TEMPOROMANDIBULAR JOINT DYSFUNCTION SYNDROME - RELATIONSHIP OF FIXED APPLIANCE ORTHODONTIC TREATMENT AS A POSSIBLE AETIOLOGICAL FACTOR.

by

K.J. MOUNTAIN BDS, FRACDS.

A treatise submitted in partial fulfilment of the requirements for the degree of

Master of Dental Surgery

DEPARTMENT OF PREVENTIVE DENTISTRY

UNIVERSITY OF SYDNEY

1988
Acknowledgements

I wish to thank Dr Ives Lopez who as my supervisor until his retirement, provided assistance and direction. Also, Associate Professor Keith Godfrey who undertook the supervision of this treatise in its final stages.

Dr Susan Howell, Head of Unit (Orthodontics) and Professor Keith Lester, Director of Dental Services, Dental Clinical School, Westmead Hospital, who kindly allowed me study time to assist in the preparation of this treatise.

To my wife Maryanne, I owe thanks for her immense patience, understanding and encouragement throughout the period of writing.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Acknowledgements</th>
<th>ii</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table of Contents</td>
<td>iii</td>
</tr>
<tr>
<td>List of Figures</td>
<td>vi</td>
</tr>
<tr>
<td>List of Tables</td>
<td>viii</td>
</tr>
<tr>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>1. Concepts of occlusion and function during diagnosis and treatment.</td>
<td>1</td>
</tr>
<tr>
<td>1.1 Concepts of occlusion.</td>
<td>4</td>
</tr>
<tr>
<td>1.2 Inter-arch relationships of teeth</td>
<td>5</td>
</tr>
<tr>
<td>1.2.1 Centric occlusion</td>
<td>5</td>
</tr>
<tr>
<td>1.2.2 Centric relation</td>
<td>7</td>
</tr>
<tr>
<td>1.3 Theories of functional occlusion</td>
<td>11</td>
</tr>
<tr>
<td>1.3.1 Bilateral balanced occlusion</td>
<td>11</td>
</tr>
<tr>
<td>1.3.2 Group function</td>
<td>12</td>
</tr>
<tr>
<td>1.3.3 Canine protected occlusion</td>
<td>12</td>
</tr>
<tr>
<td>1.4 Electromyography of the muscles of mastication - Normal muscle function</td>
<td>13</td>
</tr>
<tr>
<td>1.5 Activity of lateral pterygoid muscle</td>
<td>16</td>
</tr>
<tr>
<td>1.6 Muscle function and temporomandibular joint activity</td>
<td>16</td>
</tr>
<tr>
<td>1.7 Neurophysiological control of jaw movement.</td>
<td>18</td>
</tr>
<tr>
<td>1.7.1 Jaw reflexes</td>
<td>18</td>
</tr>
<tr>
<td>1.7.2 Neurobiology of the occlusal interface</td>
<td>19</td>
</tr>
<tr>
<td>1.7.3 Neurobiology of the temporomandibular joint interface</td>
<td>21</td>
</tr>
<tr>
<td>1.7.4 Occlusal interface versus TMJ interface</td>
<td>21</td>
</tr>
<tr>
<td>1.8 Features of an ideal occlusion</td>
<td>21</td>
</tr>
</tbody>
</table>

| 2. Temporomandibular joint dysfunction - Current concepts on aetiology. | 24     |
| 2.1 Historical note and definition | 24     |
| 2.2 Prevalence studies            | 25     |
| 2.3 Symptom mechanisms            | 31     |
| 2.3.1 Pain                        | 31     |
| 2.3.2 Sounds occurring during condylar movements | 33     |
| 2.3.3 Limitations of mandibular movements | 36     |
| 2.4 Aetiological theories         | 37     |
| 2.4.1 TMJ Dysfunction theories    | 38     |
| 1. Osteoarthritis                 | 39     |
| 2. Rheumatoid arthritis           | 39     |
3. Condylar displacement 40
   a. Trauma 40
   b. Malocclusion 41
   c. Oral habits 43

2.4.2 Masticatory muscle hyperactivity theories 44
2.4.3 Causes of masticatory muscle hyperactivity 46
   1. Local causes 46
   2. Centrally mediated causes 49

2.5 Overview 51

3. Diagnosis of temporomandibular joint dysfunction 53
   3.1 Initial screening history and examination 53
   3.2 Diagnosis of TMJ Dysfunction and MPD Syndrome 54
       1. History 55
       2. Orofacial examination 58
       3. Special tests 64
   3.3 Differential diagnosis 70

4. Possible effects of orthodontic treatment on the functional stability of the temporomandibular joint 74
   4.1 Orthodontically established occlusion - Possible iatrogenic causes of TMJ problems 75
       4.1.1 Forced mandibular positioning 75
       4.1.2 Significance of dual bite 79
       4.1.3 Occlusal interferences following orthodontic treatment 81
   4.2 Mechanical effects of orthodontic treatment - Possible iatrogenic causes of temporomandibular joint problems 87
       4.2.1 Animal experiments - Effects of intermaxillary forces on temporomandibular joints 87
       4.2.2 Skeletal and neuromuscular adaptation to orthodontic forces 90

5. Orthodontic treatment in relation to symptoms attributed to dysfunction of the temporomandibular joint - Clinical and experimental evidence 94

6. Assessing the risk, and reducing the iatrogenic effects of orthodontic treatment on the temporomandibular joints 117
   6.1 Assessing the risk 117
   6.2 Reducing the iatrogenic effects of orthodontic treatment on the temporomandibular joints 121
6.2.1 Clinical application of functional concepts—
Posterior relationships 123
6.2.2 Clinical application of functional concepts—
Anterior relationships 127
6.3 Occlusal adjustment 129
6.4 Orthodontic treatment considerations for the patient
with temporomandibular joint dysfunction symptoms 132

7. Summary and discussion 137

8. Conclusion 148

Appendix A – Anamnestic and Clinical Dysfunction Index of Helkimo (1974) 151

Bibliography 153
### LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Temporomandibular joint anatomy. (Klineberg, 1986)</td>
<td>23</td>
</tr>
<tr>
<td>2</td>
<td>Prevalence of clinical signs in children. (Solberg and Seligman, 1985)</td>
<td>28</td>
</tr>
<tr>
<td>3</td>
<td>Prevalence of clinical signs related to different age groups. (Solberg and Seligman, 1985)</td>
<td>29</td>
</tr>
<tr>
<td>4</td>
<td>Prevalence of clinical signs in young adults related to sex. (Solberg and Seligman, 1985)</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>Mechanism of anterior disc displacement with reduction. (Moloney, 1985)</td>
<td>35</td>
</tr>
<tr>
<td>6</td>
<td>Palpation of middle fibres of temporalis muscle. (Gelb, 1977)</td>
<td>58</td>
</tr>
<tr>
<td>7</td>
<td>Palpation of body portion of the masseter muscle. (Gelb, 1977)</td>
<td>59</td>
</tr>
<tr>
<td>8</td>
<td>Palpation of medial pterygoid muscle intra and extra orally simultaneously. (Gelb, 1977)</td>
<td>59</td>
</tr>
<tr>
<td>9</td>
<td>Palpation of the lateral pterygoid muscle (interior head). (Gelb, 1977)</td>
<td>60</td>
</tr>
<tr>
<td>10</td>
<td>Diagram showing orientation of condyle in fossa. (Weinberg, 1972b)</td>
<td>67</td>
</tr>
<tr>
<td>11</td>
<td>Linear tomogram of temporomandibular joint. (Solberg, 1986d)</td>
<td>68</td>
</tr>
<tr>
<td>12</td>
<td>Distribution of individuals according to symptoms of impaired TMJ function. (Janson and Hasund, 1981)</td>
<td>100</td>
</tr>
<tr>
<td>13</td>
<td>Distribution of individuals according to pain on movement. (Janson and Hasund, 1981)</td>
<td>101</td>
</tr>
<tr>
<td>14</td>
<td>Distribution of individuals according to palpation tenderness of the masticatory muscles. (Janson and Hasund, 1981)</td>
<td>102</td>
</tr>
<tr>
<td>15</td>
<td>Distribution of individuals according to the temporomandibular joint pain. (Janson and Hasund, 1981)</td>
<td>103</td>
</tr>
<tr>
<td>16</td>
<td>Relationship to age of symptoms before orthodontic treatment. (Dibbets and van der Weele, 1987)</td>
<td>111</td>
</tr>
<tr>
<td>17</td>
<td>Relationship with treatment time of three symptom categories. (Dibbets and van der Weele, 1987)</td>
<td>112</td>
</tr>
</tbody>
</table>

# LIST OF TABLES

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Differential diagnosis of non-articular conditions mimicking pain of MPD syndrome. (Laskin, 1986)</td>
<td>71</td>
</tr>
<tr>
<td>2</td>
<td>Differential diagnosis of non-articular conditions producing limitation of mandibular movement. (Laskin, 1986)</td>
<td>72</td>
</tr>
<tr>
<td>3</td>
<td>Differential diagnosis of temporomandibular joint disease. (Laskin, 1986)</td>
<td>73</td>
</tr>
<tr>
<td>4</td>
<td>Diagnosis, treatment and type of appliance used in study by Larson and Ronnerman (1981)</td>
<td>105</td>
</tr>
<tr>
<td>5</td>
<td>TMJ signs and symptoms of subjects in Illinois and Eastman studies. (Sadowsky and Polson, 1984)</td>
<td>109</td>
</tr>
<tr>
<td>6</td>
<td>Frequency of symptom categories for treatment procedures in study by Dibbets and van der Weele (1987)</td>
<td>113</td>
</tr>
<tr>
<td>7</td>
<td>Comparison of symptom frequencies in study by Dibbets and van der Weele (1987).</td>
<td>114</td>
</tr>
</tbody>
</table>
INTRODUCTION

There has been an explosion of interest in temporomandibular joint disorders over the last ten years or so. Ample evidence of this exists in the dental literature where literally hundreds of articles dealing with every aspect of the temporomandibular joint in health and otherwise, have appeared.

