

CHAPTER 2: LITERATURE REVIEW

2.1. Masticatory system

Masticatory system, also known as masticatory apparatus, comprises temporomandibular joint (TMJ) and masticatory muscle.

2.1.1. Temporomandibular joint (TMJ)

2.1.1.1. Functional anatomy of TMJ

TMJ is a diarthrodial synovial paired joint. This means that the joint has to function in pairs and the joint movement will involve both joint compartments. TMJ comprises mandibular condyle, glenoid fossa (mandibular fossa or articular fossa), articular eminence (articular tubercle), articular disc, capsule (which is lined by synovial membrane) and ligaments. Each joint involves the articular eminence and glenoid fossa above and mandibular condyle below. The articular disc divides the joint space into the upper and lower compartments. It is very important to understand the TMJ in a three dimensional structure (Gray et al., 1995) (Figure 2.1.1.1(a)-(e)).

2.1.1.1.1. Articular disc

Articular disc has been referred to as meniscus. However it is not a meniscus at all as we can find it in the knee joint (Okeson 1998). Two dimensional illustrations do not readily convey the shape of the articular disc (Rees, 1954).

Rees (1954) has described the articular disc as like a jockey's cap (Figure 2.1.1.1(a)) which overlaps the condylar head, with the border of the cap forming its attachment to the condyle and the peak attached to the tendon of the lateral pterygoid muscle. The articular disc is firmly attached to the head of the condyle medially and laterally where

it bends with the capsule. Rees (1954) described the meniscus as having four parts. The anterior band is moderately thick but narrow antero-posteriorly. The posterior band is the thickest and widest, whereas the intermediate zone is the thinnest. The interposition of the thin intermediate zone between the anterior and posterior bands gives the disc more flexibility. The bilaminar zone comprised of loose fibro-elastic tissue forming its upper part and inextensible collagenous tissue forming its lower part (Gray et al., 1995) (Figure 2.1.1.1(b)).

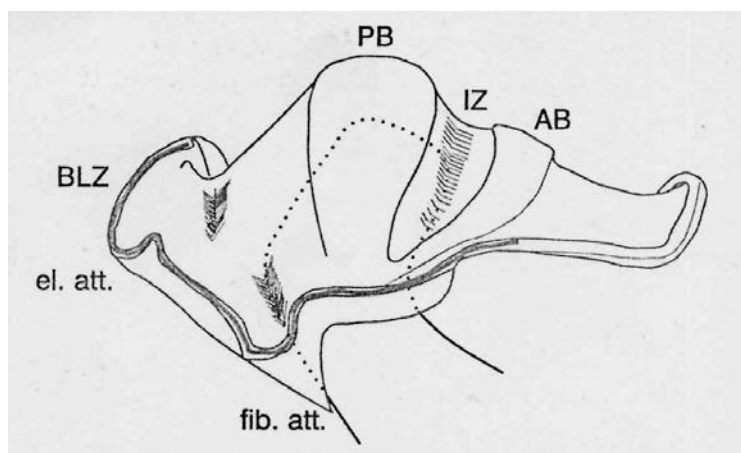


Figure 2.1.1.1(a) Line drawing of TMJ representing a Jockey's cap. Fib. att., fibrous attachment to posterior neck of condyle, el. att., elastic attachment to fossa; BLZ, bilaminar zone; PB, posterior band of articular disc; IZ, intermediate zone of articular disc; AB, anterior band of articular disc; SPt, attachment to the superior pterygoid (superior head of lateral pterygoid); IPt, attachment to the inferior pterygoid (inferior head of lateral pterygoid) (Gray et al.1995).

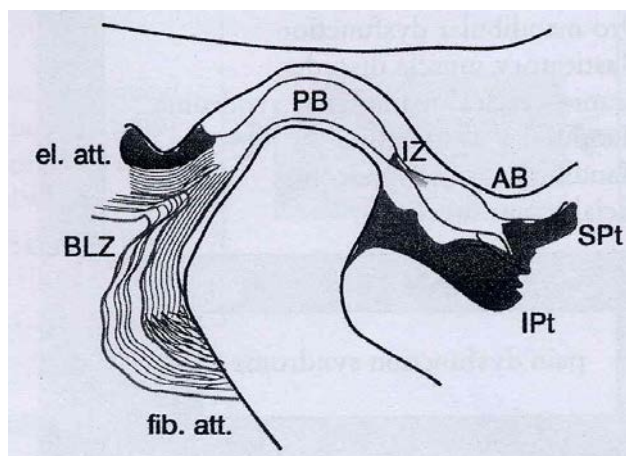


Figure 2.1.1.1(b) Line drawing showing the cross section of TMJ (Gray et al. 1995).



Figure 2.1.1.1(c) Superior aspect of the TMJ (Rees, 1954; Soames, 1995).

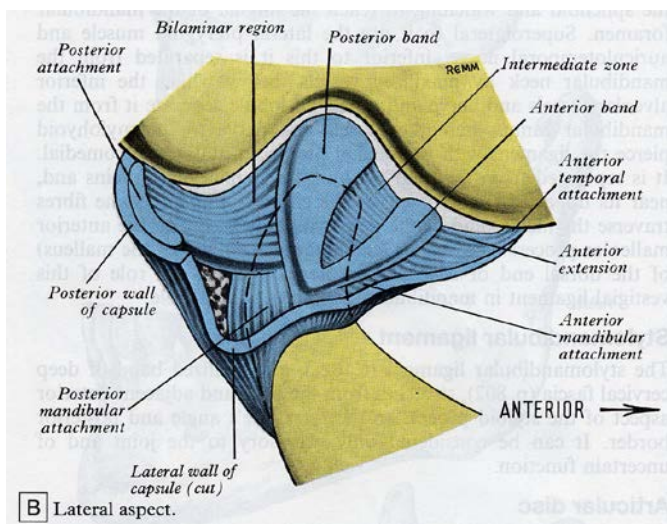


Figure 2.1.1.1(d) Lateral aspect of the TMJ (Rees, 1954; Soames, 1995).

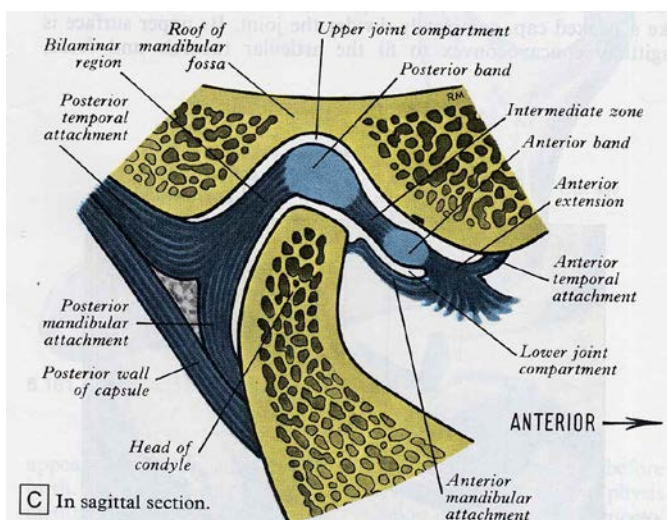


Figure 2.1.1.1(e) Form, subdivisions and thickness variations of the intra-articular disc in the TMJ, in sagittal section (Rees, 1954; Soames, 1995).

