BRUXISM

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Dedication

This thesis is dedicated unreservedly to my loving wife Jenepher who has worked with me diligently through the preparation, compilation and processing of this publication. The pain of the past year has been treated with the love of this special lady.
III.

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# TABLE OF CONTENTS

| Title Page | i |
| Dedication | ii |
| Acknowledgements | iii |
| Table of Contents | v |
| List of Tables | viii |
| List of Diagrams | x |
| List of Illustrations | xi |
| Chapter 1. Introduction | 1 |
| Chapter 2. History | 6 |
| Chapter 3. Incidence | 10 |
| Chapter 4. Aetiology | 14 |
| A. Psychic Factors | 18 |
| B. Occlusal Factors | 23 |
| C. Hereditary Factors | 29 |
| D. Allergy | 30 |
| E. Autonomic Nervous System | 31 |
| F. Pain | 32 |
| G. Other | 34 |
| Discussion | 34 |
| Chapter 5. Diagnosis of Bruxism | 39 |
| Differential Diagnosis | 48 |
| A. Attrition | 50 |
| B. Bruxism | 51 |
| C. Abrasion | 51 |
Table of Contents (cont.)

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. Erosion</td>
<td>52</td>
</tr>
<tr>
<td>E. Combination Lesions</td>
<td>52</td>
</tr>
<tr>
<td>F. The Anorexia Nervosa Sydrome</td>
<td>53</td>
</tr>
<tr>
<td>Discussion</td>
<td>61</td>
</tr>
</tbody>
</table>

Chapter 6. Bruxism as a Component of Functional Disturbances of Temporomandibular Joints and Muscles 68

Chapter 7. The Effect of Bruxism on the Temporomandibular Joint and Muscles 79

A. Muscles                                                            80
B. Temporomandibular Joints                                           84
C. Headaches                                                         86

Chapter 8. The Effect of Bruxism on the Teeth 91

A. Hard Tooth Structure                                                92
B. Fractures of Teeth and Restorations                                95
C. Dental Pulp                                                        97
D. Root Changes                                                       99
E. Endodontic Treatment                                               100
F. Restorative Materials                                              101
G. Wear                                                               109
H. Dentures                                                          117

Chapter 9. The Effect of Bruxism on the Periodontium 121

Chapter 10. Treatment of Bruxism                                       135

A. Occlusal Splints                                                   138
B. Occlusal Treatment                                                  143
C. Occlusal Reconstruction and Prostheses                              144
D. Post Treatment Splints                                              147
E. Adjunctive Therapy to Occlusal Treatment                            148
F. Psychotherapy                                                      153
<table>
<thead>
<tr>
<th>Table of Contents (Cont.)</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>G. Biofeedback</td>
<td>156</td>
</tr>
<tr>
<td>H. Massed Practice Exercises</td>
<td>158</td>
</tr>
<tr>
<td>I. Habit Change</td>
<td>158</td>
</tr>
<tr>
<td>J. Drugs</td>
<td>161</td>
</tr>
<tr>
<td>K. Adjunctive Therapy</td>
<td>161</td>
</tr>
<tr>
<td>L. Other Factors</td>
<td>162</td>
</tr>
<tr>
<td>Discussion</td>
<td>162</td>
</tr>
<tr>
<td>Chapter 11. Discussion and Conclusion</td>
<td>164</td>
</tr>
<tr>
<td>List of Conclusions</td>
<td>167</td>
</tr>
<tr>
<td>Bibliography</td>
<td>172</td>
</tr>
</tbody>
</table>
LIST OF TABLES

Table I. Aetiology of Bruxism
   Table of Other Factors 35a

Table II. Aetiology of Bruxism 38

Table III. Nadler Case History 54

Table IV. Differential Diagnosis of Bruxism
   Compared To Other Conditions That
   Effect Hard Tooth Structure Excluding
   Caries And Restorative Dentistry 64

Table V. I. Ultimate Strength of Dental Materials 106
   II. Knoop Hardness of Dental Materials 107

Table VI. I. Factors Increasing Wear 113
   II. The Relative Wear of Gold Against
       Listed Materials. 113
   III. Relative Wear of Porcelain Against
        Listed Materials 114
   IV. Comparison of Total Wear of Acrylic
       Resin in Opposition to Other Materials 114
   V. Relative Wear Rate of Like Materials 115
   VI. Clinical Comparison of Amalgam and
Table VI. (cont)  Composite in Class I. and Class II. Restorations at Three Years. 115
<table>
<thead>
<tr>
<th>Diagram</th>
<th>Aetiology of Bruxism.</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>The Ramfjord Model.</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Aetiology of Bruxism.</td>
</tr>
<tr>
<td></td>
<td>Autonomic Nervous System.</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>Aetiology of Bruxism.</td>
</tr>
<tr>
<td></td>
<td>Pain Component.</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Aetiology of Bruxism.</td>
</tr>
<tr>
<td></td>
<td>Other Factor Component.</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>Aetiology of M.P.D. Syndrome.</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>VI</td>
<td>Muscular Reactions to Psychological and Physical Stress.</td>
</tr>
</tbody>
</table>

Page

16

32

33

34

73

83
## LIST OF ILLUSTRATIONS

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1</td>
<td>The clinical picture of a bruxer.</td>
<td>26</td>
</tr>
<tr>
<td>Figure 2</td>
<td>Models of patient in Figure 1.</td>
<td>26</td>
</tr>
<tr>
<td>Figure 3</td>
<td>A closer view of the lower model.</td>
<td>27</td>
</tr>
<tr>
<td>Figure 4</td>
<td>Post adjustment tooth contact position in a 19 year old female.</td>
<td>28</td>
</tr>
<tr>
<td>Figure 5</td>
<td>Pattern faceting in a young female bruxer.</td>
<td>45</td>
</tr>
<tr>
<td>Figure 6</td>
<td>Mandibular excursion to engage bruxo-contact.</td>
<td>45</td>
</tr>
<tr>
<td>Figure 7</td>
<td>The contact position.</td>
<td>46</td>
</tr>
<tr>
<td>Figure 8</td>
<td>Bruxism: Open position.</td>
<td>49</td>
</tr>
<tr>
<td>Figure 9</td>
<td>Bruxism: Closed position.</td>
<td>49</td>
</tr>
<tr>
<td>Figure 10</td>
<td>Gross upper lingual bruxoabrasion.</td>
<td>56</td>
</tr>
<tr>
<td>Figure 11</td>
<td>Gross lower incisal bruxoabrasion.</td>
<td>56</td>
</tr>
<tr>
<td>Figure 12</td>
<td>Clinical view of bruxoabrasion.</td>
<td>57</td>
</tr>
<tr>
<td>Figure 13</td>
<td>Lower arch with confirmed bruxism and erosion.</td>
<td>58</td>
</tr>
<tr>
<td>Figure 14</td>
<td>Close up of arch in Figure 13.</td>
<td>58</td>
</tr>
<tr>
<td>Figure 15</td>
<td>Combined bruxo-erosion-abrasion facet.</td>
<td>59</td>
</tr>
<tr>
<td>Figure 16</td>
<td>Engagement of upper cusp and lower lateral incisor in bruxism.</td>
<td>77</td>
</tr>
<tr>
<td>Figure 17</td>
<td>Bruxo-engagement.</td>
<td>77</td>
</tr>
<tr>
<td>Figure 18</td>
<td>Gross disarticulation of the dentition.</td>
<td>78</td>
</tr>
<tr>
<td>Figure 19</td>
<td>Frontal view of mounted study models.</td>
<td>88</td>
</tr>
<tr>
<td>Figure 20</td>
<td>Frontal view of mounted study models.</td>
<td>88</td>
</tr>
<tr>
<td>Figure 21</td>
<td>Right lateral view of mounted study models.</td>
<td>89</td>
</tr>
<tr>
<td>Figure 22</td>
<td>Left lateral view of mounted study models.</td>
<td>89</td>
</tr>
<tr>
<td>Figure 23</td>
<td>Right condylar element.</td>
<td>90</td>
</tr>
<tr>
<td>Figure 24.</td>
<td>Bruxo-damage 60 year old male.</td>
<td>96</td>
</tr>
<tr>
<td>Figure 25.</td>
<td>Bruxo-damage 60 year old male.</td>
<td>96</td>
</tr>
<tr>
<td>Figure 26.</td>
<td>The enamel fracture of an over restored bicuspid.</td>
<td>98</td>
</tr>
<tr>
<td>Figure 27.</td>
<td>Cuspid to cuspid bruxism.</td>
<td>102</td>
</tr>
<tr>
<td>Figure 28.</td>
<td>Bruxo-engagement of cuspids.</td>
<td>102</td>
</tr>
<tr>
<td>Figure 29.</td>
<td>Precision grinding of inlay and tooth.</td>
<td>103</td>
</tr>
<tr>
<td>Figure 30.</td>
<td>Occlusal Splint.</td>
<td>139</td>
</tr>
<tr>
<td>Figure 31.</td>
<td>Occlusal Splint.</td>
<td>139</td>
</tr>
<tr>
<td>Figure 32.</td>
<td>Right lateral view of mounted study casts.</td>
<td>149</td>
</tr>
<tr>
<td>Figure 33.</td>
<td>Left lateral view of mounted study casts.</td>
<td>149</td>
</tr>
<tr>
<td>Figure 34.</td>
<td>Right lateral view of mounted diagnostic casts.</td>
<td>150</td>
</tr>
<tr>
<td>Figure 35.</td>
<td>Left lateral view of mounted diagnostic casts.</td>
<td>150</td>
</tr>
<tr>
<td>Figure 36.</td>
<td>Left view finished case.</td>
<td>151</td>
</tr>
<tr>
<td>Figure 37.</td>
<td>Right view finished case.</td>
<td>151</td>
</tr>
<tr>
<td>Figure 38.</td>
<td>Anterior view finished case.</td>
<td>152</td>
</tr>
</tbody>
</table>
INTRODUCTION
INTRODUCTION

While mastication and swallowing are now proven to involve tooth contacts and stresses upon the supporting structures of the teeth, the forces that have been thought to be the most damaging are those associated with parafunctions. "Bruxism" is the most common parafunction. The term "parafunction" was introduced by Drum to suggest a distinction between:

a. Occlusal stress exerted during mastication and swallowing, i.e. for normal functions of the masticatory system and,

b. Occlusal stresses which are brought into action outside of the normal function.

The former group of activities are called "functions"; the latter group are called "parafunctions". It is suggested by Drum to divide parafunction into the following five groups.

1. **Psyche motivated parafunctions**: This term coincides with the term Karolyi-effect bruxism and bruxomania and means more or less neurotic symptoms.

2. **Stress motivated parafunctions** are normal but exaggerated reactions to "stress", found for instance in soldiers in battle, sportsmen in races, truck drivers, workers on high buildings. etc.
3. Habitual parafunctions are bad habits generally due to the profession or trade of the person.

4. Endogenous parafunction may be caused by systemic diseases such as epilepsy, tetanus, meningitis and other infection.

5. Excessive-compensating parafunctions are involuntarily and unconsciously exaggerated reactions to occlusal interference and to disturbances of various kinds. This locally started muscle activity should be treated by selective grinding or correction of the disturbance.

In this context of "function" compared to "parafunction" Graf demonstrated that with regard to the magnitude of applied occlusal forces and their duration and direction, that mastication and deglutition provide the necessary functional stimuli for the maintenance of a healthy state of the components of the masticatory system, including the periodontal structures. Parafunctions, especially bruxism, on the other hand, can produce additional stress on the components that may be tolerated to a great extent by healthy tissues. However, due to the different characteristics of stress they produce, they may constitute a higher potential for occlusal trauma than normal functions.

In reviewing the literature, the most popularly accepted definitions of bruxism are provided by Ramfjord and Ash and Nadler. Ramfjord and Ash define bruxism as a "gnashing and
grinding of the teeth for non functional purposes" and when this is performed in eccentric excursions they term it eccentric bruxism and when it occurs in the form of clenching in centric they term it centric bruxism. Nadler in a series of publications over the years\textsuperscript{5,6,7,8,9} concerning bruxism, has defined the term fully. In the most recent publication,\textsuperscript{5} bruxism is defined as a non functional, voluntary or involuntary, mandibular movement which may occur during the day or night. It is manifested by occasional or habitual grinding, clenching or clicking of the teeth. Causes of bruxism may be local, systemic, psychological, occupational and, in children, developmental. However, this classification is not a rigid one and causes at times do overlap, such as for example, from occupational to psychological. Bruxism is a mandibular movement originating from the physiologic rest position, or from centric occlusion, to a contact position which may be momentarily maintained or continued into a modification of the original contact. There are two variations of the grinding habit.

1. The clenching habit in which pressures are brought upon the periodontal structures by the repeated clamping of the teeth.

2. The clicking habit, which is usually a rapid, repeated, rhythmic tapping of the teeth.

Bruxism is not always a single activity but may at times be performed as a combination movement. Thus, there may be a form of bruxism which is not purely a grinding or clenching but a combination
of both. In Nadler's opinion, except for early infancy, bruxism may occur virtually at any age and is almost universal in its incidence. Contrasting the different types of bruxism, clenching, because of the forceful sustained biting pressure may produce severe alveolar bone destruction. In clicking, because the pressures are not sustained, the effects are mild. Grinding, because it abrades as well as excerts pressure, may produce attrition and/or alveolar bone loss.

In so defining the terms of this review and differentiating between function and parafunction, Posselt's comments at Panel Discussion on Parafunction of the Masticatory System in Jan. 1960 still apply. "The way in which patients use their occlusion is more important than occlusion or malocclusion itself. Chewing and swallowing are carried out for a relatively short time and are protected by neuro-muscular mechanism whereas bruxism may be carried out for a long time and may not be protected by such mechanisms."

The following is a review of the literature with the author's observations and clinical results included in the substance of the text.
CHAPTER 2

HISTORY
HISTORY

The term bruxism is derived from the French "La bruxomanie" in 1907 and 1908, suggested by Marie and Pietkiewicz\textsuperscript{10} to designate the habit of gritting the teeth, which was considered to be caused by certain cortical brain lesions and, occasionally, by disturbances in the medulla and pons. This form of grinding habit in chronic cortical lesions was contrasted with that observed in nephritis and in cerebral diseases such as meningitis. Bruxomania was observed in cases of paralyses, spasmodic hemiplegia of infancy, dementia, epilepsy, chorea and in cerebral spastic infantile paralyses.

However, the problem of tooth gnashing has been known since ancient times, having been mentioned in the Old Testament. Through the ages, bruxism is indicated in literature. References have been made to the grinding habits as related to emotion, including Darwin in 1898.\textsuperscript{11} The term "bruxomania" was changed to bruxism by Frohman\textsuperscript{12} in 1931. Although Karolyi\textsuperscript{13} did not introduce the term "bruxism", he introduced most of the current concepts of this condition in 1901. He did, however, introduce the term "neuralgia traumatica" in 1906\textsuperscript{14} and out of respect of Karolyi's historical significance the term "Karolyi effect" was introduced.

Quedenfelt\textsuperscript{15} (1908) reported on 100 cases diagnosed as pyorrhea alveolares, found that 60 per cent were the result of malocclusion. He declared that certain habits, such as biting thread, holding a
pipe, finger nail biting and grinding the teeth at night, were some of the causes producing this malocclusion. Tishler\textsuperscript{16} (1928) defined this condition as "occlusal habit neuroses". Miller\textsuperscript{17} (1936) suggested differentiating between nocturnal grinding of the teeth which he called "bruxism", and the habitual grinding of the teeth in the daytime, which he called "bruxomania".

The historical evolution of the current concepts of bruxism is intimately associated with the evolutionary understanding of the pain dysfunction syndromes, because of the active involvement of bruxism in the aetiology of these syndromes. Therefore, mention should be made of the influence of Costen\textsuperscript{18} who took a conglomeration of symptoms, some unrelated clinically, some unrelated anatomically, and developed what was to become known as Costen's syndrome. In the 1940's Henry Beyson\textsuperscript{19} investigated attrition in the Australian Aborigine, and Boyens\textsuperscript{20} and Leof\textsuperscript{21} reported on the significance of bruxism in periodontal disease. In 1950 Bundgaard-Jorgensen\textsuperscript{22} reported a bruxism incidence of 88% in periodontal patients, one of the first studies to hint at the universal incidence of this condition. During the 1950's Posselt\textsuperscript{23} Brill\textsuperscript{24} and Lammie\textsuperscript{25} reported on the structure and function of the masticatory apparatus and the influence of "occlusal habits". During this period also Schwartz\textsuperscript{26} in 1955 reported that he was able to delineate "TMJ syndrome" patients whose symptoms appeared to originate on the mandibular musculature. This was a key development after the Costen Syndrome had been laid to rest by Sicher\textsuperscript{27} and Zimmerman\textsuperscript{28}.
In 1960 the Symposium on Parafunction was chaired by Posselt and in 1961 Ramford published his key E.M.G. study on bruxism. Also in 1960 Janet Travell published an article on the occurrence and treatment of muscle pain of the head and neck. These studies are of major significance in the evolution of the pain dysfunction syndromes and the involvement of bruxism in their pathogenesis. In 1962 Walter Drum published his concept of the auto destruction theory of periodontal disease. Finally in 1969 Laskin published his hypothesis on the Myo-fascial Pain Dysfunction Syndrome.

As can be seen from this historical perspective, the development of investigations and hypotheses is varied and intricate, such that, the concepts of bruxism in the 1980's are still evolutionary and complex.
CHAPTER 3

INCIDENCE
INCIDENCE

The incidence of bruxism is difficult to determine since the criteria used in diagnosing it varies. Estimates vary from about 15% based on subject awareness, to 85% based on occlusal signs.¹ Karolyi's³³ original postulation that all humans engage in bruxism at some stage in their life is difficult to prove or disprove, but many eminent workers in the field support Karolyi's hypothesis. Nadler⁶ stated his contention that bruxism is practically universal. This fact makes bruxism important. Bruxism occurs practically at all ages and may be as old as man himself. Everyone manifests bruxism, or has manifested bruxism, in one form or several forms. Drum³¹ presenting his concept of autodestruction stated that all persons afflicted by periodontal diseases exert parafunctions.

Beyron² stated that one way to diagnose the occurrence of parafunctions is to ask the patient, or members of the patients family if the patient is exerting any bruxism. This is, however, very subjective and the results gained from such subjective questioning are not too valid. Another method is to examine for atypical attrition facets. In reviewing the literature on the evidence of bruxism, the great diversity of results is found to be directly related to the method of diagnosis.

As previously mentioned, early studies of the incidence of bruxism with periodontal disease revealed a reasonably high incidence. Boyens²⁰ in 1940 reported a 78% incidence; Leof²¹ in
1944, 81%; Bundgaard-Jorgensen in 1950, 88%. A study was published in 1966 by Reding, Rubright and Zimmerman. They conducted a questionnaire survey among students of the University of Chicago Laboratory school and among all new undergraduate and graduate students (2,290). Their results were 5.1% for the university students and 15.1% for the laboratory students.

Berit Lindqvist in a series of articles on bruxism in children reported in his first paper that 47% of the children examined had atypical facets while the subjective information supplied by the parents in relation to audible grinding was 15% for grinding and 1% for clenching. In another subjective study Helkimo in 1974 reported a 42% incidence of parafunction (all types). Of the bruxers in his group 65% were aware of diurnal bruxism and 23%, nocturnal bruxism.

More recently in 1981 Nilner and Lessing noted on a random selection of school children and noted a 77% incidence of all parafunctions and Nilner on a random selection of teenagers, 74%.