It soon becomes apparent from a study of this literature that there is very little agreement among the various specialities and interest groups in dentistry about the nature and causes of temporomandibular joint dysfunction (TMJ dysfunction). The reasons for this may be as Rinchuse (1987) has stated:

"Certainly the advent of better imaging techniques and the accumulation of research data have improved our understanding of the temporomandibular joint. Nevertheless, even with the increased attention given to TMJ study, prospective longitudinal research is lacking. Perhaps, the complex biologic, physiologic, and psychologic factors involved in craniomandibular articulation make TMJ research and its interpretation more than a little difficult. Possibly there are far too many extrinsic variables impacting upon the TMJ, making it practically impossible to control cross-sectional research."

The fundamental disagreements among the various speciality groups demonstrates that dentistry is confronted by a dilemma concerning the causes of TMJ dysfunction. Orthodontics is not exempt from having its own TMJ concepts related to diagnosis, etiology and treatment.

It is the aim of this treatise to investigate the hypothesis that fixed appliance orthodontic
treatment is an aetiological factor in TMJ dysfunction. In the context of this treatise the term "fixed appliances" is defined as those orthodontic treatments using attachments fixed to the teeth and forces applied by labial arch wires or auxiliaries through these attachments. Edgewise techniques, Begg lightwire and B.E.D.T.I.O.T. techniques would be typical of the treatment methods described by this definition. The possible effects of other forms of "fixed" appliances such as the Quad helix, W arch, Herbst appliance and so on, on the aetiology of TMJ dysfunction should not be dismissed. However, in accordance with the above definition, these appliances are outside the scope of this analysis.

In order to achieve the above aim it will be necessary to:


2. Provide an overview of current theories on the aetiology of TMJ dysfunction. In view of the huge number of general articles that have appeared over recent years on this topic, a review of the more current, commonly accepted ideas will be given, with particular emphasis on the importance of occlusion.

3. Current diagnosis techniques for the identification of TMJ dysfunction symptoms in orthodontic patients before, during and after orthodontic treatment must be examined.

4. Provide an analysis of the literature examining the suggested possible ways in which orthodontic treatment may be implicated as an aetiological factor in TMJ dysfunction.
5. Detail an examination of the comparatively very few clinical studies that have attempted to investigate the degree of responsibility that may be attributed to orthodontic treatment, as an aetiological factor in TMJ dysfunction.

6. Conduct a review of the methods suggested in the literature to reduce the iatrogenic effects of orthodontic treatment on the function of the temporomandibular joints.

If the hypothesis is to be accepted as valid, evidence must exist in the literature in support of the following statements:

1. Aetiological factors responsible for initiating TMJ dysfunction may be identified, and their individual degree of responsibility quantified.

2. Clinical and statistical evidence will support the concept that fixed appliance orthodontic treatment of dental or skeletal malocclusions will increase the chance of developing TMJ disorders.
CHAPTER ONE

CONCEPTS OF OCCLUSION AND FUNCTION DURING ORTHODONTIC DIAGNOSIS AND TREATMENT

A major goal of orthodontic diagnosis and treatment has always been to achieve maximum intercuspation in a static occlusal relationship. Although this objective remains, the goals of orthodontic treatment have been extended in recent years to include relationships of the teeth that facilitate optimal occlusal function.

At present, our goals of treatment are to treat to a functional and aesthetic occlusal end point, and at the same time, strive to restore and maintain the normal seated position of the condyles (centric relation), which according to Weinberg (1974), Mongini and Schmid (1982), Mohamed and Christensen (1985), and Williamson (1985) and others, provides the greatest neuromuscular efficiency during function, and stability in the static position.

1.1 Concepts of Occlusion

Ash and Ramfjord (1982) define occlusion as:

"The functional and dysfunctional relationships between an integrated system of teeth, supporting structures, joints and neuro-muscular components."

This dynamic inter-relationship of component parts of the masticatory apparatus is stressed in Klineberg's (1986) definition:
"Occlusion is the dynamic biological relationship of all components of the masticatory system to the contacting surfaces of the teeth in function and parafunction ie. the integrated function of jaw muscles, temporomandibular joints and teeth."

The term functional occlusion, that is acceptable, is defined by Ash and Ramfjord (1982) as:

"A state of the occlusion conducive to function with the following characteristics:

1. In which the occlusal interfaces are free of interferences to smooth gliding movements of the mandible.
2. There is freedom for the mandible to close or to be guided into maximum intercuspation in centric occlusion and centric relation.
3. In which occlusal contact relations contribute to occlusal stability."

1.2. Inter-Arch Relationships of Teeth

1.2.1. Centric Occlusion (Intercuspal Jaw Position).

This was defined by Ash and Ramfjord (1982) as the position of maximum intercuspation of the teeth. It is also called intercuspal position, tooth position, acquired centric or habitual centric position.

Klineberg (1986) however, defines it as:

"The centred contact position of the occlusal surfaces of the mandibular teeth against the
occlusal surfaces of the maxillary teeth."

Klineberg (1986) makes the point that this definition does not imply maximal intercuspal contact of teeth, but this is often incorrectly assumed to be the case.

Centric occlusion may be considered as the terminal jaw position in the closing phase of chewing and swallowing (Celenza 1978, Klineberg 1986).

According to Celenza (1978) there are three elements which make a major contribution to determine the centric position. These are:

1. Temporomandibular joints.
2. The neuromuscular elements.
3. The dental units.

These elements in the healthy state with neurological control and co-ordination, produce smooth, rhythmic, cyclic mandibular movements. This is the basis of mandibular function and together with the mechanical form of the teeth surfaces, determine the approach, the final position and the stability of the stomatognathic system.

Klineberg (1986) stresses that intercuspal position is a static tooth contact position. This tooth contact position has developed in harmony with biological influences of bone growth and muscle function during development. It must be noted that this biological balance is most vulnerable to sudden alteration in form accompanying dental treatment.

Scharer (1978) notes that there is very little evidence for any specific condylar position in
intereuspal position.

On closure from postural position when standing or sitting upright with head straight, and looking straight ahead, the teeth occlude in intereuspal position, according to Klineberg (1986). Slight variations in head position significantly change the point of tooth contact. (Mohl 1971).

1.2.2.  **Centric Relation**

The definition, reproduction and significance of centric relation in dental treatment has been a controversy for many years.

The Report of the Committee on Scientific Investigation of the American Academy of Restorative Dentistry (1986), states that the definition of centric relation has never enjoyed more consistent, synonymous definitions than in the past year. According to this report, most contributors to the literature recognize centric relation as:

"The physiologic relationships of the mandible to the maxillae and the cranial base when both condyles are properly related to their articular discs and the condyle disk assemblies are stabilised against the posterior slopes of the articular eminences of the glenoid fossae."

Other suggested definitions of centric relation have included:

Ramfjord and Ash (1979) - "The most retruded position of the mandible from which opening and lateral movements can be performed comfortably."

Parker (1978) - "A situation in which the head of the condyle of the TMJ, at rest, is in a
position where all of its attending muscles and ligaments are at rest."

Geering (1974) - "The relative position of the mandible to the maxillae when the condyles are in the uppermost and rearmost position in the glenoid fossae (under healthy conditions)."

A more recent definition is given by Klineberg (1986) who believes that centric relation should be:

"The position of the mandible at tooth contact where the condyles are in a physiologically acceptable position. Such a position may be described as the jaw position at which the condyles are in an uppermost position in the glenoid fossae, and where the condyles and interarticular discs are correctly aligned (i.e. a "close packed" position) at an acceptable vertical dimension of occlusion."

The controversy involving centric relation relates not only to its definition, but also to whether it can be regarded as merely a reference point for treatment procedures, or whether it exists as a functional position.

Sicher (1956) discussed the biological significance of centric relation with regard to the occlusion of natural teeth. He contended that centric relation is the optimum physiologic position with regard to the temporomandibular joints and the teeth, and that centric relation is a functional position assumed during mastication, deglutition and respiration.

In an electromyographic study by Owens et al (1975) intended to study the activity of the lateral pterygoid muscles during mastication, it was found that the lateral pterygoid muscles were inactive for a period of time as the mandible was elevated during mastication. They argued that during the period of inactivity of the lateral pterygoid muscles in this elevating portion of the
chewing cycle, the mandibular condyles are under the influence of the elevating musculature. The masseter and its synergist, the medial pterygoid muscle, exert a combined bilateral elevating, retruding force on the condyles. The temporal muscle exerts a combined bilateral elevating, retruding force on the condyles also.