2.1.1.2. Movement at TMJ

In the first few millimetres of jaw opening, the mandibular condyle rotates. As the opening continues, the condyle glides forward and downward on the inferior surface of the articular disc, which slide in the same direction down the slope of the articular eminence but lagging slightly behind relative to condyle. In the final maximal opening, the condyle rotates again, leaving the disc unchanged in its position on the eminence, but increasing its discrepancy with the disc. In closing, the condyle moves in the reverse direction. During anterior movement of the condyle up to maximum mouth opening, the head of the condyle will have moved approximately twice as far as the articular disc (Rees, 1954; Gray et al., 1995) (Figure 2.1.1.2 (a), (b)).

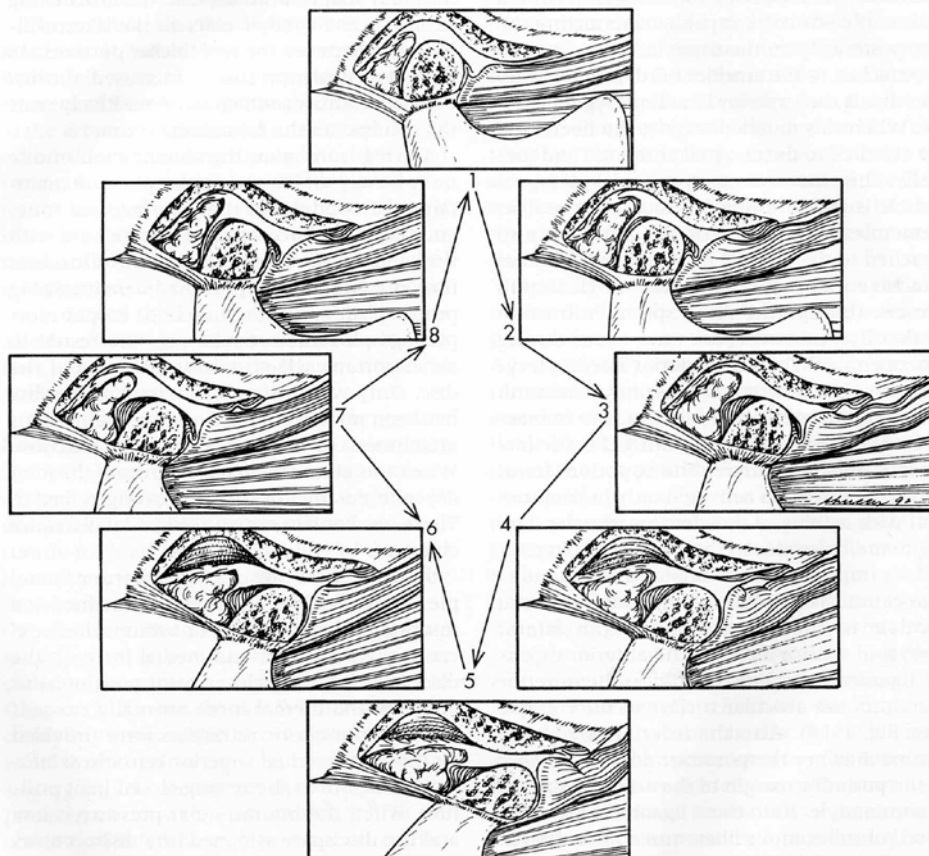


Figure 2.1.1.2 (a) Normal functional movement of the condyle and disc during the full range of opening and closing. Note that the disc is rotated posteriorly on the condyle as the condyle is translated out of the fossa. The closing movement is the exact opposite opening (Okeson, 1998).

Figure 2.1.1.2 (b) Changing relationships of the condyle of the mandible, the articular disc and the articular surface of the temporal bone during one complete opening (A-D) and closing (D-A) cycle of the mouth (Rees, 1954; Soames, 1995).

2.1.2. Masticatory muscle

Muscles of mastication consist of the masseter, temporal, medial pterygoid and lateral pterygoid muscles.

2.1.2.1. Functional anatomy of masseter and temporal muscle

2.1.2.1.1. Masseter muscle

The masseter muscle is a quadrilateral muscle consisting of two layers (Figure 2.1.2.1.1). The superficial layer, which is larger than the deep layer, arises from the maxillary process of the zygomatic bone and from the anterior two thirds of the inferior border of the zygomatic arch. Its fibers pass downward and backwards to insert into the angle and the lateral surface of the ramus. Its insertion on the mandible extends from the region of the second molar at the inferior border and extends posteriorly to include the angle. The deep layer arises from the deep surface of the zygomatic arch and inserts onto the upper part of the ramus. Its fibers run predominantly in vertical direction (Okeson, 1998; Soames, 1995).

The masseter muscle elevates the mandible to occlude the teeth in mastication. It is a powerful muscle that provides the force necessary to chew efficiently. The masseter muscle has a small effect in side-to-side movements, protraction and retraction (Okeson, 1998; Soames, 1995).

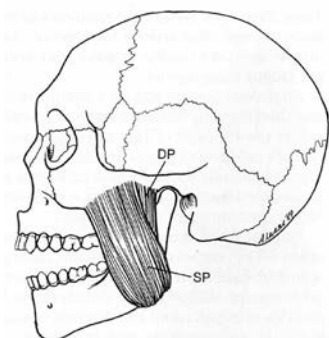


Figure 2.1.2.1.1 Masseter muscle: DP, deep layer; SP, superficial layer (Okeson, 1998).

2.1.2.1.2. Temporal muscle

The temporal muscle is a fan-shaped muscle that arises from the temporal fossa and from the deep surface of the temporal fascia. The muscle fibers converge to a tendon and passed deep to the zygomatic arch and inserts onto the coronoid process and the anterior border of the mandibular ramus almost to the third molar tooth. The anterior fibers of temporal muscle are orientated vertically, the posterior fibers almost horizontally, and the intervening middle fibers with intermediate degrees of obliquity (Okeson, 1998; Soames, 1995) (Figure 2.1.2.1.2).

The temporal muscle elevates the mandible to approximate the teeth. The anterior fibers elevate the mandible vertically. Contraction of the middle fibers elevates and retrudes the mandible. The posterior fibers retract the mandible after it has been protruded. Since the angulations of its muscle fibers vary, the temporal muscle is capable of coordinating closing movements. Thus, it is a significant positioning muscle of the mandible. The muscle is also a contributor to side-to-side grinding movements (Okeson, 1998; Soames, 1995).

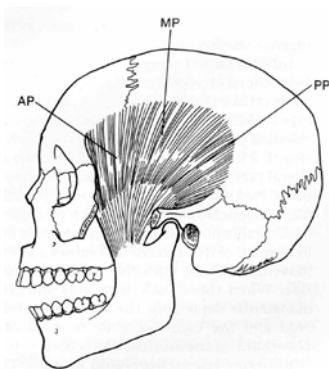


Figure 2.1.2.1.2 Temporal muscle: AP, anterior portion; MP, middle portion; PP, posterior portion (Okeson, 1998).

2.1.2.2. Neuromuscular system of the masticatory system

The neuromuscular system of the masticatory system, which is like any other neuromuscular system, is divided into the muscle fibers and the neurological structures.

2.1.2.2.1. Muscle fibers

The basic component of the neuromuscular system is the motor unit. A motor unit consists of a number of muscle fibers that are innervated by one motor neuron.

The entire muscle has three potential functions. First, when a large number of motor units in the muscle are stimulated, an overall shortening of the muscle result. This is called isotonic contraction. The action is to move the mandible. Second, when a proper number of motor units are stimulated, contraction of the muscle can occurs without shortening. This is called isometric contraction. The action is to hold or stabilize the mandible. Third, when stimulation of the motor units is discontinued, the fibers of the motor units relax and return to their normal length. This is called controlled relaxation (Okeson, 1998).