There has been no study on the incidence or epidemiology of bruxism that would satisfy a realistic quantitative assessment and the studies published to date have to be interpreted with the individual prejudice of the clinician. With this in mind, Karolyi's original contention, as mentioned at the beginning of this section is also endorsed in the 1983 edition of Ramfjord and Ash when they close their newly added section "Epidemiology of Bruxism" thus: "Karolyi's suggestion in 1902 that practically all persons may at one
time or another grind their teeth may not be far from the truth."
CHAPTER 4

AETIOLOGY

A. PSYCHIC FACTORS
B. OCCLUSAL FACTORS
C. HEREDITARY FACTORS
D. ALLERGY
E. AUTONOMIC NERVOUS SYSTEM
F. PAIN
G. OTHER
DISCUSSION
AETIOLOGY

"Most of the aetiological theories advanced are nothing but classifications of the syndrome. It is not known why human beings clench and grind their teeth." Olkinuora 1969.44

"Bruxism may be considered a common phenomenon in normal subjects, even if it may be more common in neurotic patients." Clemmensen 1960.2

These two quotations give some expression to the difficulty of adequately presenting the aetiology of bruxism. Is it possible to have any insight whatsoever regarding the etiology of this phenomenon and, further, is bruxism a normal or abnormal condition?

All discussion in aetiology should begin with Ramfjords article "Bruxism a Clinical and Electromyographic Study"29 of 1961. In discussing aetiology he cited that great emphasis has always been placed on the psychic component in the aetiology of bruxism by past workers, but others including Tishler16 and Karolyi18 had suggested a relationship between occlusal interferences and neurotic conditions. He then further mentioned, that some articles stress the psychic component while other articles stress the occlusal component. Twenty years later, what has changed?

Ramfjord's experiment29 involved a combined clinical and electromyographic study of bruxism in 34 adults, before and after occlusal adjustment. The neuromuscular mechanism of bruxism is
explained on the basis of an intimate relationship between occlusal interferences and psychic tension. In other words, when these two factors are present, regardless of the proportion or dominance of each particular factor in the organism at any one time, bruxism can be initiated.

![Diagram](image)

**DIAGRAM I**

This hypothesis has stood the test of time and is a key hypothesis in the sense that it crystallised the thinking and research up to that time and has provided a model for research to work against since. With the passage of time through to the eighties this hypothesis has been under constant challenge, but it is still standing. Perhaps its simplistic theme will not remain as further factors are inserted into the formula but it would appear, at this point in time, that the equation will remain.

The problem with much of the literature is the stressing of one factor in the causology and this is done particularly by the "central
initiation school. According to this school, psychic tension alone can induce bruxism and the theme is fueled by evidence of continued bruxism after occlusal equilibration or occlusal rehabilitation. Going back to the Ramfjord model, there is a relationship between peripheral and central factors, regardless of the proportion or dominance of each particular factor.

Rugh and Solberg discussed the psychological implications in temporomandibular joint dysfunction stress the principle of "multicausology" whereby several interlocking factors act upon the target organ at the same time, as being more appropriate in understanding aetiology and developing effective treatment strategies. Therefore the central question is not which factor is involved, but how much of each is involved?

This theme has been further developed in stating that the unitary concept (one cause - one disease) must be discarded in favour of the more applicable multifactorial concept (several harmful factors act upon an organ system at the same time). The fact that one agent does not produce active disease is man's strongest protective device keeping him from continual illness. It could perhaps, be extrapolated that if only one cause is obvious, e.g. psychic tension in the occlusally equilibrated bruxer, the other causes await scientific, discovery. But how does this compare to the Ramfjord model? The bruxism pre-existed the occlusal equilibration.

In reviewing the literature on bruxism, almost every article has some comment on aetiology. The majority contain but a small
literature review, which, in the main supports the Ramfjord model. Other principal articles, yet to be mentioned develop a particular theme on aetiology. It is therefore proposed to discuss this important aspect of the concepts of bruxism under theme headings.

A. Psychic Factors

Maintaining Ramfjord's terminology this component of aetiology has been called many things by many authors such as psychological factors, emotional factors, anxiety factors, stress factors, etc, but all will be discussed under this heading. According to Rugh and Solberg, the influence of psychological factors upon jaw function and behaviour has long been recognised. This has gradually led to the development of psycho-physiological and psychosomatic theories which maintain that emotional, behavioural and personality factors are the principal cause of muscle hyperactivity.

Learning principles may by used to understand the development of specific oral habits. Oral habits such as teeth clenching, lip and tongue biting, may be learned through modeling or imitating others, similar to the way in which language is learned in the child. Clenching the masseter musculature and presenting a strong facial expression may be a learned form of non verbal communication.

Whatmore and Kohli have provided a thorough analysis of nonfunctional muscular responses such as bruxism. These authors suggested that responses such as bruxism should be viewed as learned
errors in energy expenditure. The responses are believed to be learned during attempts to cope with or adapt to noxious stimuli which may include social situations, thoughts, emotional arousal, or tissue irritations. They further developed a successful treatment strategy which is directed at retraining the patients musculature response patterns. The patients learn to control muscular activity more effectively through electromyographic evaluation of muscular responses.

Regarding anxiety, it can be assumed that anxiety is causative in bruxism but conversely perhaps bruxism can cause anxiety (Note diagram Ramfjord model).

The factors of anxiety and muscular tension in the etiology of bruxism assumes that emotional states such as anxiety elicit muscular tension. Cameron suggested in 1944 that the types of anxiety patients are distinguishable: those who respond to anxiety with muscular tension, those who respond predominantly with the autonomic nervous system and those who display both muscular and autonomic responses (the aetiology of bruxism has been linked to both muscular hyperactivity and autonomic response). According to Rugh and Solberg, Cameron's ideas have generally proven correct and have led to the concept of "response specificity". Individuals are found to have relatively consistent, unique, physiological response patterns to a variety of stressful situations. For example a "muscle responder" will respond repeatedly with tension in the same set of muscles to a wide range of emotional stimuli. Response specificity does not exclude the possibility of several bodily
responses to stress. It merely specifies that the same set of bodily responses will be repeated regardless of the particular stressor.

The relationship between emotional stress and muscular tension has been observed outside the laboratory setting. Patients were provided with portable EMG devices which signalled the patient when masseter muscular activity exceeded a preset threshold. The patients wore the device during their normal daily activity from 4 - 7 days. All patients were surprised to find how frequently they clenched their teeth without being aware of their behaviour. Each was able to report stressful stimuli which consistently elicited the clenching.

Recent attempts to demonstrate the relationship between stress and bruxism in the natural environment have involved the use of portable EMG recording devices. Using these devices unilateral masseter activity has been recorded on sleeping bruxists for a period of up to three months. The recordings indicate that bruxist behaviour may vary greatly from night to night and is correlated with the previous day's stress level.

In a review of the literature Olkinuora questioned whether bruxists differed from non bruxists as regards to some psychic variable or variables. In a following series of three publications that have been designated the Olkinuora Hypothesis, a comprehensive examination was conducted into the psychosomatic aspect of bruxism with emphasis on mental strain and familiar predisposition factors, a factor analytic study of
psychosocial background in bruxism and psychosocial aspects of bruxists compared to non bruxists.

The research suggested that bruxists may be divided into two categories: 1, nocturnal ("non strain") grinders and 2, diurnal ("strain") clenchers. Nocturnal grinders are "true" bruxists who grind rather than clench. They grind nocturnally, independent of psychologic stress and have a higher incidence of bruxism in relatives. Diurnal clenchers, on the other hand, clench rather than grind. They clench during the day, in response to stress, and have a low rate of bruxism among family members. Further findings were that strain bruxists have significantly more emotional disturbances than non strain bruxists and that strain bruxists are more aggressive than non strain bruxists. When compared to non bruxists, bruxists are emotionally more unbalanced.

In his series of papers on bruxism in children Lindqvist \textsuperscript{35,36,37,38,39} devoted one study \textsuperscript{36} to bruxism and emotional disturbance. The results showed that stress symptoms and nervous disorders occurred more often in children with clinical evidence of bruxism.

Walsh\textsuperscript{55} on the psychogenesis of bruxism reported that bruxism may be part of an obsessional neurosis and related to the primitive idea that the world can be controlled by the mouth. Chronic anxiety states can cause considerable psychological tension and arouse defense mechanism which may be orally centred. Bruxism in adolescents thus corresponds to and may follow, thumb sucking or
nail biting in childhood. It can also be linked with defense mechanisms including auto eroticism and regression to earlier behavioural patterns. Thus the mouth as the first outlet of the infant for emotion develops into oral consciousness in the adult.

Fenichel\textsuperscript{56} considered that physical effects of being dammed up emotionally produce changes in the muscular system, either hypertonic or hypotonic or both alternately. He designated this field as "psychogenic dystonia". Muscular function in the masticatory system is rhythmical. Bruxism is thus a special example of emotional and nervous tensions manifesting themselves through the muscular system.

Thaller et.al.\textsuperscript{57} in 1967 described how mans' existence is governed by two drives essential for survival and development. The primary drive is mandatory for existence and the secondary for progress. If for any reason the fulfillment of these drives is blocked, frustration ensues with some resultant aggression. Frustration is said to occur whenever a behavioural pattern is blocked by; (a) a physical object or the action of a person; (b) a conflicting pattern within an individual; (c) the absence of environmental objects which were anticipated by the individual. It was observed that the oral cavity is of prime significance in the psycho-sexual development and behaviour of the individual. That the oral cavity can be both a source of gratification and/or an outlet for aggression or frustration is easily demonstrated in the child thumbsucking and biting. A psychological examination was performed on 42 known bruxers and 44 non bruxers at New York University College
which established a correlation between the anxiety state, reaction to frustration and bruxism.

B. Occlusal Factors

According to Ramfjord and Ash⁴ in every individual there is a limit for physiologic adaption to imperfection and disharmony in occlusal relationship. When this limit is surpassed, either because of increased occlusal disharmony or increased central nervous system tension, a hypertonic response in the masticatory muscles follows. The hypertonic response may be on the basis of facilitation of nervous impulses of occlusal origin and/or lowered threshold of neuron excitability from nervous tension or pain.

Discomfort from occlusal interferences or pain affects the central nervous system. Ramfjord and Ash⁴ give the example of a patient complaining of the maddening effect of an occlusally hypercontacting restoration. Such central nervous system irritation will lower irritability threshold associated with jaw movements as well as increase the muscle tonus through the fusimotor system directly. Fatigue and subsequent pain from sustained contractions with the jaw muscles also will lower the irritability threshold and enter into the unfavourable "feedback" mechanism.

Ramford and Ash⁴ further stated that some kind of occlusal interference will be found in every patient with bruxism. However it is often extremely difficult to locate occlusal interferences,
especially in the retrusive range in patients with hypertonic jaw muscles and bruxism. In his 1961 paper Ramfjord stated that the occlusal interferences that most trigger bruxism were firstly, those between the most retruded mandibular position and intercuspal position secondly, the balancing side interferences and thirdly, the working side and protrusive interferences. Figs. 1,2,3,4.

In support of this view Peter Dawson stated his clinical experience that bruxism can be stopped by complete elimination of all occlusal interferences. He is so certain of this result that he asks every patient to report any sign of bruxism because it is an indication that the occlusion still needs refining. His contention regarding bruxism and other occlusal treatment is that if you are totally skilled in manipulating the mandible you will firstly find the interferences and secondly remove the interferences. It is alleged that trials on which bruxism continues after occlusal equilibration, the interferences have not been totally found or removed.

In the opinion of the author, it can be reasoned that when bruxism is initiated during sleep two factors come into play regarding occlusal interferences

1. the bruxist is swallowing during sleep and swallowing more slowly than when awake. During swallowing the mandible closes into the retruded contact position. During wakefulness there is muscular accomodation to avoid this occlusal interference but during sleep this protection is abolished or diminished. Hence
the finding by Ramfjord\textsuperscript{29} that this is the most common interference.

2. During sleep stages skeletal muscle passes through cycles from activity to total paralysis. Therefore, depending on the sleeping position the mandible can fall back or to either side. As sleep lightens and mandibular muscle activity returns closure can bring it on to the occlusal interference in the range noted by Ramfjord.

Scharer\textsuperscript{59} in his paper on bruxism in 1974 listed the aetiological factors as nerual, external or internal. Occlusal interferences were grouped with malocclusion, faulty restorations, calculus and periodontics as external factors and it was stated that peripheral oral factors, especially occlusal disharmonies, have therefore to be considered as a main cause in the aetiology of bruxism and are from the dental point of view of primary therapeutic importance.

Lindqvist's\textsuperscript{35,36,37,38,39} series on bruxism in children as well as including one paper\textsuperscript{36} on bruxism and emotional disturbances, also included a paper on occlusal interferences in children with bruxism.\textsuperscript{37} This study showed statistically an almost significantly higher frequency of occlusal interferences in teeth grinders than in non teeth grinders. Occlusal interferences may therefore be of importance in the pathomechanism of teeth grinding.
Figure 1. The clinical picture of a bruxer. The lacework upper incisal line and the broad, flat, highly polished bruxofacets on the lower.

Figure 2. Models of the patient in Figure 1. with the gross bruxofacets outlined. The posterior interferences are emphasised.
Figure 3. A closer view of the lower model.

Figures 1, 2, and 3, demonstrated excessive tooth wear as a result of bruxism in a 42 year old male. Regarding aetiology, it is interesting to note the retruded contact interferences, which have obviously reprogrammed the mandible forward with the resultant gross anterior faceting.
Figure 4. The post-adjustment tooth contact position in a 19 year old female. As in figures 1, 2, and 3, the retruded contact interferences had programmed mandibular contact position forward leading to anterior tooth wear. The physiologic contact position achieved after splint therapy and occlusal adjustment has resulted in an anterior open bite.
Rugh and Solberg\textsuperscript{45} stated that the possibility of an occlusal interference activating bruxism cannot be overlooked. While the differences regarding occlusal interferences in the early bruxist and non bruxist subjects appear to be minimal, the results of occlusal treatment seem to indicate a relationship between occlusion and bruxism.

Budtz-Jorgensen\textsuperscript{60} conducted an experiment on eight Macacca monkeys. The method involved the fitting of gold cap splints, raising the vertical dimension and producing occlusal irritation which did induce bruxism in these animals.

To support the aetiological significance of occlusal factors, Dessner\textsuperscript{2} reported on the fitting of a pseudo-splint to seven occlusal dysfunction pain patients exhibiting headache who were bruxists. The splint consisted of an upper acrylic palate which did not interfere with the occlusion or muscular function at all. The plate was worn nightly for 2 months without improvement in the symptomatology. After this period the patients were issued with an orthodox occlusal splint which eventually cured four, improved one and did not improve one.

C. Hereditary Factors

There is not a great volume of literature on the significance of hereditary factors in the aetiology of bruxism but it would probably
be broadly accepted. Bunting and Hill\textsuperscript{61} in 1940 mentioned a possible association between bruxism and hereditary factors. Abe and Shimakawa\textsuperscript{62} found that 11.6% of a series of 336 three year old children were bruxists. 18.3% of the parents of these children were or had been bruxists. Reding et. al.\textsuperscript{63} in 1966 discovered a statistically significant correlation between bruxism and family history of bruxism. Olkinuora\textsuperscript{52} reported that "strain bruxists" have significantly less bruxism in relatives than "non strain bruxists"

Lindqvist\textsuperscript{35} investigated bruxism in twins. The results showed that monozygotic twins have a statistically significant higher frequency of the same facet patterns than do dizygotic twins. The study supported the hypothesis that hereditary factors are important to the genesis and pattern of bruxism.

D. Allergy

The factor of allergy in the etiology of bruxism does not occupy a paramount position in the literature on bruxism, but nonetheless, what is reported appears valid. Meyer B. Marks\textsuperscript{64} reported on a study conducted in the pediatric and pediatric allergy clinics at Jackson Memorial Hospital in Miami, Florida. In the non allergenic children the incidence was 20%. In a similar group of allergic children
matched by age, sex and colour, it was found that 60% bruxed. Thus, there was a three fold incidence of bruxism in allergic children as compared with those who were apparently non allergic. Marks concluded: Without deprecating other prominently mentioned causes of bruxism, such as psychological influences, occlusal defects and genetic factors, allergic sensitisation must be considered seriously. Intermittent allergic oedema of the Eustachian tube causes changes to occur in the tympanic cavity, reflexly initiating bruxism as a means of obtaining a patent Eustachian tube. Bruxism in allergic persons may have its origin in infancy and early childhood.

E. Autonomic Nervous System

Dubner, Sessle and Storey\(^1\) suggested that bruxism may be associated with sympathetic nervous system overactivity and this was supported by (1973) on the basis of increased heart rate and peripheral vasoconstriction during periods of tooth grinding. Clark and Rugh and Handelman\(^6\) experimented by performing nocturnal EMG recordings of masseter muscle activity on twenty bruxists and ten control subjects. Each subject collected two twenty four hour urine samples. An analysis of urinary catecholamine content was performed. A positive relationship was found between increased adrenalin content and high levels of nocturnal masseter muscle activity and Burch and Abbey\(^6\) tested the effect of blood pressure levels in normotensive and hypertensive subjects. In all groups there was an elevation in blood pressure as a result of the clenching.
There is obviously a relationship between the autonomic nervous system and bruxism but from the evidence that is available it is difficult to determine if it is aetiological, but it could be speculated that the following cycle is appropriate.

![Diagram II]

F. Pain

Pain is a definite factor in the aetiology of bruxism as well as a consequence. Dubner Sessle and Storey\(^1\) conclude that emotional states can lead to increased jaw muscle activity and this increased muscle activity can lead to pain. Pain and occlusal factors can trigger bruxism. Sternbach\(^6\) stated that pain has a reflexive, protective capacity but it is also a set of responses which consists of expressions of anxiety and is a form of stylized interpersonal behaviour. It is rare that any of these aspects are absent in normal pain behaviour. The author has observed the vicious pain cycle when
a patient with an acute periapical condition has suffered pain initiated bruxism. This is further evidenced when the tooth undergoing endodontic therapy remains painful regardless of treatment until occlusally relieved. The relationship of pain in the etiology of bruxism could be represented schematically as follows:

![Diagram III](image-url)
G. Other

This is a very broad group and could be subtitled, "name a cause". All the reported auxillary factors that have been reported as either aetiological or accessory to bruxism have been summarised in Table 1. In the majority of cases bruxism is just a side effect of the condition and not caused by the condition e.g. Marie and Pietkiewicz⁷⁰ reported bruxomania in certain cortical brain lesions. However many of the other factors would fit into the equation:

![Diagram IV](https://example.com/diagram.png)

**DIAGRAM IV**

**Discussion**

The Aetiology of bruxism would be the most controversial aspect of the concepts of bruxism. As previously discussed the Ramfjord model is still the most acceptable and this is generally accepted in the literature even though other factors are incorporated and different emphases reasoned. Scharer⁷⁹ in a key review presents aetiology under three headings (a) Neural (b) External (c) Internal
### Aetiology of Bruxism

**Table of "Other Factors"**

<table>
<thead>
<tr>
<th>Dubner, Sessle &amp; Storey&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Nader&lt;sup&gt;7&lt;/sup&gt;</th>
<th>Ramfjord &amp; Ash&lt;sup&gt;4,43&lt;/sup&gt;</th>
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<td>Brain damaged children (low mental development rather than severity of palsy is significant) amphetamines dopamine precursors – Magnesium deficiency</td>
<td>dentigerous cyst faulty eruption calculus deep overbite gastro intestinal disorder intestinal parasites– hyperacid urine Subclinical nutritional deficiencies relationship between blood sugar, blood calcium and bone calcium allergy and histamines release vesicle irritability endo disorders overactive thyroid brain damage</td>
<td>gingival flaps on third molar gingival hyperplasia periodontal disease surface irregularities on lip cheek tongue TMJ pain Premenstrual Tension traffic stress</td>
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**Scharer<sup>59</sup>**

Subclinical nutritional deficiency calcium deficiency hypovitaminoses gastro-intestinal disturbances blood pressure alteration hereditary factors

**Olkinuora<sup>44</sup>**

gastro-intestinal disorders parasites of intestine nutritional disorders endocrine disturbances allergy brain injury hereditary factors

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Table I
factors. His "neural factors" are quite complimentary to Ramfjords "psychic tension factors" and his "external factors". The "internal factors" are contained within the "other factors" complex.