These elevating and retruding vectors of force on the condyles, coupled with the inactivity of the lateral pterygoid muscles tend to indicate that, at this point during the chewing cycle, the mandibular condyles are seated in their most posterior, superior and medial unstrained position – or in centric relation.

According to Owens et al (1975), their evidence indicates that centric relation is a functional position, and the principle of centric relation should be incorporated into treatment procedures involving gross rearrangement of occlusal surfaces of teeth, as in orthodontic treatment. Their contention is that centric relation being not only a repeatable point, but also a functional entity, suggests that it is the optimal physiologic position from which the functional movements of the stomatognathic system occur.

Gilboe (1983) argues that if centric relation is to considered a functional position, it should be defined as:

"The most superior position of the mandibular condyles with the central bearing area of the disk in contact with the articular surface of the condyle and the articular eminence."

According to Gilboe (1983) this position would be consistent with load-bearing capacity of the posterior slope of the articular eminence, the adaptation of the intermediate zone of the disk for transmission of pressure, and the biomechanical stability of the joint resulting from the shape of
its components."

It is generally accepted that centric occlusion and centric relation in the normal untreated patient do not coincide (Parker 1978, Williamson 1978, Ramfjord and Ash 1979, Klineberg 1986). Indeed, Scharer (1978) has suggested that over 90% of the population have a centric relation that does not coincide with centric occlusion.

The importance of centric relation in any form of dental treatment has been summarised by the Report of the Committee on Scientific Investigation of the American Academy of Restorative Dentistry (1986):

1. Centric relation is considered a border position that may be related to the mandibular transverse axis.

2. Centric relation is therefore an anatomically and physiologically stable, repeatable posture of the mandible and can be considered a most acceptable treatment and reference position.

3. Centric relation is a position that is not commonly coincident with the maximum intercuspidation of the dentition.

A concept of long centric has been suggested by some authors as being normal. According to Ramfjord and Ash (1979) this theory states:

"The distance between centric relation and centric occlusion is about 0.1 to 0.2 mm in the temporomandibular joint, and about 0.5 mm at the level of the teeth."

Williamson (1976) clarified this concept by stating that the concept of long centric allows the freedom of the mandible to move in one plane of space from its most retracted position to a point
approximately 0.2 – 0.5 mm anterior.

1.3  **Theories of Functional Occlusion**

According to Williamson (1976) there are currently three main theories of functional occlusion. These are:

1. Bilateral balanced occlusion.
2. Group function.
3. Canine protected occlusion.

1.3.1.  **Bilateral Balanced Occlusion**

The requirements of balanced occlusion are:

a. Centric position – all teeth contact evenly when closed into centric relation. The anterior teeth contact lightly.

b. Working side – maxillary buccal cusp inclines are in even contact.

c. Balancing side – teeth opposite the working side shall have a balancing contact between the lingual cusps of the maxillary teeth and the buccal cusps of the mandibular teeth.

d. Protrusive – Incisal edges of the maxillary six anterior teeth are in contact with the incisal edges of the mandibular six most anterior teeth. There should be balancing contact between the maxillary and mandibular last molars.
It is now generally accepted that bilateral balanced occlusion should be mainly used for complete denture prosthetics. It is rarely seen in the natural dentition.

1.3.2. **Group Function – Or Unilateral Balanced Occlusion.**

The requirements of this type of occlusion are:

a. **Centric position** – the concept of long centric as described above.

b. **Working side** – the maxillary buccal cusp inclines are in even contact with the mandibular buccal cusp inclines.

c. **Balancing side** – there shall be no tooth contacts on the side opposite the working side.

d. **Protrusive** – the maxillary six anterior teeth shall contact the mandibular six most anterior teeth in an edge to edge relationship. There shall be no contacts of any teeth posterior to the above mentioned teeth.

1.3.3 **Canine Protected Occlusion – Or Mutually Protected Occlusion.**

The requirements of this type of occlusion are:

a. **Centric position** – the concept of long centric as outlined above.

b. **Working side** – the maxillary canine should contact the mandibular canine. No other teeth should contact at any point once the jaws leave the immediate centric position.

c. **Protrusive** – there shall be no posterior tooth contact when the maxillary anterior six teeth contact the six most anterior mandibular teeth in an edge-to-edge position.
Group function occlusion and canine-protected occlusion could be regarded as being "normal" functional occlusions. According to Williamson (1976), the canine-protected occlusion is the type most commonly seen in today's naturally occurring young adult dentitions.

These theories on normal occlusions have been described, as it is only with an understanding of normal functional occlusions that deviations causing mandibular dysfunction can be appreciated.

1.4 Electromyography of the Muscles of Mastication - Normal Muscle Function.

Since the early work of Moyers (1949, 1950), a great number of electromyographic (EMG) studies have been published, examining the oro-facial musculature in normal function. Basmajian (1978) presents an excellent review of the published reports on the functions of the muscles of mastication and the oro-facial muscles.

According to Plunkett (1986), most neurophysiologists now agree that electromyography shows conclusively the complete relaxation of normal human striated muscle at rest. In other words, by relaxing a muscle, neuromuscular activity can be abolished, although the muscle tone will still be present. This will become an important concept when we come to analyze abnormal EMG activity in muscles.

1.4.1 Rest Position.

According to Munro (1975a), all free movements of the jaw, as elsewhere in the body, start from the rest position. This position is relatively stable in healthy individuals (Thompson 1946) apart from minor changes due to age, malocclusion or loss of teeth (Ricketts 1952).
The rest position may be determined as the position of minimum EMG activity of the muscles of mastication (Munro 1975a). It is generally agreed that the temporalis (Moyers 1950), particularly its posterior fibres (Plunkett 1986), is most active in maintaining mandibular posture in the physiological rest position.

According to Moyers (1950) and Plunkett (1986), the masseter is not an important muscle in the habitual resting position.

1.4.2. **Elevation.**

The muscles active in mandibular elevation are the temporalis, masseter and medial pterygoid muscles. (Moyers 1950, Plunkett 1986).

1.4.3. **Protraction.**

The lateral pterygoid muscle is the main protractor of the mandible. (Moyers 1950, Hickey et al 1957, Plunkett 1986). It is assisted by the medial pterygoid, and possibly by the superficial part of the masseter. (Moyers 1950).

Activity of the anterior part of the temporalis muscle in protraction was found by McCollum (1943), Greenfield and Wyke (1956) and Moyers (1950).

Greenfield and Wyke (1956) reported that the minimal activity of the anterior temporalis muscle during protraction without occlusion is increased when dental contact occurred during the movement.
1.4.4. Retraction.

Retraction of the mandible is accomplished by the temporalis muscle, principally its posterior and middle parts. (Moyers 1950, Greenfield and Wyke 1956, Plunkett 1986).

1.4.5. Depression.

The main muscles involved in opening the mouth are the lateral pterygoid and digastric muscles. (Munro 1975a). Plunkett (1986) states that it is the lateral pterygoid which is the first active muscle during depression of the mandible, and it reaches a peak of activity before other muscles become active.

According to Plunkett (1986), the digastric muscle comes into action after lateral pterygoid during wide opening and is not as important.

1.4.6. Side to Side Movements.

Chewing and grinding movements of the mandible are effected by bilateral activity of different muscle groups, including the medial and lateral pterygoid muscles, masseter and temporalis. The contralateral group of muscles provide for condylar translation, while the ipsilateral muscle groups provide the essential activity of chewing activity on that side. (Romanes, 1978).

When one lateral pterygoid muscle acts alone, the condyle on that side is drawn forwards, the mandible pivots around the opposite joint so that the chin is slewed toward the opposite side. The medial pterygoid muscles acting alternately and assisted by the masseter and temporalis muscles, produce a grinding movement.
1.5  **Activity of Lateral Pterygoid Muscle.**

Gibbs et al (1984) in an electromyographic study, found that the EMG activity of the superior belly of the lateral pterygoid was similar but not identical to the EMG activity of the anterior fibres of the temporal muscle. They found that the superior belly of the lateral pterygoid muscle was active in clenching, especially in retruded contact. It was moderately active in ipsilateral movement, and showed little activity in other basic jaw positions.

Gibbs et al (1984) found the inferior lateral pterygoid was active in protrusive, opening, and contralateral positions. It was minimally active during clenching in retruded contact unlike the superior lateral pterygoid which achieved its greatest EMG activity during clenching.

1.6  **Muscle Function and Temporomandibular Joint Activity.**

The "correct "positioning of the meniscus at any one time is critical for the normal functioning of the temporomandibular joint. Moloney (1984) notes that the proper position of the disc is determined by the state of muscle tonus of the upper head of the lateral pterygoid muscle counterbalanced by the elastic pull of the superior retrodiscal lamina. During the initial rotatory phase of jaw opening, the upper head of the lateral pterygoid relaxes and the natural elastic recoil of the bilaminar zone slightly retracts the disc, ensuring that the posterior thick band remains at all times in a superior position in the condyle.