2.1.2.2.2. Neurological structures

The neurological structures comprise motor and sensory neurons. Sensory receptors are neurological structures that are located in all body tissues; provide information regarding the status of these tissues to the CNS via afferent neurons. The masticatory system has four major types of sensory receptors, namely muscle spindles, Golgi tendon organs, Pacinian corpuscles and nociceptors (Okeson, 1998).

A muscle spindle is a sensory receptor consists mainly of a bundle of intrafusal muscle fibers. They monitor primarily the muscle length. Contraction of intrafusal fibers or generalized stretching of the entire muscle will cause contraction of the muscle, mediated by muscle spindle (Okeson, 1998).

The Golgi tendon organs are located in the muscle tendon, and they are stimulated by contraction of the muscle. They primarily monitor muscle tension. The Golgi tendon organs protect the muscle from overcontraction by eliciting inhibitory stimuli directly to the muscle that they are monitoring. They may be more sensitive and active in reflex regulation during normal function (Okeson, 1998).

The Pacinian corpuscles are widely distributed and present in tendons, joints, periosteum, fascia, and subcutaneous tissues. They are considered to serve principally for the perception of movement and firm pressure such as in the joint structures (Okeson, 1998).

The nociceptors generally are sensory receptors that stimulated by injury and conduct injury impulses to the CNS via afferent nerve fibers. They are distributed throughout most of the masticatory system. They monitor the condition, position and movement of the tissues in the masticatory system (Okeson, 1998).

2.1.3. Biomechanics of mandible

The biomechanics of the masticatory system are determined by the morphology and structural arrangement of the TMJ, as well as the arrangement of the masticatory muscle, as they relate to the demands of function.

2.1.3.1. Analysis of forces

It was assumed that the mandible functions as a lever both during biting and the power stroke of mastication, with the mandibular condyle acting as a fulcrum. During biting and chewing, the resultant masticatory muscle force does not always pass through the bite point. The tissues of the TMJ are capable of dissipating considerable joint reaction force. The biomechanical analysis of the mandible can be done through analysis of moment (Figure 2.1.3.1(a)). Moment can be analyzed about any point since the summation of moments about any point is equal to zero under conditions of static equilibrium (Hylander, 1992).

Usually the mandible is thought to function as a Class III lever, which is overly simplistic because it implies that the various external forces acting on the mandible lie within the same plane. A better approach to modeling the biomechanics of the mandible is to analyze the magnitude and direction of all muscle and reaction force in three-dimension. However, a simplified analysis of forces and moments in both the lateral and frontal projection provides some interesting insights into patterns of reaction force along the ipsilateral and contralateral TMJ, by assumption that all muscle and reaction forces are essentially vertical and parallel to one another (Hylander, 1992) (Figure 2.1.3.1(b)).

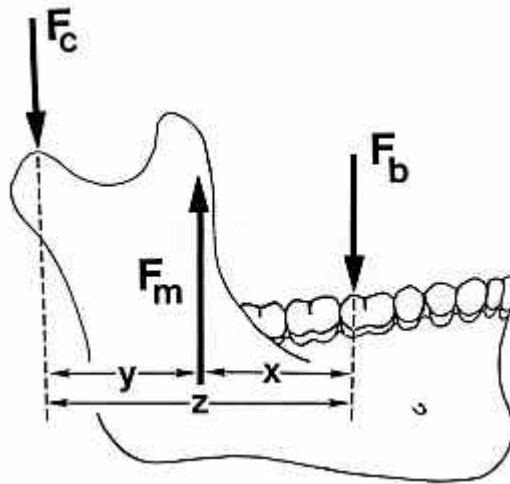


Figure 2.1.3.1(a) The human mandible functioning as a lever during biting along the first molar. Only the vertical components of the muscle and reaction forces are included in this figure. The resultant muscle force of the jaw elevators (F_m) is located posterior to the bite point. In order to maintain static equilibrium under these conditions, the muscle force is divided into reaction force along the bite point (F_b) and reaction force along the two mandibular condyles (F_c). For a given amount of muscle force, F_b can be determined by analyzing moments about F_c (i.e., $F_b = (F_m)(y)(z)$). F_c can be determined by analyzing moments about F_m (or F_b) (i.e., $F_c = (F_b)(x)/y$ or $F_c = (F_m)(x)/z$) (Hylander, 1992).

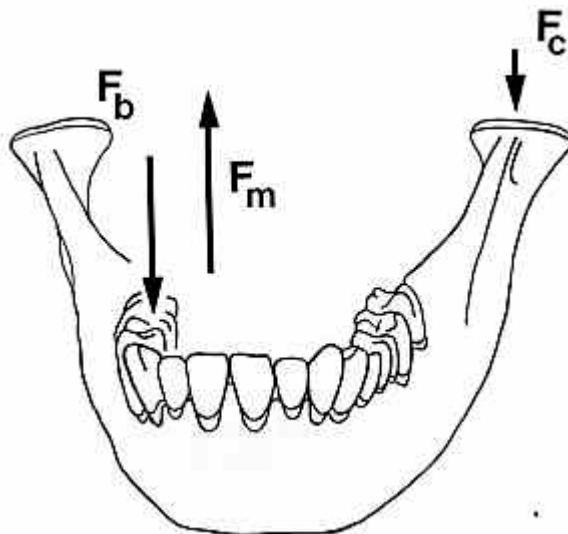


Figure 2.1.3.1(b) Forces acting along the mandible in the frontal projection. Only the vertical components of the muscle and reaction forces are included in this figure. The jaw elevators on the chewing side are generating slightly more force than the elevators on the nonchewing side. Under these conditions the resultant muscle force (F_m) is located along the ipsilateral side of the midline. F_b is the bite force and F_c is the condylar reaction force along the nonchewing side (Hylander, 1992).

2.1.3.2. Condylar force

The greatest biting force was generated in the first molar region. During biting along the second molar, the mandible functions as a class II lever in which the resistance was between the power source and the fulcrum (Mansour & Reynik, 1975) (Figure 2.1.3.2(a)). This would cause the fulcrum arm of the lever to move downward, causing a distracting force on the mandibular condyle to the extent that was limited by the temporomandibular ligament. This is because the fulcrum of the masticatory system is quite free to move. Unilateral biting against a resistant bolus between the third molar teeth widens the articular disc space ipsilaterally. The mandible functions with an axis ran obliquely between the occlusal bite point on the working side and the opposite nonworking condyle. If the resultant muscle force vectors were posterior to this axis, the working condyle remained loaded. But if the muscle force vectors lay anterior to it, the working condyle became unloaded (Bell, 1990). The position of the resultant muscle force vector is depending on the ratio of the ipsilateral to contralateral muscle force (Hylander, 1992).

On the other hand, forward to the second molar, the mandible functions as a class III lever. During biting, the fulcrum arm of the lever is directed upward into the joint (Figure 2.1.3.2(b)). The condylar force increased as the biting point move anteriorly (Figure 2.1.3.2(c)). The TMJ loading was thus increased in individual with deficient posterior tooth support (Bell, 1990).