Nadler has published five papers\textsuperscript{5,6,7,8,9} on bruxism. His first paper classifying aetiology under local causes, systemic causes, psychological causes and occupational causes still fits the above framework. Most research has been structured to examine principally the emotional or the occlusal component or both together with some associated effect. For example Olkinuora\textsuperscript{44,52,53,54} investigated the psychological aspects and considered hereditary factors as well. Lindqvist\textsuperscript{35,36,37,38,39} examined incidence, occlusal factors, psychological factors, hereditary factors and some of the consequences of bruxism. The bulk of the literature reviewed the aetiology and therefore supported the model and the remainder considered one or more of the associated factors e.g. Marks\textsuperscript{54} on bruxism in allergic children, Lehvilla\textsuperscript{68} on bruxism and magnesuim and Magee\textsuperscript{69} on bruxism and levodopa therapy.

Dubner Sessle and Story\textsuperscript{1} also raise the possiblitly of "susceptible subjects" to bruxism. Such a "subject" would fit into one or more of the appropriate emotional, behavioural and personality profiles, would have a blood relationship to a bruxer and would exhibit one or more of the local factors.

Table 2. attempts to summarise and classify the aetiological factors of bruxism.
In concluding the aetiology of bruxism, it is appropriate to include a major review of this aspect of bruxism according to Ramfjord and Ash 1983. The third edition of this famous text has only been recently released and due to the international respect accorded the authors, their up to date views on the aetiology of bruxism are most pertinent.

According to Ramfjord and Ash 1983, a close relationship, between psychic stress and bruxism has been obvious to most investigators of bruxism. Although a possible cause-and-effect relationship has been studied extensively, especially during the last decade, there are still gaping voids in the present knowledge. However, repressed aggression, emotional tension, anger, fear and frustration are continually cited as the most important or the sole factors in the aetiology of bruxism. Citing various studies, the authors comment that all of the studies are concerned with relationships between psychological factors and bruxism, but they do not establish an aetiological cause and effect relationship.

Discussing the heading, "Occlusal factors in the aetiology of bruxism", Ramfjord and Ash 1983 stated that almost all of the literature on bruxism states a dual aetiology of stress and occlusal interferences. Mentioning studies for and against occlusal factors in the aetiology of bruxism they conclude this section by stating that some kind of occlusal interference may be found in every patient with bruxism. Apparently, bruxism is precipitated by combined psychic and occlusal factors and consequently, is more apt to be present at certain times in a person's life than at other times.
Bruxism may be present due to severe occlusal interferences and a moderate degree of psychic stress, or due to very severe psychic stress and minimal occlusal interferences. Psychic stress in a persons life often varies greatly from one age to another or even from one situation to the next. Occlusal interferences that are normally avoided and thus are usually of no consequence may, in a stressful situation, assume an annoying dimension and trigger bruxism.

Therefore, according to the internationally famous and respected Ramfjord and Ash, the original Ramfjord model still applies in 1983. The "central initiation school" have yet to establish a cause and effect relationship.
Table II

<table>
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<th>AETIOLOGY OF BRUXISM</th>
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<td><strong>SCHARER</strong>&lt;sup&gt;59&lt;/sup&gt;</td>
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<td><strong>8ba Shimakawa</strong>&lt;sup&gt;62&lt;/sup&gt;</td>
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<td>Lindqvist&lt;sup&gt;35&lt;/sup&gt;</td>
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CHAPTER 5

DIAGNOSIS OF BRUXISM

Differential Diagnosis
A. Attrition
B. Bruxism
C. Abrasion
D. Erosion
E. Combination Lesions
F. The Anorexia Nervosa Syndrome

Discussion
DIAGNOSIS OF BRUXISM.

According to Ramfjord and Ash, the signs or symptoms of bruxism are not conspicuous in most instances; however, certain signs and symptoms, are indicative although not pathognomonic or diagnostic. By careful observation of these signs and symptoms severe cases of bruxism can usually be discovered. The diagnosis of bruxism is based on suggestive clinical signs and symptoms followed by a confirmatory history from the patient or other sources. Various recording devices for sound, tooth mobility and muscle action are helpful in establishing the diagnosis of bruxism.

The method of diagnosing bruxism employs the general principles of oral examination and diagnosis and this involves:

1. case history
2. clinical examination
3. study casts
4. examination of roentgenograms.

In Nadler's paper "Detection and recognition of bruxism" he details exhaustively the examination for the diagnosis of bruxism.

The case history employed by Nadler is involved as are most case histories and is included as an addendum in the form of Table 3. Clinical examination begins with:
1. **Soft tissue and musculature:** Soft tissue laceration and irregularities caused by sharp enamel edges of the bruxofacets should be looked for. Overdeveloped musculature or visual muscle contractions during examination.

2. **Gingiva:** Recession, bleeding or inflammation

3. **Teeth:** Abnormal wear on selective teeth or the entire dentition should be looked for. The presence of malocclusion, traumatic occlusion, tilted or missing teeth, sharp incisal edges, overhanging fillings and other faulty dental work should be noted and evaluated in relation to bruxism. The presence of calculus, and sensitive root surfaces also should be noted.

4. **Mobility:** Mobility may be caused by traumatic occlusion, periodontal disease or some destructive habit, such as bruxism. The mobility of the entire dentition should be checked. An attempt should be made to account for the mobility of each tooth. This information may be correlated with the subsequent examination of the roentgenograms.

5. **Type of Occlusion:** The presence of unilateral mastication, monocclusion, and hyperocclusion should be noted, as well as the presence of any bite discrepancies or faulty occlusion. The presence of a deep overbite or a locked bite should also be noted and a loss of vertical dimension and alteration of the freeway space should be looked for.
6. **Prosthesis:** Removable dentures may contribute to bruxism. Some patients have the feeling that their denture is dropping, so they clench their teeth to overcome this sensation. Ill-fitting dentures may contribute even more.

When bruxism is severe, abutment teeth may be loosened, and castings may be dislodged, particularly if they are thin. In removable prosthesis, underlying tissues may be traumatised and abutment teeth subject to undue stress. Artificial teeth have shown signs of wear and, in severe cases, have broken.

7. **Swallowing:** If the swallowing habits of the patient are abnormal, they may contribute to bruxism. If repeated enough these abnormal habits may destroy tissue by improper pressure, and thus swallowing becomes a conjunctive process to bruxism.

8. **Periodontal Disease:** Signs of periodontal disease may be noted in the examination. Pressure habits, and bruxism in particular, may be contributing factors to periodontal disease.

The nature and type of occlusion and the manifestation of wear on the occlusion may be highly significant. The individualized movements that result from the particular grinding habit of each patient, at times, causes attrition on a few teeth, rather than generalised attrition. Figs. 5,6,7.

Discrepancies in the occlusion may be seen on the study casts.
Occlusal interference, interlocking cusps and the locked bite are anatomical considerations that may initiate the bruxism.

It has been observed that as a result of wear a curve is gradually brought about in the natural dentition and a plane of wear is established. Variations from this phenomenon of attrition lead to the suspicion of occlusal dysfunction and the possibility of bruxism.

Because there is little in the present-day diet that produces serious abrasive effects, excessive wear cannot be explained in terms of normal mastication. Wear may be simulated by the selective grinding of the dentist, biting of a pipe or other foreign objects, and by the excessive mastication of food or the chewing of very hard foods. Excessive gum and tobacco chewing should also be considered.

Wear must also be considered in terms of the type of occlusion, the chronologic age and physiologic requirements of each patient. If, for example, the patient has prematurely lost the posterior teeth and wear is manifested on the incisal edges of the lower anterior teeth and the lingual surfaces of the upper anterior teeth, this amount of wear is not necessarily due to the effects of bruxism. Perhaps the demand made on these teeth in their performance of mastication is the cause. Thus it is important to distinguish between normal wear and bruxism wear.

The following factors, which will aid in the detection of bruxism, may be seen on the study casts:
Attrition on selective surfaces of the teeth or on the entire dentition.

Variation from the normal curve of wear.

Highly polished facets on slopes of cusps and matching facets.

Ridges between facets and grooves.

Well-defined paths of wear.

Ledges or grooves on the lingual surfaces of maxillary anterior teeth or thinning out of the labiolingual dimension.

Sharp and jagged incisal edges on anterior teeth.

Consistent wear on the mesial slopes of the maxillary teeth might indicate an anterior displacement of the mandible or a protrusive bruxism habit.

Scooping out of the incisal aspects of the anterior teeth.

Loss of vertical height of the clinical crown.

Spacing between teeth caused by excessive wear.

Flattening of inclines, obliteration of cusps and facets, and flattening of the teeth.

Presence of interlocking cusps or a locked bite.

Bruxism is not always destructive to the alveolar bone. When a balanced bite and uniform wearing of the dentition are produced, and the underlying forces of the dentition fall within the health tolerance of the alveolar bone matrix, a building up process may occur. This is an oversimplification of a complex question. Why is it a building up process for one and a breaking down process for another? Certainly there are many factors at work, including the nature of the bite, the particular type of bruxism (its duration, frequency, intensity, and direction of force), possibly some inherent
Figure 5. Pattern faceting in a young female bruxer.

Figure 6. Mandibular excursion to engage this bruxofacet is emphasised.
Figure 7. The contact position where the midline shift and the disarticulation of the other teeth are illustrated.
bone factor, and the inherited, systemic and emotional background. For some, bruxism seems to be stimulating just as good exercise might be to the musculature and general well-being of an individual.

Examination of the roentgenograms as a means of diagnosis in the detection of bruxism may be significant. It is important to establish the generalised picture of alveolar bone support and whether the bone is dense, average or diffuse. Where bone destruction has taken place on selective teeth, a local periodontal situation, a traumatogenic occlusion, the aging process or bruxism may be considered. When bone is lost, careful checking with the study casts about the position of a particular tooth or teeth in relation to other teeth within the arch and the particular morphology, noting inclined planes of the cusps, possibility of locking of the cusps, paths of wear, and so forth, give the dentist valuable information.

Where a selective destruction of alveolar bone is observed, bruxism should be suspected. As a result of bruxism, periodontal damage may take place and it can be observed roentgenographically. The periodontal space widens and the ultimate result may be increased mobility. Loss of alveolar bone adjacent to second molars for example, should elicit suspicion of an interceptive contact from an adopted centric position, interference on the balancing bite, or more commonly, clenching of the teeth.

The radiograph should also be checked for premature pulp aging,
pulp stones and hypercementosis, which are all pulpal reaction to bruxism.

Other diagnostic factors according to Ramfjord and Ash⁴³ are:

(a) unexpected fractures of teeth or restorations
(b) palpable tenderness in the muscles of mastication.
(c) temperomandibular joint discomfort and pain.
(d) maxillary or mandibular exostoses.
(e) audible occlusal sounds of non functional grinding.

The above lists in some detail the factors to look for when diagnosing for bruxism, however it could be stated that the principal diagnostic signs are atypical tooth wear, palpable muscle tenderness and the presence or absence of periodontal disease. Figs. 8,9.

Differential Diagnosis

A. Attrition
B. Bruxism
C. Abrasion
D. Erosion
E. Any combination of the preceding conditions
F. Anorexia Nervosa syndrome
Figure 8.

Figure 9.

Figures 8, and 9. The precise meshing of bruxofaceted teeth are illustrated.
A. Attrition

Attrition\textsuperscript{70} is the physiologic wearing away of a tooth as a result of occlusion and is associated with the aging process. The older a person becomes, the more attrition is exhibited. The dentition of children suffering from dentinogenises imperfecta or amelogenises imperfecta may exhibit pronounced attrition resulting from ordinary masticatory pressure.

Attrition in modern western society is rare or minimal due to our modern diets. It still exists in primitive tribes on primitive diets or abrasive containing diets. The work by Beyron\textsuperscript{19} in the 1940's on the Australian Aboriginals made mention of the abrasive particles in the diet from cooking within the fire on the ground. This led to advanced physiological attrition.

In differential diagnosis with bruxism the attrition facets would not be as advanced as the bruxofacets, nor would they be as highly polished. Further they would occur within the functional range of mandibular movements whereas bruxofacets would occur without that range.

In a patient exhibiting physiologic attrition there would be no atypical gingival clefts and no palpable muscle tenderness.
B. **Bruxism**

This has been fully listed under Diagnoses of Bruxism. Figs. 10,11,12.

C. **Abrasion**

Abrasion to tooth structure occurs by the interaction of foreign abrading particles and the dentition. It is a pathologic wearing away of the tooth through an abnormal mechanical process. Abrasion may occur on exposed root surfaces, as well as incisal or proximal root surfaces. Improper tooth brushing on exposed cementum or dentine can cause severe dental wear. In the "Index to Dental Literature" for the past two years the most common association for dental abrasions have been toothbrush and dentifrice abrasion and abrasion caused by removal of orthodontic bonded brackets. The differential diagnosis of abrasion or attrition and bruxism is firstly at the sight of occurrence. Abrasion of the cervical tooth structure is obvious as tooth brush abrasion and abrasion to the facial surfaces of the tooth is obvious by the dullness and abraded appearance. Abrasion at the incisal or occlusal surface is normally apparent as being outside the functional and parafunctional range. It will often be unilateral and spread off to involve another tooth surface as well. If the abrasion is advanced the lesion will be somewhat wedge shaped.
D. **Erosion.**

Erosion to tooth structure takes place by chemical means. The eroding agent may be etching solution used in clinical practice, industrial fumes or agents, foodstuff and food additives of a highly acidic nature and various secretions from within the organism. A gross sucrose attack on tooth structure as exhibited by pastry cooks can also take on the form of an erosion. Erosion in isolation can be differentiated from the other conditions in that the injury is not clearly demarcated as in a facet or a cervical abrasion. The eroded tooth surface is dull, non abraded and generally reduced only moderately in dimension but over an entire surface. The site is dependent on the agent e.g. citric foods affecting the chewing surfaces, industrial fumes the labial surfaces, regurgitated gastric acid the lingual surfaces.

E. **Combination lesions.**

This is perhaps more common particularly for abrasion and erosion. It is common to see abrasion and bruxism when the abrasion is caused by foreign objects such as pipes etc. and also when a tooth exhibits incisal bruxofaceting and cervical abrasion. It is also common for erosion and bruxism to occur simultaneously. The normal bruxofacet is smooth, flat, highly polished. In the bruxer exhibiting superimposed erosion, most commonly from citric foods or food agents, the bruxofacet is cupped out from the acid erosion on the dentine. It is commonly seen also as facets in posterior teeth.
when the bruxofacet appears like an enamel dimple and this is most commonly caused by the erosion of carbonated beverages on bruxofacets. In very advanced bruxofaceting, the cupping out of the dentine is normally caused exclusively by the bruxism. It is only in early to moderate faceting that the superimposed erosion is clearly evident. Figs. 13, 14, 15.

F. The Anorexia Nervosa Syndrome.

Whilst discussing the differential diagnosis of conditions that effect the hard tooth structure, consideration should be given to this syndrome, which has received extensive media and medical coverage and is now appearing more commonly in the dental literature. Anorexia Nervosa can be defined\(^1\) as a chronic psychosomatic illness manifesting itself primarily as an eating disorder. The use of the term anorexia with this disease is actually a misnomer since there is no real appetite loss. The reverse usually occurs, the patient claims to have appetite loss, but alternates food avoidance with overeating and follows this with induced vomiting.

The cause and effect relationship between regurgitation of gastric acid and loss of enamel and dentine was first reported in the literature by Hellstrom in 1974.\(^2\) The dental manifestation of Anorexia Nervosa include the following:

1. Perimyelolysis as defined by Hellstrom is a loss of enamel and dentine on the lingual surfaces of the teeth as a result of
Table III

NADLER CASE HISTORY

Local factors that can be included in the questionnaire are:

1. Do you grind, clench or click your teeth during the day or night?
2. Do you awake with your jaws clenched?
3. Do your jaws feel tired when you awaken?
4. Do you awake with a feeling of tooth consciousness?
5. Do you notice any bleeding when you awaken?
6. Do your jaws ever "get out of joint?"
7. Do you have pain when opening or closing your mouth?
8. Do your jaws ever click or lock?
9. Do you ever have pain in front of your ears?
10. Do you have teeth that feel high or long?
11. Do your jaws feel tired at the end of the day?
12. Do you grind, clench or click your teeth during sleep? Has anyone said that you do?
13. Do your teeth ever feel loose?
14. Do you think your teeth are moving or drifting?
15. Are you wearing dentures or removable bridges? If yes, are they comfortable? Are they loose?

Systemic and psychological factors which should be considered when diagnosing bruxism and may be included in the questionnaire are:

1. Do you have frequent headaches?
2. Do you suffer from constipation?
3. Have you ever noticed a twiching in any part of your body?
4. Do your parents grind, clench or click their teeth?
5. Did your parents grind, clench or click their teeth?
Table III (cont.)

6. Do you smoke more than one pack of cigarettes per day?
7. Do you drink more than six cups of coffee or tea per day?
8. Do you eat at regular hours? If no, explain?
9. Do you take vitamins?
10. Do you grind, clench or click your teeth when you are nervous?
    When you are angry?

The occupational factors which may be include in the questionnaire are:

1. What type of work do you do?
2. Does your work make you nervous or tense?
3. What type of work have you done in the past?
4. Do you do any work with sand or gravel?
5. Do you work with acids?

The concomitant factors which are important in the diagnosis of bruxism and may be included in the questionnaire are:

1. Do you chew gum? If yes, for how long, How many hours per day?
2. Do you smoke cigars? If yes, do you clench them?
3. Do you smoke a pipe? If yes, for how long? How many hours per day?
   Do you support the pipe with your fingers?
4. Do you bite your fingernails?
5. Are you in the habit of biting any foreign objects such as bobby
   pins, pencils, tooth-picks, and so on? Elaborate,
6. Do you have any difficulty in swallowing? If yes, explain.
7. Do you use many citrus fruits such as lemons or oranges in your
   diet?
8. Do you chew tobacco? If yes, for how long? How many hours per
   day?
9. Are you aware of a habit such as pushing your tongue against your
    teeth? Or biting your lip?
Figure 10. Gross upper lingual bruxoabrasion.

Figure 11. Gross lower incisal bruxoabrasion.
Figure 12. The progressive stage of this abrasion is emphasised by the translucency of the remaining enamel.

Figures 10, 11, 12, demonstrated tooth faceting outside the "functional range" which is diagnostic of bruxism versus attrition.
Figure 13. A lower arch where combined bruxism and erosion are demonstrated.

Figure 14. The combined lesion is illustrated by the flat enamel bruxofaceting and the dental cupping induced by the superimposed erosion. The erosion is also demonstrated on the gingival tooth structure.
Figure 15. A combined bruxo-erosion abrasion facet is highlighted. The added abrasion factor was caused by the introduction of marginal amalgam and enamel particles to the bruxo-slurry.
"The lack of abrasiveness in the modern diet is probably conducive to the development of restricted masticatory movements. Although the frequency of lateral tooth contacts is not significantly altered by the type of food being eaten, it is quite likely that hard foods, such as raw fruits and vegetables, and fibrous or dry meat, are more effective in eliminating the influence on total occlusal guidance than soft foods. Tests on biting strength show that really heavy biting is more comfortable done close to centric occlusion than in lateral or protrusive positions of the jaw, so that the crushing of very hard food probably involves very limited lateral excursions."

Ramfjord and Ash.43
chemical and mechanical effects caused mainly by regurgitation of gastric contents and activated by movements of the tongue.