On closing, the upper head of the lateral pterygoid contracts along with the levator muscles and overcomes the posterior elastic traction of the bilaminar zone. The disc thus rotates slightly forwards and the "self-correcting wedge" effect of the posterior thick band ensures that the condyle
is in firm contact with the central articular zone. The chief function of the superior lateral pterygoid muscle is to supply strong tractive force on the disc to maintain joint stability.

Bell (1984) agrees with this concept of the disc rotating on the head of the condyle in jaw movements. By rotating on the condyle, thicker portions of the disc can be correctly positioned to compensate for varying articular pressures. For the disc to occupy any other position, particularly anterior displacement, implies stretching of the tissue of the bilaminar zone, and abnormal function of the superior head of the lateral pterygoid muscle. (Moloney, 1984)

Williamson (1978, 1981a, 1981b) has found in EMG studies that the temporalis muscle and the superior head of the lateral pterygoid muscle predominate in seating the condyles bilaterally in a stable position prior to any posterior tooth contacts. He found also that the masseter and medial pterygoid muscles, however, do not seem to contract until the posterior teeth are in contact.

In investigations using monkeys, McNamara (1972, 1973a) found that the superior head of the lateral pterygoid muscle is the first to show electromyographic activity upon closure of the mandible. Williamson (1978, 1981) has corroborated McNamara's findings in human subjects.

Williamson (1985) notes that when the mandible is moved to the working side, in a canine-protected form of occlusion, there is an immediate cessation of the temporalis, masseter and medial pterygoid function on the non-working side. However, when anterior guidance is missing, the posterior teeth contact and these muscles contract. Under these circumstances, excessive loading to the teeth may occur.

According to Isberg et al (1985), masticatory muscle activity can be provoked by TMJ disc displacement. They found this activity ceased when the disc position was normalised on mouth
opening, only to occur again each time the disc became displaced on mouth closure. These authors note that when a disc has become anteriorly displaced, a prerequisite for normalization is that the condyle can move downward and forward, passing over the posterior thick part of the disc. Muscle activity in the elevator muscles will oppose the downward movement of the condyle pulling against the tubercle. During opening, the anteriorly displaced disc thus acts as a wedge rendering correction of disc position more difficult.

In joints with such disc displacement, according to Isberg et al (1985), it is likely that nonfunctional muscle activity of the masseter and temporal muscles during opening aggravates the development of a closed lock condition because such activity may prevent the condyle from passing over the posterior band of the disc to restore normal disc/condyle relationships.

1.7 Neurophysiological Control of Jaw Movement

The significance of tooth contacts in jaw movements, and the association between contact interferences and the development of jaw muscle dysfunction is poorly understood, although there is a close association between occlusal morphology and functional jaw movements. For more detailed coverage of the anatomy and neurophysiological controls involved in this very complex area of research, the reader is referred to texts by Anderson and Matthews (1976), Sessle et al (1976), and Dubner et al (1978).

1.7.1 Jaw Reflexes

The jaw reflexes are usually considered to reflect reflex activity in the important jaw moving muscles i.e. masseter, temporalis, pterygoids and digastric. All are supplied by motor axons
originating from motorneurones in the trigeminal (V) motor nucleus, with the exception of the posterior belly of the digastric which is supplied by the facial nerve, and the motorneurones of this muscle lie in the accessory facial nucleus.

The simplest reflex arc is one with a single synapse between the afferent and efferent neurons. Such arcs are monosynaptic, and reflexes occurring in them are monosynaptic reflexes e.g. the jaw-closing reflex. Reflex arcs in which one or more interneurons are interposed between the afferent and efferent neurons are termed polysynaptic. The activity of polysynaptic reflex arcs are modified by spatial and temporal facilitation and the effects of other modifying influences. The reflex arcs arising in muscle spindles of the muscles controlling articulation of the teeth are generally accepted to be polysynaptic in nature. (Ganong, 1983).

1.7.2 Neurobiology of the Occlusal Interface.

Dellow and Lund (1971) have suggested that the control of jaw muscle function is centrally programmed by a brain stem pattern generator which is activated by adequate inputs from certain higher centres in the brain. Neurophysiological control of jaw muscle activity is also influenced by peripheral afferent activity arising from receptor groups in various anatomic structures such as muscles (polysynaptic reflex arcs arising from muscle spindles, inverse stretch reflex mediated through Golgi tendon organs), periodontal, mucosal and periosteal (various specialized and non-specialized nerve endings), and the temporomandibular joints (free nerve endings, Golgi tendon organs, encapsulated and non-encapsulated nerve endings.) (Dubner et al 1978, Klineberg 1986).

The functional articulation of the teeth is not simply determined by occlusal contact relations alone, but also by factors such as the anatomy of the joint structures, the restraints of the
ligaments, and the shape and orientation of the occlusal plane. Storey (1985) notes that the functional articulation of the maxillary and mandibular teeth is determined by both passive (or nonreflex) and active (reflex) interactions. By this interpretation, the "normal" occlusal interface is considered to be passively determined. Under certain conditions, to be identified later, abnormal occlusal contacts can lead to reflex alteration in movement patterns at the occlusal interface.

The physical features of tooth guidance vary with tooth arrangement and inter-arch relationships of anterior and posterior teeth. Klineberg (1986) notes that anterior guidance is provided by the overbite and overjet arrangement of anterior teeth and posterior guidance by the relationships of inclines of bicuspid and molar supporting cusps. The effect of posterior guidance may be increased by factors such as tilting and drifting of teeth and variations in the occlusal plane antero-posteriorly (curve of Spee) and laterally (curve of Wilson).

Schaerer, Stallard and Zander (1967) were among the first to differentiate the effects of passive and active guidance at the occlusal interface in electromyographic experiments. Bridges incorporating switches that would signal tooth contact in the intercuspal position and EMG activity of working and balancing side interferences during mastication were used. They found that on contact with an interference, muscle activity stopped for about 20 milliseconds ("silent period") followed by asymmetric activity of the muscles ("lateral jaw reflex") leading to an avoidance of the interference.

These experiments demonstrated that under normal circumstances occlusal guidance was passive, while balancing side interferences caused closure patterns which were reflex modulated as a consequence of occlusal feedback. Storey (1985) believes that at first the response to initial contact of the teeth is unlearned or conditioned, then following multiple contacts on the interference,
the offending contact is avoided through conditioning.

1.7.3 Neurobiology of the Temporomandibular Joint Interface.

Guidances for function of the temporomandibular joint may also be considered as either passive or active reflex activity. Anatomical constraints on condylar position provide the passive guidance while active reflex guidance is derived from afferent influences initiated in receptors innervating the joint, the periodontium and the musculature. Occlusal interferences may unilaterally or bilaterally cause protraction of one or both condyles. Storey (1985) notes that if small, these displacements are described as "slides in centric", and if large as "functional malocclusions".

1.7.4 Occlusal Interface Versus TMJ Interface.

Under certain circumstances it is possible for the occlusal and joint interface to be under active guidance, and these reflexes to be working to different ends. For example, reflexes working to position the condylar assembly in an ideal location may be opposed by reflexes trying to achieve maximal intercuspation of the teeth. In such a situation the joints, occlusion and muscles are not in harmony. In many cases these disharmonious relationships are asymptomatic; occasionally there are symptoms of pain and dysfunction associated, as will be discussed in more detail in the following chapters.

1.8 Features of an Ideal Occlusion.

Williamson (1985) summarizes the features of an ideal occlusion after any form of
treatment as:

1. A static (centric) occlusion in which the mandibular condyles are seated on the posterior slope of the articular eminence with the meniscus properly interposed. Figure 1.

2. A dynamic occlusion in which the cuspids and incisors disclude the posterior teeth in any eccentric movement.

3. A minimum level of muscular contraction during function.

In other words, a static and dynamic occlusion that gives the patient the ideal skeletal and muscular function and yet minimizes muscle contraction during parafunctional cyclic jaw movements, would seem to offer the best probability for optimum oral health.
TEMPOROMANDIBULAR JOINT ANATOMY

MANDIBULAR FOSSA

ARTICULAR TUBERCLE

ANTERIOR SLOPE

POSTERIOR SLOPE

CREST

BILAMINAR ZONE

ARTICULAR TISSUE (0.3–0.5 mm thick) – FIBROUS CONNECTIVE TISSUE AND FIBROCARDILAGE

INTERARTICULAR DISC – CENTRAL BEARING SURFACE

CONDYLAR FOVEA

LATERAL PTERYGOID MUSCLE - LOWER HEAD

LATERAL PTERYGOID MUSCLE - UPPER HEAD - SPHENOMENISCAL MUSCLE

Figure 1 - Diagrammatic representation of a mid-sagittal profile of the human temporomandibular joint in centric occlusion, with central bearing surface correctly aligned between condylar head and posterior slope of articular tubercle.


See also Rees A. (1954), British Dental Journal, 96:125–133.
CHAPTER TWO
TEMPOROMANDIBULAR JOINT DYSFUNCTION – CURRENT CONCEPTS ON AETIOLOGY

2.1 Historical Note and Definition.

It is usually considered that Costen (1934) was the first to describe the symptoms associated with the temporomandibular joint. Using a sample of eleven subjects, he noted a number of symptoms including impaired hearing, dizziness, tinnitus, headache, popping noises in the temporomandibular joint, earache and dry mouth. This grouping of symptoms came to be known as Costen's Syndrome, and according to Costen (1934), the condition was due to the loss of molar support resulting in damage to the TMJ and associated structures.