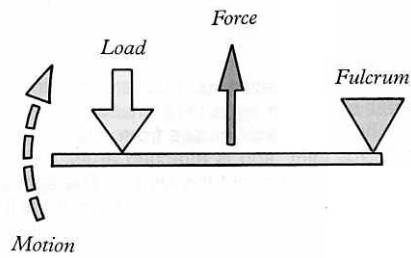


Figure 2.1.3.2(a) Class II lever. (Adapted from Soames, 1995)

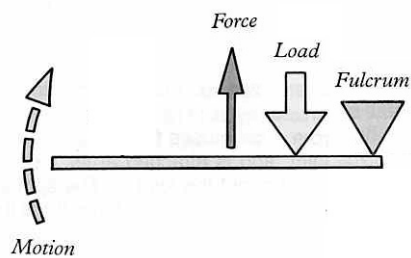


Figure 2.1.3.2(b) Class III lever. (Adapted from Soames, 1995)

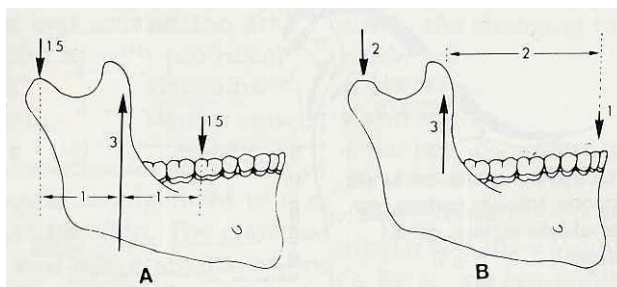


Figure 2.1.3.2(c) Biomechanics of the mandible in lateral projection during biting along the first molar and central incisor. A: The perpendicular distance between the resultant muscle force and the bite force is 1 unit. The perpendicular distance between the resultant muscle force and the condylar reaction force is also 1 unit. During biting along the first molar, 3 units of muscle force yield 1.5 units of both bite force and condylar force. B: The perpendicular distance between the resultant muscle force and the bite force is 2 units. During biting along the central incisor, 3 units of muscle force yield 1 unit of bite force but 2 units of condylar force. (Modified from Bell, 1990)

2.2. Terminology of temporomandibular disorders

2.2.1. Terminology

Temporomandibular disorders (TMD) are a type of musculoskeletal system disorders involving TMJ and the masticatory musculature. Muscle disorders result especially when functional activity involves such factors as proprioceptive and sensory feedback

mechanisms, while joint disorders relate primarily to biomechanical factors such as loading and movement (Bell, 1990). Bell (1970) actually advocated the term TMD since 1970, which has gained popularity. This term does not suggest merely problems that are isolated to the joints but includes all disturbances associated with the function of the masticatory system. The American Dental Association (Griffiths, 1983) adopted the term TMD for better communication and coordination by avoiding wide variety of terminology. TMD is a general term used to include all functional disturbances of the masticatory system.

2.2.2. History of terminology

It was used to think that dysfunctions of the masticatory system comprise a “syndrome”. The dysfunction of the masticatory system was initially referred as Costen’s syndrome or the temporomandibular syndrome (Costen, 1934). Schwartz (1955) later introduced the temporomandibular joint pain-dysfunction syndrome. Then the term, myofascial pain-dysfunction syndrome was introduced by Laskin (1969).

According to Stedman’s Medical Dictionary (Pugh, 2000), myofascial pain-dysfunction syndrome is the dysfunction of the masticatory apparatus related to spasm of the muscles of mastication precipitated by occlusal disharmony or alteration in vertical dimension of the jaws, and exacerbated by emotional stress; characterized by pain in the preauricular region, muscle tenderness, popping noise in the TMJ, and limitation of jaw motion; synonyms to temporomandibular joint pain-dysfunction syndrome.

The syndrome concept has contributed to the one disease-one treatment approach. The clinical signs and symptoms displayed by masticatory disorders are much too varied to be classified as a “syndrome”. According to Dorland’s Illustrated Medical Dictionary

(Anderson, 2003), a “syndrome” is a set of symptoms which occur together, a symptom complex; while a “disorder” is a derangement or abnormality of function, a morbid physical or mental state. The term “disorder” applies not to symptoms but to conditions.

Bell (1990) advocated that the general term TMD should designate the conditions that comprise complaints of the masticatory system involving the craniomandibular articulation and its musculature, which stresses the multifactorial nature of such conditions.

2.3. Epidemiology

TMD represents a significant problem in the general population. The prevalence of signs and symptoms associated with TMD can best be appreciated by examining epidemiologic studies. Dorland's Medical Dictionary describes epidemiology as "the science concerned with the study of the factors determining and influencing the frequency and distribution of disease, injury, and other health-related events and their causes in a defined human population for purpose of establishing programs to prevent and control their development and spread" (Anderson, 2003).

Epidemiologic studies can be descriptive or analytic. Descriptive studies usually deal with prevalence which involves retrospective evaluation of the number of cases with any disease or associated factor. Analytic studies usually deal with incidence which involves prospective longitudinal evaluation of the number of cases acquiring a disease or an associated factor over a specified time (Okeson, 1996). Few studies have been reported on the incidence of TMD, whereas numerous epidemiologic studies have examined the prevalence of TMD in given populations. However, the majority of studied populations are cross sectional samples, and this is not necessarily representative of general population.

Okeson (1996, 1998), Solberg (1987) and Burakoff (1991) have widely reviewed studies regarding epidemiology of TMD and their findings are generally concurrent.

2.3.1. Prevalence

Okeson (1998) had summarized several epidemiologic studies that examined the prevalence of TMD in given populations (Table 2.3.1). In each study patients were questioned regarding symptoms and then examined for common clinical signs associated with TMD. The prevalence number represents the percentage of patients who had at least one clinical symptom or one clinical sign that related to TMD. These studies reported that an average of 45% of these populations shows at least one symptom associated with TMD, whereas 58% showed at least one clinical sign. It is probably safe to assume that a similar percentage also exists in the general population since these studies ranged through many age and sex distributions. According to these studies, the percentage of people in the general population with at least one detectable sign of TMD is between 40% and 60%. A study by Schiffman et al. (1990) found prevalence of 33% with articular disorders and 41% with masticatory muscle disorders in general population.

In a study by Solberg et al. (1979), 739 university students aged 18 to 25 were recruited. A questionnaire was given to be completed and a short clinical examination was performed to identify any sign related to TMD. A sign was considered to be any clinical finding associated with a TMD. A symptom was any reporting associated with a TMD which the patient was aware of. The clinical examination revealed that 76% of the students had one or more signs associated with TMD. However, the questionnaire revealed that only 26% of the students reported having a symptom that was related to TMD. It was also reported that only 10% of the total group had symptoms that were severe enough to cause the patient to seek treatment. Only 5% of the total group would attend dental offices as typical TMD patients with severe problems. In other words, this study may reflect that 50% of the general population had a sign that were not reported

as symptom or regarded as subclinical. About one quarter of the general population will report some awareness of TMD symptom; whereas less than 10% of the populations feel that their problem is severe enough to seek treatment.

Table 2.3.1 Signs and symptoms of TMD in investigated populations.

Author	No. of individuals	No. of women/men	Age (year)	Population	At least one symptom	At least one clinical sign
Nilner and Lassing (1981)	440	218/222	7-14	Swedish children	36	72
Egermark-Eriksson et al. (1981)	136	74/62	7	Swedish children	39	33
	131	61/70	11	Swedish children	67	46
	135	59/76	15	Swedish children	74	61
Gazit et al. (1984)	369	181/188	10-18	Israeli children	56	44
Nilner (1981)	309	162/147	15-18	Swedish children	41	77
Swanljung and Rantanen (1979)	583	341/256	18-64	Finnish workers	58	86
Solberg et al. (1979)	739	370/369	19-25	American University students	26	76
Pullinger et al. (1988)	222	102/120	19-40	Dental hygiene and dental students	39	48
Rieder et al. (1983)	1040	653/387	13-86	American private practice	33	50
Ingervall et al. (1980)	389	0/389	21-54	Swedish reservists	15	60
Osterberg and Carlsson (1979)	384	198/186	70	Retired Swedish workers	59	37

The references were referred to those as reviewed by Okeson (1998).

