-catcher tongue), lip pursing, sucking movements and puffing of the cheeks (Crane 1973, Paulson 1975, Brightman 1983, Fahn 1984, Bassett et al 1986, The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c). These rhythmical movements of the lips, side-to-side shifts of the chin and flicking of the tongue present the classical '**bucco-lingual-facial**' dyskinetic movements of TD. In TD the orofacial region is usually the first site of presentation and can look like a form of mannerism (McSwiggan 1991). TD does not necessarily involve the limbs, trunk, neck and orofacial region all at the same time. Orofacial movements can exist independently of the choreoathetoid movements which involve other anatomical regions (Paulson 1975, Brightman 1983, Fahn 1984, Bassett et al 1986, Glazer et al 1988). Of the different anatomical areas involved in TD, the mouth is the most commonly affected region (Fahn 1984, Scott and Humphreys 1987). Gentle side-to-side chin movements, intermittent shrugging of the neck and face, and grimacing movements of the lower part of the face, can also present together with the dyskinetic movements of the mouth (including the tongue) (Paulson 1975). In the early stages of TD, the movements are often not observed except surreptitiously (Felder et al 1988).

9.4 Prevalence of tardive dyskinesia

TD has been referred to as a late-appearing movement disorder that occurs in patients who have taken certain neuroleptics (McLean and Casey 1978, Bassett et al 1986, Glazer et al 1988). However, many other studies have produced slightly different

results. In some patients TD occurs after much shorter treatment. Ayd noted that the earliest onset of TD was between 3 and 6 months after the beginning of therapy whilst Degkwitz reported that 9 out of 247 dyskinetic patients developed symptoms in less than 6 months of treatment (cited by Crane 1973). Paulson (1975) states that most severe cases of TD have received antipsychotics (phenothiazines) for longer than 6 months and this period could be up to 3 to 4 years. There have been a few exceptions where patients who develop TD have been exposed to these agents for only a few weeks (Fahn 1984). This writer feels that it is appropriate to say that patients who are medicated with certain neuroleptics will be at risk to develop TD.

In 1973, Crane published a review on the number of patients with TD reported in the literature between 1967 and 1971. He found that there were approximately 1,200 cases of TD. He also analysed surveys by 17 investigators and found that the incidence of TD varies from 0.5 % to 40 % of those patients on neuroleptics. Crane (1973) felt that this discrepancy can be attributed to a number of factors, some of which include the definition of the syndrome, the methods used in collecting information, the type of patient population and the clinical assessment and evaluation of symptoms.

Jeste and Wyatt (1981) combined data from the 36 studies published on the prevalence of TD among chronically ill neuroleptic treated psychotic patients from 1960 through 1980. They found that:

1. the overall mean prevalence of TD among these patients is 17.5 %.
2. the overall mean prevalence TD based on 19 studies from 1960 to 1970 was 13.6 %.
3. the above figure has increased to 23.3 % since 1971 (based on 17 studies).

4. 13 of the last 17 studies individually indicated a prevalence exceeding 22 %.
5. the mean prevalence in the 11 studies published from 1976 through 1980 is 25.7 %.

Kane and Smith (1982) reviewed 56 prevalence surveys of TD in neuroleptic treated patients between 1959 and 1979. They found the prevalence to be an average of 20 %. They reported an increase in the prevalence of TD during the past two decades of their study. From these surveys, the prevalence of TD can be said to be significant among people who are on prolonged use of certain neuroleptics.

9.5 Risk factors associated with tardive dyskinesia

Among neuroleptic treated patients it has been indicated that there is a significant risk of disturbance of the extrapyramidal system. As indicated by the numerous studies on TD there are many other risk factors involved. Perhaps one of the most intriguing risk factors would be the amount of neuroleptic medication that is required to trigger off this movement disorder.

In his review of literature on TD from 1967 to 1971, Crane (1973) stated that the task of determining the amount of drug intake necessary to produce the disorder presents unsurmountable difficulties. Some reasons for these difficulties include finding out about the trend of drug administration during any particular period and the various types of neuroleptics prescribed. He reviewed 8 studies and felt that it was understandable that the results were inconclusive. He reported that 5% to approximately 40% of asymptomatic patients develop dyskinesia only after discontinuation of treatment.

Turek and his colleagues (1972) picked 56 patients from an original sample of chronic hospital in-patients who exhibited symptoms of TD, for observation over a period of approximately one year. They aimed to clarify the relationship between TD and drug dosages (cessation as well as increases in neuroleptics dosages and possible control of this movement disorder with antiparkinson agents). They could not determine any relationship between severity of the symptoms and the dosage. However, they observed that the dyskinetic symptoms were lower during the neuroleptic drug treatment phases and increased during subsequent neuroleptic drug free periods.

Kane and Smith (1982) reviewed 18 studies which explored the relationship between cumulative drug dose and the prevalence of TD. Four of these studies reported a significant positive relationship (i.e. the risk of TD development increases with increasing drug exposure), whilst fourteen did not. Kane and Smith suggested that the reason for this conflicting result is that certain patients may be more vulnerable to the development of TD.

In their comparisons on studies of TD and Levodopa induced dyskinesia, Karson and his colleagues (1983) found that the severity of TD was not related to duration of neuroleptic treatment.

It seems that the dosage and length of neuroleptic treatment needed to produce TD depends on the individual's vulnerability. It has been found that TD is more common among the elderly (Crane 1973, Jeste and Wyatt 1981, Jeste and Wyatt 1982, Kane and Smith 1982, Brightman 1983). However, the presence of TD in adolescent patients treated with neuroleptic drugs has also been reported (McLean and Casey 1978, Pickar 1978, Jeste and Wyatt 1981).

In children, TD seldom involves the orofacial region but presents as dyskinetic movement of the limbs and trunk (Glazer 1988). Similarly, there are more females than males who are affected with TD (Crane 1973, Jeste and Wyatt 1981, Jeste and Wyatt 1982, Kane and Smith 1982, Brightman 1983). Many reasons have been proposed. Jeste and Wyatt (1981) felt that the higher prevalence of TD in females may be due either to differences in certain biological characteristics (e.g. brain neurotransmitter concentrations, role of different hormones), or may just be a reflection of differences in treatment. Kane and Smith (1982) voiced similar views and also stated that since the highest rates of TD are reported in post-menopausal women, the loss of ovarian function may perhaps play a part.

Another risk factor may be the treatment practices carried out. Intermittent drug treatment (i.e. periods where neuroleptics are not taken) do not seem to prevent TD (Jeste and Wyatt 1982, Kane and Smith 1982). Jeste and his colleagues (1979) felt that the repeated 'on-off' manipulation of the neurochemical system by stopping the neuroleptic treatment and then starting the medication again may even increase the persistence of TD in susceptible persons through a possible 'kindling' effect.

9.6 How persistent is tardive dyskinesia?

TD may develop while the patient is on neuroleptic treatment but paradoxically, in some patients, TD develops after neuroleptic treatment has been reduced or stopped (Carruthers 1971, Fahn 1984, Basset et al 1986). Fahn (1984) and Jeste and Wyatt (1982) felt that a period of up to 3 months after the discontinuation of neuroleptic treatment should be allowed and if dyskinesia appears after that, another aetiology may be suspected.

Withdrawal of neuroleptic treatment during the early stage of development of TD may stop the dyskinetic movements (Quitkin et al 1977) but in many patients, this does not happen. In fact, the dyskinetic movements may worsen (The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c).

It has been found that the use of neuroleptics reduces or suppresses the dyskinetic movements that may develop after neuroleptic treatment has been stopped (Kennedy 1969, Roxburgh 1970) and the dose required is lower than those commonly employed in the treatment of schizophrenia (Jeste and Wyatt 1982). Kennedy (1969) and Roxburgh (1970) found that an increase of dosage of neuroleptic can lessen or suppress the dyskinetic movements in patients who continue to receive neuroleptics.

In some patients, TD will gradually fade and disappear over several months, but in others, it may take longer and worst of all, in some, it may be permanent (The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c). Fahn (1984) suggested that TD can be classified as permanent if it lasts for at least five years without remission. Smith and Baldessarini (1980) found that full or partial recovery occurred in 64 % of patients but this improvement was seen mostly in younger patients. Jeste and Wyatt (1982) reviewed 285 treatment studies involving more than 3000 patients with neuroleptic-induced TD. They found that this movement disorder is reversible for more than one third (37 %) of all patients who discontinued the drug treatment. It was also found that the likelihood of recovering from TD is greater in younger patients and increases with the length of time the patient has been off the drug treatment (Jeste and Wyatt 1985). Unfortunately for 30 % of cases TD is irreversible even when the medication is stopped (Marsden et al 1975 cited by Scott and Humphreys 1987).

9.7 Other movement disorders induced by the D2 receptor blockers

Besides TD, neuroleptics (the D2 receptor blockers) can also induce other movement disorders. This variety of movement disorders include (Paulson 1975, Fahn 1984, Bassett et al 1986, Harris 1988a, The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c):

1. Pseudoparkinsonism, where symptoms resembling that of Parkinson's disease such as immobile facial expression, 'pill-rolling' tremors of the fingers at rest etc, are present.
2. Dystonia, where muscle spasms can produce bizarre postures of face, jaw, neck, and trunk, similar to TD. (Dystonic reactions disappear hours after stopping the offending drug).
3. Akathisia, where an inner or motor restlessness is evident, showing complex motor signs such as pacing up and down the hall, getting up from the chair intermittently and walking about etc.

The use of antiparkinsonian drugs such as Cogentin and Artane can often prevent these movement disorders (McSwiggan 1991).

9.8 Dental implications of tardive dyskinesia

Pain is not usually a presenting complaint or symptom of TD and in many mild cases of TD, the patients are not even aware of it until alerted by observers (Paulson 1975, Bassett et al 1986). However, orofacial pain can result from chronic trauma between denture bearing mucosa and dentures which occurs during the repetitive abnormal movements of the mouth and tongue when TD involves the orofacial region. Bassett

and his co-workers (1986) reported two such cases. In some cases of TD, difficulty in wearing dentures can result from the persistent chewing movements of the mouth and jaw, and the repetitive flicking actions of the tongue (Evans 1965, Brightman 1983, Langer 1984, Felder et al 1988). The writer has found it difficult to get a good impression of the denture bearing tissues in patients with TD (when the orofacial region is involved) on the first attempt.

Complaints of pain involving the tongue (due to lingual dyskinesia) rubbing against teeth and palate have also been reported (Evans 1965, Hierholzer 1989). The writer feels that this pain is probably from trauma imposed on the tongue during the repetitive flicking movements of the tongue over other surfaces which can strip off the top most layer of oral epithelium on the tongue. The movements can interfere with speech and eating (The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c). A recent case of severe degenerative changes of the temporomandibular joint secondary to the effects of TD has been reported (Osborne et al 1989).

From the various reports above, this writer recognises that pain may be a presenting symptom of TD as a result of chronic trauma between the tongue and other oral structures and/or dentures, and the denture bearing tissues during the orofacial dyskinetic movements of TD. The writer also feels that routine procedures involved in restorative dentistry may prove to be a challenge to the clinician when orofacial TD is present. Extractions and surgical removal of teeth when indicated may even have to be carried out under general anaesthesia.

9.9 Overview

Antipsychotics such as fluphenazine, perphenazine haloperidol etc., have been recognised to have the ability to ameliorate the course of schizophrenia (Hollister 1980, Harris 1988a). Tardive dyskinesia (TD) involving the orofacial region has been reported to be a better known extrapyramidal syndrome resulting from treatment with antipsychotics (neuroleptics).

The diagnosis of TD cannot be confirmed by any specific laboratory tests or neurological examination. TD can only be identified by sight (Paulson 1975). Two other factors distinguish TD from other similar dyskinetic movements. A history of present or past (a period of up to three months absence of neuroleptics has been suggested by Fahn (1984) and Jeste and Wyatt (1982)) neuroleptic medication is one of the two factors essential to making the diagnosis of TD. Secondly, the movements involved in TD should be rapid, brief, repetitive, and complex (Fahn 1984).

Lingual-facial-buccal dyskinesias which sometimes arises spontaneously in the elderly (Kane and Smith 1982, D'Allessandro et al 1986) and the chorea-like movements in Sydenham's and Huntington's chorea (Paulson 1975, Fahn 1984, Bassett et al 1986) may sometimes be mistaken for TD. The two factors mentioned in the earlier paragraph will help to distinguish TD from these similarly alike dyskinetic movements.

Risk factors which increases the vulnerability to developing TD are old age and the female gender. Patients with organic brain damage have also been found to be more predisposed to TD (Crane 1973, Kane and Smith 1982, Bassett et al 1986). Surprisingly, many workers in the field of TD have found the lack of an apparent cumulative dose effect in the development of this movement disorder.