Schwartz (1959) was one of the first to view the condition as a pain-dysfunction syndrome. He regarded emotional tension as the most important factor in producing muscle spasm while malocclusion and premature tooth contacts were considered of minor importance. The opposite view was taken by Sarnat (1964) and Ramfjord and Ash (1971), who were of the opinion that occlusal interferences and/or mandibular displacement were the most important predisposing causes.

The term Costen's Syndrome eventually fell into disuse and was replaced by the term TMJ dysfunction syndrome. Other terms for the condition have included TMJ syndrome, TMJ Pain Dysfunction Syndrome, craniomandibular syndrome, TMJ arthritis, myofascitis, myalgia and fibrositis (Foreman 1985).

Rugh and Solberg (1976) in their review of the literature at the time, found there was consensus on three symptoms which are now held to comprise TMJ dysfunction syndrome:
1. Pain and tenderness of the muscles of mastication and temporomandibular joint.

2. Sounds during condylar movements eg. popping, clicking or crepitus of the jaw.

3. Limitations of mandibular movements.

Greene et al (1969) note that other symptoms such as subluxation/dislocation of the mandible, tinnitus and dizziness may also be present, but are not considered necessary for the diagnosis of TMJ dysfunction syndrome.

The term Myofacial Pain Dysfunction Syndrome (MPD) was originally proposed to identify a subgroup of TMJ patients which Laskin (1986) considered represented the majority of patients reporting pain and dysfunction of the masticatory system. According to Laskin (1986), MPD syndrome may be defined as the presence of one or more of the above three symptoms, with the exception that the pain is always unilateral. In addition, two other conditions must be noted:

1. Absence of clinical or radiographic evidence of organic changes in the temporomandibular joints.

2. Lack of tenderness in the temporomandibular joints when palpated in the external auditory meatus.

It has been noted by a number of authors (Moss and Garrett 1984, Foreman 1985), that the terms TMJ dysfunction syndrome and MPD syndrome have often been used in the literature synonymously, leading to much confusion.

2.2 Prevalence Studies.

A number of studies have evaluated the prevalence of specific symptoms in various
populations. In a group of 269 young dental nurses, Posselt (1971) found that 41% exhibited TMJ sounds, 15% had headaches and 6% reported facial pain.

In a larger sample, Hansson and Nilner (1975) examined 1,069 employees of a Swedish shipyard, ranging in age from 20 – 65 years. They reported that 7% had some temporomandibular joint or related muscular symptoms, and 30% had two or more such symptoms. The most frequent symptom was clicking of the TMJ, which occurred in 65% of the subjects.

In a study of 389 Swedish men aged 21 – 54 years with a mean age of 32 years, Ingervall et al (1980) noted impaired chewing function in about 10% of the sample, different types of parafuction in 26%, frequent headaches in 5%, TMJ or muscle pain in 3% and difficulty in mouth opening in 10% of the men. In this study, 60% of the men had one or more clinical symptoms of dysfunction.

A study of 285 seventeen year old Swedish adolescents by Wanman and Aerberg (1986) revealed 20% of the boys and 25% of the girls had TMJ sounds. 32% of the boys and 45% of the girls experienced tenderness of the lateral pterygoid muscle. Their results were in agreement with the above studies that the most frequent signs were muscular tenderness to palpation and clicking sounds from the TMJ.

Most studies have reported that the majority (65 – 85%) of patients with symptoms are female. (Campbell 1958, Weinberg and Lager 1980, Foreman 1985). Greene and Marbach (1982) are critical of many of these studies, arguing that many of them were not conducted according to statistical principles. Widely different criteria and research design make the studies difficult to compare with one another or with subsequent studies. Although this argument may be true when applied to the actual numerical results of these studies, the overall trend these studies
show is a high percentage of dysfunction symptoms in the general population, and this cannot be ignored.

The predominant age group appears to be aged 20 - 40 years according to Carraro et al (1969), Weinberg and Lager (1980). However, one important study by Helkimo (1976) concluded that no apparent sex or age differences were evident, apart from a slightly greater frequency in older individuals.
Figure 2 - Clinical signs in children using the clinical dysfunction index of Helkimo (1974), showing all grades of dysfunction increased significantly with age. (From Egermark-Eriksson et al 1981).

Index Code for Figures 2, 3 and 4. Clinical signs assessed using the clinical index of Helkimo (1974)(see Appendix 1), where 0 = no signs; 1 = mild signs; and 11/111 = moderate or severe signs.

Figure 3 - Clinical signs in children (Egermark-Eriksson et al 1981), young adults (Pullinger et al, unpublished data) and elderly men and women (Osterberg and Carlsson 1979), showing a relatively even prevalence of signs among the various groups.

Figure 4 - Clinical signs in young adults, indicating that over twice as many men are free of positive signs as women (Pullinger et al, unpublished data.)

2.3 Symptom Mechanisms.

2.3.1 Pain.

Dubner et al (1978) describe pain as:

"A complex experience that includes the sensations evoked by tissue damaging or noxious stimuli and the reactions to such stimuli. Attentional, cognitive, motivational, and emotional variables modify behaviours elicited by noxious stimuli."

Early theorists believed that pain was a primary or specific sensation with its own specialized peripheral receptors and central pathways. This theory cannot explain some of the characteristics seen with clinical pain, and is no longer held to be a satisfactory explanation of the mechanism of pain.

The more recent Gate Control Theory (Melzack and Wall, 1965), provides a general conceptual framework in which to view the multidimensionality of the pain experience. Their theory suggests that nerve impulses from afferent fibres to spinal chord transmission or relay neurons are modulated by a spinal "gating" mechanism, presumably in the substantia gelatinosa layer of the spinal chord dorsal horn. A similar gating mechanism is believed to exist in the trigeminal system. The term "gate" refers only to the relative amount of inhibition or facilitation that modulates the activity of the transmission cells carrying information about noxious stimuli.

In addition, this gating mechanism is influenced by descending control mechanisms from the brain that relate to cognitive, motivational, and affective processes. When the output of the transmission exceeds a critical level (known as central summation), neural systems are activated.
or accessed that allow for avoidance of the tissue damaging stimuli. (Dubner et al, 1978)

The actual physiological mechanism by which muscle spasm may arise and be experienced as pain in the presence of an occlusal interference has been proposed by Moller (1976). He believes that if shortening of an elevator muscle is arrested, gamma efferent activity will impose stretch upon the nuclear bag region of the intrafusal fibres of the muscle spindles in the involved muscle. The result is increased facilitation of the alpha motorneurons in the muscle spindle, resulting in stronger contraction of the muscle to overcome the resistance. Such a situation could occur during jaw closing when the mandible is stopped by an occlusal interference and the resultant hyperactivity leads to localized muscle spasm and it is this failure of the muscles to relax that leads to a reduction in blood flow and an accumulation of metabolic byproducts leading to pain.

Pain would appear to be the most common complaint of patients presenting with TMJ dysfunction. The pain is most often reported to be unilateral in origin, although bilateral pain is also common. The quality of pain is usually a dull ache although it can become sharp and acute (Solberg 1986a, Laskin 1986).

According to Laskin (1969), the pain is usually described by the patient as a dull ache felt in the ear or pre-auricular area, that may radiate to the angle of the mandible, the temporal area, or the lateral cervical area. The pain can be relatively constant, or may be worse on arising in the morning, or else is relatively mild in the morning but gradually worsens as the day progresses.

Muscle tenderness is also a common finding. Although not usually reported by the patient, this symptom is easily determined by the examiner. Laskin (1969) notes that the most frequent areas of tenderness are over the neck of the mandible and in the region distal and superior to the maxillary tuberosity.
The masticatory muscles implicated as being involved with pain include the masseter, temporal, medial pterygoid and lateral pterygoid muscles. (Moss and Garrett 1984).

An alternative to the muscle spasm hypothesis is the theory proposed by Yemm (1985), that only a part of the muscle is damaged. He suggests that some evidence is provided by the observation that the clinical abnormality is frequently limited to a small part of the muscle, whereas the muscle spasm theory would suggest that the tenderness might be expected to be distributed along the whole length of the muscle.

Although many theories have been suggested to account for the pain in MPD and TMJ dysfunction syndromes, a review of the literature reveals that few definite conclusions have been reached in relation to the physiological mechanisms responsible for this pain.

Moss and Garrett (1984) point out that each theory must be viewed cautiously since it is improbable that one explanation can accommodate the wide range of reported differences in the location, quality and temporal variations in the pain.

2.3.2 Sounds Occurring During Condylar Movements.

The sounds that occur in the temporomandibular joint are best observed by listening with a stethoscope to the TMJ while the patient opens and closes his/her mouth. The noises can be clicking, popping and/or crepitus. (Gale and Gross 1986).

According to Weinberg (1980), joint noise (clicking, popping, crepitus) is found in approximately 85% of acute TMJ dysfunction pain patients.
Other epidemiologic studies report an incidence of about 50% of joint sounds in the normal population. (Reider 1983, Gross and Gale 1983).

Solberg and Seligman (1985) describe joint noises as being due to an internal derangement of the TMJ i.e. a mechanical disturbance of the joint which interferes with smooth joint function. Shore (1976) believes this may occur due to muscle inco-ordination.