2.3.2. Age and Sex Distribution

Epidemiology studies revealed that the most TMD symptoms are reported among population aged 20 to 40 years (Von Korff et al., 1988; Dworkin et al., 1990). Patients who are 60 years of age or older rarely complain of TMD symptoms. Well-defined studies by Helkimo (1974a, 1974b, 1974c) revealed that the recorded symptoms of dysfunction were equally common among men and women and varied only slightly with age, with but few exceptions. Helkimo had used a population of 321 Finnish-Lapps aged 15-65 years as the basis sample for his studies. A study on sample group aged 6-18 years done by Mintz (1993), reported the prevalence of signs and symptoms of TMD is the same in children and adolescents as in adults.

However, the percentage of female constitute of those seeking treatment for TMD can be as high as 85%, reported by Howard (1991) (Figure 2.3.2). The predominance of female seeking treatment much more often may be due to their greater health awareness (Randolph et al., 1990). In spite of the high ratio of women to men in TMD patient population, initial investigations did not document more symptoms in women (Agerberg & Carlsson, 1972; Helkimo, 1974c). In the other words, there is a great difference in the prevalence of female TMD patient among TMD patient population and among nonpatient population.

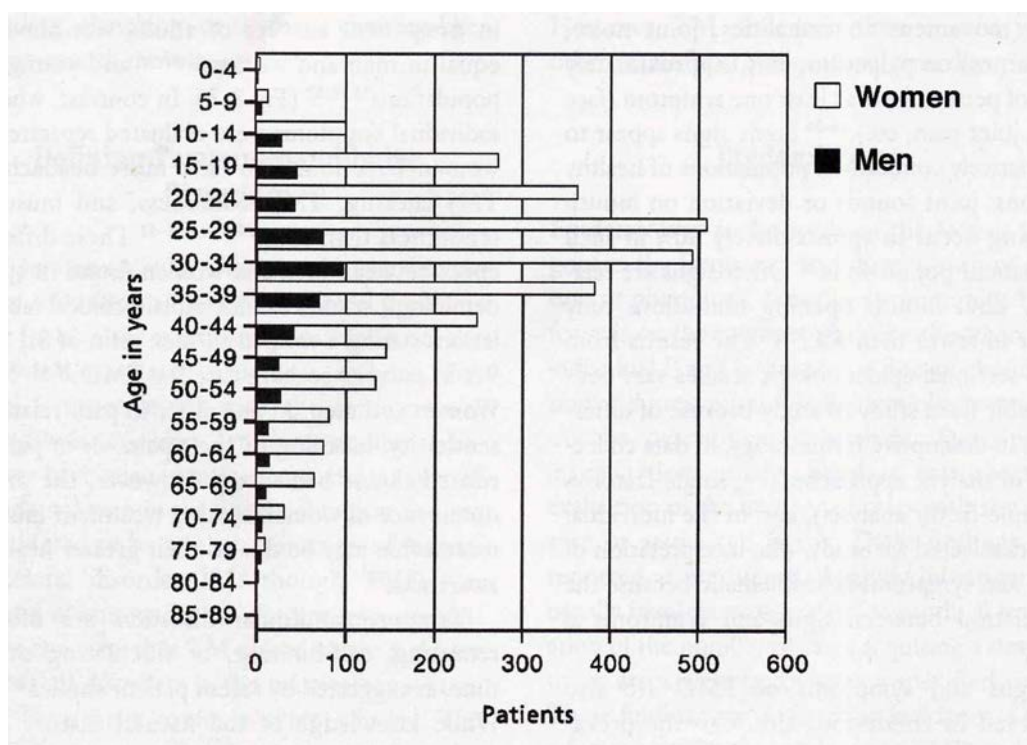


Figure 2.3.2 Age and sex distribution of 3,428 TMD patients presenting with TMD at a Seattle-based health maintenance organization of 360,000 enrollees. The mean ages of the women and men were 34.2 years and 33.8 years, respectively, and, of those seeking treatment, 85.4% were women (Howard, 1991; Okeson, 1996).

2.3.3. Social and psychological factors

In a large study of five separate groups, the frequency of temporomandibular dysfunction was 1.4 times higher in groups with psychoemotional tension (Wigdorowicz-Makowerowa, 1979). In a case control study using psychometric

analysis, all of the members of the control group were relatively free of anxiety, whereas half of the symptom group showed clinical signs of anxiety (Solberg, 1972). The lack of demonstrated differences in occlusal abnormalities or psychological characteristics between symptomatic men and women makes the predominance of female TM disorders patients difficult to explain on the basis of these two popular etiological theories (Solberg, 1987).

2.3.4. Relationship between the signs and symptoms of TMD and occlusion

Okeson (1998) had summarized several epidemiologic studies to evaluate the relationship between the signs and symptoms of TMD and occlusion (Table 2.3.4). Some studies revealed a positive relationship, whereas others did not. Moreover in the studies that found a significant positive relationship, no consistent type of occlusal condition emerged. Thus these studies exemplify the reason that confusion and controversy concerning the relationship between occlusion and TMD continues even today.

Table 2.3.4 Studies that investigated the relationship between the signs and symptoms of TMD and occlusion (Okeson, 1998).

Author	No. of individuals	No. of women/men	Age (year)	Population	Relationship occlusion and TMD	Type of occlusal condition related
Williamson and Simmons (1979)	53	27/26	9-30	Orthodontic patients	No	None
DeBoever and Adriaens (1983)	135	102/33	12-68	Patients with TMJ pain/dysfunction	No	None
Egermark-Eriksson et al. (1983)	402	194/208	7-15	Random sample of children	Yes	Occlusal interferences, anterior open-bites, anterior cross-bite, Class II and III
Gazit et al. (1984)	369	181/188	10-18	Israeli school children	Yes	Class II and III, cross-bite, open-bite, crowding
Brandt (1985)	1342	669/673	6-17	Canadian school children	Yes	Overbite, overjet, open-bite

Table 2.3.4 , continued

Author	No. of individuals	No. of women/men	Age (year)	Population	Relationship occlusion and TMD	Type of occlusal condition related
Nesbitt et al. (1985)	81	43/38	22-43	Growth study patients	Yes	Class II, open-bite, deep-bite
Thilander (1985)	661	272/389	20-54	Random sample in Sweden	Yes	Class III, cross-bite
Bernal and Tsamtsouris (1986)	149	70/79	3-5	American preschool children	Yes	Anterior cross-bite
Nilner (1986)	749	380/369	7-18	Swedish children adolescents	Yes	Centric slides, non-working contacts
Stringert and Worms (1986)	62	57/5	16-55	Subjects with structural and functional changes of TMJ vs. control	No	None
Riolo et al. (1987)	1342	668/667	6-17	Random sample of children	Yes	Class II
Gunn et al. (1988)	151	84/67	6-18	Migrant children	No	None
Dworkin et al. (1990)	592	419/173	18-75	HMO members	No	None
Pullinger et al. (1988)	222	102/120	19-41	Dental and dental hygiene students	Yes	Class II division 2, lack of RCP-ICP slide, asymmetric slide
Seligman and Pullinger (1989)	418	255/159	18-72	Patients and nonpatient control subjects	Yes	Class II division 1, asymmetric slide, RCP-ICP slide >1 mm, anterior open-bite
Pullinger and Seligman (1991)	319	216/103	18-72	Patients and asymptomatic control subjects	Yes	Increased overjet and anterior open-bite with osteoarthritis
Wanman and Agerberg (1991)	264	Not given	19	Swedish young adults	Yes	Reduced number of occlusal contact in ICP, long slide length
Cacchiotti et al. (1991)	81	46/35	19-40	Patients and nonpatient control subjects	No	None
Shiau and Chang (1992)	2033	872/1161	17-32	Taiwanese university students	Yes	Balancing supracontacts, molar contact in guidance
Pullinger and Seligman (1993)	418	287/131	18-72	Patients and asymptomatic control subjects	No	None (attrition)

The references were referred to those as reviewed by Okeson (1998). ICP, intercuspal position; RCP, retruded contact position.