From the literature reviewed, neuroleptic withdrawal during the early stages when TD is detected seems to be the logical first line of treatment of TD. However, the importance of management of psychotic symptoms may overshadow the presence of the movement disorder. The decision to stop antipsychotic treatment is a joint decision for the patient and clinician. To date there is no satisfactory treatment for TD (The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee 1989c).

The dental implications of TD have been recognised. Dentures traumatising denture bearing tissues and the tongue moving repetitively against other oral structures can cause orofacial pain in TD. TD not only makes the use of removable oral prosthesis difficult, it also makes communication and eating a chore. Simple dental procedures can prove to be difficult for the dental surgeon.

It is important that the dentist is aware of the relationship between neuroleptic treatment and the presence of TD as he may be the first clinician to come across this movement disorder. Referral to the appropriate clinician for assessment and possibly control of this movement disorder is indicated.

10 Xerostomia and the Dental Sequela

Xerostomia refers to the subjective feeling of oral dryness (Glass et al 1984, Fox et al 1985, Sreebny and Valdin 1988). This symptom is fundamentally due to a marked decrease in the flow of saliva. The aetiology of xerostomia can be divided into 3 main categories – radiation induced, pharmacologically induced, and as a symptom with systemic conditions not related to radiation or drugs.

The hyposalivation in xerostomia has many implications to oral health as saliva plays an important role in the maintenance of oral homeostasis (Mandel 1989). Of relevance to this treatise is xerostomia brought about either by medication prescribed for mental illness or as a symptom related to an existing mental illness.

10.1 Xerostomia associated with mental illness

Xerostomia is a symptom of a number of diseases and conditions, of which diabetes mellitus, cystic fibrosis, and autoimmune diseases such as Sjogren's syndrome are commonly known (Sreebny 1989). For a long time, researchers have recognised that psychic stimuli can affect the flow of saliva (Bushfield et al 1961). Since the pioneering work of Winsor (in the early 1930s) and Strongin and Hinsie (in the later 1930s), many other studies have conclusively shown that disturbances of salivation occur in depressed patients (cited by Bushfield et al 1961).

In 1961, Bushfield and Wechsler carried out a comparison study of salivation rates in 45 depressed hospitalised patients, 42 non-depressed hospitalised patients and 50 normal controls (non-patients). It was found that the depressed patients salivate the least and that the normal controls salivate the most.

In the same year, Gottlieb and Paulson (1961) reported on a longitudinal study on the salivary output of 18 patients at the Dorothea Dix Hospital (a State mental hospital). They found that the depressed patients showed a uniformly lower rate of salivary excretion compared with the 8 normal control subjects.

Busfield and Wechsler also collaborated with Barnum to investigate if the salivary flow rate can demonstrate a psychological difference between reactive and endogenous depression (Busfield et al 1961). From this study on 109 severely depressed hospitalised patients, they found that patients with reactive depression had significantly higher rates of salivation as compared with patients with endogenous depression. It was also found that the salivation rate did not vary with severity of depression in any of the diagnostic groups and was lower than in normal controls.

Mathew, Weinman and Claghorn (researchers from the Texas Research Institute of Mental Science, Houston) studied salivation in 51 depressed patients and in a normal control group matched for age and sex (Mathew et al 1979). Interestingly, they found that both dry mouth and excessive salivation (less frequently) are seen in the depressed patients. These patients are not medicated.

In some of the later studies on salivation and depressive illness, many interesting ideas have evolved. One of these interesting theories is that proposed by Russ and Ackerman (1989). Appetitive factors such as the desire to eat and food intake are frequently disturbed in depressed patients. Since these factors and others such as caloric preloading and dietary history influence salivary secretion, Russ and Ackerman feel that diminished salivary flow in patients with depressive illness may be significantly accounted for by the appetitive disturbances in depression. The appetitive disturbances could be a result of an unpleasant taste in the mouth (Miller and Naylor 1989). Miller and Naylor considered this to be a neglected symptom in depression

when 19 of the 47 depressed patients in their study had this complaint. This symptom appeared unrelated to previous drug treatment. Nishino and his colleagues (1989) studied 58 patients (42 diagnosed as having major depressive disorder, 16 diagnosed as having minor depressive disorder) and 39 healthy control subjects. Their results showed that salivary prostaglandin concentration was highest in patients with major depressive disorder and showed a correlation with the severity of the depression. They suggested that salivary prostaglandin concentrations may be possible indicators for the state of major depression.

The various studies carried out on salivation and depressive illnesses have indicated that hyposalivation and xerostomia are associated with depressive illness. It has also been shown that the diurnal pattern of salivary flow in depressed patients is the reverse of that of normal controls (Palmai and Blackwell 1965). In controls, maximum salivary flow occurred in the morning and reached its lowest levels in the evening. In the depressed patients, decreased salivary flow in the morning increased till the evening when it reached the maximum level. During treatment, the salivary flow of the depressed patients increased progressively until it approached that of controls when they were close to being discharged from the hospital. As mentioned earlier, the study carried out by Mathew and his co-workers (1979) has shown that sialorrhoea (hypersalivation) is also sometimes present. Alias and his co-workers (1983) have published a case report of a 62 year old woman (with a 35 year history of bipolar affective disorder) who developed sialorrhoea during the manic phase of her illness. The finding is similar to that of Jenner and his co-workers (1967) who reported on a male patient showing hypersalivation during the manic phase of a 48 hour manic-depressive cycle. The researchers suggested that sialorrhoea could perhaps be considered a sign of mania in bipolar affective disorders (manic-depressive disorders).

10.2 Xerostomia induced pharmacologically by medication prescribed for mental illness

In psychiatry, medications prescribed for mental illness are referred to as psychotropic drugs. The four main classes of psychotropic drugs are antidepressants, the antipsychotic drugs, mood-stabilising drugs, and the anti-anxiety and hypnotic compounds (The Psychotropic Guidelines Sub-Committee Victorian Drug Usage Advisory Committee, 1989a). These psychotropic drugs all have xerostomia (of varying degrees) as a side effect. Dry mouth is usually worse in the first few weeks of medication (McSwiggan 1991).

10.2.1 Xerostomia Due to Antidepressants

As the name suggests, antidepressants are used primarily to treat depression and also prove to be effective for a wide variety of other psychiatric conditions such as panic attacks and bulimia (Hollister 1981, Harris 1988b). According to their different chemical structures, antidepressants can be categorised into the tricyclic antidepressants, the atypical antidepressants, and the monoamine oxidase inhibitors.

10.2.1.1 The tricyclic antidepressants

Tricyclic antidepressants boost the activity of norepinephrine and serotonin at the synaptic cleft by inhibiting their reuptake at the presynaptic junction (Mörnstad et al 1986, Harris 1988b). They are also anticholinergic and dry mouth is a consequence. Tricyclic antidepressants such as amitriptyline, imipramine, nortriptyline, and others, are well known xerogenic drugs (Blackwell 1981a, Mörnstad et al 1986, Harris 1988b). Tricyclic antidepressants are by far the most popular medication for depression.

10.2.1.2 The atypical antidepressants

The atypical antidepressants (the non-tricyclic antidepressants) are also referred to as the '**second generation antidepressants**' (Blackwell 1981b, Hollister 1981). This group of antidepressants offer no great therapeutic advantages over the older ones but may act more quickly and have a more acceptable profile of side effects (Blackwell 1981b, Hollister 1981). Drugs such as maprotiline, trazodone, and mianserin, are claimed to have fewer anticholinergic side effects than most tricyclics (Hollister 1981). Fluoxetine (an atypical antidepressant) may still cause dry mouth (Harris 1988b).

10.2.1.3 The monoamine oxidase inhibitors (MAOIs)

MAOIs inhibit the enzyme monoamine oxidase which breaks down norepinephrine and serotonin, thus promoting activity of these two amines (Blackwell 1981a, Harris 1988b). Increased amine activity at the synapse lifts the patients depression – this is facilitated both by antidepressants and MAOIs.

The MAOIs were very popular in the early 1960s but due to their drug interaction and stringent dietary restrictions, their use became less widespread (Blackwell 1981a). Today, the MAOIs are used if tricyclic antidepressants are unsuccessful (The Psychotropic Guidelines Sub-Committee Victorian Drug Usage Advisory Committee 1989d). The MAOI phenelzine has the noxious side effect of dry mouth (Harris 1988b).

10.2.2 Xerostomia Due to Antipsychotics

Schizophrenia and other psychotic symptoms are treated with antipsychotic drugs. Antipsychotics can be classified either according to their chemical structure or by their

potency (Harris 1988a). Antipsychotics such as chlorpromazine, thioridazine and trifluoperazine have a side effect of that of xerostomia (The Psychotropic Guidelines Sub-Committee Victorian Drug Usage Advisory Committee, 1989c).

10.2.3 Xerostomia Due to Mood-Stabilising Drugs (notably lithium)

Lithium and carbamazepine are the 2 mood-stabilising drugs used to prevent recurrences of depression and mania (The Psychotropic Guidelines Sub-Committee Victorian Drug Usage Advisory Committee, 1989b). Both these drugs have the adverse side effect of a dry mouth. In a study by Bone and co-workers (1980), it was found that the perception of the side effect of dry mouth was significantly increased when patients were treated with both lithium and other psychotropic medications (tricyclics, antipsychotics, and tranquilisers).

Polyurea and polydipsia, are side effects of lithium which can produce thirst. Patients often use soft drinks to quench their thirst (McSwiggan 1991). The xerogenic effect of lithium together with the excessive soft drink intake can lead to increased caries.

Markitziu and co-workers (1988) found in their study that two thirds of their patients on lithium suffered from hyposalivation. They suggested that since hypothyroidism develops in 20 to 30 % of patients treated with lithium carbonate there is the possibility that thyroid function level might be one of the many factors involved in xerostomia connected with lithium medication.

Salivary dysfunction associated with lithium therapy is generally accepted to be that of hyposalivation although patients have also been known to exhibit normal salivation. However, a case of sialorrhoea (excessive flow of saliva) as a side effect of lithium has been reported by Donaldson (1982). A 37 year old male patient (with a history of

mania and episodic alcohol abuse) developed sialorrhea secondary to salivary gland enlargement while being treated with lithium carbonate. The excessive salivary flow and associated salivary gland hypertrophy diminished when the lithium therapy was discontinued. However, the lithium therapy was restarted when the manic episodes recurred. Hypersalivation was reported after 1 week of therapy and salivary gland enlargement was present.

10.2.4 Xerostomia Due to Anti-Anxiety and Hypnotic Compounds

Most anti-anxiety (anxiolytic) and hypnotic drugs used today are benzodiazepines, of which diazepam is the most popular (The Psychotropic Guidelines Sub-Committee Victorian Drug Usage Advisory Committee, 1989e). Dry mouth is also a known side effect of benzodiazepines.

10.3 The dental sequela of xerostomia

Psychotropic drugs with xerostomia as a side effect can affect dental health in many ways. Of greatest concern is that which results in the increase of dental caries. Caries and other dental sequela of xerostomia will be elucidated in the following section of this chapter.

It is important to be aware of the multifunctions of saliva in order to fully appreciate the consequences to oral health when there is considerable decrease in salivary flow as in dry mouth or xerostomia.

10.3.1 Functions of Saliva

Saliva is a rich complex mixture of water, proteins and non protein constituents, such as phosphorus, potassium, uric acid etc. Saliva has many functions (Mandel 1987,1989, Fox 1989). It is an effective system for lubricating oral soft and hard tissues. Numerous antimicrobial systems in the saliva contribute towards the maintenance of a balanced oral ecological environment and also protects the oral tissues. Supersaturation of calcium and phosphate in saliva enhances the remineralisation of the tooth. The buffering capability of saliva is effective in maintaining pH in the oral cavity and this in turn helps to regulate plaque pH. The water content in saliva acts as a mechanical cleansing agent in the mouth. A range of digestive enzymes such as amylase, lipase, and nucleases, present in saliva aids the formation of the food bolus and prepares the food for digestion in the stomach. The rheological properties (such as low solubility, high viscosity, and elasticity) of saliva contributes to maintenance of mucous membrane integrity.

In brief, the main functions of saliva are that of lubrication, antimicrobial, remineralisation (of teeth), cleansing, buffering, digestive, and maintenance of mucosal integrity. The presence of a less than acceptable 'normal' amount of saliva (as in xerostomia) will interfere with many of the roles of saliva in maintaining oral homeostasis.

10.3.2 Oral Pathology Due to Xerostomia

The dental ramifications of xerostomia include dental caries, soft tissue damage and infection, poor tolerance of dental prostheses, disturbance of eating, digestion, and speech, and general oral discomfort (Bahn 1972, Glass et al 1984, Tetrault and

Weintraub 1984, Handelman et al 1986, Sreebny and Schwartz 1986, Sreebny and Valdini 1987, Mandel 1989).