In normal opening, the condylar head always holds a constant relationship to the central part of the disc such that the disc moves forward with the condylar head during the opening of the mouth. (Moss and Garrett 1984). (See also 1.6 Muscle Function and Temporomandibular Joint Activity).

Solberg and Seligman (1985) believe that in the clicking TMJ, the posterior aspect of the articular disc is not positioned over the crest of the mandibular condyle, but rather, is displaced anteriorly. The clicking sound is produced as the condyle bumps over the thickened posterior border of the articular disc on opening and on closing as the disc and condyle once again destabilizes to the prolapsed disc relationship.

Bell (1984) notes that it appears that clicking occurs as the direct result of mechanical obstruction of the moving parts, which tend to wedge the articular surfaces apart as movement is arrested. As additional force is applied and the obstruction is overcome, the noise accompanies a renewed burst of movement.
**Figure 5** - Anterior disc displacement with reduction.  

a. The posterior thick band lies anterior to the condylar head when the teeth are in centric occlusion.  
The movement from b to c produces an audible or palpable click as the condyle snaps forward under the posterior thick band.  
At maximum opening, d, the disc and condyle are in normal relationship.  
On closing, the disc again subluxes forwards, the click is produced somewhere between e and f.  

Weinberg (1980) reports that a crepitus sound is associated with perforations in the disc. There is general agreement that crepitus sounds are associated with degenerative joint disease. (Shore 1976, Solberg and Seligman 1985).

Although there appears to be consensus on the mechanism involved in joint sounds, the direct causes of changes in disc motion and the disc itself have not been conclusively demonstrated. (Weinberg 1980). A number of authors believe that joint sounds may be a sign of an active or potential pathologic process that can be progressive and debilitating. (Weinberg 1980, Solberg and Seligman 1985).

A survey of 173 authorities in the areas of orthopaedics, rheumatology and arthritis by Gale and Gross (1986), attempted to ascertain the importance each individual attached to joint sounds. The general response of these experts indicated that clicking in the absence of other symptoms is not important. Clicking in the presence of other symptoms may be important; crepitus in the absence of other symptoms may be important; and crepitus in the presence of other symptoms is important. They advised caution in treating clicking when it is the only sign.

Solberg and Seligman (1985) advise that it is essential to include a history of past clicking in the evaluation of temporomandibular symptoms. Even if these symptoms are no longer present, past clicking episodes and their progression should be noted. They note that if clicking occurred in the past, the possibility of latent derangement of the TMJ should be suspected, even though this condition may not seem clinically apparent.

2.3.3 Limitations of Mandibular Movements.

Moss and Garrett (1984) believe that the symptom of limited mandibular movements can be
divided into two categories:

1. Restricted mouth opening (trismus).
2. Deviations during mandibular movements.

Toller (1976) feels that trismus may be closely related to the mechanisms involved in TMJ sounds. He contends that trismus may result from the failure of a full forward sliding of the articular disc in the upper joint compartment. Thus restricted mandibular movement results from the failure of the disc to slide fully down and over the articular eminence.

Solberg and Seligman (1985) believe that clicking may progress to painful displacement of the articular disc, with arrest of condylar motion along the functional path, a condition known as "locking" because it involves jaw limitation at midopening.

Bell (1969) suggests some other possible causes of trismus apart from jamming of the articular disc. These include:

1. Shortened elevator muscles due to spasm, inflammation or contracture.
2. Ankylosis due to fibrous adhesions or calcifications that join the articular surfaces.
3. Capsular ligament restriction due to inflammation or capsular fibrosis.

2.4 Aetiological Theories.

A review of the literature reveals a number of theories that have been proposed to account for the constellation of symptoms observed in the TMJ and MPD syndromes. These theories fall
generally into two groups:

1. Theories advocating the TMJ as the causal factor.
2. Theories based on muscular hyperactivity as the primary originating factor.

2.4.1 TMJ Dysfunction Theories.

According to Sutherland (1982), it is almost universally accepted that there is a functional circle involving the occlusion, the periodontal tissues (proprioceptors), the central nervous system and the musculature in the aetiology of temporomandibular joint disorders.

Treatment therefore involves interference in the effect this circle has on the dysfunctional state. The orthodontic implication of this view is that alterations to the occlusion, either temporarily or permanently, affect the afferent impulses from the periodontal receptors to the central nervous system, and could possibly lead to a dysfunctional state.

A number of intracapsular conditions have been implicated as causal factors of TMJ dysfunction. Moss and Garrett (1984) list the following conditions as possibly being involved in the aetiology of TMJ dysfunction:

1. Osteoarthritis.
2. Rheumatoid arthritis.
3. Condylar displacement. This is proposed to result from:
   a. Trauma.
   b. Malocclusion.
   c. Oral habits such as bruxism.
Osteoarthritis.

Carlsson (1980) defines osteoarthritis as:

"A primarily non-inflammatory disease characterized by both deterioration and abrasion of the articular soft tissue surface and by simultaneous remodelling processes in the underlying bone."

The disease process usually affects the centre of the joint and spreads peripherally. In cases with TMJ problems, osteoarthritis is most often unilateral, according to Gurainick (1984). In a study of 491 patients with TMJ dysfunction, Shwartz and Cobin (1957), found that arthritis and neurologic conditions were present in only 8% of the sample.

In another study by Kopp and Rockler (1978), of patients seeking treatment for TMJ dysfunction, it was found that radiographic signs of osteoarthritis were present in 20% of those examined. By contrast, Ericson and Lundberg (1968) found a comparable percentage of symptom free patients i.e. with no symptoms of TMJ dysfunction, who showed radiographic evidence of osteoarthritis in the temporomandibular joints.

On the basis of these studies no definite conclusion can be drawn on the implication of osteoarthritis as a cause of TMJ dysfunction syndrome.

Rheumatoid Arthritis.

Rheumatoid arthritis is an inflammatory systemic disease that produces destructive changes, customarily in more than a single joint. According to Weinberg (1979a), the conversion
of synovial cells to panus cells which secrete an enzyme capable of eroding the articular surface, is the characteristic feature of rheumatoid arthritis.

Ogus (1975) notes that its occurrence in the temporomandibular joints is relatively common, although it does not always affect this joint even in patients with severe involvement of several other joints. It is also noteworthy that the disease is not always bilateral in the temporomandibular joints.

Guralnick (1984) notes that obvious distortion of the occlusion may be seen with rheumatoid arthritis, but it is only rarely present in degenerative joint disease. Further, in cases of juvenile rheumatoid arthritis, there is such extensive damage to the condyle that growth of the lower jaw may be seriously impaired by the development of ankylosis. Fortunately, not all rheumatoid arthritis patients suffer such crippling disease.

Diagnosis is established by both laboratory and radiographic studies. A positive rheumatoid factor, particularly in the presence of multiple joint involvement, is fairly decisive in establishing diagnosis. Radiographic findings may be less positive, for there is often similarity to degenerative joint disease. (Guralnick 1984).

Carlsson (1980) notes, however, that early diagnosis of rheumatoid arthritis is often difficult since erosive changes are usually not visible radiologically during the first year of the disease.

Condylar Displacement.

a. Trauma.
Reade (1984) believes that TMJ dysfunction begins with, or is initiated by trauma to one or both temporomandibular joints. The traumatic incident may be something as simple as a wide uncontrolled yawn, a vigorous kiss, a long dental appointment, or a more obvious event such as a general anaesthetic or a blow to the mandible.

The argument is proposed by Reade (1984) that this initiating trauma injures the soft parts of the joints i.e. the capsule, ligaments, meniscus or synovial membranes, rather than the bony components. Reade (1984) believes that this concept of a traumatic initiating event explains how a TMJ dysfunction can occur at any time in a patient’s life, and how it can occur with any degree or type of malocclusion.

b. Malocclusion.

It is apparent in a review of the literature that an effort is being made in dental research, on an epidemiologic basis, to determine those morphologic components of malocclusion which are potentially dangerous to the integrity of the stomatognathic system.

Malocclusion has been one of the most frequently cited causes of both condylar displacement and masticatory muscle disorders. The relationship between malocclusion and muscle disorders will be discussed in a later section.

A number of cross sectional morphologic studies have been performed which attempt to analyse whether different types of malocclusion can be implicated in the cause of TMJ dysfunction. These characteristics include:
1. **Angle Classes of Malocclusion.**

Egermark-Eriksson et al (1983) with a random sample of 402 children of three ages, 7, 11 and 15 years, studied the influence of dental status, occlusal interferences, occlusal anomalies, on the clinical signs of mandibular dysfunction. They found that a positive correlation existed between Class II and Class III occlusions and mandibular dysfunction in the children studied.

Mohlin et al (1980) studied the relation between malocclusion and symptoms of mandibular dysfunction and cuspal interferences in a group of 389 men aged 21 – 54 years with a mean age of 32 years. They concluded that there was a strong correlation between mandibular dysfunction and Angle Class III malocclusion. By contrast, no correlation was found between Angle Class II malocclusion and clinical symptoms of dysfunction.