2.4. Classification of TMD

2.4.1. History of classifications of TMD

For years, there exists a lot of confusion in the classification of TMD. The advancement in understanding TMD required a well accepted taxonomy. The first serious attempt to classify TMD in the dental literature was done by Bell (1970). However, the classification entailed implications of etiology, making it not very useful as a diagnostic tool.

In 1982, Bell (1990) had presented a classification that logically categorizes these disorders, on the President's Conference on the Examination, Diagnosis and Management of Temporomandibular Disorders sponsored by the American Dental Association. It was then adopted by American Dental Association with few changes (Griffiths, 1983). However it still continued to reflect etiologic considerations. Again Bell (1990) emphasized that a truly useful classification of TMD should be based on clinical symptoms, so that it can serve as a sort of diagnostic "road map" to help guide the examiner toward accurate identification of the patient's complaint. In 1989, Okeson (1998) had later further made some modification on it and the classification is summarized in Figure 2.4.1(a).

On the other hand, since 1980, the American Academy of Orofacial Pain (AAOP), formerly the American Academy of Craniomandibular Disorders (AACD) proposed an elaborate classification of masticatory disorders that has been well received. (McNeill, 1993). It is however complex and is not based on symptomatology.

In 1996, AAOP chaired by again Okeson (1996) has made changes to the classification, incorporating Bell's classification. This classification is summarized in Figure 2.4.1(b).

I. Masticatory muscle disorders	i. Osteoarthritis
1. Protective co-contraction	ii. Osteoarthritis
2. Local muscle soreness	iii. Polyarthritides
3. Myofascial pain	d. Inflammatory disorders of associated structures
4. Myospasm	i. Temporal tendinitis
5. Centrally mediated myalgia	ii. Stylomandibular ligament inflammation
II. Temporomandibular joint disorders	III. Chronic mandibular hypomobility
1. Derangements of the condyle-disc complex	1. Ankylosis
a. Disc displacements	a. Fibrous
b. Disc dislocation with reduction	b. Bony
c. Disc dislocation without reduction	2. Muscle contracture
2. Structural incompatibility of the articular surfaces	a. Myostatic
a. Deviation in form	b. Myofibrotic
i. Disc	3. Coronoid impendence
ii. Condyle	IV. Growth disorders
iii. Fossa	1. Congenital and developmental bone disorders
b. Adhesions	a. Agenesis
i. Disc to condyle	b. Hypoplasia
ii. Disc to fossa	c. Hyperplasia
c. Subluxation (hypermobility)	d. Neoplasia
d. Spontaneous dislocation	2. Congenital and developmental muscle disorders
3. Inflammatory disorders of the TMJ	a. Hypotrophy
a. Synovitis/ Capsulitis	b. Hypertrophy
b. Retrodiscitis	c. Neoplasia
c. Arthritides	

Figure 2.4.1(a) Classification system used for diagnosing TMD (Okeson, 1998).

11.7 Temporomandibular joint articular disorders	11.7.5 Osteoarthritis (noninflammatory disorders)
11.7.1 Congenital or developmental disorders	11.7.5.1 Osteoarthritis: primary
11.7.1.1 Aplasia	11.7.5.2 Osteoarthritis: secondary
11.7.1.2 Hypoplasia	11.7.6 Ankylosis
11.7.1.3 Hyperplasia	11.7.7 Fracture (condylar process)
11.7.1.4 Neoplasia	
11.7.2 Disc derangement disorders	11.8 Masticatory muscle disorders
11.7.2.1 Disc displacement with reduction	11.8.1 Myofascial pain
11.7.2.2 Disc displacement without reduction	11.8.2 Myositis
11.7.3 Temporomandibular joint disorders	11.8.3 Myospasm
11.7.4 Inflammatory disorders	11.8.4 Local myalgia-unclassified
11.7.4.1 Capsulitis/ Synovitis	11.8.5 Myofibrotic contracture
11.7.4.2 Polyarthritides	11.8.6 Neoplasia

Figure 2.4.1(b) Recommended diagnostic classification for TMD by AAOP (Okeson, 1996). The numbering order was established by the AAOP in cooperation with the International Headache Society.

2.4.2. Masticatory muscle disorders

Masticatory muscle disorders is a collective term that broadly includes all functional disturbances of the masticatory muscle. Functional disturbances of masticatory muscles are probably the most common TMD complaint among patients who seek treatment in the dental practice.

Masticatory muscle disorders were described by Laskin (1969) as myofascial pain dysfunction syndrome. Myofascial pain dysfunction syndrome had been used in Dentistry as a general term to denote primarily masticatory muscle disorder. The term myofascial actually simply means pertaining to or involving the fascia surrounding and associated with muscle tissue (Anderson, 2003). The term myofascial was used by Travell and Rinzler (1952) to first described myofascial trigger point pain, which was adopted by McNeill (1993) and Okeson (1998). However, it is worthwhile to note that their description was different from the Laskin's description of myofascial pain dysfunction syndrome, using the term myofascial as well. Therefore, both sides' descriptions should not be confused.

Dworkin & LeResche (1992), who advocated Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD), had classified the muscle disorders in much simple form, after ruling out muscle spasm, myositis and contracture, into:

1. Myofascial pain
2. Myofascial pain with limited opening

According to their definition, myofascial pain simply means pain of muscle origin, including a complaint of pain as well as pain associated with localized areas of tenderness to palpation in muscle. Their definition is similar to Laskin's description of myofascial pain dysfunction syndrome.

2.4.3. Temporomandibular joint disorders

Temporomandibular joint disorders is a collective term that broadly includes all functional disturbances of the TMJ. Functional disorders of the TMJ are probably the most common findings in patient examined for masticatory dysfunction. RDC/TMD (Dworkin & LeResche, 1992), which presented a classification that based mainly on the classification advocated by Truelove et al. (1992), had divided disc displacements into:

1. disc displacement with reduction (which is similar to disc displacement (Figure 2.4.3(a)) and disc dislocation with reduction (Figure 2.4.3(b)) as classified by Okeson, (1998)).
2. disc displacement without reduction, with limited opening (which is similar to disc dislocation without reduction (Figure 2.4.3(c)) as classified by Okeson, (1998); and also similar to acute disc displacement without reduction as classified by McNeill (1993)).
3. disc displacement without reduction, without limited opening. (which is similar to chronic disc displacement without reduction as classified by McNeill (1993)).

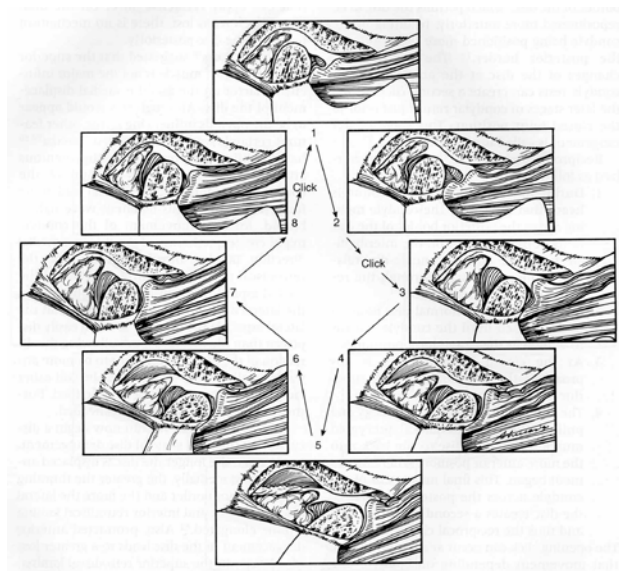


Figure 2.4.3(a) Functional movement at the TMJ with disc displacement. Between position 2 and 3, a click is felt as the condyle moves across the posterior border of the disc. Normal condyle-disc function occurs during the remaining opening and closing movement until the closed joint position is approached. Then a second click is heard as the condyle once again moves from the intermediate zone to the posterior border of the disc, between position 8 and 1 (Okeson, 1998).