10.3.2.1 Dental caries

The incidence of dental caries increases when the flow of saliva is markedly decreased as in xerostomia (Aldous 1964, Mandel 1989). The dental literature has numerous reports on drug related destruction of teeth secondary to xerostomia. Winer and Bahn (1967) were among the first to report of extensive dental caries which resulted in a loss of 10 teeth over a 12 month period in a 61 year old man. He had xerostomia caused by antidepressant (imipramine, trihexiphenidyl) therapy for depression.

Stevens and Wilkinson (1971) reported on a woman in her 30's who developed rampant caries after 8 months of psychotropic medication (thioridazine, amitriptyline, and benzotropine) therapy. She eventually had to have all her teeth extracted as the caries was extensive. The case report of a 43 year old woman who developed 28 carious lesions following the use of first, a combination drug (containing phenothiazine and a tricyclic antidepressant) and then a tricyclic antidepressant alone, was published by Bassuk and Schoonover (1978). These carious lesions developed over a period of 11 months. The similarity between the 3 individual case reports described is that the patients all complained of a dry mouth as a result of psychotropic medication.

Rundegren and others (1985) made a study of the salivary function and presence of oral pathology (caries and oral mucosa lesions) in 32 patients who had taken tricyclic antidepressant drugs for an average of 5.5 years. Four complained of dry mouth although 50% of the patients had a normal secretion rate for stimulated saliva. Out of the 32 patients, 12 were edentulous. Fifty five percent of the patients with teeth had

new carious lesions at the time of examination. Only two of the patients had not received dental care for more than a year before the examination. Caries risk evaluation was carried out and the researchers concluded that about 50% of the patients with teeth had a high caries risk. The researchers felt in these patients, the anticholinergic effect of the antidepressants is amplified by combination with other medication e.g. anti-hypertensive medication.

Von Knorring and Wahlin (1986) carried out a study on frequency of dental caries among children suffering from enuresis treated with antidepressants. Their findings indicated that the mean caries activity was higher in the children treated with antidepressants for one month or longer compared to both controls and the enuretic children not treated pharmacologically.

Increased caries susceptibility has also been linked with lithium carbonate therapy (Gillis 1978, Markitziu et al 1988). The positive correlation between xerostomia due to psychotropic medications (including other such anticholinergic medication) and increased caries susceptibility cannot be refuted. At present, two of the most recent studies on dental condition prevalent among people on long term psychotropic medication reinforces this statement (Friedlander and Birch 1990, Stiefel et al 1990). These two studies will be discussed in the next chapter.

10.3.2.2 Soft tissue oral pathology

Candidiasis appears to be a frequent complication of psychotropic drug treatment. Kane (1962, 1963) reported three cases of oral moniliasis (candidiasis) following the use of chlorpromazine therapy. Kane and Anderson (1963) then discovered a fourth case of oral candidiasis at the North Carolina Memorial Hospital during therapy with

chlorpromazine followed by thioridazine. Chlorpromazine and thioridazine (both phenothiazines) are antipsychotics.

It was not until the extensive study carried out by Pollack and co-workers (1964) that the frequent occurrence of oral candidiasis associated with psychopharmacological treatment is correlated to xerostomia. The subjects of the study were 3,000 psychiatric patients (hospitalised and out-patients) receiving psychotropic drug treatment (phenothiazine and antidepressants) and 723 control patients from a country hospital. This large scale study indicated that the presence of xerostomia (secondary to the drug therapy) was associated with diffuse redness of the oral mucosa, denture stomatitis, cracking of lips and corners of the mouth, candida infection of the tongue, and varying degrees of atrophy of the fungiform papillae of the tongue. Candidiasis was found to be more prevalent in patients with dentures. Loosened dentures was also found to be a common complaint among these patients.

Rundegren and co-workers (1985) reported that 72% (23 out of 32 patients) of their subjects who had been on antidepressant drug therapy for an average of 5.5 years showed pathological alteration of their oral mucosa, mostly affecting the tongue and lips.

Other oral pathology (besides candidiasis and dental caries) related to xerostomia include increase in susceptibility of the oral mucosa to infection and ulceration, acute parotitis and parotid gland enlargement (Glass et al 1984, Tetrault and Weintraub 1984, Handelsman et al 1986, Sreebny and Valdin 1987, Sreebny and Valdin 1988, Widdop 1991). This oral pathology will bring about many oral symptoms and dental problems. These include the following:

1. soreness and burning of the oral mucosa especially the tongue.

2. difficulty with mastication, swallowing, and speech.
3. impairment or loss in taste acuity.
4. difficulty with wearing dentures, especially full dentures.
5. dry, cracked lips and angular cheilitis.

10.4 Overview

Of relevance to this treatise is xerostomia as a result of psychogenic factors such as depression and the xerogenic properties of psychotropic drugs. The protective role that saliva plays in maintaining the health of soft and hard dental tissues can be demonstrated clearly by the oral pathology arising in xerostomia regardless of the aetiology. The numerous complaints that arise from the oral pathology is not only distressing but can also impair the patient's quality of life.

The writer feels it is important for the competent dentist when treating this group of patients to be aware of xerostomia which is related to both psychogenic factors and the result of psychotropic drugs.

11 Discussion

As mentioned in previous chapters of this treatise, certain types of dental disease are strongly linked with the presence of a particular psychiatric disorder in some individuals. For example, perimylolysis (severe destruction of tooth enamel) has been recognised to be common among anorexic and bulimic individuals who vomit or regurgitate food in an attempt to reduce and maintain low body weight.

In the Western world today, these eating disorders are on the increase (Wolf 1991). Some of the people suffering from them could become dental patients. It is important for the dentist to recognise the dental 'tell-tale' signs of these eating disorders. The writer feels that liaison and/or referral to a psychiatrist is imperative in these cases. Referral to a psychiatrist should be communicated through the general medical practitioner.

The various presentations of psychogenic facial pain if encountered by an unsuspecting dentist can be perplexing and time consuming for both clinician and patient. To do the right thing by the patient, the dentist needs to be aware of this phenomenon of psychogenic facial pain. This will prevent many unnecessary dental procedures for the patient. Time need not be wasted and cost will be saved for the patient.

Although self-induced or factitious lesions are seldom seen in the oral cavity (Shira et al 1959), oral self-mutilation has been reported to occur in people with mental illness. The writer feels that as in psychogenic facial pain, oral self mutilation could be seen as a silent cry for help. The dentist can be instrumental in helping these patients if he has a knowledge regarding these entities.

Many other orofacial manifestations of certain mental illness have been reported and recognised. Rampant caries as a result of xerostomia brought on by certain psychotropic medications has also been documented. Accounts of types of dental disease and the psychiatric disorders have mostly been case reports or a study to look at just a particular dental disease among individuals with a certain psychiatric disorder. To date, the writer has found 3 recent studies which look into dental disease in general (not any particular dental problem) with the presence of mental illness.

Study 1 (This has been previously discussed briefly in Chapter 10)

Rundegren and co-workers (1985) studied the oral condition of 32 patients who had received long-term treatment (median of 5.5. years) with tricyclic antidepressant medication. Twenty of these patients had teeth while 12 were edentulous. Only 4 complained of a dry mouth even though a normal secretion rate for stimulated saliva was reported in only about 50% of the patients. Rundegren and co-workers felt that some of these patients who used fruit syrup, juice, or frequently ate to relieve their dryness, were providing a more powerful stimulant to overcome the anticholinergic inhibition of the tricyclic antidepressants. This has been supported by an earlier study of Von Knorring and Mörnstad (1981) who found that the anticholinergic inhibition of antidepressant medication could be decreased by using a more powerful stimulant consisting of increasing concentration of citric acid. Most of these patients had low values of salivary buffering capacity and high numbers of streptococci mutans and lactobacilli in the saliva. Oral hygiene among these patients was poor and even though most (except 2) of them had received dental treatment a year or less before the time of the examination, 55% of the patients with teeth had new carious lesions. Caries risk tests were carried out on the patients with teeth and it was evaluated that half of them had a high caries risk. Seventy-two percent of all the patients showed some form of

mucosal lesions mostly affecting the tongue and lips. Five of the twelve patients with full dentures had denture stomatitis.

Study 2

Stiefel and co-workers (1990) compared the oral status of noninstitutionalised adults with chronic mental illness with a similar group (without a history of mental illness) and evaluated relative risk factors such as xerostomia, diet, hygiene and poverty. The sample studied consisted of 37 subjects with chronic mental illness and 29 control subjects without mental illness. Dental, medical and social history were assessed and it was found that the 2 groups were similar in socioeconomic level, education, dental history and home care. All patients with chronic mental illness had received psychotropic medications (average of 3.8 drugs for 10.3 years). These patients reported a higher incidence of dry mouth, consumed more carbonated beverages and had more lesions on the mucosa, lip, and tongue. Although oral hygiene was poor for both groups, the patients with chronic mental illness showed a significantly higher caries prevalence (a mean of 6.4 carious surfaces compared to 2.1 carious surfaces for the group without mental illness). Presence of calculus, gingivitis, and loss of periodontal attachment, were also higher in the group with chronic mental illness. Among this group of subjects, those who were treated with lithium were more prone to plaque accumulation and showed significantly greater severity of gingivitis than those who were on other psychotropic medications.

Study 3

Friedlander and Birch (1990) examined the dental conditions in patients with bipolar disorder (manic depressive disease) who are on long term lithium therapy. The sample population consisted of 38 males and 2 females who were admitted to a

neuropsychiatric institution in West Los Angeles with a diagnosis of bipolar disorder. Seventy-three percent of these patients claimed to have been treated with lithium for 12 or more years. Eight of these patients had also been treated with a short course (less than 6 months) of tricyclic antidepressant medication. Oral hygiene was poor and accumulations of supragingival and subgingival calculus were high amongst these patients. Extensive caries and a large number of teeth missing were other findings in these patients. Friedlander and co-workers felt that this high level of dental disease was due to the nature of the mental illness which influences both salivary flow and compliance with oral care. The deleterious effects of xerostomia (due to depression and lithium) on the dentition is magnified in those patients who responded to their dry mouth by an increased intake of cariogenic food stuffs. All of the 29 patients who complained of xerostomia responded to this symptom by increased use of candy and chewing gum to promote salivation. They also admitted to an increased consumption of cariogenic fluids to satisfy their thirst.

From these 3 studies, the level of dental disease (caries and soft tissue pathology) is found to be high among patients with chronic mental illness. These findings emphasize the importance of dental care and dental health education among these individuals. The writer feels that supportive family members, friends, and care-givers of individuals with mental illness should be informed of some of these dental implications and given the appropriate dental health education in order to help look after these people. It is also important that the dentist who treats patients with chronic mental illness (and others who may be on psychotropic medications for minor or acute psychiatric disorders) is aware of the high level of dental disease among this population.

The level of dental disease among patients with chronic mental illness is reported to be high. These patients have a unique set of factors that can lead to the development of

advanced dental disease. For example, during periods of depression there is significant disregard of personal hygiene resulting in poor oral hygiene. There may also be a craving for sweets and cariogenic food stuff (including drinks) due to impaired taste perception or to relieve dry mouths. During periods of acute mania these patients may become overzealous with their tooth brushing or other dental health regimes (flossing, use of interdents etc.) which may cause considerable abrasions to the teeth and soft tissues. Patients with chronic schizophrenia have little perception of the need for dental health. In general they neglect their health and are difficult patients (McSwiggan 1991). Patients with chronic mental illness usually belong to a lower socioeconomic group and may have difficulty gaining access to dental treatment (McSwiggan 1991).

The nature of mental illness and xerogenic side effects of psychotropic medications have been recognised to play a large part in this high dental disease level. Other side effects of psychotropic medications such as tardive dyskinesia or orthostatic hypotension can complicate dental treatment.

Mental illness has been said to be an '**invisible disability**' which include conditions that may not be immediately apparent but may result in handicap and stigma when revealed (Harding 1990). The writer feels that the patients with chronic mental illness who are edentulous but unable to adapt to, or wear dentures, will be doubly handicapped. Their edentulous appearance can cause them to be more stigmatised. The quality of life will be lessened for these patients.

The significance of psychological factors in dental treatment have been recognised by many workers (Moulton 1955, Shira et al 1959, Sinick 1964, Miller 1970, Harris and Davies 1980, Jackson 1980, McSwiggan 1991). All these workers stress the importance of a good dentist-patient relationship in overcoming many of these problems. The writer supports this wholeheartedly. The importance of the mouth, and

its significance may have a strong psychological influence for certain individuals. They may feel that their whole being is under attack during dental procedures. This can complicate dental treatment considerably.

The writer feels that the psychiatric morbidity in dentistry has to be recognised. The McGill Pain Questionnaire and the Hospital Anxiety and Depression scale has been recommended for use by both the medical and dental professions (Lamey and Lamb 1989, Zakrzewska and Feinmann 1990). These 2 scales can be used to assess pain and its effects on the patient's well being. The psychological morbidity in dental practice can be measured in this way.