2. Dental Caries: An excessive carbohydrate intake and poor oral hygiene may result in an increase in dental caries in these patients.

The differential diagnoses of perimyolysis and dental erosion should be fairly apparent due to the perimyolysis being principally lingual and so severe. Any superimposed abrasions and bruxofacets can be diagnosed by the aforementioned criteria.

Discussion.

A general history guide to aid in the differential diagnosis of tooth damaging conditions excluding dental caries and restorative dentistry is suggested by Nadler. 7

1. Physiology - heavy jaws and strong muscular development; acid eructation incident to digestive disorders; deep intercuspatation; a deep bite or cross bite occlusion.

2. Unusual diet - hard or gritty foods.

3. Habit - some people chew their food longer than others. Nadler has observed the attrition where the patient chewed gum for several hours per day for 30 years; similarly in the case of
tobacco chewers - due to the silex and other gritty substances, the use of a pipe, biting of thread.

4. **Chemical action** - excessive and prolonged use of lemon juice may produce etching on the labial and lingual surfaces of the teeth and in some cases may even involve the occlusal surfaces. The low pH of the juice coupled with ardent toothbrushing may bring about such an effect; the action of other natural fruit acids.

5. **Excessive and improper brushing** - utilizing abrasive dentifrices.

6. **Occupational environment** - the action of acid fumes; sand, gravel and other abrasives (masonry for example) where entrance into the oral cavity might conceivably produce abrasion or etching.

7. **Selective grinding** by the dentist may simulate wear.

8. **Age** - a certain amount of greater wear is normal as age increases.

9. **Inadequate dentition** - lack of a complete dentition may produce greater wear on the remaining teeth.

Clinically it has been observed, by the author that a tooth exhibiting bruxism induced trauma can be differentially diagnosed by
enquiring whether there has been a history of pain when chewing grain bread or muesli and a hard component of the food bolus is engaged in functional tooth contact. This pain at times can be quite exquisite and is caused by a wedge effect on the tooth that has been occlusally traumatised during bruxism. It is normally a tooth exhibiting an occlusal interference and does not become tender, except for extreme cases, on normal closure and some times even clenching, due to the developed avoidance pattern in the awake mandibular musculature.

The differential diagnosis is summarised in Table IV.
**DIFFERENTIAL DIAGNOSIS OF BRUXISM**

**COMPAORED TO OTHER CONDITIONS THAT EFFECT HARD TOOTH STRUCTURE**

**EXCLUDING CARIES AND RESTORATIVE DENTISTRY**

<table>
<thead>
<tr>
<th>Table IV</th>
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<tr>
<td><strong>ATTRITION</strong></td>
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<tr>
<td>FACETING</td>
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<td>Within functional range.</td>
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<td>OCCLUSAL WEAR PATTERN</td>
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<td>When accelerated in indigent groups still within normal occlusal range.</td>
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<td>TOOTH SURFACE AFFECTED</td>
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<td><strong>TOOTH MOBILITY</strong></td>
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<td><strong>PULPAL SENSITIVITY</strong></td>
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<td><strong>HABITS</strong></td>
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<td><strong>AGE</strong></td>
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(cont.)
<table>
<thead>
<tr>
<th>PERIODONTAL FACTORS (cont.)</th>
<th>Attrition</th>
<th>Bruxism</th>
<th>Abrasion</th>
<th>Erosion</th>
<th>Perimyloysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Can be gross periodontal damage (non inflammatory) as a result of bruxism.</td>
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<thead>
<tr>
<th>MUSCLE PALPATION</th>
<th>Attrition</th>
<th>Bruxism</th>
<th>Abrasion</th>
<th>Erosion</th>
<th>Perimyloysis</th>
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<tr>
<td>Independent of condition.</td>
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<tr>
<td>May be gross muscle development in certain indigent tribes because of fibrous diet, but no palpable tenderness.</td>
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<tr>
<td>Some palpable muscle tenderness is common. Overdeveloped muscles such as masseters common. Difficulty in manipulating mandible because of muscle splinting.</td>
<td>Independent of condition.</td>
<td>Independent of condition.</td>
<td>Independent of condition.</td>
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CHAPTER 6

BRUXISM AS A COMPONENT OF FUNCTIONAL DISTURBANCES OF
TEMPOROMANDIBULAR JOINTS AND MUSCLES
BRUXISM AS A COMPONENT OF FUNCTIONAL DISTURBANCES OF TEMPOROMANDIBULAR JOINTS AND MUSCLES

According to Ramfjord and Ash4, functional disturbances include acute traumatic arthritis, muscle spasm, chronic traumatic arthritis and osteoarthritis. One or more of these conditions may be present at a given time and their manifestations may be limited only to the joints and adjacent structures; however, manifestation may involve the entire masticatory system and extend even to other parts of the head and neck.

The aetiology of functional disturbances as described by Ramfjord and Ash4, stated that considering all the theories that have been advanced it is becoming more and more evident that the most common underlying factor is an abnormally increased muscle tonus plus some form of bruxism.

The tissues within the temperomandibular joints, as well as other parts of the masticatory system, are normally protected by basic neuromuscular reflexes, and through co-ordination of muscle function and forces, by the neuromuscular system. Injury to the temporomandibular joints, except that due to external trauma, is therefore the result of an abnormal muscle action and a related imbalance in the alignment of the various parts of the masticatory system. Anything that might increase the basic muscle activity or tonus, such as frustration, psychic stress, emotional tension,
occlusal interferences or pain, may lead to functional disturbances and pain in the tempormandibular joints and adjacent muscles.

There is minimal stress upon the components of the tempormandibular joints in "empty movements" provided a harmonious relationship exists between the occlusion and joints and the person has physiologic muscle tonus. "Empty movements" refer to tooth contact during swallowing or tooth contact without anything between the teeth. Although heavy stress may occur on the balancing side in the tempromandibular joint when biting on hard food or other objects, the joint is normally protected from damaging forces by neuromuscular co-ordination of the biting forces and protective reflexes. However, when there is an abnormal increase in muscle tonus and response to stimulus, there exists the potential for traumatic injury to the joint, as well as to muscles and ligaments. Such an increase in basic muscle activity is associated with psychic tension. Intense reticular stimulation will not only tend to initiate heavy contraction in the masticatory and facial muscles, but at the same time render the nociceptive inflow less effective than with low reticular stimulation. This reduction in efficiency of protective reflexes by way of central nervous system overstimulation may in part explain the interplay between the masticatory system and the central nervous system in the etiology of tempromandibular joint dysfunction. The increased activity is found to a greater extent in the masticatory and facial muscles than in most other muscles of the body, since the facial and jaw muscles are involved normally in expression of emotions such as anger, fear and aggression.
After an injury has been established, the pain from the injured tissues has a tendency to increase muscle activity, which in turn may increase the injurious forces and produce additional trauma. This vicious cycle of "feedback" between muscle tension and injury usually is expressed in one or another form of bruxism and plays an exceedingly important role in the development of traumatic temporomandibular joint arthritis and associated functional disturbances.

It is not the purpose of this review to discuss in any detail the occlusal dysfunction syndromes as major subjects of this nature as reviews unto themselves. The only purpose in including this heading is to clearly establish that bruxism is an aetiological factor in the pathogenesis of functional disturbances of the masticatory apparatus, that it is a major aetiological factor and that it has an almost universal involvement. This does not mean to say that it is present as an aetiological factor in such inflammatory condition as Rheumaroid Arthritis of the Temporomandibular Joint. This discussion is limited to functional disturbances as defined by Ramfjord and Ash.

Rugh and Solberg discussing the psychological implication of temporomandibular joint dysfunction stated that emotional factors such as anxiety, fear, frustration and anger play a significant role in the aetiology of T.M.J. disorders, in that they elicit muscular tension and oral habits.
De Boever\textsuperscript{28} in reviewing the aetiology of functional disturbances of the temporomandibular joint stated that clenching or grinding are very common phenomena in almost all pain dysfunction patients. These parafunctions cause muscle spasm and pain in joints and muscles.

Laskin\textsuperscript{32} in his key papers defining the Myofascial Pain Dysfunction Syndrome (M.P.D.) stated that although adverse mechanical factors leading either to muscle overextension or overcontraction may cause some instances of M.P.D. syndrome, it is believed the most common cause to be muscle fatigue produced by chronic oral habits such as clenching or grinding of the teeth. These habits can be initiated by annoying "dental irritation" like an improperly occluding restoration or an overhanging margin. Generally, however, we believe that they are an involuntary tension-relieving mechanism. Since this explanation for most cases of M.P.D. syndrome implicated emotional rather than mechanical factors as prime etiologic agents, it has been termed the psychophysiologic theory. Laskin's schematic diagram of the aetiology of myofascial pain dysfunction syndrome is reproduced.
TENSION → ORAL HABIT ← DENTAL IRRITATION

MUSCULAR FATIGUE

MUSCULAR OVEREXTENSION → MYOSPASM ← MUSCULAR OVERCONTRACTION

MYOFACIAL-PAIN-DYSFUNCTION

CONTRACTION ← SYNDROME → OCCLUSAL DISHARMONY ← DEGEN. ARTHRITIS

ALTERED CHEWING PATTERN

DIAGRAM V
In a study to relate the frequency of parafunctional occlusal habits compared with the incidence of mandibular displacement, Rieder found that the prevalence of occlusal habits in all patients was high but became greater with the increased complexities of the mandibular displacement.

Lehman, discussing the aetiology of pain dysfunction syndrome, stated that psychological factors play a vital role. Emotionally based stress contributes to the development of the syndrome initially by increasing the overall activity of the masticatory muscles, mainly through bruxing and clenching. Once the syndrome has developed, the pain and other symptoms may then cause the patient to become anxious and tense about its future course. This, in feedback fashion, increases further both the masticatory muscle activity and the severity with which the patient reacts to the symptoms, so that the syndrome tends to perpetuate itself.

Further, psychologically induced and stress connected bruxing and clenching increases the impact of unequal distribution of motor unit activity enormously. It is held then, that frequent bruxing or clenching and an occlusal disharmony are both necessary to produce this extremely disproportionate activity. One without the other does not and the syndrome does not develop.

Clarke et al. conducted an experiment on eighty five subjects during night time grinding. Their results supported the hypothesis that prolonged jaw closing muscle hyperactivity was correlated with the symptoms of jaw dysfunction. More specifically, the greater the
level of nocturnal E.M.G. a subject had the more likely he was to have signs and symptoms of jaw dysfunction.

There is a definite consensus in the literature that bruxism is an important component in the multifactorial aetiology of functional disturbances of the masticatory apparatus. The only part of discussion is its frequency of occurrence. It could be speculated that bruxism always adds to any pre-existing, or causes, muscle hyperactivity but muscle hyperactivity does not always cause functional disturbance in the masticatory apparatus. Figs. 16,17,18.
"The cause of the functional disturbances and the pain is a combination of psychic tension and occlusal disharmony resulting in muscular hyperactivity and producing traumatic injury to the joint structures, tendons, and muscles of the masticatory system.

"Distal displacement, overclosure, and loss of occlusial vertical dimension are not specific causes of dysfunctional pain. The pathological changes in the joints are the results of direct trauma rather than direct degeneration associated with any of these often implicated factors."

Ramfjord and Ash.⁵³
Figure 16. The engagement of an upper cuspid and a lower lateral incisor in bruxism is emphasised.

Figure 17. The bruxo-engagement is illustrated and the precise meshing is emphasised. The gross excursion of the mandible is also noted.
Figure 18. The gross disarticulation of the dentition is demonstrated. The obvious strain on the muscles, ligaments and joints can be imagined.

Figures 16, 17, 18, demonstrated an extreme bruxism position. Not an incisal contact position between an upper and lower cuspid but the more extreme upper cuspid to lower lateral incisor. The figures demonstrated clearly that this position was engaged frequently and ferociously enough to have induced the degree of contact wear exhibited. The physics of the strain inflicted on the entire masticatory apparatus can be appreciated.
CHAPTER 7

THE EFFECT OF BRUXISM ON
THE TEMPEROMANDIBULAR JOINT AND MUSCLES

A. MUSCLES
B. TEMPEROMANDIBULAR JOINTS
C. HEADACHES
THE EFFECT OF BRUXISM ON
THE TEMPOROMANDIBULAR JOINT AND MUSCLES.

This is not to be confused with the previous heading which discussed bruxism as an aetiological component in functional disturbances of the masticatory apparatus, but the aim is now to discuss the direct effect of bruxism on unit tissue, whether syndromes exist or not.

A. Muscles

Scharer\textsuperscript{59} stated that the most regularly observed symptom of bruxistic disorder is tenderness of specific jaw muscles, especially on palpation. In addition, patients can complain of muscular tiredness upon awakening or of general tension.

Christensen\textsuperscript{77} who has conducted many experiments on masticatory muscle clenching stated that maximal voluntary isometric contractions of the elevator jaw muscles, with the mandible in the position of maximal intercuspation of the natural teeth, can induce fatigue and weak as well as severe pains in the jaw muscles of children and adults. In an earlier experiment he produced myalgic pain two hours after artificial initiation of bruxism with patients grinding their teeth for 30 minutes over periods of five minutes alternating with rest periods between.
Milton Arnold in the 1981 Dental Clinics on Occlusion discussed the effect on the masticatory muscles thus: In bruxism the masticatory muscles are involved with the teeth in a vicious circle. The muscles are responsible for the teeth coming together and the teeth, by coming together, are responsible for increasing the tension in the muscles. Tension may, therefore accumulate in these muscles from unexpressed emotions or from the inefficient discharge of psychological stress, with the teeth receiving some of the tension for discharge and reflecting the remainder back to the muscles.

Continued working of the muscles results in various signs and symptoms. The earliest symptom is usually a feeling of tiredness. A fatigued muscle shows a loss of irritability due to an accumulation of lactic acid from a blood supply that can not bring enough oxygen or carry away the products of catabolism. Constant working of muscles, even when fatigue does not occur, may result in hypertrophy.

A muscle that is continually worked without adequate rest may develop spasm, and involuntary painful contraction. In the mouth, this may be seen as trismus, a limitation of opening, where the patient may not be able to separate his jaws more than 1 to 2 cm. Other manifestations of spasm include incoordination (where the patient moves his mandible from one side to the other as he opens), deviation (where the mandible's midline is not lined up with the maxillary mid-line in the fully open or closed position), and clicking (a snapping sound heard as the two separate insertions of the external pterygoid, on the condyle and the other on the disc, move asynchronously).
During bruxing, a muscle may be stretched. This will happen when a tense, shortened muscle is forced into an elongated position (for example, in a class 2 occlusion with a marked overbite where the mandible is forced posteriorly as it closes within the confines of the perimeter of the maxillary teeth). Normally, when the mandible is elevated or resting, the external pterygoid muscle is relaxed with the head of the condyle close to the posterior slope of the articular eminence. In the event that this muscle is tense, with its insertion in a more anterior position as a result of the mouth being kept open for a prolonged period (such as for a long dental procedure), the muscle will be stretched as the mandible is forced backward into occlusion, carrying its head deeper into the glenoid fossa. In a similar manner the elevators of the mandible, masseters, temporals, and internal pterygoids may be stretched during a long dental visit if they had been made tense during the night with prolonged bruxing. The significance of this stretching of a tense muscle is that it will frequently result in reflex spasm with the attendant pain, tenderness, limitation, deviation, incoordination, or joint noise.

Bruxing with the mandible in a deviated position as a result of cuspal interference may also predispose to muscular spasm. In this case, the muscles are asymmetrically contracted to begin with, and a superimposition of additional metabolic products of contraction may be enough to make the difference. One might say there was a muscle spasm diathesis.
Glaros and Rao\textsuperscript{80} describe how chronic bruxism has been implicated as a primary cause of hypertrophy of the masticatory muscles, particularly the masseter muscle. The overdeveloped masseter muscles appear to be swollen and in the region of the angles of the mandible, are pointed in appearance. Hypertrophy of the masseter muscles may seriously interfere with the maintenance of a constant rest position of the mandible. In severe instances, the hypertrophied muscles may cease spontaneous contractions of the facial musculature, trismus and an alteration of the occlusion. This alteration has been described as a loss of vertical dimension and a diminished inter-occlusal distance. Finally, the muscular
involvement may alter the opening and closing pattern of the jaws both in mastication and in speech habits.

Ahlgren et. al.\textsuperscript{81} described three patients (men aged 14 - 18 years) in two of whom hypertrophy caused by bruxism occurred bilaterally and in one, unilaterally. They found that the maximal muscle tension recorded electromyographically, was twice as great as they found in normal individuals of the same age. Additionally the patients did twice as much work in chewing their food. When a muscle biopsy was performed, the fibre diameter of the muscles was twice the normal size. The authors reported that all three subjects had a small gonial angle, which they accounted for by hypothesizing a functional adaptation to the increased mechanical stress on the mandibular insertion of the masseter muscle.

Leof\textsuperscript{82} noted that bruxism may produce more powerful muscles of mastication, resulting in hypertonicity of the masticatory muscles with little effect on the depressor muscles.

B. Temporomandibular Joints

Weinberg\textsuperscript{83} notes that changes that occur in the T.M.J. as a result of bruxism are closely related to the changes that occur in the masticatory musculature. He suggested that the position of the condyles in the fossae when the teeth are in maximum occlusion can influence whether the effect of bruxism will be concentrated on the joint structure or on the musculature.
According to Glaros and Rao\textsuperscript{80} bruxism created T.M.J. disorders commence with asymptomatic joint and disc injury and advance to conditions such as clicking and crepitus in the joint, locking of the jaw, restriction of mandibular movement, difficulty in chewing and incoordination in opening or closing the jaws and subluxations. In more severe cases, actual luxations have been reported. In these patients, fibrous and/or bony tissue changes occur in the T.M.J. The disc becomes worn, the cartilage thins out and the capsular ligaments become stretched. This is followed by a cracking and a fibrillation of the cartilage, leading to changes in the bone. This degenerative process affects the whole face as the condyle shortens and the chin recedes.

According to Nadler\textsuperscript{9} hypertonicity of the masticatory muscles that is developed from severe grinding may seriously interfere with the maintenance of a constant rest position of the mandible. Alteration and closure of the maxillo-mandibular relationship and diminution of the free-way space may occur. In severe bruxism, temporomandibular disturbances may result from trauma or excessive closure due to abnormal wear inadequately compensated for by continuous eruption.

Mongini\textsuperscript{84} examined one hundred crania from male and female subjects, aged 25 - 53 years at death, with complete but variously abraded dentitions. Condylar morphology was individually assessed in terms of the slope, extent and direction of their joint surfaces, as well as the inclination of the transverse condylar diameter relative to the frontal plane. A relationship between the abrasion pattern
and condylar shape was noted. Extensive generalised abrasion was associated with slanting lateral and medial slopes. When working abrasion predominated either or both condylar slopes tended to be flattened.

Hansson stated that biomechanical loading during function and parafunction plays a dominant role in the interaction and development of tissue changes in the T.M.J. The ability of the articulating tissues of the joints to resist pressure and wear and tear depends largely on the physical and chemical properties. A number of factors: genetic, endocrine, metabolic and vascular, which may vary with age and sex, can alter the properties of the joint tissues and the joint fluids and therefore influence the development and progress of degeneration. The author proceeded to discuss the results of examination of T.M.J. from post mortem material. The point of interest is the high incidence of gross change in joint form. Approximately 50% of joints in the 20-40 years age group exhibited some change in form. Various speculations were advanced regarding the cause of these degenerative changes, but the high incidence in young healthy people indicates that the joint can be affected by occlusal factors and it has been clearly demonstrated that bruxism is a destructive occlusal habit. Figs. 19,20,21,22,23.