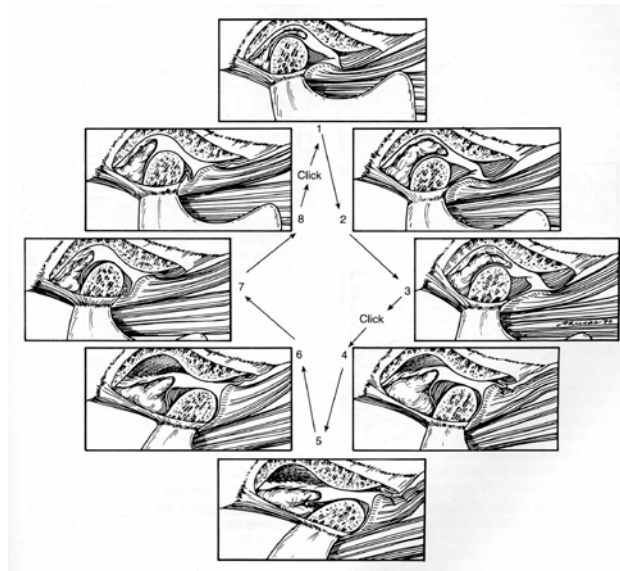


Figure 2.4.3(b) Functional movement at the TMJ with disc dislocation with reduction. Note that during opening, the condyle passes over the posterior border of the disc onto the intermediate area of the disc, thus reducing the dislocated disc (Okeson, 1998).

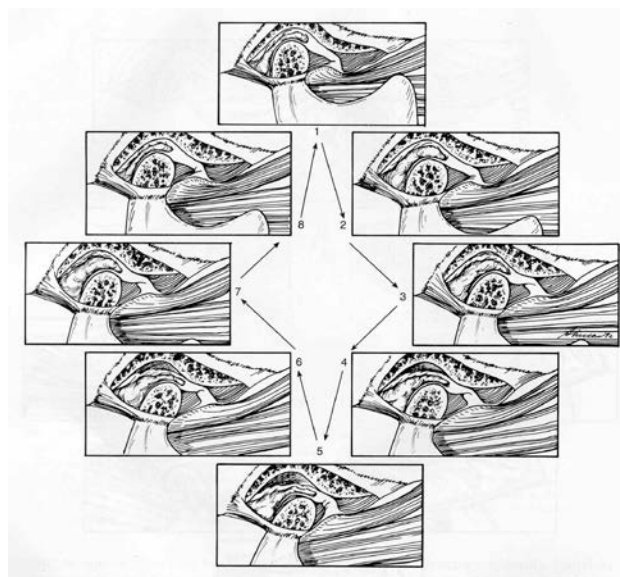


Figure 2.4.3(c) Functional movement at the TMJ with disc dislocation without reduction. Note that the condyle never assumes a normal relationship on the disc but instead causes the disc to move forward ahead of it. This condition limits the distance it can translate forward (Okeson, 1998).

2.5. Pathogenesis of TMD

2.5.1. Etiology of TMD

To date, a variety of causes has been suggested for TMD. The contributing factors of TMD are divided into predisposing factors, precipitating factors and the perpetuating factors. Generally, individual factors may serve any or all of these roles. A comprehensive examination and diagnostic approach is needed to identify all the possible contributing factors in a prioritized list.

The contributing factors can drive the balance between components of masticatory system toward physiologic health and function, or towards pathology and dysfunction (Paker, 1990). Bone and TMJ soft tissue remodeling, and muscle tone regulation are adaptive physiologic responses to contributing factors. Loss of structural integrity, altered function, or biomechanical strains and stresses can reduce the adaptive capacity of the masticatory system. Okeson (1996) had summarized the contributing factors according to trauma, anatomy, pathophysiology and psychosocial issues.

2.5.1.1. Trauma

Trauma is described as any force applied to the masticatory structures that exceeds that of normal functional loading. They are generally divided into direct trauma, indirect trauma and microtrauma (Okeson, 1996).

2.5.1.1.1. Direct trauma

Direct trauma usually produces injury to the temporomandibular structures via direct impact caused by sudden and usually isolated blow. Direct trauma generally causes more localized symptoms within 24 to 72 hours after the event. Stretching, twisting, or compressing forces during eating, yawning, yelling, or iatrogenic trauma from oral

intubation or dental procedures, may become a direct trauma to temporomandibular structures (Okeson, 1996).

2.5.1.1.2. Indirect trauma

Indirect trauma is associated with a sudden force but without direct contact to the affected structures. This is common in acceleration-deceleration accident (Okeson, 1996).

2.5.1.1.3. Microtrauma

Microtrauma to the temporomandibular structures produces injury as a result of prolonged, repeated force over time. Harmful loading of masticatory system from adverse usage and parafunctional habit can result in microtrauma (Okeson, 1996). Microtrauma is associated with functional overloading. It has been suggested that some postural habits may create muscle and joint strain and lead to musculoskeletal pain, including headache (Travell & Simon, 1983).

2.5.1.1.3.1. Functional overloading

Functional loading comprised muscle wearing and joint loading. The joint starts to be loaded only when the teeth are biting into something or when the teeth are in contact. Similarly, the muscles start to contract forcefully only when resistance is met. When contraction of muscle is prolonged, the muscle will then start to become weary as most of the motor units had been recruited. In the resting state of the muscle, even with the increased postural muscle activity, the muscle is less likely to become weary as motor units are recruited alternately while only few motor units are recruited at each time. Functional overloading was caused by parafunctional habits and adverse usage of masticatory system.

2.5.1.1.3.1.1. Parafunctional habits

Parafunctional habits refer to those activities that are not considered as normal functional activities. Parafunctional activities during the day are often performed without awareness of the individual. Parafunctional habits include clenching, grinding, and many other oral habits such as tongue thrusting, cheek and lip biting, finger sucking, and object biting such as biting on fingernails, pen and tooth pick. Parafunctional habits also includes unusual postural habits such as resting the jaw on the hand (Goldman, 1991), and many occupation-related activities such as biting on pencils, pin, or nails to hold them; and holding telephone under the chin or known as phone bracing.

Generally, activities of muscle could be divided also into postural activity and functional activity. Postural activity usually referred to the resting stage of the masticatory muscle whereby there was no occlusal contact of the teeth. At time when there was obvious occlusal contacting of the teeth, even there was no conscious voluntary action of masticatory muscle; it was regarded as functional activity such as subconscious clenching rather than postural activity.

It is important to stress that clenching and grinding can occur either during sleep or during the daily life. Clenching especially could occur commonly during daily activities. Daily activities that can be associated with clenching include driving, reading, writing, typing, washing and lifting heavy object. Some individuals may place their teeth together and apply force, which is irrelevant to the task at hand (Rugh & Robbins, 1982). This is especially true for those tasks that involve force application. Glaros et al., (1998) concluded in their study that chronic, low-level parafunctional clenching may be a factor in the cause of TMD pain.