The writer feels that the dentist should be aware of the possible extensive Hepatitis B virus carrier status among institutionalised people with mental illness. Information regarding the prevalence of the Hepatitis B virus carrier status in Australia is lacking (Reed 1987). However, statistics of the high incidence of Hepatitis B virus carrier status in institutionalised patients with mental illness overseas suggest the need for this group to be screened routinely before dental treatment (Reed 1987). The writer considers that the presence of the Hepatitis B virus carrier status should not affect the type of dental treatment required by a patient with mental illness. However, the appropriate infection control procedures, as for all Hepatitis B virus carriers, will have to be adopted during dental treatment.

The writer has not discussed the various treatment modes for the different types of dental problems associated with mental illness as this is not within the scope of this treatise. The writer has not discussed drug and alcohol abuse and their dental implications in this treatise as she feels that this type of psychiatric disorder has a different significance both psychosocially and dentally.

12 Conclusions

Mental illness (psychiatric disorders) and dental disease are the most prevalent health problems of modern society (Jackson 1980). Thus, it is not hard to envisage that many psychiatric patients have dental problems (some of which may be the result of the mental illness or side effects of the psychotropic medications) and that a certain section of dental patients have psychiatric problems. The psychiatric morbidity in general illness has been recognised and has resulted in the development of consultation-liaison psychiatry. The psychiatric morbidity in dentistry has to be recognised too.

Similar to many other dentists who work together with the medical profession in the management of certain patients with mental illness (Beck et al 1979, Friedlander and Brill 1986a, Friedlander and Brill 1986b, Friedlander and Birch 1990, Friedlander and Jolyon 1991), the writer recognises the importance of understanding the nature of the psychiatric disorder concerned before commencement of dental treatment. People with undiagnosed mental illness may often be unaware of their disorder and may initially seek treatment from a dentist (Friedlander and Jolyon 1991). Dentists must become familiar with signs and symptoms of some common mental illnesses such as depression and schizophrenia, to be able to recognise these indicators. Prompt referral of the patient for definitive diagnosis and treatment before complex dental procedures commence, will be necessary. The chapter on mental illness in this treatise may not be sufficient but will help the dentist begin to understand the complex nature of mental illness. The writer has recommended a book on psychiatry to be kept for reference.

The common orofacial manifestations (psychogenic facial pain, oral self-mutilation, perimyololysis, parotid gland enlargement) of mental illness have been discussed. Psychological factors which can bring about parafunctional behaviour such as bruxism

and/or cause failure of full prosthetic treatment have been considered. Psychiatric disorders which may be encountered in general dental practice have been reviewed. These psychiatric disorders include dental phobia, hypochondriasis and non-accidental injury of children. The side effects of psychotropic medication that affect the delivery of dental treatment (primarily orthostatic hypotension and excessive sedation) and/or cause dental problems (most significantly, xerostomia and tardive dyskinesia) for the patient have been corroborated. The extensive level of dental disease frequently associated with mental illness has been highlighted. The writer feels that this treatise has fulfilled its aims. She feels that she has established the need to recognise the psychiatric morbidity in dentistry. She has also demonstrated the need for psychiatric input into dental education as well as dental input into the education of others in the medical professions so that the concept of the psychiatrist and dentist working together as a team in the management of certain patients will not be so far fetched.

13 References

- Abrams RA, Ruff JC. Oral signs and symptoms in the diagnosis of bulimia. *J Am Dent Assoc* 1986; 113:761-764.
- Aldous J. Induced xerostomia and its relationship to dental caries. *J Dent Child* 1964; Second Quarter: 160-162.
- Alias AG, Shen WW, Parwatikar SD. Cyclic sialorrhea unrelated to lithium in a patient with bipolar affective disorder. *Am J Psychiatry* 1983; 140:1538-1539.
- Allan DN. Dental erosion from vomiting. *Br Dent J* 1969; 127:311-312
- Altom RL, DiAngelis AJ. Multiple autoextractions: Oral self-mutilation reviewed. *Oral Surg Oral Med Oral Pathol* 1989; 67:271-4.
- Andreasen NC, Bardach J. Dysmorphobia: Symptom or disease? *Am J Psychiatry* 1977; 134:673-676.
- Andrews FFH. Dental erosion due to anorexia nervosa with bulimia. *Br Dent J* 1982; 152:89-90.
- Andrews G. The anxiety disorders. In: Beumont PJV, Hampshire RB eds. *Textbook of Psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:99-104.
- Arenillas L. Amitriptyline and body-weight. *Lancet* 1964; 1:432-433.
- Arnold M. Bruxism and occlusion. *Dent Clin North Am* 1981; 25:395-407.
- Attanasio R. Nocturnal bruxism and its clinical management. *Dent Clin North Am* 1991; 35:245-252.
- Australian Bureau of Statistics. Australian Health Survey 1977-78: Chronic condition (illness and permanent disabilities). Catalog No. 4314.0:22.
- Australian Bureau of Statistics. Australian Health Survey 1983: Illness conditions experienced. Catalog No. 4356.0:21.

- Australian Bureau of Statistics. Census of population and housing 30th June 1986: Characteristics of In-patients of health institutions in Australia. Catalog No. 4347.0:5.
- Australian Bureau of Statistics. Census of mental health and long-stay in-patients in hospitals and nursing homes. New South Wales 1987. Catalog No. 4310.1:iv-v.
- Ayer WA, Levin MP. Self-mutilating behaviours involving the oral cavity. *J. Oral Med* 1974; 29:4-7.
- Badger GR. Caries incidence in child abuse and neglect. *Pediatr Dent* 1986; Special Issue 1:101-102.
- Bahn SL. Drug-related dental destruction. *Oral Surg* 1972; 33:49-54.
- Bar Nathan EA, Brenner S, Horowitz I. Nonspecific stomatitis due to lithium therapy. *Am J Psychiatry* 1985; 142:1126-1127.
- Barrett AP, Buckley DJ. Covert self-mutilation of oral tissues and skin by mechanical and chemical means. *Oral Surg Oral Med Oral Pathol* 1988; 65:685-688.
- Barrow G. Consultation and liaison psychiatry. In: Beumont PJV, Hampshire RB eds. Textbook of psychiatry 1989 edition. Australia: Blackwell Scientific Publications 1989:243-245.
- Basker RM, Sturdee DW, Davenport JC. Patients with burning mouths. *Br Dent J* 1978; 145:9-16.
- Basset A, Remick RA, Blasberg B. Tardive dyskinesia: An unrecognised cause of orofacial pain. *Oral Surg Oral Med Oral Pathol* 1986; 61:570-572.
- Bassuk E, Schoonover S. Rampant dental caries in the treatment of depression. *J Clin Psychiatry* 1978; 39:163-165.
- Batsakis JG, McWhirter JD. Nonneoplastic diseases of the salivary glands. *Am J Gastroenterology* 1972; 57:238.
- Beaumont PJ, Beardwood CJ, Russell GFM. The occurrence of the syndrome of anorexia nervosa in male subjects. *Psychol Med* 1972; 2:216.

- Bebbington PE. Monosymptomatic hypochondriasis, abnormal illness behaviour and suicide. *Br J Psychiatry* 1976; 128:475-478.
- Beck FM, Kaul TJ, Weaver II JM. Recognition and management of the depressed dental patient. *J Am Dent Assoc* 1979; 99:967-971.
- Becker DB, Needleman HL, Katelchuck M, Child abuse and dentistry: Orofacial trauma and its recognition by dentists. *J Am Dent Assoc* 1978; 97:24-28, 447.
- Berggren U, Meynert G. Dental fear and avoidance: Causes, symptoms and consequences. *J Am Dent Assoc* 1984; 109:247-251.
- Bernard JD, Shearn MA. Psychogenic Pseudo-Sjögren's Syndrome. *Western J Med* 1974; 120:247-248.
- Berry DC. Mandibular dysfunction pain and chronic minor illness. *Br Dent J* 1969; 127:170-175.
- Bianchi GN. Patterns of hypochondriasis: A principal components analysis. *Br J Psychiatry* 1973; 122:541-548.
- Blackwell B. Adverse effects of antidepressant drugs. Part 1: Monoamine oxidase inhibitors and tricyclics. *Drugs* 1981a; 21:201-219.
- Blackwell B. Adverse effects of antidepressant drugs. Part 2: 'Second generation' antidepressants and rational decision making in antidepressant therapy. *Drugs* 1981b; 21:273-282.
- Blanton PL, Hunt WC, Largent MD. Oral factitious injuries. *J. Periodontol* 1977; 48:33-37.
- Blasberg B, Remick RA, Miles JE. The psychiatric referral in dentistry: Indications and mechanics. *Oral Surg Oral Med Oral Pathol* 1983; 56:368-371.
- Boakes AJ, Laurence DR, Lovel KW, O'Neil R, Verrill PJ. Adverse reactions to local anaesthesia/vasoconstrictor preparations. *Br Dent J* 1972; 133:137-140.
- Bolender CL, Swoope CC, Smith DE. The Cornell Medical Index as a prognostic aid for complete denture patients. *J Prosthet Dent* 1969; 22:20-29.
- Bone S, Roose SP, Dunner DL, Fieve RR. Incidence of side effects in patients on long-term lithium therapy. *Am J Psychiatry* 1980; 137:103-104.

- Borland LR. Odontophobia - Inordinate fear of dental treatment. *Dent Clin N Am* 1962; 1:683-695.
- Brady JP. Parotid enlargement in bulimia. *J Fam Practice* 1985; 20:496-502.
- Brady WF. The anorexia nervosa syndrome. *Oral Surg Oral Med Oral Pathol* 1980; 50:509-516.
- Brightman VJ. Tremors and other abnormal involuntary jaw movements. In: Rose LF, Kaye D eds. *Internal medicine for dentistry* 1st ed. CV Mosby Company, 1983:870-871.
- Brooke RI, Stenn PG, Mothersill KJ. The diagnosis and conservative treatment of myofascial pain dysfunction syndrome. *Oral Surg Oral Med Oral Pathol* 1977; 44:844-852.
- Brooke RI. Atypical odontalgia. *Oral Surg Oral Med Oral Pathol* 1980; 49:196-199.
- Brown JP, Smith IT. Childhood fear and anxiety states in relation to dental treatment. *Aust Dent J* 1979; 24:256-259.
- Browning S, Hislop S, Scully C, Shirlaw P. The association between burning mouth syndrome and psychological disorders. *Oral Surg Oral Med Oral Pathol* 1987; 64:171-174.
- Burton RC. The problem of facial pain. *J Am Dent Assoc* 1969; 79:93-101.
- Bushfield BL, Wechsler H, Barnum WJ. Studies of salivation in depression II: Physiological differentiation of reactive and endogenous depression. *Arch Gen Psychiatry* 1961; 5:472-477.
- Bushfield BL, Wechsler H. Studies of salivation in depression. A comparison of salivation rates in depressed, schizoaffective depressed, non depressed hospitalised patients and in normal controls. *Arch Gen Psychiatry* 1961; 4:36-41.
- Butler RN. Psychiatry and the elderly: An overview. *Am J Psychiatry* 1975; 132:893-900.
- Carlsson A. Antipsychotic drugs, neurotransmitters and schizophrenia. *Am J Psychiatry* 1978; 135:164-173.

- Carruthers SG. Persistent tardive dyskinesia. *Br Med J* 1971; 3:572.
- Cash RG. Bruxism in children: Review of the literature. *J Pedod* 1988; 12:107-127.
- Casper RC, Eckert ED, Halmi KA, Goldberg SC, Davis JM. Bulimia. *Arch Gen Psychiatry* 1980; 37:1030-1035.
- Cawson RA, Spector RG. Depressants of cerebral function. In: *Clinical pharmacology in dentistry*, 5th edn. United Kingdom: Churchill Livingstone 1989:91-92.
- Chamberlain BB, Chamberlain KR. Depression: A psychological consideration in complete denture prosthodontics. *J Prosthet Dent* 1985; 53:673-675.
- Chancellor A, Mant A, Andrews G. The general practitioner's identification and management of emotional disorders. *Aust Family Physicians* 1977; 6:1137-1143.
- Chellapah NK, Vignehsa H, Milgrom P, Lo GL. Prevalence of dental anxiety and fear in children in Singapore. *Community Dent Oral Epidemiol* 1990; 18:269-271.
- Colquitt T. The sleep-wear syndrome. *J Prosthet Dent* 1987; 57:33-41.
- Connolly FH, Gipson M. Dysmorphobia - a long term study. *Br J Psychiatry* 1978; 132:568-570.
- Corah NL, Gale EN, Illig S. Assessment of a dental anxiety scale. *J Am Dent Assoc* 1978; 97:816-819.
- Corah NL, O'Shea RM, Bissell GD. The dentist-patient relationship: Perceptions by patients of dentist behaviour in relation to satisfaction and anxiety. *J Am Dent Assoc* 1985a; 111:443-446.
- Corah NL, O'Shea RM, Ayer WA. Dentist's management of patients' fear and anxiety. *J Am Dent Assoc* 1985b; 110:734-736.
- Crane G. Persistent dyskinesia. *Br J Psychiatry* 1973; 122:395-405.
- Crisp AH, Palmer RL, Kalucy RS. How common is anorexia nervosa? A prevalence study. *Br J Psychiatry* 1976; 128:549-554.