C. Headaches

Some mention should be made of headaches of a dental origin. There has been recent media coverage of "migraine" being cured by
dentists, utilising splints among other therapies. Professionally diagnosed "migraine" is a vascular disease and it will not respond in any favourable way to dental treatment. There may be occasions when dental factors are superimposed in a case of migraine and the successful dental treatment of these other factors will aid the patients well being, but will in no way treat the migraine.

However, head pain and headaches can be caused by bruxism and by functional disorders of the masticatory apparatus. The effect that bruxism has on the masticatory and surrounding tissues and the role it plays in the aetiology of functional disturbances has been discussed. The role of bruxism and dysfunction syndromes in the pathogenesis of "headache" is but an extension of this discussion.
Figure 19. Frontal view of mounted study models engaged in bruxistic contact position.

Figure 20. Similar view where the articulation is illustrated.
Figure 21. Right lateral view with the disarticulation of the teeth and the displacement of the condylar element being illustrated.

Figure 22. Left lateral view.
Figures 19, 20, 21, 22, 23. A mechanical representation of what happens at the joints during bruxism is demonstrated. The mounted models have been engaged into the bruxistic pattern and the displacement of the articulator condylar elements gives a reasonable representation of the abnormal joint position, relative to the forces exerted, during bruxism.

Figure 23. Right condylar element.
CHAPTER 8

THE EFFECT OF BRUXISM ON THE TEETH

A. HARD TOOTH STRUCTURE
B. FRACTURES OF TEETH & RESTORATIONS
C. DENTAL PULP
D. ROOT CHANGES
E. ENDODONTICS
F. RESTORATIVE TREATMENT
G. WEAR
H. RESTORATIVE TECHNIQUE
I. DENTURES
A. Hard Tooth Structure

To a certain extent the effect that bruxism exerts on tooth structure has already been discussed under Diagnosis and Differential Diagnosis of Bruxism. It was noted that bruxism causes accelerated loss of hard tooth structure and the imprinting of the tooth with bruxofacets.

The mechanism of excessive tooth wear associated with bruxism, according to Uhlig,\textsuperscript{2} is based on the loosening and crushing of enamel prisms between contacting enamel surfaces, which provides the grit necessary for rapid wear of the enamel. According to Arnold,\textsuperscript{79} the effect of bruxing on the tooth itself is dependent on a number of factors: quantity, quality, location (maxilla or mandible), position in arch, jaw relationship, number of teeth, degree of calcification or hardness, cusp height or angulation, mobilitly and proximal contacts. It should be obvious that what takes place is related primarily to the quantity and quality of the bruxism. Since the mandible moves during grinding movements, varying amounts of energy will be dissipated during that action. The fact that the teeth in the maxilla are fixed in position while the lower teeth move against them may have a bearing on the amount of force that is received. Teeth in the posterior part of the mouth receive more force. A class 3 relationship of the jaws may remove some teeth from occlusion and place others in crossbite. A greater number of teeth receiving a
force will divide that force more adequately. A tooth that is well calcified and, as a result, hard will absorb the force less than one that is soft and abradable. The harder tooth will pass the forces on, whereas the softer one will have its tooth structure reduced and dissipate the force in the process. The greater the height and angulation of the cusp, the greater the possibility of torque being produced or for clenching to occur, due to the locked-in effect. Teeth that are anchored in bone of good quality will be more subject to wear than those that are more mobile; on the other hand, the mobile ones are in greater danger of having their mobility increased. Teeth that have proximal contacts will be able to share their load with their neighbours and consequently will be less subject to movement as will those that do not share contact and so may receive the full brunt and be tipped into a proximal space.

Grossly a tooth may move in a number of directions as a result of receiving the forces from bruxism. This movement may be labially or buccally, lingually or palatally, as well as mesially and distally. Teeth may also be intruded and kept from erupting with consequences for the developing occlusion. Many cases of dual levels of occlusal heights are the result of this.

Frequently, instances of fractured (or damaged) restorations, restored teeth, orthodontic and prosthetic appliances may be the result of these noxious occlusal forces. Undermined enamel and nonvital teeth are particularly vulnerable to breakage. The more movement of these structures, the less the chance they will be broken; the tighter and firmer the structure, the greater the chance
of fracture. Teeth themselves may chop or break so that the occluding parts approximate in the manner of a lock and key.

The enamel of the tooth is the first tissue that receives the bruxing force. If this is from an isometric contraction, the probability is that little wear will take place and that the bulk of this force will remain in the muscles or be passed on to other structures. If wear does occur from grinding or rubbing, a shiny, planed off area of varying size will be seen. The abrasive lesion from the intervention of a coarse, hard food or substance between the teeth may be similar in appearance on the enamel except for the possible presence of scratch marks or a dull look. Bruxing and abrasion may occur on the same teeth, presenting the frustrating and impossible task of trying to determine how much each contributed to the facet.

Once the dentine is reached through attrition or abrasion, a difference is usually discernible. In bruxism, one still sees the planed surface, a central core of dentine surrounded by a rim of enamel of varying thickness, with both tissues on the same level. By contrast, in abrasion there is a central cupping of the dentine because it is softer and therefore wears differently. Because of the type of movement of bruxing, the rim of enamel protects the dentine; in abrasion the dentine is not as protected, especially if the coarse substance is small.

The pulp may react to the bruxing force once the dentine is affected. The unprotected dentinal tubules are more subject to
irritation and therefore may be stimulated to lay down secondary or tertiary dentine. The probability is that the formation of protective dentine will keep pace with the rate of attrition so that the likelihood of a pulp exposure is low. On the other hand, if abrasion takes place concomitantly with bruxism, a pulp exposure is a distinct possibility.

Glaros and Rao\textsuperscript{80} state that wear generally occurs on the incisal edges of the anterior teeth and as facets on the cusps and restorations of the posterior teeth. The grinding forces are extremely destructive, because they are lateral rather than vertical and they produce an uninterrupted, mainly horizontal load lasting for several minutes, as measured by a strain gauge between antagonistic first molars in a study by Graf.\textsuperscript{3} The amount of abrasive damage is a function of the intensity, frequency, direction, duration and type (grinding or clenching) of the bruxing habit and the individual differences in resistance. The characteristic shiny occlusal or incisal wear facets with sharp edges may be caused by bruxism exclusively. The antagonistic pairs of facets can be matched and used to diagnose the direction of the bruxing. Severe bruxism may result in teeth that are worn below the contact areas. This harsh abrasion may produce spacing, especially between the anterior teeth. Figs. 24,25.

B. Fractures of Teeth and Restorations

According to Ramfjord and Ash,\textsuperscript{43} splitting or fracturing is a common sequela, which may occur in intact teeth, but is found mainly
Figures 24, 25. Severe bruxo damage to the teeth in a 60 year old male is illustrated. Cervical abrasion is also evident. The cupping of the dentine in this instance is exclusively bruxism. The advanced state of the abrasion has resulted in the tooth contact being totally on a flat dentinal surface with no anterior disclusion being provided. As a consequence the dentine is worn at a faster rate and the facet is dished rather than cupped as with erosion.
with occlusal wearing of the central fossa of soft restorations, leaving hard cusp tips in occlusal interference. Fracturing of restorations and teeth may also occur outside the functional range of occlusion in patients with bruxism during episodes of extreme forced malposition of the jaws. Fig. 26

Rosenberg\textsuperscript{70} talks of bruxism potentiating the "cracked tooth syndrome". He adds that the most common symptom of a cracked tooth is discomfort or pain elicited at the initiation or release of chewing pressure. The pain tends to intensify in time and frequency and terminates with an excruciating toothache. Incomplete fractures of teeth present difficult diagnostic problems, for they may respond normally to thermal sensitivity tests, radiographic evaluation and electrical vitality responses. Important clues to the diagnosis would be the description of pain occurring only on chewing or release, as well as on percussion from only one direction.

C. Dental Pulp

According to Rosenberg,\textsuperscript{70} irritants have a cumulative effect on the pulp and the pulps reaction at any time is the result of a variety of factors: severity and duration of the insult, caries experience, restorations, periodontal state of the surrounding tissues and dental habits such as bruxism. The pulps of teeth that have experienced occlusal abrasion and attrition as well as erosion, extensive caries, operative procedures and periodontal lesions appear to undergo retrogressive and atrophic changes that is
Figure 26, The enamel fracture of an over-restored bicuspid in a bruxing patient is illustrated. Obvious on the adjacent bicuspid is a bruxofacet at the tip of the cusp, which certainly would have been causative.

Figure 26.
typical of aging. The term "induced pulp aging" is used to
describe the effect of such injuries inflicted on the pulp. The
degenerative changes associated with chronological or induced aging
of the pulp probably result in tissue with a reduced defensive
capacity. This factor must be considered when contemplating complex
restorative procedures for teeth that have undergone induced aging.

Abnormal tooth wear induces exposure of the dentinal tubules and
subsequent irritation of odontoblastic processes resulting in
formation of secondary dentine pulpal to the primary dentine. While
this acts to prevent or postpone pulpal exposure, the pulp undergoes
the retrogressive changes associated with induced aging. In some
cases pulps may not be sufficiently protected by secondary dentine
and the pulp becomes exposed. However, the condition is closely
correlated to the degree of tooth wear and to the speed with which
the wear has proceeded.

D. Root Changes

Rosenberg⁷⁰ mentions that occlusal trauma including bruxism has
led to pulpal changes such as increased pulp stones, pulpitis, root
stumping, root resorption and ankylosis. When the root
resorption has taken place it is permanent and it must be
appreciated that bruxistic induced root modification can advance or
accompany chronic apical ankylosis. In these cases the contents of
the root canal are necrotic and endodontic treatment should be
instituted.
Internal and External Resorption may also be associated by bruxistic induced occlusal pathology. In some instances the resorption originates in the periodontal membrane rather than in the pulp. Some teeth undergo ankylosis during the reparative process.

Hypercementosis or clubbing of the apical areas of the teeth can occur as a result of bruxism. In some instances both hypercementosis and root resorption may be seen in the tooth. It has been noted that hypercementosis will increase the surface area of the root and allow for the attachment of an increased number of periodontal fibres, enabling the tooth to withstand an increased functional load. However, hypercementosis or dystrophic calcification may obliterate the apical portion of the root canal and complicate endodontic treatment.

E. Endodontic Treatment

Endodontic treatment may be complicated by bruxism in that pericementitis may become a recurrent problem. Such patients report that they are awakened in the morning or during the night due to severe pain in the tooth undergoing treatment. Typically, pericementitis due to night grinding will improve or disappear as the day progresses. In pericementitis due to an endodontic exacerbation, pain does not dissipate as the day goes on. In bruxing patients it is essential to take the tooth undergoing treatment out of occlusion. In these patients, an endodontically treated tooth has a reduced potential for periapical repair. In evaluating endodontic failures,
despite apparently sound therapy, bruxism should be considered as a potential factor interfering with normal repair.

It has been clinically observed by the author that bruxers who have received endodontic treatment can complain of an apical tenderness up to a year after the endodontics were performed. The fitting of a splint has relieved this tenderness. It has also been observed that when great difficulty has been experienced in obtaining apical anaesthesia there is usually an overlying bruxism problem. Further, the so called "cementum sensitivity" after total root canal instrumentation can also be ascribed to bruxism or occlusal trauma.

F. Restorative Materials

According to Arnold,⁷⁹ abrasion or bruxism occur simultaneously when restorative material occludes with tooth structure. Not only is there the one to one relationship of wear on tooth and abrasion on restorative material but the mixture of tooth debris and restorative particles forming an abrasive slurry. Further particulation occurs with e.g. amalgam margins and fractured enamel margins being caught in the meshing of the bruxistic activity. It is therefore important to consider the effect of bruxism on individual restorative materials. It is further important to consider the material of choice in a bruxing patient. Figs. 27,28,29.

Table V. reproduces three tables from Arnold;⁷⁹ "Strength of Dental Materials", and "Knoop Hardness of Dental Materials". Arnold⁸⁰
Figure 27. The cuspid to cuspid bruxism is emphasised.

Figure 28. The bruxoengagement with wear of the gold inlay is illustrated.
Figure 29. The precision grinding of the inlay and tooth is illustrated. Secondary abrasion is demonstration on the inlay caused by the bruxoslurry of tooth and restoration particles and fragments.
considers other factors regarding occlusion that should influence material selection e.g. a harder material will pass forces on to underlying structures more readily than a softer and that can absorb the occlusal forces. A softer material may wear or yield to the point of breaking a cement seal, or even of becoming deformed.

Phillips\textsuperscript{87} discussing "Abrasion Resistance" stated that hardness has often been used as an index to the ability of a material to resist abrasion. However, abrasion is an exceedingly complex mechanism, involving an interaction between numerous factors. For this reason the reliability of hardness as a predictor of abrasion resistance is limited. Often it is valid for comparing materials within a given classification. However, it may be invalid when evaluating different classes of materials, such as a metallic material with a synthetic resin. Abrasion resistance is an exceedingly elusive characteristic to evaluate. This incongruity of a harder material having a greater wearing factor was demonstrated in the clinical utilisation of composite resins in posterior teeth. The resistance to wear was abysmal compared to amalgam. This is an enigma when considering the compressive strength of the composite. It might be speculated that bruxism was an ingredient in this failure.

The average composite placed and finished in a posterior cavity would not have a surface finish of a high standard, particularly if it had been accurately finished for occlusal contacts. It could be theorised that the average posterior composite that was well finished was in hypercontact or if occlusally adjusted bore a poor finish.
Significant damage from bruxism is often greater to the crown of the teeth than to the periodontium. Wearing away of the teeth from bruxism may result in an unsightly reduction in the length of the crown and disturbances in interproximal contact relationships; it may lead to pulpitis, pulp exposure, or pulp death. Sharp, irritating enamel margins, fractured or split teeth or restorations, and even apical strangulation of the pulp are other possible dental sequelae of bruxism.

Ramfjord and Ash.43
Table V - I

ULTIMATE STRENGTH OF DENTAL MATERIALS

<table>
<thead>
<tr>
<th>Material</th>
<th>Tensile Strength (PSI)</th>
<th>Compressive Strength (PSI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human Dentine</td>
<td>7,000</td>
<td>43,000</td>
</tr>
<tr>
<td>Human Enamel</td>
<td>1,500</td>
<td>58,000</td>
</tr>
<tr>
<td>Dental Amalgam</td>
<td>7,000-10,000</td>
<td>45,000-55,000</td>
</tr>
<tr>
<td>Gold Alloys</td>
<td>60,000-120,000</td>
<td></td>
</tr>
<tr>
<td>Composite Plastics</td>
<td>6,000-8,000</td>
<td>30,000-40,000</td>
</tr>
<tr>
<td>Unfilled Acrylic Plastics</td>
<td>4,000</td>
<td>14,000</td>
</tr>
<tr>
<td>Material</td>
<td>Knoop Hardness Number (KG/MM)</td>
<td></td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------------------------</td>
<td></td>
</tr>
<tr>
<td>Human Enamel</td>
<td>343</td>
<td></td>
</tr>
<tr>
<td>Human Dentin</td>
<td>68</td>
<td></td>
</tr>
<tr>
<td>Human Cementum</td>
<td>110</td>
<td></td>
</tr>
<tr>
<td>22K Gold Alloy</td>
<td>85</td>
<td></td>
</tr>
<tr>
<td>Unfilled Acrylic Plastic</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Composites</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Porcelain</td>
<td>460</td>
<td></td>
</tr>
<tr>
<td>Zinc Phosphate Cement</td>
<td>40</td>
<td></td>
</tr>
</tbody>
</table>
Add bruxism to the equation and an abrasive slurry of tooth and ground off composite filler particles and the wear factor on such a composite would be enormous. If such a restoration acted as an occlusal trigger because of its hypercontact or its coarse finish, to the occlusally sensitive bruxer, the wear would be characteristically magnified.

Lundeen, discussed occlusal morphologic consideration for restorative dentistry and stated that an analysis of the forces exerted on the natural teeth during chewing indicated that the occlusal contacts have a relatively benign effect on the teeth compared with bruxism.

Phillips discussed wear on composite resins and stated that in time, the resins show definite evidence of wear. This observation may seem somewhat surprising, since abrasion resistance tests employing brushing of the resin by a toothbrush and an abrasive slurry show superior wear resistance for the composite as compared with amalgam. The exact causative factor involved in this loss of material in stress bearing restorations has not been identified but it probably involves a number of mechanisms. Unquestionably, some of it may be attributed to a loss or "plucking" out of the filler particles under masticatory attrition. Since the wear pattern is usually typified as a general erosion of the entire occlusal surface, it is possible that a chemical corrosion of some type also occurs in the resin binder. This leads to a loss of the resin itself.
It could be assumed that if this "erosion factor" if it did exist, surely it would attack the composite at all sites, not just the occlusal surface.

G. Wear
In their article "Wear: Dental Materials and Hard Tissue", Moon and Draughn define wear as a time dependent removal of material from surfaces that are in motion relative to each other. In reference to the mechanisms of wear, the authors stated that wear phenomena of hard dental tissue and restorative materials are among the least understood of the physical characteristics that are significant in the performance and function of dental structures. Guidance in understanding clinical wear can be exacted from studies of industrial wear, which have led to the definition of several conditions and mechanisms considered to control wear processes. The predominant processes appear to include adhesive wear, abrasive wear, wear enhanced by chemical degradation of the materials, and wear resulting from mechanical fatigue.

Abrasive wear occurs when a hard, rough body slides over a softer material. Substance is removed from the softer material as asperities on the harder surface cut onto the softer material. This process is minimized if the harder material is smooth. Abrasive wear also occurs if hard particles are present between sliding surfaces. The abrading particles in this three-body wear situation may originate from external sources or may be formed by other wear processes acting in the system. Thus, adhesive wear processes can
generate particles which cause wear by a three-body abrasion process.

Resistance to wear is generally decreased when rubbing structures are exposed to a chemically reactive environment such as the oral cavity. For instance, when polymers are subjected to sliding in a reactive environment, the attendant deformation of molecular bonds can make the polymer more susceptible to chemical degradation processes. Some metallic materials, such as stainless steel and other chromium-containing alloys, form surface films of corrosion products which, in the absence of sliding, protect the material from further corrosion. If sliding action is present, the protective films may be worn away so that corrosive attack can continue.

Mechanical fatigue may be a very important consideration in the wear of restorative materials. As two surfaces slide across one another, cyclic stresses, due to loading and unloading, cause a lowering of the mechanical properties of the materials. For a long time there may be no change in material surfaces, but when the materials have been sufficiently weakened by the cyclic stresses, a fragment can be removed from a surface by abrasion or adhesion mechanisms. This forms a flaw in the surface which is susceptible to further damage. Depending upon their nature, the particles produced may cut into adjoining structures and cause abrasive wear.

The dominant processes of physiologic wear have not been established. The oral environment is complex, consisting of fluids with changing composition and pH, cyclic thermal conditions, and
cyclic mechanical forces exerted during mastication, tooth brushing, and bruxing. It is probable that in such a complex environment, all of the wear mechanisms are active with the predominant mechanism changing as the conditions of the environment and materials combinations change.

Factors affecting wear: The recognition of factors that affect wear is important in proper selection of materials and in the development of a treatment plan to establish occlusal conditions that minimize clinical wear. Factors which should be considered are; occlusal force, contact area, slide distance, surface roughness, frequency of contact, and environment. The effects of these factors are summarized in Table VI-I.

Force: The greater the magnitude of the force exerted during chewing and contact between teeth, the greater is the resulting wear. The heavy forces generated by the posterior teeth as compared to those of the anterior teeth enhance the wear of the posterior teeth and posterior occlusal restorations. Patients with heavier biting forces should be more prone to wear. Females, older persons, and patients with removable prostheses tend to exert lower occlusal forces, which should lower the wear rate.