Lieberman et al (1985) sought to determine an association between certain morphologic characteristics of occlusion such as Angle classification, overbite, open bite, cross bite and crowding, in a sample of 369 Israeli schoolchildren aged 10 – 18 years. They were unable to find a correlation between Angle Class II or Class III malocclusions and dysfunction symptoms.

2. **Cross-bite.**

Egermark-Eriksson et al (1983) found a significant correlation between molar crossbite and muscle sensitivity. However, Lieberman et al (1985) could not find a significant correlation between crossbite and dysfunction syndromes.

3. **Frontal Open Bite.**

A positive correlation between frontal open-bite and dysfunction was reported by Mohlin and
Kopp (1978) in an adult sample, and similarly in a sample of children by Williamson (1977). The study by Lieberman et al (1985) also found a positive correlation between overbite less than 1 mm and dysfunction.


In Williamsons (1977) sample of 304 patients aged 6 - 16 years, he found that 107 were symptomatic, and of these 72% had either deep bite or an open bite. By contrast, the studies by Egermark-Eriksson et al (1983) and Mohlin et al (1980) found no correlation between deep bite and dysfunction symptoms. Liebermann et al (1985) reported a positive correlation where the deep bites were greater than 5 mm.

Weinberg (1979a) maintains that the conflicting nature of this research is due to the lack of an operational definition of malocclusion, arguing that the criteria for malocclusion have never been satisfactorily determined. In other words, the lack of uniformity may be attributed to the lack of uniformity in quantifying both aetiological factors and the symptoms themselves.

Better evidence may be provided for a correlation between malocclusion and TMJ dysfunction symptoms if future studies clearly define malocclusion and include radiographic evaluations of each condyle's location in relation to the glenoid fossae as a function of mandibular position. Thus, the existence of condylar displacement in relation to malocclusion could be better determined. (Weinberg 1979a).

c. Oral Habits.

The existence of destructive oral habits have also been proposed as causes of condylar
displacement and thus potential causes of TMJ dysfunction.

Habits such as bruxism, lip and lateral tongue biting, chewing of pens, pencils or a pipe, cupping the chin in the hand or resting the head with the hand on the side of the face, and playing certain musical instruments such as the violin, clarinet, trumpet etc. which require certain mouth and chin positions, have all been suggested as being correlated with TMJ dysfunction symptoms. (Moss and Garrett 1984).

Only the habit of bruxism has received any degree of empirical support as being involved with TMJ dysfunction. Toller (1979) estimated 40% of TMJ dysfunction patients exhibited nocturnal bruxism, while Ramfjord (1961) suggested a 100% correlation between the two disorders.

Occlusal interferences continue to be cited as aetiological factors in bruxism, according to Ramfjord and Ash (1983), although some authors such as Egermark-Eriksson (1983) would disagree with this concept. The relationship between bruxism and TMJ problems remains inconclusive.

2.4.2 Masticatory Muscle Hyperactivity Theories.

Theories related to muscular hyperactivity are, by definition associated with Myofacial Pain Dysfunction Syndrome. According to Haber et al (1983) support for muscular involvement in facial pain has been drawn from studies in three basic areas:

1. Studies which have induced pain similar to that of MPD syndrome in non-MPD subjects
via sustained contraction of the masseter and lateral pterygoid muscles.

Christensen (1971) investigated the effects of unilateral tooth grinding in normal subjects. Following a period of 30 minutes of tooth grinding, subjects frequently experienced a variety of facial pain and headaches. Both the pain and its locations were similar to that reported by MPD syndrome subjects.

Scott and Lundeen (1980) examined the effects of thrusting the mandible forward for a five minute period, in a group of non-MPD syndrome subjects. This experiment was specifically designed to hyperactivate the lateral pterygoid muscles. On comparison with a non-exercise control group, it was found the exercise group reported significantly more pain. The pain was reported to be similar in quality and location to that of MPD syndrome patients. However, other signs associated with MPD syndrome such as tenderness upon palpation, limitation of jaw function and sounds during condylar movement were absent.

2. Electromyographic assessment studies which have reported elevated EMG activity in the masticatory muscles of TMJ patients. (Munro 1975b, McCall et al 1978, Skiba and Laskin 1981). For example, the inhibitory response following initial tooth contact (silent period), has been shown to be longer in symptomatic patients compared to non-symptomatic patients. (Bessette et al 1974, Munro 1975b, McCall et al 1978)

When these patients are treated with acrylic splints, their symptoms remit and the length of the silent period is reduced, indicating a more normal muscle function. (Beemsterboer et al 1976).

3. Studies which have employed treatments such as EMG biofeedback to reduce masticatory muscle activity levels. For example Clarke and Kardachi (1977) attempted to reduce
MPD problems in seven patients by treating their nocturnal bruxism. The treatment consisted of administering analogue auditory feedback from the masseter and temporals muscles. As the feedback did not interfere with sleep, it was given to patients while they slept. Four patients reported improvement following treatment.

Other EMG biofeedback studies showing some positive results have been conducted by Carlsson et al. (1975), Peck and Kraft (1977) and Olson (1977).

2.4.3 Causes of Masticatory Muscle Hyperactivity.

The reasons for increased and sustained masticatory muscle activity have been generally divided in the literature into two categories:

1. Local causes.
2. Centrally mediated causes.

1. Local Causes. (See also 1.7.2 Neurobiology of the Occlusal Interface, and 2.3.1 Pain.)

Occlusal interferences have often been claimed to play an important part in the aetiology of mandibular dysfunction. Ramfjord and Ash (1971) define occlusal interferences as occlusal contacts hampering or hindering smooth gliding harmonious jaw movements with the teeth maintaining contacts.

It is generally seen that the majority of patients with disturbances of the masticatory system present with some form of occlusal disharmony. However, not everyone with occlusal disharmony
experiences discomfort or pain, or shows evidence of trauma from occlusion, such as muscle
dysfunction or TMJ dysfunction.

Geering (1974) suggested that the combination of a slide from centric relation to centric
occlusion with a lateral component, and balancing interferences on one or both sides seem to have
the most disturbing effects on the masticatory system.

In a study of 70 patients with the above interferences, Geering (1974) found 70% of these
patients had pain and/or symptoms of disturbance of the masticatory system. By contrast, in a
group of 35 patients with a slide but with no lateral component and no balancing interferences,
Geering (1974) found 60% had no pain symptoms at all.

In a study of 389 Swedish men by Ingervall et al (1980), a faulty relation between centric
relation and centric occlusion due to non-working side interferences was found in about every fifth
individual. This type of interference was positively correlated to TMJ sounds and muscle
tenderness, providing further evidence that a faulty relation between the two positions leads to a
predisposition for symptoms of dysfunction.

In a similar study by De Boever and Adriaens (1983), 135 patients seeking treatment for
pain and dysfunction of the TMJ were investigated. A weak correlation between non-working side
interferences and TMJ symptoms was found.

In a study by Randow et al (1976) seven out of eight subjects developed subjective symptoms
of TMJ dysfunction after creation of an interference in both centric relation and centric occlusion.
Six subjects exhibited one or more clinical signs of dysfunction, and all subjects had considerable
changes in the EMG recordings.
Magnusson and Enbom (1984) conducted a double blind study on two groups of young, healthy women without signs or symptoms of mandibular dysfunction. Each group contained 12 individuals. In one of the groups, balancing side interferences were applied bilaterally, whereas the application was simulated in the other group. The participants were re-examined after two weeks.

Ten individuals in the experimental group reported one or more subjective symptoms during the two weeks, whereas seven exhibited clinical signs of dysfunction. The commonest symptom was headache, and the commonest clinical sign was muscles tender to palpation. In the control group, three individuals reported subjective symptoms, and three had clinical signs of dysfunction. One week after elimination of the interferences, all signs and symptoms had disappeared in all individuals but two. In these two subjects it took six weeks before pre-experimental conditions were restored.

Magnusson and Enbom (1984) concluded from their study that there is no simple relationship between interferences and signs and symptoms of dysfunction. How the individual reacts to local factors depends to a large extent on his or her psychic condition. In some individuals addition of balancing side interferences is sufficient to create dysfunction. Their findings thus illustrate the importance of local factors in the aetiology of mandibular dysfunction, but show that a relationship is not obligatory.

Klineberg (1986) proposes that if a supracontact interference is so severe that it cannot be absorbed by periodontal and articular tissue resiliency, there will be immediate changes in jaw muscle activity, altering evoked reflexes and resulting in muscle asynchrony and hyperactivity.

The altered neuromuscular control provoked by tooth contact interferences alters the overall programming of cyclical jaw movement. Such changes may not be in the best long term interest of
co-ordinated jaw muscle function, and provide the basis upon which jaw muscle hyperactivity may develop and the associated symptoms of myofacial pain dysfunction may thus arise. (Klineberg 1978).

2. Centrally Mediated Causes.

The most commonly suggested cause of central activation of masticatory muscle hyperactivity is psychological and/or physical stress.

Haber et al (1983) defines stress as:

"An innate response that helps us adjust to the everday demands of life."

Stress is both necessary and desirable for our bodies to react to various demands calling for adaptation and adjustment. The disruption of an individuals normal response to stress, may result in a breakdown of homeostatic processes.