- D'Alessandro R, Benassi G, Cristina E, Gallassi R, Manzaroli D. The prevalence of lingual-facial-buccal dyskinesias in the elderly. *Neurology* 1986; 36:1350-1351.
- Dallos V, Heathfield K. Iatrogenic epilepsy due to antidepressant drugs. *Br Med J* 1969; 4:80-82.
- Dawson J, Jones C. Vomiting-induced hypokalaemic alkalosis and parotid swelling. *Practitioner* 1977; 218:267-268.
- De Martino BK. Anxiety. Dental overview. *Dent Clin N Am* 1987; 31:11-16.
- Delaney JF. Atypical facial pain as a defense against psychosis. *Am J Psychiatry* 1976; 133:1151-1154.
- DenBesten PK, McIver FT. Oral self-mutilation in a child with congenital toxoplasmosis: A clinical report. *Pediatr Dent* 1984; 6:98-101.
- Dettmar DM, L'Estrange. The effects of stress release in indices of mandibular dysfunction. *Aust Dent J* 1987; 32:39-41.
- Domoto PK, Weinstein P, Melnick S, Ohmura M, Uchida H, Ohmachi K et al. Results of a dental fear survey in Japan: Implications for dental public health in Asia. *Community Dent Oral Epidemiol* 1988; 16:199-210.
- Donaldson SR. Sialorrhea as a side effect of lithium: A case report. *Am J Psychiatry* 1982; 139:1350-1351.
- Donlon WC, Kaplan H, Javid B, Harness M, Schultz P, Rome H et al. Multifactorial facial pain - differential diagnosis: A case report. *J Am Dent Assoc* 1990; 120:315-320.
- Dreiling DA, Noronha M, Nacchiero M, Pieroni P, Wolfson P. The parotid and the pancreas. *Am J Gastroenterology* 1978; 70:627-634.
- Dura JR, Torsell AE, Heinzerling RA, Mulick JA. Special oral concerns in people with severe and profound mental retardation. *Spec Care Dent* 1988; November-December:265-267.
- Dworkin SF, Burgess JA. Orofacial pain of psychogenic origin: Current concepts and classification. *J Am Dent Assoc* 1987; 115:565-571.

- Edwards JG. Antidepressants and convulsion. *Lancet* 1979; 2:1368-1369.
- Eklfeldt A, Hugoson A, Bergendal T, Helkimo M. An individual tooth wear index and an analysis of factors correlated to incisal and occlusal wear in an adult Swedish population. *Odontol Scand* 1990; 48:343-349.
- Evans JH. Persistent oral dyskinesia in treatment with phenothiazine derivatives. *Lancet* 1965; 1:458-460.
- Evans L. Psychiatry and general practice. In: Beumont PJV, Hampshire EB eds. *Textbook of psychiatry* 1989 edition. Australia: Blackwell Scientific Publications. 1989:255.
- Fabisch W. Psychiatric aspects of dermatitis artefacta. *Br J Dermatology* 1980; 102:29-34.
- Fahn S. The tardive dyskinesias. In: Mathews WB, Glaser GH eds. *Recent advances in clinical neurology*. Vol 4. Edinburgh: Churchill Livingstone Press, 1984:229-260.
- Fann WE, Sullivan JL, Richman BW. Dyskinesias associated with tricyclic antidepressants. *Br J Psychiatry* 1976; 128:490-493.
- Faulkner KDB. Bruxism: A review of the literature. Part I. *Aust Dent J* 1990; 35:266-276.
- Feinmann C, Harris M. Psychogenic facial pain. Part 1: The clinical presentation. *Br Dent J* 1984a; 156:165-168.
- Feinmann C, Harris M. Psychogenic facial pain. Part 2: Management and prognosis. *Br Dent J* 1984b; 156:205-208.
- Feinmann C, Harris M, Cawley R. Psychogenic facial pain: Presentation and treatment. *Br Med J* 1984; 288:436-438.
- Feinmann C. Psychogenic facial pain: Presentation and treatment. *J Psychosomatic Res* 1983; 27:403-410.
- Felder RS, Millar SB, Henry RH. Oral manifestations of drug therapy. *Spec Care Dent* 1988; 8:119-124.

- Fine EW. Psychological factors associated with non-organic temporomandibular joint pain dysfunction syndrome. *Br Dent J* 1971; 131:402-404.
- Finlay-Jones R. Psychiatric Illness and their classification. Part 1. Disease and illness. In: Beumont PJV, Hampshire RB eds. *Textbook of Psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:1-7.
- Fox PC, Weiffenbach JM, Van der Ven PF, Baum BJ, Sonies BC. Xerostomia: Evaluation of a symptom with increasing significance. *J Am Dent Assoc* 1985; 110:519-525.
- Fox PC. Saliva composition and its importance in dental health. *Compend Contin Educ Dent* 1989; Suppl No. 13:457-460.
- Freeman RE. Dental Anxiety: A multifunctional aetiology. *Br Dent J* 1985; 159:406-408.
- Friedlander AH, Birch NJ. Dental conditions in patients with bipolar disorder on long term lithium maintenance therapy. *Spec Care Dent* 1990; 10:148-151.
- Friedlander AH, Brill NQ. The dental management of patients with schizophrenia. *Spec Care Dent* 1986a; 6:217-218.
- Friedlander AH, Brill NQ. The dental management of patients with bipolar disorder. *Oral Surg Oral Med Oral Pathol* 1986b; 61:579-581.
- Friedlander AH, Jolyon L. Dental management of the patient with major depression. *Oral Surg Oral Med Oral Pathol* 1991; 71:573-578.
- Friedman AP. Differential diagnosis of facial pain. *Dent Clin North Am* 1966; November:545-551.
- Friedman N, Landesman HM, Wexler M. The influence of fear, anxiety, and depression on the patient's adaptive responses to complete dentures. Part I. *J Prosthet Dent* 1987; 58:687-689.
- Friedman N, Landesman HM, Wexler M. The influence of fear, anxiety, and depression on the patient's adaptive responses to complete dentures. Part II. *J Prosthet Dent* 1988a; 59:45-48.

- Friedman N, Landesman HM, Wexler M. The influence of fear, anxiety, and depression on the patient's adaptive responses to complete dentures. Part III. J Prosthet Dent 1988b; 59:169-173.
- Frisch J, Katz L, Ferreira AJ. A study on the relationship between bruxism and aggression. J Periodontol 1960; 31:409-412.
- Gale EN, Ayer WA. Treatment of dental phobias. J Am Dent Assoc 1969; 78:1304-1307.
- Gecker LM, Weil RB. Bruxism - a rationale of therapy. J Am Dent Assoc 1963; 66:14-18.
- Gelbier S. Self-extraction of lower incisors in a boy. Dent Pract 1963; 13:291-292.
- Gelder M, Gath D, Mayou R. Classification in psychiatry. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain: Butler and Tanner Ltd 1989a:73-78.
- Gelder M, Gath D, Mayou R. Neurosis Part 1. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain: Butler and Tanner Ltd 1989b:154-155.
- Gelder M, Gath D, Mayou R. Neurosis Part 1. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain: Butler and Tanner Ltd 1989c:164-168.
- Gelder M, Gath D, Mayou R. Aetiology. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain: Butler and Tanner Ltd 1989d:97.
- Gelder M, Gath D, Mayou R. Neurosis: Part II. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain. Butler and Tanner Ltd 1989e:191.
- Gelder M, Gath D, Mayou R. Neurosis: Part II. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain. Butler and Tanner Ltd 1989f:183-186.
- Gelder M, Gath D, Mayou R. Psychiatry and medicine. In: Oxford textbook of psychiatry. 2nd edn. Oxford: Oxford University Press 1989g:416-417.
- Gelder M, Gath D, Mayou R. Psychiatry and medicine. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain. Butler and Tanner Ltd 1989h:417-418.

- Gelder M, Gath D, Mayou R. Paranoid symptoms and paranoid syndromes. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain. Butler and Tanner Ltd 1989i:340.
- Gelder M, Gath D, Mayou R. Drugs and other physical treatments. In: Oxford textbook of psychiatry. ELBS edition. 2nd edn. Great Britain. Butler and Tanner Ltd 1989j:633-673.
- Giddon DB. Psychologic aspects of prosthodontic treatment for geriatric patients. J Prosth Dent 1980; 43:374-379.
- Gillis A. Lithium carbonate and dental caries. Br Med J 1978; 2:1717.
- Glaros AG, Rao SM. Effects of bruxism: A review of the literature. J Prosthet Dent 1977; 38:149-157.
- Glaros AG. Incidence of diurnal and nocturnal bruxism. J Prosthet Dent 1981; 45:545-549.
- Glass BJ, Van Dis ML, Langlais RP, Miles DA. Xerostomia: Diagnosis and treatment planning considerations. Oral Surg 1984; 58:248-252.
- Glazer WM, Morgenstern H, Niedzurieki D, Hughes J. Heterogeneity of tardive dyskinesia, a multivariate analysis. Br J Psychiatry 1988; 152:253-259.
- Goldberg D. The recognition of psychiatric illness by non-psychiatrists. Aust NZ J Psychiatry 1984; 18:128-133.
- Goldberg DP, Blackwell B. Psychiatric illness in general practice. A detailed study using a new method of case identification. Br Med J 1970; 2:439-443.
- Golden S, Chosach A. Oral Manifestations of a psychological problem. J Periodontol 1964; 35:69-70.
- Goldhamer PM. Psychotherapy and pharmacotherapy: The challenge of integration. Can J Psychiatry 1983; 28:173-177.
- Goldstein IC, Dragon AI. Self-inflicted oral mutilation in a psychotic adolescent. Report of a case. J Am Dent Assoc 1967; 74:750-751.

- Goodman P, Greene CS, Laskin DM. Response of patients with myofascial pain-dysfunction syndrome to mock equilibration. *J Am Dent Assoc* 1976; 92:755-758.
- Gottlieb G, Paulson G. Salivation in depressed patients. *Arch Gen Psychiatry* 1961; 5:468-471.
- Gould AR, Farman AG, Corbitt D. Mutilation of the dentition in Africa: A review with personal observations. *Quintessence* 1984; 1:89-94.
- Graf H. Bruxism. *Dent Clin North Am* 1969; 13:659-665.
- Green RM, Green A. Patients' perception of their overall level of anxiety about dental care. *Br Dent J* 1987; 162:155-16.
- Greene CS, Laskin DM. Long-term evaluation of conservative treatment for myofascial pain-dysfunction syndrome. *J Am Dent Assoc* 1974; 89:1365-1368.
- Greene CS, Lerman MD, Sutchter HD, Laskin DM. The TMJ pain dysfunction syndrome: Heterogeneity of the patient population. *J Am Dent Assoc* 1969; 79:1168-1172.
- Gross SM, Vacchiano RB. Personality correlates of patients with temporomandibular joint dysfunction. *J Prosthet Dent* 1973; 30:326-329.
- Groves BJ. Self-inflicted periodontal injury. *Br Dent J* 1979; 147:244-246.
- Grushka M, Sessle BJ. Burning mouth syndrome. *Dent Clin North Am* 1991; 35:171-184.
- Grushka M. Clinical features of burning mouth syndrome. *Oral Surg Oral Med Oral Pathol* 1987; 63:30.
- Grzesiak RC. Psychologic considerations in temporomandibular dysfunction. *Dent Clin North Am* 1991; 35:209-225.
- Halestrap DJ. Indigenous dental practice in Uganda. *Br Dent J* 1971; 131:463-464.
- Hall N, Edmondson HD. The aetiology and psychology of dental fear. *Br Dent J* 1983; 154:247-252.