Slide Distance: An increase in the length of the sliding contact between teeth will increase wear. Thus, the existing occlusion will be a factor in wear. Moon and Draughn stated that wear facets are not present in wear. Anterior teeth that contact while acting as guide planes to closure may often show
appreciable wear resulting from the long contact distances. Clinical evidence of this wear is most observed when porcelain anterior crown contact with natural teeth, especially canines.

As corollaries to the effect of force and contact distance, wear would be expected to be minimal on teeth that do not contact and greatest in malocclusion on the few teeth contacting with the greatest slide distances. Also, greater wear would be expected on restorations placed in supraocclusion.

**Contact Area:** The area of contact determines the stress level in opposing surfaces and thereby influences wear. A given masticatory force acting over a small area results in higher stress than the same force exerted over a larger area. The greater the stress, the greater the chance the surface will be damaged and wear will result. A sharp tooth cusp has a small contact areas which would promote wear and more rapid change in occlusal dimension. The effect of a change in area can be seen in laboratory tests as a change in the wear rate with time. Initially, the wear rate may be at its maximum because only the high spots may be in contact, creating a high surface stress. As the bearing surfaces wear in, a greater contact areas results and the wear rate drops. A similar change in the wear rate may exist with changes in a patients dentition. As the occlusal contact area of the teeth increased, the rate of loss of vertical dimension from wear decreased. An advantage of monoplane artificial teeth may be a larger occlusal contact area and an attendant lower stress.
Table VI - I

Factors Increasing Wear

- Increasing occlusal force
- Decreasing contact area
- Increasing sliding distance
- Increasing surface roughness
- Increasing contact frequency
- Increasing chemical action

Table VI - II

The Relative Wear of Gold Against the Listed Materials

<table>
<thead>
<tr>
<th>Materials</th>
<th>Relative Wear Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acrylic</td>
<td>0.3</td>
</tr>
<tr>
<td>Polished enamal</td>
<td>0.5 to 1.0</td>
</tr>
<tr>
<td>Gold</td>
<td>1.0</td>
</tr>
<tr>
<td>Polished porcelain</td>
<td>2.0</td>
</tr>
<tr>
<td>Rough glazed porcelain</td>
<td>3.0</td>
</tr>
<tr>
<td>Rough porcelain</td>
<td>5.0</td>
</tr>
</tbody>
</table>
Table VI - III$^{89}$

Relative Wear of Porcelain Against the Listed Materials

<table>
<thead>
<tr>
<th>Materials</th>
<th>Relative Wear Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gold (glazed porcelain)</td>
<td>0.6</td>
</tr>
<tr>
<td>Gold (rough porcelain)</td>
<td>2.1</td>
</tr>
<tr>
<td>Porcelain</td>
<td>5.0</td>
</tr>
<tr>
<td>Gold (polished porcelain)</td>
<td>6.5</td>
</tr>
<tr>
<td>Enamel</td>
<td>6.5</td>
</tr>
</tbody>
</table>

Table VI - IV$^{89}$

Comparison of Total Wear of Acrilic Resin in Opposition Other Materials

<table>
<thead>
<tr>
<th>Materials</th>
<th>Relative Wear Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acrylic vs gold</td>
<td>2.5</td>
</tr>
<tr>
<td>Acrylic vs acrylic</td>
<td>9.0</td>
</tr>
<tr>
<td>Acrylic vs enamel</td>
<td>28.0</td>
</tr>
<tr>
<td>Acrylic vs porcelain</td>
<td>29.0</td>
</tr>
</tbody>
</table>
Table VI - V89
Relative Wear Rate of Like Materials

<table>
<thead>
<tr>
<th>Materials</th>
<th>Relative Wear Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gold vs gold</td>
<td>1</td>
</tr>
<tr>
<td>Porcelain vs porcelain</td>
<td>5</td>
</tr>
<tr>
<td>Acrylic vs acrylic</td>
<td>9</td>
</tr>
</tbody>
</table>

Table VI - VI89
Clinical Comparision of Amalgam and Composite in Class I and Class II Restorations at Three Years

<table>
<thead>
<tr>
<th>Materials</th>
<th>% of Restorations to Lose Anatomic Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amalgam</td>
<td>5% to 10%</td>
</tr>
<tr>
<td>Composite</td>
<td>60% to 70%</td>
</tr>
</tbody>
</table>
Surface Roughness: A corollary of the effect of contact area is the increase in wear produced by increasing roughness of the surfaces. Rough surfaces increase wear because only the high spots are in contact, producing high localized stresses. The effect of a rough surface is evident when opposing dentition is worn by porcelain which has not been glazed or polished after adjusting the occlusion. A simple and highly practical procedure for polishing a restoration or natural tooth after adjusting the occlusion with a rough stone or diamond bur should help minimize the wear, which could result if the surface were left rough. In fact, wear is reduced if porcelain is polished or given a relatively smooth surface before glazing. A porcelain surface left rough after grinding will have the sharp scratches rounded over upon glazing, but the rounded ripples and grooves left in the surface after glazing will reduce the contact area, increase local stress, and thereby increase wear (see Tables VI-II, VI-III, VI-IV).

Frequency of Contact: The wear of materials and dental tissue takes on added importance in patients exhibiting bruxism. These patients increase wear because they are capable of exerting greater forces with their developed musculature and more frequent contact. Wear is increased with the accumulated number of occlusal contacts, as shown by the increased wear with age.

Environmental Factors: Environmental factors, such as low salivary pH, a coarse diet, exposure to abrasive atmospheres, lack of salivary flow, and poor or excessive oral hygiene may have marked effects on the wear of dental tissue and materials. The
chemical and mechanical environment in the mouth can cause decreases in the mechanical properties of restorative materials as time passes after placement. Reduced mechanical properties lead to accelerated wear.

There are other less defined factors that probably affect clinical wear. Among these factors are the coefficient of friction between contacting materials, properties of teeth as affected by genetics and nutrition, and mechanical properties of materials.

When Table VI-V is compared to the other tables, some justification is apparent for a clinical rule of thumb that like materials should oppose each other where possible. The total wear of like materials in opposition is usually less than the total wear of different combinations. The idea of using like materials in opposition agrees with the results of abrasive wear tests which have shown a rapid decrease in the wear rate as the hardness of the abrasive particles was decreased toward the hardness of the surface being abraded. There, the mode of wear is changing from an abrasion to fatigue and adhesive processes which are slower. Abrasion wear is evident when a rough hard surface of porcelain slides against softer acrylic or gold. Also, porcelain is harder than enamel and will abrade it if rough. Tables VI-II, VI-III, and VI-IV support these statements about abrasive wear.
H. Restorative Technique

As well as considering the choice of restorative material in the bruxing patient, the clinician has to give some recognition to restorative technique. Lundeen stated that during chewing with a "normal" arrangement of natural teeth, the muscles return the mandible to the intercuspal position with repeated accuracy. This position is a remembered tooth position which is constantly reinforced through acquired neuromuscular reflexes. A tooth with a "high" restoration may be avoided reflexly during chewing, but it can develop wear facets through bruxing. The chewing forces generated on the natural teeth, in the absence of advanced periodontal disease, are not likely to produce pathologic changes within the masticatory system. Therefore chewing activities seem to play a secondary role in establishing a basis for the occlusal morphologic design of restorations for the natural teeth.

Bruxing movements can produce tooth contacts with sustained forces occurring throughout the range of horizontal mandibular movement the condylar portion of the movements characteristics are simulated by adjustable dental articulators. These are the movements that are consulted to detect occlusal interferences that can lead to increased muscle activity and pathologic changes. They are of primary importance in the occlusal morphologic design of fixed restorations for the natural dentition.

Therefore it is understood that the appropriate dental restorative material is utilised, its occlusal morphology is
developed with an appreciation of bruxistic activity, in particular, no occlusal interference is introduced including this extreme range reached in bruxism and it is finished to a high standard for the benefit of the physical properties of the material and also to provide no occlusal trigger for bruxism.

Schulman and Pentel\textsuperscript{90} discussed the use of materials when occlusion is being considered, stressed basic operative techniques such as cavity design, bulk of material, correct handling of the material etc. It can be understood that, whereas, many mouths contain amalgam restoration of a very average or less than average standard, these restorations will come to grief with the pathologic load of bruxism e.g. the class II amalgam fracturing at the isthmus because of inadequate bulk of material. The frequently observed clinical sign of bruxism, the amalgam with badly fractured margins involving fracturing of the cavo surface enamel as well as the amalgam margin. Would this have still occurred if all the unsupported enamel was trimmed during the cavity preparation and if the marginal amalgam was appropriately manipulated, condensed and finished?

This theme is continued by Dunn\textsuperscript{91} in his paper on advanced operative dentistry. The emphasis is in technique being that when performing restorative dentistry on a patient with a history of bruxism, it's back to basics.
I. Dentures

The effect of bruxism on dentures is related to the effect of bruxism on teeth. If the habit is principally grinding and the artificial teeth are porcelain, they will be fractured, worn or split. If the teeth are acrylic, they will be excessively abraded. If the habit is primarily clenching, there will be a loss of vertical dimension, and inflammation of the denture bearing tissues, excessive resorption in that the lower denture is being forced through the mandible.

Klein stated that bruxism may be involved whether the dentures are well or poorly constructed, but where the dentures are well constructed, bruxism is unmasked. A loose denture and constant clenching to seat it, soreness with the tongue subconsciously lifting a denture to unseat it and greater clenching to seat it are symptoms indicating bruxism. No tissue conditioning can help unless the cause of the problem is eliminated.

In the partially edentulous dentition, it is a combination of the effect on the natural teeth and on the prosthesis. With tissue borne acrylic dentures there is excessive shrinkage and gross damage and inflammation to the gingival tissues associated with the dentures. With distal extension partial dentures there is a rocking between the support frame of the denture and the edentulous saddle, even with frequent relining. Further, with chrome cobalt cast partial dentures there is constant fracturing of the clasp arms as they are bruxed upon directly, or the denture as a whole, is stressed laterally during parafunction.
CHAPTER 9

THE EFFECT OF BRUXISM ON THE PERIODONTIUM
THE EFFECT OF BRUXISM ON THE PERIODONTIUM.

According to Ramfjord and Ash,\(^4\) any tissue changes associated with traumatic occlusion may, of course, be the result of bruxism. However, since these tissue changes are confined mainly to the periodontal tissues apical to the alveolar crest, it is generally believed at the present time that bruxism does not initiate gingivitis or pocket formation. The role of bruxism and associated traumatic occlusion in the aetiology of periodontal disease is controversial and not fully understood.

It is emphasized that bruxism does not necessarily lead to pathologic changes in the periodontal tissues. In most individuals with fairly normal periodontal support, the common sequelae of bruxism are compensatory hypertrophy of the periodontal structures, thickening of the alveolar bone, increased trabeculation of the alveolar process, wider than normal periodontal membrane made up of heavy collagenous fibres, and a well-developed fibre attachment to the cementum. The effect of severe bruxism, at least in most younger individuals, closely resembles the adaptive periodontal reaction to heavy function reported in Eskimos and Australian aborigines.

The potential for periodontal injury from bruxism is generally dependent upon the factors that predispose to traumatic occlusion. The greatest amount of periodontal damage from bruxism occurs in patients with steep cusps when lateral stress is applied to the tips of these cusps. Stress on the tip of a cusp has a longer arm of
leverage than stress applied to the central fossa, and the stress on
the cusp is often directed outside the supporting tissues of the
tooth. Buccolingual stress is also of greater significance than
mesiodistal stress if the teeth have good interproximal support from
normal contacts. If the stress is placed on a few teeth, either
because of loss of teeth or severe occlusal disharmony, the potential
for injury from bruxism increases. The same potential for injury is
present when there is lost periodontal support resulting from
advanced periodontal disease. It is conceivable, although not
proved, that bruxism increases the potential for periodontal injury
in such systemic conditions affecting the collagenous support of the
teeth as scurvy and protein deficiency, especially in young
individuals. The significance of bruxism in the cause of periodontal
disease depends on whether bruxism results in trauma from occlusion.
Trauma from occlusion may accelerate the progress of destructive
periodontal, thus, bruxism may play a role in loss of attachment for
patients with periodontitis. Advanced periodontal breakdown due to
bruxism may also necessitate splinting of the teeth.

It has been claimed that periodontal disease predisposes an
individual to bruxism by increasing the tonus of jaw muscles. Oral
discomfort and movement of the teeth associated with gingival and
periodontal inflammation may initiate occlusal interferences and
thereby provide a "trigger" for bruxism. The increased muscle tonus
from the discomfort associated with the inflammation increases the
likelihood for this triggering factor to precipitate bruxism. Under
these circumstances, it may be said that periodontal disease initiates
bruxism. Recent studies have failed to support these claims.
Arnold states that the periodontal ligament (peridental membrane) is the highly vascularized narrow band of connective tissue that is the buffer between the tooth and the alveolar bone. It is well suited to this function because of the bands of collagenous fibres that go from the cementum to the bone. These fibres (Sharpey's fibres) absorb a great deal of the occlusal stresses, both physiologic and parafunctional. It is aided in this function by lymph and blood. To the extent that the ligament can absorb the occlusal forces and can convert them to stimulating tension on the lamina dura, it may have a salutary effect on the bone. When the physiologic limits are reached and exceeded there may be hemorrhage, thrombosis, and degeneration. This will be manifested as thickening in the radiograph. This widened ligament may be uniform or in the shape of an hourglass, with the thinnest part in the midroot area around which the root is rotating. Clinically, we may see an increased mobility.

This ligament is the home for cementoblasts, cementoclasts, odontoclasts, and other cells. It is not far fetched to believe that root resorption and cementum resorption and deposition could be initiated by traumatic occlusal forces.

Cementoblasts, which are present in the apical third of the root, are undoubtedly responsible for the clublike deposition of cementum called hypercementosis. Hypercementosis and cementomas are probably responses to occlusal forces delivered to the periodontal ligament and, by way of the collagenous fibres, to the cementum.
Lamina Dura and Alveolar Bone: The bone immediately surrounding the teeth involved in bruxism may show effects both from the pathologic changes in the pulp and the direct pressures of these teeth through the periodontal ligament on the lamina dura.

A pulp that has become necrotic as a result of occlusal trauma will send the breakdown products out by way of the ligament to the periapical bone, or the result will be a lysis of the bone with a radiolucent lesion, a granuloma, or abscess. Less frequently, a radiopacity such as condensing osteitis from a partially vital pulp may be seen.

The forces transmitted to the lamina dura directly will either be pressure or tension depending on the type, quality, and quantity, and the intervening fibres. Pressure stimulates the osteoclasts, whereas tension will affect the osteoblasts. The results may be seen as either a thinning or loss of lamina dura or a thickening even to the point of bone condensation. Very often what will be seen is the aforementioned hourglass effect where the lamina dura is thick around the apical and gingival portions of the tooth with a thin small area somewhere in between. This is due to the tendency of the tooth to rotate under pressure with the thin band acting as a fulcrum.

The clinical signs or the effects of bruxism may be seen on the radiograph as localized or general radiolucency or as a condensation, or they may be seen by the effects on the teeth, such as tilting, migration, and mobility. Another element difficult to evaluate predictably is the genetic or bone factor. The net effect on bone is
a product of both the stress and the ability to react to it. Precocious bone loss, fenestration, and bone whorls may very well be examples of a combination of factors.

There has been some controversy about the origin of infrabony pockets, with most periodontists arguing that occlusal factors cannot cause them without the presence of local irritants (calculus). On the other hand, there are some who still implicate occlusal factors, with bruxism as the prime culprit.

**Gingivae:** The effect of parafunctional habits on the gingivae has also been fraught with a great deal of controversy. One group argues that dystrophic changes such as bleeding, congestion, recession, gingival festoons and clefts, blunting, and even periodontal abscesses are closely involved with bruxism. The feelings about this cover the spectrum from no effect to partial contribution to total acceptance.

Glaros and Rao⁸⁰ in their literature review on the effects of bruxism reported that isometric muscle contraction cannot be the only cause of periodontitis. Ramfjord⁴³ has interpreted this finding that periodontal disease increased the tonic contraction of the jaw muscles as indicating that periodontal disease predisposes individuals to bruxism. On the other hand it has also been stated the the role of bruxism in periodontal disease is minimal. It was stated that bruxism does not produce the periodontal pocket and has no effect upon the gingival unit. For periodontal disease to occur, two separate lesions must be produced; one of the margin (gingival
unit) and the other of the attachment apparatus. Bruxism affects only the attachment apparatus and thus is only a contributing factor to periodontal disease. It is further stated that traumatic breakdown occurred without pocket formation or apical migration of the gingival margin. It appears that .... bruxism in itself cannot initiate the periodontal lesion. However, the phenomenon can aggravate and contribute to the existing disease process.

Resorption of the alveolar bone has also been considered a common symptom of bruxism.\textsuperscript{80} An experimental study is reported, in which rats and hamsters were mechanically stressed to stimulate the forces on the dentition created by bruxism. Histologic examination revealed extensive ostoporosis of alveolar bone.

Along with alveolar bone loss, bruxism may also contribute to the loosening of the teeth in the alveolus. Research on this relationship has been carried out by O'Leary et. al.\textsuperscript{93} Three bruxist and four nonbruxists were subjected to stress-inducing conditions for thirty days. The subjects had to carry out complex work situations daily within set time intervals, and they were confined to small areas. The authors recorded tooth mobility measurements daily. They found that mean tooth mobility increased significantly for the bruxists, while the nonbruxists did not display any changes. Withdrawal from stress-inducing conditions reduced tooth mobility in bruxists to the pre-experimental level, but mobility increased again six days later when the subjects were given a battery of psychological tests. Two days after the administration of the tests, tooth mobility readings once more returned to the pre-experimental
level, thus suggesting that the forces associated with bruxism are a source of tooth mobility.

According to Graf,\textsuperscript{3} analysis of tooth mobility measurement during applications of small and big forces of short and long duration point to the important factors which are responsible for the mechanical resistance of the periodontium against functional stress. For small forces of short duration, a hydrodynamic system which is in part dependent on the vascularisation seems to be of primary significance. With medium transient forces, the resistance seems to depend on the number and state of the collagen fibres and on the degree of polymerization of the ground substance. Long and excessive forces on the tooth produce deformation of the bony socket and the dentinal nucleus. The stress on tissues may have irreversible sequelae.

Posselt\textsuperscript{2} stated that tooth wear during empty contact movement may result if movements are carried out during a long period of time and by relatively low pressure whereas loosening of the teeth may be initiated if high pressure is exerted during shorter periods. The total amount of force may be the same in both cases, only the result is different.

Wank and Kroll discussing the periodontal aspects of occlusal trauma\textsuperscript{46} stated that occlusion has been suspected as an aetiologic factor in the pathogenesis of periodontal disease since the beginning of the 20th century. More recently, in the past 20 years, bacterial plaque has been shown to play a dominant role in the initiation and
progression of gingivitis and periodontitis. These two major aetiologic agents, occlusion and local factors, may act independently or together.

The local toxic entity that is primarily responsible for marginal gingivitis is bacterial plaque, although dental calculus and materia alba may also be involved. Iatrogenic factors such as ill fitting margins may result in increased accumulation of plaque and calculus.

Excessive occlusal overloading is characterised by dystrophic changes in the periodontal ligament, alveolar bone and cementum. These changes are reversible in the absence of inflammatory agents such as toxins. When periodontal inflammation has caused an alteration of the attachment apparatus, in the presence of occlusal overloading, a condition known as secondary occlusal traumatism results.

Pathologic occlusion may result in impaired masticatory function, poor phonetics, non-inflammatory periodontal disease, positive occlusal sense, and tooth migration. These patients can also exhibit retrograde wear, myofacial pain syndrome, and pulp disease. Retrograde wear signifies a pathologic situation when the rate of occlusal wear increases beyond the capacity of the dentition to compensate for such wear. Pulpal degeneration may result from occlusal trauma when excessive force is applied to teeth that remain firmly seated in the periodontium. In such a situation the lack of tooth mobility has a deleterious effect. This is in contrast to the
postulated harmful effects that tooth mobility may have in certain cases.