One area of support for a stress related concept of TMJ dysfunction syndrome is based on the belief that TMJ dysfunction patients show a higher incidence of other 'psychosomatic' disorders. Berry (1969) noted an incidence rate of migraine headache and backache which was ten times higher than that reported in the normal population. In another study, Gold et al (1975) found that 135 MPD syndrome patients had more frequent low back and neck pain, nervous stomach, asthma and history of ulcers, than did a control group of subjects.

The search for distinct personality correlates of patients reporting facial pain and mandibular dysfunction has resulted, however, in confusing and contradictory findings. Although
specific personality profiles have been reported for MPD patients by Schwartz (1974) and Shipman (1973), other studies have failed to show any support for distinct personality profiles. (Moss and Adams 1984).

One current view proposed by Greene et al. (1982) is that no one particular personality type is predisposed to the development of TMJ and MPD syndromes. Rather, the development of symptoms appears to be caused by a combination of a physiologic predisposition to develop dysfunction in the masticatory musculature, current levels of psychological and physical stress, and the individual's ability to cope with such stresses.

Greene et al. (1982) suggests that research conducted over the last thirty years has shown a strong relationship, in general, between stress and various physiological responses such as increased heart rate, increased respiration, and increased muscle tension. This normal psychophysiological relationship occurs in all individuals, but usually one type of response predominates over the others in a particular individual. This response is described as 'response specificity'.

In addition, the degree or magnitude of each response varies considerably from one person to another, so that in some individuals a response may become pathologic. As a result, it is suggested by Greene et al. (1982) among others, that they may develop somatic symptoms such as headache, duodenal ulcer, dermatitis or masticatory muscle hyperactivity.

Mercuriet et al. (1979) attempted to evaluate the physiological effects of stress in twenty MPD patients and twenty non-MPD controls. Each subject listened to a 75 decibel white noise, performed a card sort task, responded verbally to words denoting neutral, sexual and personal themes, and engaged in a pain tolerance test. During these tests, the authors recorded heart rate,
skin resistance and EMG activity in the frontalis, right gastrocnemius and both masseter muscles.

Their results indicated that the MPD patients exhibited significantly higher EMG levels in the masseter and frontalis muscles, than the controls. No significant differences of heart rate or skin resistance were noted between the groups. The authors concluded that the results supported the existence of a response specific reaction to stress in MPD patients.

A final area of study related to centrally mediated causes of TMJ syndrome is the evaluation of threshold/tolerance levels of experimentally induced pain. Using noxious electrical stimulation which was increased in discrete steps, Molin et al (1973) reported quantitative scores of comparisons between MPD patients and non-MPD controls. They found that MPD patients demonstrated significantly lower pain thresholds than did the controls. However, there did not appear to be significant differences in pain tolerance between the groups.

2.5 Overview

In summary, it would appear that in our present state of knowledge, TMJ and MPD disorders are best viewed as a result of a number of interrelated factors of neurophysiological, psychological, occlusal, and occasionally, pathological origin. The importance of skeletal and dental malocclusion is still doubtful or lacking conclusive documentation.

Attempts have been made to integrate the various theories concerning the aetiology of TMJ dysfunction; one of the more comprehensible versions is given by Dubner et al (1978). The pain of the syndrome can arise either in the joint itself or in the masticatory muscle from which it may be referred to other sites. The generalized muscle hyperactivity or localized muscle spasm may be
the result of emotional stress, protective reflexes, or changes in muscle length. Whether the muscle response to emotional stress occurs depends on the magnitude of the stress and the psychological makeup of the patient. Protective reflexes can occur protecting the joint, muscle or dentition. Whether the muscle response to occlusal interferences occurs depends on a large number of factors that modulate the sensory input. Pain in a muscle can give rise to protective reflexes leading to hyperactivity in the same or another muscle. This sequence then acts as a "vicious circle". Protective reflexes may lead to hyperactive muscle spasm only when the stimulus that gives rise to the reflex persists eg. inflammation in the joint does not abate or a tooth interference is not removed.

A multifactorial view of aetiology would seem to be the most useful and reasonable approach taken by most current authors (Moss and Garrett 1984, Foreman 1985), with occlusion and malocclusion being only one aetologic factor in a very complex problem.
CHAPTER THREE

DIAGNOSIS OF TEMPOROMANDIBULAR JOINT DYSFUNCTION

A systemic approach should be used for the differential diagnosis of temporomandibular joint dysfunction pain syndrome and all other forms of craniomandibular pain.

In addition to obtaining a comprehensive medical and dental examination of every patient, a brief screening history and examination pertinent to the temporomandibular disorders should be done to enable the practitioner to determine the need for a more detailed evaluation.

Should the screening history and examination result in positive findings, a second more comprehensive assessment specifically related to the evaluation of temporomandibular disorders can be completed.

3.1 Initial Screening History and Examination

A simple and practical screening history and examination was proposed by the President of the American Dental Association's 1983 Conference on the Examination, Diagnosis, and Management of Temporomandibular Disorders. (Griffiths 1983.) It was suggested that this preliminary screening history should be part of the routine health history asked of all patients, and should include asking patients the following questions:

- Do you have difficulty opening your mouth?
- Do you hear noises from the jaw joints?
- Does your jaw get "stuck", "locked" or "get out"?
- Do you ever have pain in or about the ears or cheeks?
- Do you have pain on chewing or yawning or wide opening?
- Does your bite feel uncomfortable or unusual?
- Have you ever had an injury to your jaw, head or neck?
- Have you ever had arthritis?
- Have you ever previously been treated for a temporomandibular disorder? If so, when, what, how, and by whom?

A brief clinical examination for temporomandibular disorders should include:

1. Inspection for facial symmetry.
2. Evaluation of jaw movements (details will be given on this in a later section).
3. Palpation - designed to detect any masticatory muscle tenderness and joint tenderness, incoordinations, clicking and crepitus.

Should positive findings emerge from the above simple screening history and examination, a more comprehensive evaluation should be performed.

3.2 Diagnosis of TMJ Dysfunction and MPD Syndrome.

Satisfactory management of all pain problems depends on a correct diagnosis. A comprehensive history, a careful clinical examination and correlated special tests are essential requirements for any diagnosis.
A standardised approach employing detailed history and examination techniques allows a systematic analysis of pain problems, and minimises the likelihood of omitting important details.

A standard comprehensive history and examination format should investigate the following areas:

1. History
   1.1 Chief complaint
   1.2 Medical
   1.3 Dental
   1.4 Personal history
2. Orofacial Examination
   2.1 Clinical occlusal analysis
3. Special Tests
   3.1 Study casts
   3.2 Radiographs
   3.3 Arthrography
4. Other
   4.1 Electromyographic - jaw reflexes.

1. History.

   a. Chief Complaint

   The patient's chief complaint should be elicited, together with the location, duration and type
of pain present. Changes in the intensity or character of the pain in response to factors such as movement, swallowing, talking time of day, fatigue, and stress should also be noted, along with the response to any drugs taken for the pain. (Foreman 1985)

b. Medical History

A thorough history on past and present medical conditions relating to the TMJ is required. Gelb (1977) proposed the following list of conditions which could be of significance:

i. Musculoskeletal diseases - External traumatic arthritis, osteoarthritis, rheumatoid arthritis, scleroderma, bone dyscrasias.

ii. ENT Symptoms - Parotitis, middle ear infection, nasopharyngeal conditions, osteomyelitis, sinusitis, cysts, polyps.

iii. Vascular diseases and blood dyscrasias.

iv. Traumatic disorders - To TM joints or to other parts of the head.

v. Headaches and head pain (location, character, frequency, duration). - Migraine, neuralgias.

vi. Medications - According to Foreman (1985), certain medications may produce or exacerbate orofacial pain, and therefore the patient must be questioned on all drugs, prescribed or not, which are currently being taken. For example Kraak (1967), found that chlorpromazine (Largactil, May and Baker) and phencyclidine (Stemetil, May and Baker), may produce facial pain as a result of hyperirritability of the lateral pterygoid muscles, causing them to shorten. This muscular contraction positions the condyles forward in the glenoid fossa so that the posterior teeth contact prematurely. Further closing causes posterior deviation of the mandible, stretching the already irritated lateral pterygoids and thus producing spasm and pain.

It has also been reported that arthralgia has been noted in women taking hormonal
contraceptives (Spiera and Plotz, 1969), and this possibility should be considered when making a diagnosis in patients taking these drugs.

c. **Dental History**

i. Evaluation of previous dental experiences.

ii. Previous TMJ treatment.

iii. Pain in specific teeth.

iv. Oral symptoms other than pain eg. bruxism, jaws clenched upon awakening, muscle fatigue.

v. Ear symptoms – Weinberg (1980) notes the most common complaints are stuffiness and/or loss of equilibrium and tinnitus

d. **Personal History**

Geib and Bernstein (1983) suggest an evaluation of personal factors such as marital status (past and present), children, in-laws, parents, sickness in the family, occupation, sleeping habits to determine potential areas of stress.

The usual habits of biting objects, clenching, bruxism, hand pressure, and sleeping positions should be queried. Also, Weinberg (1980) emphasises the need to explore a detailed life-style history as sometimes the main aetiology may fall into this category and not under stress, occlusion or joint pathology. For example, a patient may play a musical instrument, such as the violin or a wind instrument, which may require unusual jaw positions for extended periods of time.