- Halmi KA. Anorexia nervosa: Demographic and clinical features in 94 cases. *Psychomatic Med* 1974; 36:18-25.
- Handelman SL, Baric JM, Espeland MA, Berglund KL. Prevalence of drugs causing hyposalivation in an institutionalised geriatric population. *Oral Surg Oral Med Oral Pathol* 1986; 62:26-31.
- Harding M. Invisible disability. *Br Dent J* 1990; 169:304.
- Harris E. The antipsychotics. *Am J Nursing* 1988a; 88:1508-1511
- Harris E. The anti-depressants. *Am J Nursing* 1988b; 88:1512-1518.
- Harris M, Davies G. Psychiatric disorders. In: Jones JG. Mason MK eds. *Oral manifestations of systemic disease*. Philadelphia: Saunders, 1980:439-453.
- Harris M. Psychogenic aspects of facial pain. *Br Dent J* 1974; 136:199-202.
- Harris M. Psychosomatic disorders of the mouth and face. *Practitioner* 1975; 214:372-379.
- Hasler JF, Schultz WF. Factitial gingival traumatism. *J Periodontol* 1968; 39:56-57.
- Hasler JF. Parotid enlargement: A presenting sign in anorexia nervosa. *Oral Surg Oral Med Oral Pathol* 1982; 53:567-573.
- Hay GG. Dysmorphobia. *Br J Psychiatry* 1970; 116:399-406.
- Hellström I. Anorexia nervosa - odontologiska problem. *Swed Dent J* 1974; 67:253.
- Hellström I. Anorexia/bulimia nervosa: Dental problems. *Acta Psychiatrica Scand* 1990; 82 Suppl. 361:18.
- Hellström I. Oral complications in anorexia nervosa. *Scand J Dent Res* 1977; 85:71.
- Henderson AS, Rosenman SJ. Psychogeriatrics. In: Beumont PJV, Hampshire RB eds. *Textbook of psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:226.
- Herzog DB, Copeland PM. Eating disorders. *New Engl J Med* 1985; 313:295-303.
- Hierholzer RW. Tardive dyskinesia with complaints of pain. *Am J Psychiatry* 1989; 146:802.

- Hoffman HA, Baer PN. Gingival mutilation in children. *Psychiatry* 1968; 31:380-386.
- Hollister LE. Current antidepressant drugs: Their clinical use. *Drugs* 1981; 22:129-152.
- Hollister LE. Psychiatric disorders. In: Avery GS ed. *Drug treatment. Principles and practice of clinical pharmacology and therapeutics*. 2nd edn. Sydney and New York: Adis Press 1980:1059.
- Hughes AM, Hunter S, Still D, Lamey PJ. Psychiatric disorders in a dental clinic. *Br Dent J* 1989; 166:16-19.
- Humphreys M, Scott J. Psychiatric aspects of dentistry II. *Br Dent J* 1987; 163:85-88.
- Huon GF, Broun LB. Review. Bulimia: The emergence of a syndrome. *Aust NZ J Psychiatry* 1984; 18:113-126.
- Hurst PS, Lacey JH, Crisp AH. Teeth, vomiting and diet: A study of the dental characteristics of 17 anorexia nervosa patients. *Postgrad Med J* 1977; 53:298-305.
- Jackson E. Psychiatry and dentistry. In: Kaplan HI, Freedman AM, Sadock BJ eds. *Comprehensive textbook of psychiatry II*. Baltimore: Williams and Wilkins, 1980:2069-2077.
- Jankovic J. Orofacial and other self-mutilation. *Advances in Neurology* 1988; 49:365-381.
- Jastak JT, Yagiela JA. Vasoconstrictors and local anaesthesia: A review and rationale for use. *J Am Dent Assoc* 1983; 107:823-830.
- Jenner FA, Gjessing LR, Cox JR, Davies-Jones A, Hullin RP, Hanna SM. A manic depressive psychotic with a persistent forty-eight hour cycle. *Br J Psychiatry* 1967; 113:895-910.
- Jensen ØE, Featherstone JDB, Stege P. Chemical and physical oral findings in a case of anorexia nervosa and bulimia. *Oral Path* 1987; 16:399-402.
- Jeste DV, Potkin SG, Sinha S, Feder S, Wyatt RJ. Tardive dyskinesia - reversible and persistent. *Arch Gen Psychiatry* 1979; 36:585-590.

- Jeste DV, Wyatt RJ. Changing epidemiology of tardive dyskinesia: An overview. *Am J Psychiatry* 1981; 138:297-309.
- Jeste DV, Wyatt RJ. Prevention and management of tardive dyskinesia. *J Clin Psychiatry* 1985; 46:14-18.
- Jeste DV, Wyatt RJ. Therapeutic strategies against tardive dyskinesia. Two decades of experience. *Arch Gen Psychiatry* 1982; 39:803-816.
- Johnson G. The biological therapies. Part I - pharmacotherapy. In: Beumont PJV, Hampshire RB eds. Text book of psychiatry 1989 edition. Australia: Blackwell Scientific Publications 1989:323-354.
- Jones IH. Schizophrenia. In: Beumont PJV, Hampshire RB eds. Textbook of psychiatry 1989 edition. Australia: Blackwell Scientific Publications 1989:74-84.
- Joy ED, Barber J. Psychological, physiological and pharmacological management of pain. *Dent Clin North Am* 1977; 21:577-593.
- Judd FK, Burrows GD. Affective disorders. In: Beumont PJV, Hampshire RB eds. Textbook of psychiatry 1989 edition. Australia: Blackwell Scientific Publications 1989:85-98
- Kakizaki G, Sasahara M, Soeno T, Shoji S, Ishidate T, Senoo A. Mechanism of the pancreas-parotid gland interaction. *Am J Gastroenterology* 1978; 70:635-644.
- Kane FJ, Anderson WB. A fourth occurrence of oral moniliasis during tranquiliser therapy. *Am J Psychiatry* 1963; 120:1199-1120.
- Kane FJ. Oral moniliasis following the use of thorazine. *Am J Psychiatry* 1963; 120:187-188.
- Kane FJ. Severe oral moniliasis complicating chlorpromazine therapy. *Am J Psychiatry* 1962; 119:890.
- Kane JM, Smith JM. Tardive dyskinesia, prevalence and risk factors, 1959 to 1979. *Arch Gen Psychiatry* 1982; 39:473-480.
- Karson CN, Jeste DV, LeWitt PA, Wyatt RJ. *Am J Psychiatry* 1983; 140:1504-1506.

- Katsilambros L. Asymptomatic enlargement of the parotid glands. *J Am Med Assoc* 1961; 178:513.
- Keith O, Flint S. Lingual abscess in a patient with anorexia nervosa. *Br Dent J* 1989; 167:71-72.
- Kempe CH, Silverman FN, Steele BF, Droegemueller W, Silver HK. The battered child syndrome. *J Am Med Assoc* 1962; 181:17.
- Kennedy PF. Chorea and phenothiazines. *Br J Psychiatry* 1969; 115:103-104.
- Kent G, Warren P. A study of factors associated with changes in dental anxiety. *J Dent Res* 1985; 64:1316-1318.
- Kent G. Memory of dental pain. *Pain* 1985; 21:187-194.
- Kent GG. Thinking about anxiety. *Br Dent J* 1990; 169:133-135.
- Kenyon FE. Hypochondriasis: A clinical study. *Br J Psychiatry* 1964; 110:478-488.
- Kenyon FE. Hypochondriasis. *Br J Hosp Med* 1976; 16:419-428.
- Kittle PE, Richardson DS, Parker JW. Examining for child abuse and child neglect. *Pediatr Dent* 1986; Special Issue 1:80-82.
- Kleier DJ, Aragon SB, Averbach RE. Dental management of the chronic vomiting patient. *J Am Dent Assoc* 1984; 108:618-621.
- Kleinknecht RA, Klepac RK, Alexander LD. Origins and characteristics of fear in dentistry. *J Am Dent Assoc* 1973; 86:842-848.
- Koper A. Why dentures fail. *Dent Clin North Am* 1964; November:721-734.
- Kunzelmann K-H, Dünninger P. Dental fear and pain: Effect on patient's perception of the dentist. *Community Dent Oral Epidemiol* 1990; 18:264-266.
- LaBanc J, Epker BN. Lesch-Nyhan Syndrome: Surgical treatment in a case with lip chewing. *J Oral Maxillofac Surg* 1981; 9:64-67.
- Lamey PJ, Allam BF. Vitamin status of patients with burning mouth syndrome and the response to replacement therapy. *Br Dent J* 1986; 160:81-83.

- Lamey PJ, Lamb AB. The usefulness of the HAD scale in assessing anxiety and depression in patients with burning mouth syndrome. *Oral Surg Oral Med Oral Pathol* 1989; 67:390-392.
- Lamey PJ, Lewis MAO. Oral medicine in practice: Burning mouth syndrome. *Br Dent J* 1989; 167:197-200.
- Lange BM, Entwistle BM, Lipson LF. Cerebral palsy. In: *Dental management of the handicapped ; approaches for dental auxiliaries*. Philadelphia: Lea and Febiger 1983:131.
- Langer A. Chemoppsychotherapy and its role in prosthodontic failures in elderly patients. *J Prosthet Dent* 1984; 52:14-19.
- Lascelles RG. Atypical facial pain and depression. *Br J Psychiatry* 1969; 112:651-659.
- Laskin DM. Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 1969; 79:147-153.
- Lautch H. Dental phobia. *Br J Psychiatry* 1971; 119:151-158.
- Lavender S. Vomiting and parotid enlargement. *Lancet* 1969; 1:426.
- Leader. Dysmorphobia. *Br Med J* 1978; 2:588.
- Lesch M, Nyhan WL. A familial disorder of uric acid metabolism and central nervous System functions. *Am J Med* 1964; 36:561-570.
- Levenson AJ. Psychiatric implications in the treatment of the geriatric patient. *Spec Care Dent* 1983; January-February:4-12.
- Levin PA, Falko JM, Dixon K, Gallup EM, Saunders W. Benign parotid enlargement in bulimia. *Annals Int Med* 1980; 93:827-829.
- Lindqvist B. Bruxism and emotional disturbance. *Odont Revy* 1972; 23:231-242.
- Lindsay SJE, Humphris G, Barnby GJ. Expectations and preferences for routine dentistry in anxious adult patients. *Br Dent J* 1987; 163:120-124.
- Lipowski LJ. Current trends in consultation-liaison psychiatry. *Can J Psychiatry* 1983; 28:329-338.

- Little JW, Falace DA. Dental management of the medically compromised patient. 3rd edn. Missouri: CV Mosby Company 1988a:22,216,217.
- Little JW, Falace DA. Dental management of the medically compromised patient. 3rd edn. Missouri: CV Mosby Company 1988b:418-419.
- Loudon ISL. Chlorosis, anaemia and anorexia nervosa. Br Med J 1980; 281:1669-1675.
- Lowe O. Tourette's syndrome: Management of oral complications. J Dent Child 1986; 53:456-460.
- Lowental U, Pisanti S. The syndrome of oral complaints: Etiology and therapy. Oral Surg Oral Med Oral Pathol 1978; 46:2-6.
- Lupton DE. Psychological aspects of temporomandibular joint dysfunction. J Am Dent Assoc 1969; 79:131-136.
- Mack PJ. Hairy dentures: A monosymptomatic hypochondriacal psychosis. Br Dent J 1985; 158:50-51.
- MacLean G, Robertson BM. Self-enucleation and psychosis. Report of two cases and discussion. Arch Gen Psychiatry 1976; 33:242-249.
- Main DMG, Basker RM. Patients complaining of a burning mouth. Br Dent J 1983; 154:206-211.
- Mandel ID. The function of saliva. J Dent Res 1987; 66:623-627.
- Mandel ID. The role of saliva in maintaining oral homeostasis. J Am Dent Assoc 1989; 119:298-303.
- Marbach JJ, Varoscak JR, Blank RT, Lund P. "Phantom Bite": Classification and treatment. J Prosthetic Dentistry 1983; 49:556-559.
- Marbach JJ. Phantom bite syndrome. Am J Psychiatry 1978b; 135:476-479.
- Marbach JJ. Phantom tooth pain. J Endodont 1978a; 4:362-372.
- Markitziu A, Shani J, Avni J. Salivary gland function in patients on chronic lithium treatment. Oral Surg Oral Med Oral Pathol 1988; 66:551-557.

- Mathew RJ, Weinman M, Claghorn JL. Xerostomia and sialorrhea in depression. *Am J Psychiatry* 1979; 136:1476-1477.
- McCall CM, Szmyd L, Ritter RM. Personality characteristics in patients with temporomandibular joint symptoms. *J Am Dent Assoc* 1961; 62:694-698.
- McKenna PJ. Disorders with overvalued ideas. *Br J Psychiatry* 1984; 145:579-585.
- McLean P, Casey DE. Tardive dyskinesia in an adolescent. *Am J Psychiatry* 1978; 135:969-971.
- McSwiggan W. 1991. Personal communication.
- Meares R. Somatoform and dissociative disorders and related conditions. In: Beumont PJV, Hampshire RB eds. *Textbook of psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:116.
- Merskey H, Lau CL, Russell ES, Brooke RI, James M, Loppano S et al. Screening for psychiatric morbidity. The patterns of psychological illness and premorbid characteristics in four chronic pain populations. *Pain* 1987; 30:141-157.
- Merskey H. Too much pain. *Br J Hosp Med* 1984; 31:63-66.
- Meyer BC. Some psychiatric aspects of surgical practice. *Psychosom Med* 1958; 20:203-214.
- Mikami DB. A review of psychogenic aspects and treatment of bruxism. *J Prosthet Dent* 1977; 37:411-419.
- Milgrom P, Fiset L, Melnick S, Weinstein P. The prevalence and practice management consequences of dental fear in a major US city. *J Am Dent Assoc* 1988; 116:641-647.
- Miller AA. Psychological considerations in dentistry. *J Am Dent Assoc* 1970; 81:941-946.
- Miller SM, Naylor GJ. Unpleasant taste – a neglected symptom in depression. *J Affective Disorders* 1989; 17:291-293.
- Milosevic A, Slade PD. The orodental status of anorexics and bulimics. *Br Dent J* 1989; 167:66-70.