The major factors that determine whether occlusal forces will injure the periodontium are the existence of parafunctional habits such as clenching and bruxing and the status of periodontal health during the episode of occlusal malfunction. Moreover, the patient's health and age in general must be considered when evaluating the repair capability of the host.

**Primary occlusal trauma:** from occlusion is believed by some to result from nonfunctional activities. In this situation the masticatory muscles may contract isometrically, yielding a rather large force (up to 250 PSI). The periodontium can be healthy or diseased. In the absence of inflammation this trauma has been referred to as pure primary occlusal traumatism. Primary occlusal trauma is reversible.

**Secondary occlusal trauma:** is caused by the direction of masticatory forces on teeth that are held with a severely compromised attachment apparatus (overfunction). In this instance, the muscle may contract isotonically, resulting in a physiologic force of approximately 2 to 15 PSI. Secondary occlusal trauma is irreversible.

In essence, primary occlusal trauma is caused by excessive occlusal forces (that is, forces that are greater than those experienced in normal function) applied to teeth that are held within
adequate supporting structures. Secondary occlusal trauma is the result of "normal" occlusal forces directed on teeth that are inadequately supported by the periodontium.

It has been noted that excessive occlusal forces cause changes in the periodontal tissues apical to the alveolar crest of the involved area. Gingivitis is characterized by supraperiosteal inflammation. Because this area of gingival inflammation does not directly communicate with the region apical to the alveolar crest, it is not surprising that trauma from occlusion does not seem to affect the magnitude of gingivitis. Moreover, evidence shows that periodontal pocket formation is not caused by occlusal trauma.

It is asserted that occlusion affects the development of periodontal disease and that periodontal inflammation can exist with or without occlusal pathology; however, the two are related. It has been suggested that traumatic occlusion can influence the pattern and magnitude of tissue destruction resulting from periodontitis. The pathway of inflammation observed in monkeys and humans was thought to be altered by trauma from occlusion. The resultant injury was characterized by angular bone loss and infrabony pockets. In a subsequent study, it was suggested that the severity of occlusal trauma and inflammation might determine whether infrabony pockets and angular osseous defects would result. Also, it is believed that the injury due to occlusal trauma must be located immediately subjacent to the periodontal inflammation in order to result in codestructive effects.\(^{94}\) "Gingival inflammation and trauma from occlusion are different types of pathologic processes which participate in a single
disease, periodontitis. Together they exert a combined codestructive effect which produces angular bone defects and infrabony pockets."

Results that support the "co-destruction" hypothesis are stated.\textsuperscript{94} Work with Beagles, on experimentally induced trauma from occlusion (jiggling forces) did not result in the loss of connective tissue attachment or the formation of periodontal pockets in animals with normal gingivae or even in animals with marginal gingivitis. However, in dogs with rapidly progressing periodontitis, occlusal trauma resulted in the induction of increased tooth mobility and an increased rate of tissue destruction in the periodontium as well as angular osseous defects and infrabony pockets.

In contrast to the "co-destruction theory", Drum\textsuperscript{31} advanced the theory of "autodestruction" as mentioned in an earlier section of this review. The concept, according to Drum, is that both inflammatory and non inflammatory dystrophic types of periodontal diseases began with a non-infectious traumatic destruction of alveolar bone, caused by traumatizing parafunctions and followed by changes in the gingiva. The latter may or may not be complicated by superimposed infectious inflammatory process. A new concept to fit these facts is proposed: the concept that periodontal diseases are autodestructive processes in the masticatory system, with or without superimposed infectious inflammation. Acceptance of the proposed concept requires evidence that:

1. traumatizing forces exist in the masticatory system
2. traumatic destruction of alveolar bone occurs
3. changes in the gingiva follow destruction of alveolar bone
4. all persons afflicted by periodontal diseases exert parafunctions

It is further proposed that the traumatising forces of parafunction act on the tooth, the root of the tooth and the periodontal tissues. They do not act directly on the gingiva, because the gingival tissues are connected with the tooth in form of a cuff, which is firm, yet mobile, yielding to mechanical forces. This subterranean attack on the gingiva can lead to

1. retraction of gingiva
2. McCall's festoons

The genesis of pocket formation under this concept is again via the initial lesion in the alveolar bone leading to the dystrophic changes in the gingiva and the pocket is an extension of the same thesis.

An attempt has been made in this section to present a range of hypotheses on the effect that bruxism has on the periodontal structures. It is to be remembered that when bruxistic trauma afflicts the dental component of the masticatory apparatus, that trauma will be manifested in the teeth or the supporting structures or to a lesser extent both. The minor manifestation will be minor tooth wear or minor mobility. In a major manifestation in an either/or case there would be exhibited very firm, very worn teeth or
very loose, hardly worn teeth; individual tissue response. In the periodontal cases the range of damage will progress from slight mobility to gross periodontal disease either inflammatory or infective. The initial discussion as presented in this section explained the obvious physical development of this extension with a cross section of literature support. Information was then provided of the "Co-destructive theory", parafunction plus local factor. This concept appears reasonable in the light of our increasing knowledge on plaque. There are many examples in the literature of solving the problem with scrupulous plaque control.

The universal formula:-

\[
\text{GENERAL DISEASE} \times \text{LOCAL FACTOR} = \text{ORAL MANIFESTATION}
\]

We can't always remove the general disease but when plaque is the local factor and we have the patient who is able to practice total plaque management, we then eliminate our oral manifestation of the condition.

The auto-destructive concept would not receive universal acceptance, even though it is fascinating because of the emphasis it places on parafunction as the aetiologic factor in periodontal disease. The very common incidence of periodontal and occlusal factors might merit such a concept, but bruxism is almost universal anyway, definitely more universal than periodontal disease. However, with the emerging knowledge of the role that the auto-immune system plays in periodontal disease, predisposition may generate future interest in the concept of auto-destruction.
CHAPTER 10

TREATMENT OF BRUXISM

A. OCCLUSAL SPLINTS
B. OCCLUSAL TREATMENT
C. OCCLUSAL RECONSTRUCTION & PROSTHESES
D. POST TREATMENT SPLINTS
E. ADJUNCTIVE THERAPY TO OCCLUSAL SPLINTS
F. PSYCHOTHERAPY
G. BIOFEEDBACK
H. MASSED PRACTICE EXERCISES
I. HABIT CHANGE
J. DRUGS
K. ADJUNCTIVE THERAPY
L. OTHER FACTORS

DISCUSSION
TREATMENT OF BRUXISM

According to Ramfjord and Ash,\textsuperscript{43} bruxism is of extreme clinical importance in treatment planning. It is essential to successful treatment of any disorder of a dysfunctional nature that the aetio logic factors be recognized under the diagnostic procedures and that the causative factors be eliminated. The complexity of the aetiology of bruxism and the diagnostic problems already discussed make it easy to understand the current state of confusion and controversy that exists about the treatment of bruxism. Because of a lack of conclusive diagnostic criteria it is also very difficult to prove satisfactorily whether bruxism has been eliminated by any given treatment procedure.

From a clinical and practical standpoint bruxism should be reduced below the level at which it is capable of producing recognizable harm to the teeth, the periodontium or any other part of the masticatory system. This result does not necessarily mean that the individual never clenches or grinds his teeth; it does indicate that the vicious cycle between habitual bruxism and increasing muscle tension (the neuromuscular "feedback" mechanism) has been broken and bruxism eliminated as a pernicious habit.

Since bruxism has a dual cause that includes psychic and local occlusal factors, a rational treatment should include the elimination of both disturbing aetiological factors. And, since both psychic tension and a local trigger factor have to be present to initiate
bruxism, this dysfunctional habit can be eliminated by either psychic or local therapy. It has been shown conclusively that this is true for grinding or eccentric bruxism, but it is not entirely clear whether local therapy has the same degree of importance in the elimination of clenching or centric bruxism as does psychic therapy.

Another confusing aspect of bruxism is related to the threshold values for tolerance of occlusal interference. Depending on variations in the patient's state of psychic stress, the same occlusal interference that acts as a very potent trigger factor for bruxism one week may or may not bother the patient or precipitate bruxism the next week. The identical occlusal interferences may trigger bruxism in one individual and be of no consequence in another, again depending on the degree of psychic stress. In order to eliminate bruxism, one has either to lower the threshold for neuromuscular irritability below the point where the patient's occlusal interference does not act as a trigger for bruxism, or enough occlusal interference has to be removed to get within the tolerance limit for the patient's neuromuscular mechanism. The very best treatment of bruxism is to influence both the psychic and occlusal factors in a favourable way.

It is proposed to discuss the different treatment methods separately and distinctively without, at this stage, entering the debate as to which is the most appropriate treatment modality.
A. Occlusal Splints.

According to Ramfjord and Ash the purpose of an occlusal bite splint include:

1. the control of trauma from occlusion and muscle hyperactivity,
2. the elimination of pain and discomfort associated with TMJ-muscle pain dysfunction,
3. the control of bruxism and prevention of excessive tooth wear,
4. the reduction of muscle hyperactivity in order to facilitate registration of jaws in centric relation,
5. the differential diagnosis of disorders causing symptoms similar to those related to the TMJ-muscle pain syndrome,
6. the retention of maxillary teeth following orthodontics,
7. the disclusion of teeth during orthodontic treatment, and
8. the stabilization of hypermobile teeth.

The design of the occlusal splint is based on mechanisms proposed for its function and ease for adjustment. The principles and rules are related to:

1. the absence of surface characteristics that would interfere with mandibular movement for optimal positioning of the condyles and closure of the mandible in a stable position in swallowing, bracing for physical exertion, and "tap centric"; and

2. the presence of surface characteristics that provide for cuspal guidance to eliminate balancing and working interferences and
Figure 30. The occlusal splint.

Figure 31. The Occlusal Splint.
prevent contact of mandibular incisors in protrusive movements. In effect, there should be no premature contacts in centric, no contact of posterior teeth in lateral or protrusive movements, no contact of incisors in lateral or protrusive movements, no working or balancing contacts away from "splint centric," and freedom in splint centric of approximately 1-2mm, depending on contact relations of the natural occlusion (anterior and lateral slide).

The thickness or the vertical dimension of a splint is determined by biomechanical as well as physiological and psychological factors. In order to assure the principles listed previously, the vertical dimension depends largely on the curve of Spee, the curve of Wilson, balancing side contacts, and the vertical overlap of the maxillary anterior teeth. The more pronounced the curve of Spee, the greater the vertical dimension. The greater the balancing side contact, the greater the vertical dimension of the cuspid rise. The greater the cuspid rise, the narrower the splint may be. Furthermore, the greater the vertical overlap of the maxillary anterior teeth, the thicker the splint must be to avoid entrapment of the mandibular incisors. The vertical dimension should be greater or lesser than that at which clicking, locking, or tipping of the joint occurs. While the splint should be as thin as possible, the thickness must be consistent with swallowing, speaking, and lip seal; thus, a compromise must often be sought between the thickness of the splint and the height of the cuspid rise.

Of considerable importance in the design of the splint is the need to prevent entrapment by incisal guidance. It is usually easier
to increase the vertical dimension to the approximate level of the maxillary incisor edges than to greatly increase cuspal guidance to avoid incisal guidance. Such a vertical dimension is necessary for patients who brux on anterior teeth.

According to Scharer the insertion of an occlusal splint prior to occlusal adjustment is logical. Because of "muscle splinting", very often the original border relationship cannot be obtained and any immediate occlusal adjustment could be inaccurate. The use of occlusal splints as a first step in bruxism treatment is advisable because such an appliance should remove all tooth contact relationships which could have acted as a trigger for bruxism.

Mejias and Mehta subjectively and objectively evaluated the effectiveness of short term bite splint therapy on bruxistic behaviour. A simple bruxism monitoring device was used to check pre treatment and post treatment nocturnal bruxism. They reported that short term splint therapy seems to be effective in the reduction and elimination of mild to moderate symptoms of pain and dysfunction caused by nocturnal grinding. In this study, all subjects reported feeling better and being free of pain at the end of therapy. Occlusal analysis revealed that there was a definite shift in the maxillo-mandibular relationship in favour of a more distal position of the mandible by about 0.5 - 1.0 mm. This tendency seems to be brought about by progressive relaxation and re-organisation of muscle groups involved in the positioning of the mandible.
In a key study Solberg, Clark and Rugh\textsuperscript{96} investigated eight confirmed bruxists using portable E.M.G. equipment. The results indicated that nocturnal bruxism can be significantly reduced through maxillary stabilisation splint therapy. Although the reduction appears immediate it is not permanent.

Kopp\textsuperscript{97} states that occlusal disturbances may be temporarily eliminated by occlusal splints. This "occlusal" treatment has the advantage of being reversible. The splint works through the prevention of tooth to tooth contact, thereby changing sensory impulses from the periodontal receptors and stabilizing the occlusion and the position of the mandible against the maxilla.

Fuchs\textsuperscript{98} in a sleep investigation mentioned the clinical acceptance of occlusal splint success. He adds it is probable that in those patients successfully treated the increase of activity of the chewing muscles during sleep was responsible for their dysfunction. The forces that affect, for example, the periodontal ligament in the conscious person are normally maintained by sensory receptors which ensure that tissue damage does not occur. At night these protective controls are reduced or even absent.

In a further nocturnal E.M.G. study Clark, Solberg, Rugh and Beemsterboer\textsuperscript{99} investigated twenty-five patients and observed a decreased nocturnal E.M.G. activity for 52\% of patients. A return to pretreatment E.M.G. levels after removal of the splint was noticed in 92\% of the patients.
As a point of interest Lehman\textsuperscript{100} reports on a "hydrastatic" splint that uses a fluid filled flexible splint with appropriate success. Apparently such a device has been marketed for sale in pharmacies so that you can fit your own splint.

B. Occlusal Treatment.

It is understood that splint therapy precedes occlusal treatment. It is necessary to utilise the reversible nature of splint therapy to test the diagnosis and further to optimise the maxillo mandibular relationship prior to adjusting the teeth. It appears that the elimination of the occlusal trigger areas (occlusal interferences) works favourably in the clinical treatment of bruxism. Ramfjord and Ash\textsuperscript{43} state that combined clinical and E.M.G. studies have shown that bruxism may be eliminated, or at least controlled beyond the stage when it poses a clinically recognisable problem, by precise occlusal adjustment. About 75 patients have been observed over various periods of time up to 5 years and the results indicate success. A few of the patients have experienced temporary relapses to bruxism associated with re-occurrence of occlusal interference, in most instances as a result of new dental restorations. Minor occlusal adjustment has again alleviated their bruxism.

A primary objective of occlusal adjustment is improvement of the functional relations of the dentition in such a way that the teeth and the periodontium will receive uniform functional stimulation and the occlusal surfaces of the teeth will be exposed to an even
physiologic wear. The masticatory system is a functional unit, and proper functional stimuli are of the utmost importance for the development and maintenance of a strong and healthy periodontium with high functional capacity and optimal resistance to injury. The functional self-cleansing of the surfaces of the teeth, with prevention of marginal gingivitis from plaque retention, is also enhanced by normal multi-directional occlusal function. It has been shown that occlusal adjustment may induce multi-directional functional pathways if the adjustment results in equally convenient and efficient functional relations in the various directions.

Sheppard and Price⁴¹ mention the need to remove all rough surfaces on tooth enamel and restorations so as not to leave any occlusal trigger factors. Krogh-Poulsen and Olsson⁴² stress the need of reshaping the bruxofacets so as to alter the proprioceptive impulses in the periodontal tissues of the involved teeth and thus cause a change in the muscle function pattern and a shift of the mandible in such a way that the involved muscles relax and functional disturbances disappear.

C. Occlusal Reconstruction and Prostheses

According to Ramfjord and Ash⁴³ restorative dentistry is indicated in the treatment of bruxism when a stable, well-balanced occlusion cannot be established through occlusal adjustment alone. Occlusal restorations may also be indicated in order to substitute for or prevent excessive loss of tooth substance by bruxism.
for esthetic and technical reasons the occlusal vertical dimension has to be raised, the increase in vertical dimension should be kept to a minimum. Also, in centric the restorations should have occlusal contact with all of the teeth in the opposing dental arch in order to maintain a stable result. It is essential that the occlusal pattern in such restorations be as ideal as possible in order to minimize the tendency for bruxism and to prevent future occlusal wear. The restorations should also be of the same degree of hardness to prevent uneven wear. It is advisable to let the patient wear an occlusal splint for 2 to 3 months before the final recording of centric relation is made. There is often a very marked change in jaw relations following the use of an occlusal splint. Reconstruction done after such repositioning of the mandible is usually successful because the discrepancy between centric relation and the abnormal position associated with bruxism has been eliminated. If the patient's occlusal wear pattern from bruxism is simply duplicated in the restorations, as has been recommended, the bruxism and excessive wear will continue. A faulty occlusal pattern in oral reconstruction for patients with bruxism may lead to increased bruxism, destruction of the restorations, and temporomandibular joint and muscle pain.

What has been said about bruxism in individuals with natural teeth also applies to patients with removable partial and complete dentures. Instead of adapting to a less than perfect denture occlusion by establishing learned convenience patterns for the masticatory movements, patients will seek out occlusal interferences and start playing with their appliances as an expression of bruxism. The results may be "denture sore mouth," excessive wear of both
dentures and opposing teeth, breakage of appliances, and temporomandibular joint and muscle discomfort or pain.

Several patients with bruxism have been seen in whom good natural teeth have been extracted and complete maxillary dentures made with the hope of improving the bruxism and providing relief for oral or temporomandibular joint discomfort. Unfortunately, such patients usually end up with a series of ill-fitting dentures and increasing discomfort and occlusal problems that could have been handled much more satisfactorily if the natural teeth had not been extracted.

When a patient has complete dentures and severe bruxism, it is very difficult to assure a correct centric relationship unless the dentures are left out of the mouth for some days or a temporary flat acrylic splint is made on top of the occlusal surface of the teeth. The splint is then adjusted and worn for 2 or 3 weeks until muscle relaxation can be obtained and a reliable recording of centric relation secured.

Occlusal adjustment of dentures for patients with bruxism can usually be done better with a good mounting on an articulator than in the mouth. The presence of tense jaw muscles and poor coordination of jaw movement, combined with the resiliency of the mucosa, makes intraoral adjustment highly problematic for such individuals.

Within the ambit of restorative dentistry to the bruxistic patient is any other necessary dental treatment to optimize and
stabilise the occlusion. This in various cases might include orthodontic, periodontic or surgical procedures.

D. Post Treatment Splints.

Scimone\textsuperscript{103} mentions that the life expectancy of existing amalgam restorations can be further extended by a more wide spread use of acrylic resin nightguards, worn on either upper or lower arches. Arches can be alternated at intervals, so that wear can be distributed more evenly. Scimone regularly constructs nightguards for all patients with extensive porcelain fused to metal crowns or fixed bridgework. This minimizes the occlusal wear and reduces the risk of porcelain fracture.

As has been previously stated effective therapy will correct or minimise the habit of bruxism. Few clinicians would claim total success. Further it is unusual to have a patient accept total refurbishing of the mouth, matching restorative materials etc. Further again, major reconstruction work normally involves porcelain fused to metal and gold restorations, not always perfectly matched. For these reasons, having the patient continue wearing the splint while sleeping is good insurance. Having been through splint therapy and occlusal adjustment, the patient has grown quite used to the splint and appreciates the added comfort afforded. The continued use of a splint will maintain the muscle harmony, minimise wear and destruction to teeth and restorations and complex bridgework if any bruxism should continue, albeit at a reduced level. Some clinicians modify this treatment by the continuing splint usage only in times of
stress.