- Mock D, Frydman W, Gordon AS. Atypical facial pain: A retrospective study. *Oral Surg Oral Med Oral Pathol* 1985; 59:472-474.
- Molin C, Levi L. A psycho-odontologic investigation of patients with bruxism. *Acta Odont Scand* 1966; 24:25-387.
- Molony H. Mental retardation. In: Beumont PJV, Hampshire RB eds. *Textbook of Psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:270-279.
- Mörnstad H, Von Knorring L, Forsgren L, Holmgren S. Acute effects of some different antidepressant drugs on saliva composition. *Neuropsychobiology* 1986; 15:73-79.
- Moulton RE. Emotional factors in non-organic temporomandibular joint pain. *Dent Clin North Am* 1966; November:609-620.
- Moulton RE. Psychiatric considerations in maxillofacial pain. *J Am Dent Assoc* 1955; 51:408-414.
- Muniz CE, Berghman DH. Contact stomatitis and lithium carbonate. *J Am Med Assoc* 1978; 239:2759.
- Munro A. Monosymptomatic hypochondriacal psychosis. *Br J Hosp Med* 1980; 24:34-38.
- Myers JK, Weissman MM, Tischler GL, Holzer III CE, Leaf PL, Orvaschel H et al. Six-month prevalence of psychiatric disorders in three communities. *Arch Gen Psychiatry* 1984; 41:959-967.
- Nadler SC. Bruxism, a classification: Critical review. *J Am Dent Assoc* 1957; 54:615-622.
- Nadler SC. Detection and recognition of bruxism. *J Am Dent Assoc* 1960; 61:472-479.
- Nadler SC. The effects of bruxism. *J Periodontol* 1966; 37:311-319.
- Needleman HL. Orofacial trauma in child abuse: Types, prevalence, management, and the dental profession's involvement. *Pediatr Dent* 1986; Special Issue 1:71-79.
- Negus TW, Todd JO. Bulimia nervosa in a male. *Br Dent J* 1986; 160:290-291.

- Nielson AC, Williams TA. Depression in ambulatory medical patients. *Arch Gen Psychiatry* 1980; 37:999-1004.
- Nishino S, Ueno R, Oshishi K, Sakai T, Hayaishi O. Salivary prostaglandin concentration: Possible state indicators for major depression. *Am J Psychiatry* 1989; 146:365-368.
- O'Shea RM, Corah NL, Ayer WA. Sources of dentists' stress. *J Am Dent Assoc* 1984; 109:48-51.
- Oldham L. Facial pain as a presentation in Von Munchausen's Syndrome: A case report. *Br J Oral Surg* 1974; 12:86-90.
- Osborne TE, Grace EG, Schwartz MK. Severe degenerative changes of the temporomandibular joint secondary to the effect of tardive dyskinesia: A literature review and case report. *Cranio* 1989; 7:58-62.
- Palmai G, Blackwell B. The diurnal pattern of salivary flow in normal and depressed patients. *Br J Psychiatry* 1965; 111:334-338.
- Parker G, Boyce P. Personality disorders. In: Beumont PJV, Hampshire RB eds. *Textbook of psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:137.
- Pattison EM, Kahan J. The deliberate self-harm syndrome. *Am J Psych* 1983; 140:867-872.
- Pattison GL. Self-inflicted gingival injuries: Literature review and case report. *J Periodontol* 1983; 54:299-304.
- Paulson GW. Tardive dyskinesia. *Ann Rev Med* 1975; 26:75-81.
- Pavone BW. Bruxism and its effect on the natural teeth. *J Prosthet Dent* 1985; 53:692-696.
- Paykel ES, Mueller PS, De La Vergne PM. Amitriptyline, weight gain and carbohydrate craving: A side effect. *Br J Psychiatry* 1973; 123:501-507.
- Petty F. Southwestern Internal Medicine Conference: Depression and medical illness. *Am J Med Sciences* 1989; 298:59-68

- Pickar D, Davies RK. Tardive dyskinesia in younger patients. *Am J Psychiatry* 1978; 135:385-386.
- Pilling LF. Emotional aspects of prosthodontic patients. *J Prosthet Dent* 1973; 30:514-515.
- Pilowsky I. A general classification of abnormal illness behaviours. *Br J Med Psychol* 1978; 51:131-137.
- Pilowsky I. Primary and secondary hypochondriasis. *Acta Psychiatrica Scandinavica* 1970; 46:273-285.
- Pitts WC. Difficult denture patients: Observations and hypothesis. *J Prosthet Dent* 1985; 53:532-534.
- Plainfield S. Psychological considerations in prosthetic dentistry. *Dent Clin North Am* 1962; November:669-681.
- Plessett DN. Auto-extraction. *Oral Surg Oral Med Oral Pathol* 1959; 12:302-303.
- Pollack B, Buck IF, Kalnins L. An oral syndrome complicating psychopharmacotherapy: Study II. *Am J Psychiatry* 1964; 121:384-386.
- Poporad GA, Kaye D. Diseases of presumed microbial origin. In: Rose LF, Kaye D eds. *Internal medicine for dentistry*. 1st edn. Missouri: CV Mosby Company 1983:302.
- Quitkin F, Rifkin A, Gochfeld L, Klein DF. Tardive dyskinesia: Are first signs reversible? *Am J Psychiatry* 1977; 134:84-87.
- Quock RM. Clinical complications in the psychiatric dental patient. *Compend Cont Educ* 1985; VI:333-345.
- Ramfjord SP. Bruxism, a clinical and electromyographic study. *J Am Dent Assoc* 1961; 62:21-44.
- Ranga Rama Krishnan K, France RD, Pelton S, McCann UD, Davidson J, Urban . Chronic pain and depression I. Classification of depression in chronic low back pain patients. *Pain* 1985a; 22:279-287.
- Ranga Rama Krishnan K, France RD, Pelton S, McCann UD, Davidson J, Urban . Chronic pain and depression II. Symptoms of anxiety in chronic low back pain

- patients and their relationship to subtypes of depression. *Pain* 1985b; 22:289-294.
- Rankin JA, Harris MB. Dental anxiety: The patient's point of view. *J Am Dent Assoc* 1984; 109:43-47.
- Reading A, Raw M. The treatment of mandibular dysfunction pain. *Br Dent J* 1976; 140:201-205.
- Reding GR, Rubright WC, Rechtschaffen A, Daniels RS. Relation between bruxism and dreaming. *Dent Abstracts* 1964; 9:707.
- Reding GR, Rubright WC, Zimmerman SO. Incidence of bruxism. *J Dent Res* 1966; 45:1198-1204.
- Reed B. Hepatitis B Virus carrier groups and the Australian community. In: *The implications of Hepatitis B for dental practice, a MDS Treatise*. Sydney: University of Sydney 1987:242-243.
- Rees RT, Harris M. Atypical odontalgia. *Br J Oral* 1979; 16:212-218.
- Reilly TM, Beard AW. Monosymptomatic hypochondriasis. *Br J Psychiatry* 1976; 129:191-192.
- Remick RA, Blasberg B, Barton JS, Campos PE, Miles JE. Ineffective dental and surgical treatment associated with atypical facial pain. *Oral Surg Oral Med Oral Pathol* 1983b; 55:355-358.
- Remick RA, Blasberg B, Campos PE, Miles JE. Psychiatric disorders associated with atypical facial pain. *Can J Psychiatry* 1983a; 28:178-181.
- Remick RA, Fine SH. Antipsychotic drugs and seizures. *J Clin Psychiatry* 1979; 40:78-80.
- Rich BM, Kabcenell JL. A new clinical approach to the problem denture patient. *J Prosthet Dent* 1987; 58:451-454.
- Riding BEJ, Munro A. Pimozide in monosymptomatic psychosis. *Lancet* 1975; 1:400-401.
- Robertson DM, Barbor P, Hull D. Unusual injury? Recent injury in normal children with suspected non-accidental injury. *Br Med J* 1982; 285:1399-1401.

- Roberts MW, Li S-H. Oral findings in anorexia nervosa and bulimia nervosa: A study of 47 cases. *J Am Dent Assoc* 1987; 115:407-410.
- Robinson JE, Reding GR, Zepelin H, Smith VH, Zimmerman SO, Nocturnal teeth-grinding: A reassessment for dentistry. *J Am Dent Assoc* 1969; 78:1308-1311.
- Rosenbaum C, McDonald RE, Levitt EE. Occlusion of cerebral palsied children. *J Dent Res* 1966; 45:1696-1700.
- Roxburgh PA. Treatment of phenothiazine-induced oral dyskinesia. *Br J Psychiatry* 1970; 116:277-280.
- Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. *J Prosthet Dent* 1984; 51:548-553.
- Rundegren J, Van Dijken J, Mörnstad H, Von Knorring L. Oral conditions in patients receiving long term treatment with cyclic antidepressant drugs. *Swed Dent J* 1985; 9:55-64.
- Russ MJ, Ackerman SH. Salivation and depression: A role for appetitive factors. *Appetite* 1987; 8:37-47.
- Russell J. Part 2. Classification in psychiatry. In: Beumont PJV, Hampshire RB eds. *Textbook of psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:8-17.
- Sandlin PD. Anatomy of anxiety - a physician's overview. *Dent Clin N Am* 1987; 31:1-9.
- Schizophrenia Australia. Melbourne: Produced by the Board of Management of Schizophrenia Australia 1986; March.
- Schizophrenia Fellowship of New Zealand and New South Wales Association for Mental Health. About schizophrenia. Sydney: Health Information and Translation Services. Department of Health, N.S.W. State Health Publication No (HP)85-003.
- Schmahl SR. Depression in the geriatric population - implications for the dental practitioner. *Spec Care Dent* 1984; 4:16-18.
- Schmitt BD. Physical abuse: Specifics of clinical diagnosis. *Pediatr Dent* 1986b; Special Issue 1:83-87.

- Schmitt BD. Types of child abuse and neglect: An overview for dentists. *Pediatr Dent* 1986a; Special Issue 1:67-70.
- Schuurs AHB, Düvenvoorden HJ, Van Velzen SKT, Verhage F, Eijkman MAJ, Makkes PC. Sociodemographic correlates of dental anxiety. *Community Dent Oral Epidemiol* 1985; 13:212-215.
- Scott DS, Hirschman R. Psychological aspects of dental anxiety in adults. *J Am Dent Assoc* 1982; 104:27-31.
- Scott J, Humphreys M. Psychiatric aspects of dentistry 1. *Br Dent J* 1987; 163:81-84.
- Scott PD. Non-accidental injury in children. *Br J Psychiatry* 1977; 131:366-380.
- Scully C, Cawson RA. Psychiatric disease. In: *Medical problems in dentistry*. 2nd edn. Wright I.O.P. Publishing Ltd 1987:379-405.
- Seligman DA, Pullinger AG, Solberg WK. The prevalence of dental attrition and its association with factors of age, gender, occlusion and TMJ symptomatology. *J Dent Res* 1988; 67:1323-1333.
- Shear CS, Nyhan WL, Kirman BH, Stern J. Self-mutilative behaviour as a feature of the de Lange syndrome. *J Pediatrics* 1971; 78:506-509.
- Shee JC. Dangerous potentiation of pethidine by iproniazid and its treatment. *Br Med J* 1960; 2:507-509.
- Shepherd, Cooper, Brown. Minor mental illness in London - some aspects of a general practice survey. *Br Med J* 1984; 2:1359-1363.
- Shira RB, Bernier JL, Cahn LR. Clinical Oral Pathology Conference Proceedings. *Oral Surg Oral Med Oral Pathol* 1959; 12:36-61.
- Shore D, Anderson DJ, Cutler NR. Prediction of self-mutilation in hospitalized schizophrenics. *Am J Psych* 1978; 135:1406-1407.
- Silverman M, Perkins RL. Bilateral parotid enlargement and starch ingestion. *Annals Int Med* 1966; 64:842-846.
- Silverman S, Silverman SI, Silverman B, Garfinkel L. Self-image and its relation to denture acceptance. *J Prosthet Dent* 1976; 35:131-141.