Another situation that recommends continuous splint use is the protection of porcelain jacket crowns. Frequently, aesthetics demand porcelain over porcelain to metal and if there are obvious signs of bruxism, the porcelain would be best protected by a nightguard. Some porcelain enthusiasts claim the observation of compensating wear on porcelain offsetting bruxistic fracture. This is more a reflection of the wearing of porcelain as demonstrated in the section on the effect of bruxism on restorative materials. In this instance the porcelain needs to be protected from both wear and fracture.

E. Adjunctive Therapy to Occlusal Treatment.

Brief mention should be made of aids that assist the relaxation of muscles, or removal of pain such that splint therapy and occlusal adjustment are increased in accuracy and reduction in muscular hyperactivity accelerated. Within this classification will not be included major treatment modalities such as biofeedback etc. which will be discussed separately. Appropriate adjuvants would include:

a. physiotherapy to joints and musculature.
b. trans cutaneous nerve stimulation for pain.
c. local anaesthesia, cortico-steriods and saline injection for pain.
d. iso-kinetic exercises.
Figure 32. Right lateral view of mounted study casts where gross occlusal deformity is illustrated.

Figure 33. Left lateral view. Treatment by occlusal adjustment cannot result in this case being completed.
Figure 34. Diagnostic casts, right lateral view.

Figure 35. Diagnostic casts, left lateral view.
Figure 36. Left view finished case. (mirror reversed.)

Figure 37. Right view finished case. (mirror reversed.)
Figures 32 - 38 demonstrated a simple restorative case following splint therapy and occlusal adjustment. The occlusal deformity required further treatment for stable occlusal balance and oral reconstruction was not desired by the patient. The combination of pre occlusal treatment, extraction, gross pre prosthetic tooth preparation and fabrication of partial upper and lower dentures has resulted in an acceptable result. The cast partials were waxed on mounted models and an acrylic platform was provided for lower anterior contact and function.

Figure 38. Anterior view, finished case.
e. myofunctional therapy and speech therapy for habit correction

F. Psychotherapy

Consideration will now be given to major treatment modalities aside from occlusal treatment. Many clinicians would employ one or more of these modalities as adjuvant therapy to their occlusal treatment. Other operators again would focus their entire treatment on this non-occlusal approach.

Psychotherapy aimed at lowering emotional or psychic tension has been suggested by some operators. However, a number of patients with bruxism have deep seated emotional or psychic disturbances which the dentist is not trained to evaluate or to treat. According to Bundgaard-Jorgensen,\textsuperscript{22} dentists should be very careful about getting involved in any kind of psychotherapy beyond counseling when he reported 3 suicides in a group of 50 adult patients being treated for bruxism. Twenty of these 50 patients sought psychiatric treatment and half were treated successfully. Psychoanalysis has also been recommended for patients with bruxism.

According to Ramfjord and Ash,\textsuperscript{45} there is no doubt that psychotherapy properly executed may reduce tension and at least temporarily eliminate bruxism. However, this is a complex and time-consuming therapy which should be reserved for patients who are in obvious need of such treatment. These patients should be told firmly that they need help from somebody who is better qualified to
help them with their problems than the dentist. Probably less than 1 per cent of all individuals with bruxism need psychotherapy, but the dentist can save himself and the patient from unnecessary disappointment and even harassment if he, during the examination and diagnostic procedures, can single out such patients.

The overwhelming majority of patients with bruxism do not need complex psychotherapy. Thus, in the majority of cases such therapy is impractical and of dubious value since a large number of patients can be helped through simple counseling by the dentist. An attempt should be made to explain to the patient the relationship between the bruxism and his emotional or nervous tension. The idea that the bruxism is an outlet for nervous tension is usually rejected vehemently by the patient and should never be argued. However, when the patient has had time and opportunity to think it over, it is usually stated during subsequent appointments that what was said about the bruxism was probably correct and has helped him to understand his own problems better. A dentist should be very careful not to probe too deeply into a patient's emotional problems since this approach may aggravate the instability of the psychoneurotic individual.

Rugh and Solberg\textsuperscript{45} discussed psychological management and suggested that when searching for a psychological component to the occlusal problem, the dentist need ask the patient but one question. "What does this problem stop you from doing?" Sometimes it will then be realised that the patient will be avoiding social life, work, motherhood, or a variety of other responsibilities. Thus, the
behaviour of the patient in face of his symptoms becomes an important aspect of treatment. Further if anxiety, hostility, fear or anger are detected, attempts should be made to determine if the emotional state is predominantly a result of the disorder or a cause of the disorder. Questions regarding work pressures, social adjustment, and traumatic changes in life style may help identify emotional antecedents.

The authors further state, when commenting on the success of different aspects of treatment that one factor common to all therapies is that of increasing the patients awareness of the oral cavity. Increased attention to oral behaviour may help the patient control or eliminate damaging oral habits. Another hypothesis is that involvement in any treatment may distract the patients attention from his normal stressful life style thereby reducing anxiety and relieving oral habits. Regardless of choice of treatment, it may be found that those clinicians who are successful demonstrate a "psychologic awareness" and accept the necessity of listening to the patients problems.

Another aspect of psychological treatment is to explain the genesis of the condition of the patient. A simplified discussion of tension and anger as it relates to elevated muscle tonus may be valuable. It should be explained that these reactions are typical of any individual experiencing stress. Understanding the conditions and being aware that there is nothing sinister associated with it is stress relieving for the patient.
G. Biofeedback.

Kardachi and Clarke\textsuperscript{104} define biofeedback as the voluntary control of internal psychological and physiological states. This has become an intensive field of research in recent years made possible by the rapid development in electronics. It has been used successfully to control stress related problems such as tension headache, increases and decreases in heart rate, changes in vasomotor tone and to obtain blood pressure control. In an experiment on nine subjects the results obtained showed that masticatory parafunctional activity may be significantly reduced by an appropriate biofeedback technique. The design criteria insured that the warning signal given to the subject was proportional in intensity, frequency, and duration to the bruxing activity, providing true feedback to the parafunctional activity. The authors summarise that a highly significant reduction in bruxism has been obtained using a biofeedback system. The concept that the aetiology of bruxism is related to emotional stress is supported as biofeedback has been successful in controlling other stress related parameters. The form of biofeedback used was an audible tone derived from amplified electromyographic data, relayed to the subject via an earpiece.

In a later paper, Kardachi, Bailey and Ash\textsuperscript{105} conducted a study on twenty subjects for the purpose of:

1. Determining the effects of an occlusal adjustment on muscle activity in people who brux.
2. Compare these effects to those of biofeedback.

They summarised that within the limits of the study, the results suggested that the effect of biofeedback in reducing E.M.G. activity is more consistent than an occlusal adjustment.

A study by Rugh and Johnson\textsuperscript{106} in 1981 states that several studies have demonstrated that nocturnal bruxism and related symptoms can be relieved through nocturnal E.M.G. feedback. The effects, however, are not always long lasting and the mechanism of suppression is not understood. It was the object of this study to examine more closely the manner in which nocturnal feedback works in suppressing bruxism. Chart recordings were made of nocturnal masseter E.M.G. activity in five bruxist subjects. Baseline recordings were made in the subjects home followed by 10 or more nights of feedback treatment. Treatment involved sounding a 300mw tone when E.M.G. activity exceeded about 20uv for more than 1 second. All subjects showed a decrease in the duration of bruxism. The decrease in bruxism was due to a reduction in the duration of bruxism episodes rather than a change in the number of episodes, i.e. rather than reducing the probability of an event starting, nocturnal feedback appears to simply suppress the activity once it is initiated. This provides little evidence of learning.
H. Massed Practice Exercises.

Hellen and Forgione,\textsuperscript{107} applied behaviour modification techniques of mass negative practise and relaxation training to separate groups of individuals who had been diagnosed as bruxers by multiple criteria. When assessed by an objective index of grinding, neither technique was found to have reduced bruxism significantly. Grinding and state of anxiety scores of bruxers were significantly higher than those of non bruxing controls. Bruxers did not differ from non bruxers on the trait anxiety measures.

Studies by Ayer and Gal\textsuperscript{8} and Ayer and Levin\textsuperscript{109} disagree with the previous study. Their results indicate a favourable improvement by massed practice therapy. With their approach the patients nocturnal grinding habits were treated by having him repeatedly clench his teeth together during the waking hours. At a one month post-treatment appointment the patients indicated they were still free of the habit.

I. Habit Change.

Rugh and Solberg\textsuperscript{45} state that the behaviourist views maladaptive behaviours as habits which are learned in the same way any behavioural response is learned. Treatment is directed at the specific habit or response rather than at a hypothetical internal disorder. In the case of bruxism, therapy would be directed at the behaviour of bruxism. Traditional psychoanalytical therapy on the
other hand would be directed at attempts to help the patient gain insight into unconscious suppressed desires or conflicts which the bruxism symbolises.

Zedlow\textsuperscript{110} talked about habit retraining thus: "The replacement of a bad habit with a good one is done by the brain and involves time, effort, patience, and reinforcement. Since the components of the masticatory system are under the control of the central nervous system, the contacting of the teeth for the purpose of clenching or bruxing becomes the conscious or unconscious "trigger" that institutes a sensimotor reflex. Frequently, recognition and awareness of this long existing habit will cause it to be removed autonomically and, over a period of time, a new reflex will cause a new habit to be introduced through a new fixed anatomic pathway. The newly instituted habit of functioning will eventually take precedence over the clenching or bruxing habit, making the use of tranquilizers, muscles relaxants, or splints unnecessary, except where a large number of teeth may be missing.

Although the institution of the new habit requires effort of both the patient and the clinician, the patient must realize that the problem is his and that his cooperation is mandatory. Retraining the patient into a new habit pattern involves three steps.

First, the patient must be made aware of his covert efforts resulting in clenching and bruxing. The signs that would indicate the problem are reviewed; and the patient and spouse are questioned about the clenching and bruxing since the patient is not always aware
of his habit. Past or potential damage is evaluated.

Second, the patient must learn to control the old habit by becoming instantly aware of occlusal forces, both while awake and asleep. As part of this training he must learn to relax his frontalis muscle, tongue, and masseter muscle and maintain a free-way space. This retraining program must be carried into his consciousness and subconsciousness day and night.

To control the habit during his sleep, the patient is instructed to tell himself just before dropping off to sleep, "If my teeth come together (trigger) for the purpose of clenching or bruxing, I will become aware of it and my teeth will part slightly." He is told to repeat this to himself several times (reinforcement) to develop a subconscious awareness of the control pattern in the brain which is active during sleep. Eventually the contacting of the teeth for clenching and bruxing will subconsciously trigger the reflex and maintain the freeway space and relaxation of the associated musculature.

Third, patients must learn to avoid misdirected efforts by continually reinforcing the sensimotor reflex in both the consciousness and subconsciousness. In this way a new reaction is instilled and a new habit is learned. Once the patient has been made aware of the problem and its treatment and realizes that the extent to which treatment is successful depends on his own effort, he should develop in his mind, and maintain a "self-portrait" to continually
reinforce the effort. It is this reinforcement of awareness that assumes primary importance.

Changes of habits by the brain require time. Small increments of change over a long time period are more effective than massive changes over a short time period. Habit retraining requires the patient's total cooperation, understanding, and continuous practice.

J. Drugs.

Drugs can be employed in bruxotherapy but as a general principal it is better to treat without drugs except maybe analgesics for pain, if such a condition presents in early treatment. If drugs for emotional or stress factors are deemed appropriate in a particular case, that case probably requires psychotherapy and in this instance a teamwork approach to the drug regimen can be established with the therapist. Drug treatment can also be utilised in reverse by withdrawing patient dependencies such as caffeine, tobacco, and alcohol that not only exert an effect on the musculature and the nervous system but also affect sleep patterns. Further, other addictive agents could also be considered.

K. Adjunctive Therapy.

In the non occlusal range of treatment modalities the following adjunctive therapies may be useful.
a. Autosuggestion
b. Hypnosis
c. Structural relaxation programmes
d. Aerobic exercise

L. Other Factors.

Table I listed a number of factors that may be associated with the etiology of bruxism. If, in any case it was decided that one or more of these factors may be operative, then the necessary treatment should be administered, if possible (e.g. nothing extra could be done about a brain damage factor). This is the principal of treating cause and effect. If, for example, a magnesium deficiency or and avitaminooses existed, the appropriate steps would be taken in consultation with the physician.

Discussion

Understandably, bruxism is predominately treated by dentists and dentists tend to treat dental factors only. Therefore, the bulk of treatment for bruxism has been occlusion oriented exclusively, with other approaches being taken by different disciplines or dental academics. In the main, occlusal treatment has been more than satisfactory but it has not been totally successful. In the introduction to this treatment section the attitudes quoted by Ramfjord and Ash support the view: Local treatment will work, but
total treatment is better. Determine the total aetiology and treat accordingly.

Unfortunately most of the debate in the literature is advancing a particular treatment form in competition to other therapies on the basis of case success. Blank\textsuperscript{111} stated quite appropriately: Often, proponents of one of these "philosophies" of occlusion seems to insist that only their method is the correct one and suggests that other methods are doomed to failure. How can this be true when advocates of different approaches can each present cases treated quite differently and yet apparently successfully? The answer seems to be in case selection. Many occlusal problems may respond to more than one approach.

In assessing treatment success, long term success must be the true measure, obviously. In this regard the placebo effect must not be forgotten. According to Rugh and Solberg\textsuperscript{45} nonspecific placebo effects appeared to be one of the most potent, but largely underrated therapeutetic modalities available to every clinical situation. The placebo effect has been estimated at about 30%. Regarding treatment philosophy, the authors noted that while breaking just one link in the causal chain may produce a cure, the chance of successful therapy becomes greater when more links are discovered and broken.
CHAPTER 11

DISCUSSION AND CONCLUSION

LIST OF CONCLUSIONS
DISCUSSION AND CONCLUSION

Any clinician with experience over the years in a subject of interest, who suffers from that condition himself, who has experienced various treatments for that condition, who observes that condition in his parents, spouse and children and who has extensively reviewed this subject, must have very definite views on such a subject. These views support the almost universal incidence of bruxism in western society, the Ramfjord model of the aetiology of bruxism and the view that treatment should be occlusally directed with other treatment modalities as supportive.

Some abstract curiosities also bear consideration. Is bruxism pathological or is its effects on the imperfect occlusion only pathological? If its incidence is so universal, why is it universally so pathologic? In modern man, without excessive physiologic abrasion, have we been able to observe some perfect natural dentitions, free of all occlusal imperfection, some exhibiting bruxism and some not, and finding that bruxing in this enviroment exhibited no pathology?

Arnold\textsuperscript{79} stated that in a way bruxism is a unique habit. It gives the individual a means of discharging his tensions while, at the same time, allowing him to agress against himself by regressing to an earlier fixated stage. This universal combination of psychologically fulfilling factors is probably why bruxism is so universally practiced.
If a perfect dentition evolved with bruxism, might it prove to be a healthy, stress releasing mechanism? We cannot duplicate this evolution by adjusting a dentition. It is then too late. The muscular hypertonus has been established, abnormal wear pattern, perhaps disc changes and who knows what transmission pathways. But if such a dentition did evolve would it be regarded as a healthy stress releasing habit? We can't remove the stress from modern society, we have to cope with it and of the coping methods that man has for dealing with stress, is bruxism pathological even though it is vegetative.

The development versus the pathogenesis of bruxism might then be described as: infant oral awareness - to teething with clenching and biting for pain relief and therefore stress relieving - through to proprioception - reflex and conscious oral functions and bruxism. Or conversely we retro-evolve to a society that is less stressful and the diet equilibrates the dentition; and the secondary dentine has a chance.

Regarding splint therapy, author's subjective assessment demonstrated that when a splint was worn full time to relieve myospasm and this full time wearing continued through splint and occlusal adjustment, there was a great reduction in bruxism as measured by grind marks in the splint. After occlusal adjustment and the relegation of the splint to nocturnal use, bruxism increased. It is speculated that full time wear gave a free way space accommodation that part time wear denied. With the splint impinging on free way space it is a stimulus to bruxism, even though the teeth and muscless
are protected by the adjusted splint. There has been an hypothesis advanced that long term splint usage will modify transmission pathways and eventually eliminate bruxism, but this does appear very hypothetical.

The use of biofeedback and massed practice therapy has not been evaluated on the basis of sleep physiology. If a biofeedback device is emitting signals that are effecting bruxistic behaviour, what are these signals doing to the balance of normal sleep stages in that individual, particularly long term and what are they doing to the psyche? Orthodontic experience can be extrapolated regarding the unwise modification of thumb sucking habits and the effect this had on the emotional development of some children. Drugs can be administered that will supress the sleep stages in which bruxism occurs, but it can be demonstrated that the sleep disorientation after drug dependence or drug withdrawl is dramatic. The attempted elimination of bruxism by means of sleep rhythm disturbance would appear most unwise.

CONCLUSIONS

1. The incidence of bruxism is almost universal.

2. Bruxism is induced by a combination of occlusal interferences and psychic stress and the proportion of each aetiological component will vary in the individual from time to time.
3. Some kind of occlusal interference will always be found in a patient with bruxism.

4. Severe episodes of bruxism are always stress related.

5. Bruxism can usually be diagnosed by atypical wear facets on the teeth, outside the functional range.

6. Conversely in the periodontally affected bruxer, the bruxism is diagnosed as tooth mobility. That is, firm teeth, excessive wear, loose teeth, minimal wear.

7. Functional disturbances of the temporomandibular joints and musculature will not result from every case of bruxism but bruxism is a component in the aetiology of every such functional disturbance.

8. The extreme positions adopted by the mandible during bruxism, the tremendous forces involved and the fact that these forces are applied to only a few teeth at a time and not equilibrated over the entire dentition, places profound strain on all units of the masticatory apparatus.

9. The fact that endodontic treatment can be complicated by bruxism should always be kept in mind.

I. In differential diagnosis of dental pain, the bruxed tooth will give a history of pain to hard components in the
food bolus such as grain bread and muesli.

II. The difficulty of obtaining adequate anaesthesia of the contents in the apical quarter of the root canal.

III. The continuation of pain after total root canal instrumentation.

IV. The complaint of mild apical pain for up to twelve months after apparently successful endodontic treatment.

V. Cementum sensitivity after total root canal instrumentation.

VI. Post operative endodontic tooth and root fracture in the non crowned case.

10. The effect of bruxism on restorative materials and restored teeth such as fractured enamel and amalgam margins, fractured and worn porcelain and the failure of posterior composites should always be kept in mind with restorative dentistry. Where average dentistry may survive in many cases for many years, it will not survive under the excessive loading of bruxism. Therefore the highest standards must be practiced regarding cavity preparation and finish and choice of material, manipulation and finish.
11. Denture wearers who brux must leave their dentures out at night. This particularly applies to cast metal partial dentures as the bruxism will result in fractured or loosened clasps.

12. Regarding the involvement of bruxism in periodontal disease, the consensus of opinion is that bruxism in itself cannot initiate the periodontal lesion. However, it can aggravate and contribute to the existing disease process.

13. The concept of co-destruction is most commonly accepted. Occlusal trauma plus dental plaque contribute to periodontal disease.

14. The incidence of bruxism in periodontal disease is probably underdiagnosed as it is very difficult to recognise clinically with the lack of tooth wear that occurs in periodontally involved cases. However, it has been clinically observed by the author, that when a periodontal case has been occlusally treated without apparent success, the success rate improves dramatically when the occlusal treatment has been extended to include the parafunctional range of tooth contact.

15. The treatment of a bruxism patient should always involve occlusal treatment regardless of the philosophy of the clinican. Local treatment will work but total treatment is better. As bruxism has a multifactorial aetiology. The treatment of any one factor will break the chain but if any
treatment is not totally successful it is best to leave the patient bruxing on a refined occlusion than the converse. This will be less damaging on the masticatory apparatus.

16. Post treatment nocturnal splints are recommended.
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