- Simmons MS, Grayden SK, Mitchell JE. The need for psychiatric-dental liaison in the treatment of bulimia. *Am J Psychiatry* 1986; 143:783-784.
- Simpson MA. Self-mutilation. *Br J Hosp Med* 1976; 16:430-438.
- Sims APT. Non-accidental injury in the child presenting as a suspected fracture of the zygomatic arch. *Br Dent J* 1985; 158:292-293.
- Sinick D. Psychologic factors in dental treatment. *J Prosthet Dent* 1964; 14:506-513.
- Slawson PF, Davidson PW. Hysterical self-mutilation of the tongue. A case study. *Arch Gen Psychiatry* 1964; 11:581-588.
- Smith DP, Pilling LF, Pearson JS, Rushton JG, Goldstein NP, Gibilisco JA. A psychiatric study of atypical facial pain. *Can Med Assoc J* 1969; 100:286-291.
- Smith JM, Baldessarini RJ. Changes in prevalence, severity and recovery in tardive dyskinesia with age. *Arch Gen Psychiatry* 1980; 37:1368-1373.
- Smith M, Greener A, The Sydney Manic-depressive Self-help Group, Boyce P, Reeves E, The NSW Association for Mental Health, Johnson G. Living with mood swings. Manic-depressive illness. Sydney: Health Information and Translation Services. Department of Health, N.S.W. State Health Publication No (HP)86-027.
- Smith M. Measurement of personality traits and their relation to patient satisfaction with complete dentures. *J Prosthet Dent* 1976; 35:492-503.
- Smith SM, Hanson R, Noble S. Parents of battered babies: A controlled study. *Br Med J* 1973; 4:388-391.
- Smith T, Getz T, Milgrom P, Weinstein P. Evaluation of treatment at a dental fears research clinic. *Spec Care Dent* 1987; 7:130-134.
- Smith TA, Kroeger RF, Lyon HE, Mullins MR. Evaluating a behavioural method to manage dental fear: A 2-year study of dental practices. *J Am Dent Assoc* 1990; 121:525-530.
- Sneddon I, Sneddon J. Self-inflicted injury: A follow-up study of 43 patients. *Br Med J* 1975; iii:527-530.
- Sobel RS. Child abuse: Case report. *Pediatr Dent* 1986; Special Issue 1:93-95.

- Sohlberg S. Personality, life stress and the course of eating disorders. *Acta Psychiatrica Scand* 1990; 82 Suppl 361:29-33.
- Solberg WK, Flint RT, Brantner JP. Temperomandibular joint pain and dysfunction: A clinical study of emotional and occlusal components. *J Prosthet Dent* 1972; 28:412-422.
- Sopher IM. The dentist and the battered child syndrome. *Dent Clin North Am* 1977; 21:113-122.
- Sosnow I. The emotional significance of the loss of teeth. *Dent Clin North Am* 1962; November:637-650.
- Speculand B, Goss AN, Hughes A, Spence ND, Pilowsky I. Tempero-mandibular joint dysfunction: Pain and illness behaviour. *Pain* 1983; 17:139-150.
- Speculand B, Hughes AO, Goss AN. Role of recent stressful life events experience in the onset of TMJ dysfunction pain. *Community Dent Oral Epidemiol* 1984; 12:197-202.
- Sreebny LM, Schwartz SS. A reference guide to drugs and dry mouth. *Gerodontology* 1986; 5:75-99.
- Sreebny LM, Valdini A. Xerostomia, a neglected symptom. *Arch Int Med* 1987; 147:1333-1337.
- Sreebny LM, Valdini A. Xerostomia. Part I: Relationship to other oral symptoms and salivary gland hypofunction. *Oral Surg Oral Med Oral Pathol* 1988; 66:451-458.
- Sreebny LM. Salivary flow in health and disease. *Compend Contin Educ Dent* 1989, Suppl No. 13:461-469.
- Stege P, Visco-Dangler L, Rye L. Anorexia nervosa: Review including oral and dental manifestation. *J Am Dent Assoc* 1982; 104:648-652.
- Stevens JB, Wilkinson EG. Drugs, dry mouth and dental disease. A case report. *Psychosomatics* 1971; 12:310-312.
- Stewart DJ, Kernohan DC. Self-inflicted gingival injuries. Gingivitis artefacta, factitial gingivitis. *Dent Pract* 1972; 22:418-426.

- Stewart DJ. Minor self-inflicted injuries to the gingivae. Gingivitis artefacta minor. J Clin Periodontol 1976; 3:128-132.
- Stiefel DJ, Truelove EL, Menard TW, Anderson VK, Doyle PE, Mandel LS. A comparison of the oral health of persons with and without chronic mental illness in community settings. Spec Care Dent 1990; 10:6-12.
- Stouthard MEA, Hoogstraten J. Dental anxiety: A comparison of dentate and edentulous subjects. Community Dent Oral Epidemiol 1990; 18:267-268.
- Svirsky JA, Sawyer DR. Dermatitis artefacta of the paraoral region. Oral Surg Oral Med Oral Pathol 1987; 64:259-263.
- Swoope CC. Identification and management of emotional patients. J Prosthet Dent 1972; 27:434-439.
- Swoope CC. Predicting denture success. J Prosthet Dent 1973; 30:860-865.
- Tate RJ. Facial injuries associated with the battered child syndrome. Br J Oral Surg 1972; 9:41-45.
- Tenzer JA, Orozco H. Traumatic glossectomy. Report of a case. Oral Surg Oral Med Oral Pathol 1970; 30:182-184.
- Teo CS, Foong W, Lui H, Vignehsa H, Elliott J, Milgrom P. Prevalence of dental fear in young adult Singaporeans. Int Dent J 1990; 40:37-42.
- Tetrault GA, Weintraub M. How to handle drug-related dry mouth. Drug Therapy 1984; Feb:159-166.
- Thaller JL, Rosen G, Saltzman S. Study of the relationship of frustration and anxiety to bruxism. J Periodontol 1967; 38:193-197.
- Thaller JL. The use of the Cornell Index to determine the correlation between bruxism and the anxiety state: A preliminary report. J Periodontol 1960; 31:138-140.
- The Concise Oxford Dictionary 7th edn. Sykes JB ed. 4th Reprint. Oxford: The University Press 1984a:497.
- The Concise Oxford Dictionary 7th edn. Sykes JB ed. 4th Reprint. Oxford: The University Press 1984b:765.

The Concise Oxford Dictionary 7th edn. Sykes JB ed. 4th Reprint. Oxford: The University Press 1984c:736.

The New South Wales Association for Mental Health. Taking the mystery out of mental illness. Sydney: Health Information and Translation Services. Department of Health, N.S.W. State Health Publication No (HP)84023.

The New South Wales Council for Intellectual Disability. Intellectual disability. Some questions and answers. From the media kit "People are People" 1991.

The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee. Introduction. In: Psychotropic drug guidelines. Victoria: Interprint Services Pty Ltd 1989a:1.

The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee. Drugs used in the treatment of affective disorders. In: Psychotropic drug guidelines. Victoria: Interprint Services Pty Ltd 1989b:56-60.

The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee. Drugs used in the treatment of schizophrenia. In: Psychotropic drug guidelines. Victoria: Interprint Services Pty Ltd 1989c:26-29.

The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee. Drugs used in the treatment of affective disorders. In: Psychotropic drug guidelines. Victoria: Interprint Services Pty Ltd 1989d:53.

The Psychotropic Guidelines Sub-committee Victorian Drug Usage Advisory Committee. Drugs used in the treatment of neurotic and sleep disorders. In: Psychotropic drug guidelines. Victoria: Interprint Services Pty Ltd 1989e:78-80

Thomas MR. Factitious illness and the ENT surgeon. J Laryngology Otology 1989; 103:882-884.

Thomson H. Mandibular dysfunction syndrome. Br Dent J 1971; 130:187-193.

Toone BK, Fenton GW. Epileptic seizures induced by psychotropic drugs. Psychological Med 1977; 7:265-270.

- Touyz SW, Beumont PJV. Anorexia and bulimia nervosa, the dieting disorders. In: Beumont PJV, Hampshire RB eds. Textbook of psychiatry 1989 edition. Australia: Blackwell Scientific Publications 1989:119-128.
- Turek I, Kurland AA, Hanlan TE, Bohen M. Tardive dyskinesia: Its relation to neuroleptic and antiparkinson drugs. Br J Psychiatry 1972; 121:605-612.
- Unsworth N. Anxiety. Br Dent J 1984; 156:299-300.
- Van der Ploeg HM, Van der Waal N, Eijkman MAJ, Van der Waal I. Psychological aspects of patients with burning mouth syndrome. Oral Surg Oral Med Oral Pathol 1987; 63:664-668.
- Vervoorn JM, Duinkerke ASH, Luteijn F, Van de Poel ACM. Assessment of dental anxiety in edentulous subjects. Community Dent Oral Epidemiol 1989; 17:177-179.
- Vogan WI. Traumatic gingival recession. A case report. Br Dent J 1969; 127:175-176.
- Von Knorring L, Mörmstad H. Qualitative changes in saliva composition during short term administration with imipramine and zimelidine in healthy volunteers. Scand J Dent Res 1981; 89:313-320.
- Von Knorring AL, Wahlin YB. Tricyclic antidepressants and dental caries in children. Neuropsychobiology 1986; 15:143-145.
- Wagner GN. Bitemark identification in child abuse cases. Pediatr Dent 1986; Special; Issue 1:96-100.
- Walsh JP. The psychogenesis of bruxism. J Periodontol 1965; 36:417-420.
- Walter-Ryan WG, Shiriff JR. Self-mutilation by tooth extraction. South Med J 1985; 78:1519-1520.
- Wardle J. Fear of dentistry. Br J Med Psychology 1982; 55:119-126.
- Waters B, Brennan J. Psychiatric syndromes of childhood. In: Beumont PJV, Hampshire RB eds. Textbook of psychiatry 1989 edition. Australia: Blackwell Scientific Publications 1989:186-197.
- Watson GD, Chandarana PC, Merskey H. Relationships between pain and schizophrenia. Br J Psychiatry 1981; 138:33-36.

- Webb HE, Lascelles RG. Treatment of facial and head pain associated with depression. *Lancet* 1962; 1:355-356.
- White DK, Hayes RC, Benjamin RN. Loss of tooth structure associated with chronic regurgitation and vomiting. *J Am Dent Assoc* 1978; 97:833-835.
- White RT, Beumont PJV. Organic mental syndromes and disorders. Part 1. Delirium, dementia and other organic mental syndromes and disorders. In: Beumont PJV, Hampshire RB eds. *Textbook of psychiatry* 1989 edition. Australia: Blackwell Scientific Publications 1989:49-67.
- Whittle JG, Grant AA, Worthington HV. The dental health of the elderly mentally ill: A preliminary report. *Br Dent J* 1987; 162:381-383.
- Widdop FT. The problem of dry mouth in the elderly. In: Widdop FT ed. *The greying of Australia: Impact on dental services*. Sydney: Australian Dental Association 1991:18-19.
- Wigdórowicz-Makówerówa N, Grodzki C, Maslanka T, Plouka K, Palacha A. Epidemiologic studies on prevalence and etiology of functional disturbances of the masticatory system. *J Prosthet Dent* 1979; 41:76-82.
- Winer JA, Bahn S. Loss of teeth with antidepressant drug therapy. *Arch Gen Psychiatry* 1967; 16:239-240.
- Wolcott RB, Yager N, Gordon G. Dental sequelae to the binge-purge syndrome (bulimia): Report of cases. *J Am Dent Assoc* 1984; 109:723-725.
- Wolf N. Hunger. In: *The Beauty Myth*. Vintage edition. Great Britain: Cox and Wyman Ltd 1991:181-196.
- Woodrow M. Preauricular and head pain caused by bruxism. *Dent Abstracts* 1964; 7:583.
- Woody RC, Eisenhauer G. Tooth extraction as a form of self-mutilation in Tourette's Disorder. *South Med J* 1986; 79:1466.
- World Health Organization. Organization of dental public health services. Report of a WHO Expert Committee on dental health. Geneva: World Health Organization Technical Report Series 1965; 298:4.

- Yesavage JA. Relationships between measures of direct and indirect hostilities and self-destructive behaviour by hospitalised schizophrenics. *Acta Psychiatrica Scand* 1983b; 168:345-350.
- Yesavage JA. Relationships between measures of direct and indirect hostilities and self-destructive behaviour by hospitalised schizophrenics. *Br J Psychiatry* 1983a; 143:173-176.
- Zakrzewska JM, Feinmann C. A standard way to measure pain and psychological morbidity in dental practice. *Br Dent J* 1990; 169:337-339.
- Zegarelli DJ. Burning mouth: An analysis of 57 patients. *Oral Surg Oral Med Oral Pathol* 1984; 59:34-38.
- Zeldow LL. Treating clenching and bruxing by habit change. *J Am Dent Assoc* 1976; 93:31-33.
- Zilli C, Brooke RI, Lau CL, Merskey H. Screening for psychiatric illness in patients with oral dysaesthesia by means of the General Health Questionnaire - twenty-eight item version (GHQ-28) and the Irritability, Depression and Anxiety scale (IDA). *Oral Surg Oral Med Oral Pathol* 1989; 67:384-389.
- Zucker AH. A psychiatric appraisal of tongue symptoms. *J Am Dent Assoc* 1972; 85:649-651.