The parafunctional activity is associated with CNS. Fear, anxiety, frustration or anger can increase the muscle tonicity and also create irrelevant muscle activity that may be responsible for nervous habits such as fingernail or pencil biting, clenching or grinding. The intensity and frequency of parafunctional activity may be aggravated by stress and anxiety, sleep disorders, and medications such as neuroleptics and alcohol (Okeson, 1998).

Most parafunctional activities occur at a subconscious level. The identification of parafunctional habit is usually through means of self-reporting, questionnaires, reporting by bedroom partner or tooth wear assessment. However, identification of habits such as clenching can be easily missed by patients themselves. Patients may need gradual establishment of self awareness. This was in line with the study by Glaros (1996), which suggested TMD patients with myofascial pain would more likely to engage in high levels of parafunctional oral activity without awareness compared to healthy subjects. Direct measurements of parafunctional activity through portable electromyography, sleep laboratory and direct observation are preferred (Okeson, 1996).

In general population, the prevalence of bruxism (clenching and grinding) reported by Melis and Abou-Atme (2003) (n=1014) was 27.2%; Kerdpon and Sirirungrojying (1999) (n=609), 27.3%; Michalowicz et al. (2000) (n=494), 14.6%. Besides, Kononen et al. (1987) (n=166) reported that the prevalence of grinding was 18%, clenching 6%, cheek or lip biting 23%, fingernail biting 49%, biting on foreign objects 35%. Meanwhile, Miyake et al. (2004) (n=3557) reported that the prevalence of grinding was 7.6%, clenching 5.5%, tongue, cheek or lip biting 11.5%, fingernail biting 6.8%, biting on foreign objects 5.5%, resting jaw on hand 44.8%, gum chewing 22.2%. These parafunctional habits were suggestive of the possible etiologic cause for TMD.

2.5.1.1.3.1.2. Adverse usage

Adverse usage of masticatory system includes heavy mastication, gum chewing, wide yawning and singing, poor sleeping posture especially sleeping on the stomach (Elfving et al., 2002), playing some musical instruments, and biting on the mouthpiece during diving. They usually referred to those activities that can be considered as normal functional activity but exaggerated.

Some adverse loadings of masticatory system are very closely related to the task being accomplished such as musician playing certain musical instruments, which in this study playing tuba (Howard, 1991). Yawning, yelling and prolonged mouth-opening had been reported to trigger or aggravate TMD (Harkins & Marteney, 1985). These activities exert its effect mainly onto the joint structure.

Anderson (1956) had demonstrated that different food consistencies had great difference in bite force requirement. He reported that chewing carrots produced approximately 30 pounds of force on the teeth whereas chewing meat produced only 16 pounds. There are possible associations between gum chewing and fatigue and pains in the jaw muscles. In the study by Christensen et al. (1996), during 10 minutes of one-sided gum chewing at a rate of 1.2 Hz, the majority of subjects (75%) experienced weak jaw muscle fatigue. They suggested that prolonged unilateral gum chewing and previous pain-releasing maximum voluntary clenching caused some sensitization of muscle nociceptors which, in turn, aggravated subsequent isometric jaw muscle pains elicited through maximum voluntary clenching.

Adverse loading of the masticatory system can occur in poor sleeping posture through external exertion of force. Hibi and Ueda (2005) showed that the habitual body posture

during sleep was a possible contributing factor to the anterior disc displacement in the TMJ. It was suggested that habitual body posture allows the ipsilateral condyle to displace posteriorly and this posterior position causes relative anterior disc displacement.

2.5.1.1.3.1.3. Magnitude and duration of force

In evaluating the loading, two factors must be considered and this includes the magnitude and the duration of the force applied to the masticatory system. A reasonable way to study the magnitude of force during loading is by evaluating the amount of force placed on the teeth. On the other hand, to study the loading onto the masticatory system, it should be appreciated that forces could be exerted internally by the masticatory muscle and also externally by any kind of external force.

Gibbs et al. (1981) had estimated that during each chewing stroke, an average of 58.7 pounds of force is applied to the teeth, while 66.5 pounds of force is applied to the teeth during each swallowing. Rugh and Solberg (1975) reported that the muscle activity during bruxing is greater than those used merely in swallowing and are sustained for a second or more. Jaw muscles are supposed to be active only during chewing, talking or swallow. It was estimated that the total duration of teeth touch in a 24 hour period during these activities was about 15 to 30 minutes per day. Therefore the jaw muscles should be at rest for the remaining 23½ hours. However, most of the parafunctional activities easily last for more than half an hour. Okeson (1998) compared tooth contact during functional and parafunctional activity. Since there are normally no tooth contact during speech, only chewing and swallowing activities were evaluated. It has been estimated that each chewing stroke applied teeth contact for 115 milliseconds while 1800 chews occur during an average day. Therefore the total duration of tooth contact

for chewing is 3 minutes and 27 seconds. It was estimated that tooth contact during each swallowing is 522 milliseconds while an individual swallow some 146 times a day during eating. Therefore the total duration of tooth contact for swallowing is 1 minute and 16 seconds. On the contrary, Trenouth (1979) reported that the bruxism group spent 38.7 minutes with their teeth together during the 8-hour sleeping period.

Most functional activity consists of well-controlled and rhythmic contraction and relaxation of the muscles during jaw function. This isotonic activity permits adequate blood flow to supply oxygen to the tissues and to eliminate metabolic by-products accumulated at the cellular level. In contrast, the parafunctional activity often results in sustained muscle contraction over long periods. This type of isometric activity inhibits normal blood flow within the muscle tissue. Consequently, there is an increase in by-products within the muscle tissues, leading to fatigue, pain or spasms of the muscles (Okeson, 1998). Christensen and Mohamed, (1984) has demonstrated that pain was produced in jaw muscles of subjects after 20 to 60 seconds of voluntary clenching.

2.5.1.2. Anatomical factors

Anatomical factors that associated with TMD usually refer to structural relationship of the joint and occlusion. They can be of genetic, developmental or iatrogenic origin (Okeson, 1996).

2.5.1.2.1. Structural relationship of the joint

The steepness of the articular eminence is a possible etiologic factor in TMD (Nickel & McLachlan, 1994). There is less surface congruity during protrusive biting when the eminence steepness is less, this could cause a greater concentration of occlusal forces on a smaller area of the eminence, enhancing osteoarthritic changes (Okeson, 1996).

2.5.1.2.2. Occlusal relationship

Occlusion has long been regarded as a primary etiologic factor for TMD (Okeson, 1996). However recent reviews do not strongly support the role of occlusion in the etiology of TMD. Pullinger et al. (1993), using multifactorial analysis to investigate interaction of 11 occlusal factors concluded that no single occlusal factor was able to differentiate patients from healthy subjects. However, there were four occlusal features that occurred mainly in patients with TMD, including presence of a skeletal anterior open bite, discrepancies between retruded contact position and intercuspal contact position that is greater than 2 mm, overjet of greater than 4 mm, and five or more missing posterior teeth that were not rehabilitated. A single isolated occlusal factor is rarely associated with TMD, but combination of factors is thought more likely to cause TMD (Okeson, 1996).

Other factors include non-working side interferences, Angle malocclusion, deep overbite, severe attrition, absence of anterior or canine guidance, and increased occlusal vertical dimension, anterior crossbite and bilateral posterior crossbite are suggested to be associated with TMD; however there is lack of evidence (Okeson, 1996). Anyway, unilateral posterior crossbite was found to be more common in TMD patient. Skeletal anterior open bite has been associated with condylar changes and osteoarthritic changes (Tegelberg & Kopp, 1987). Severe skeletal malformations can also cause inter-arch or intra-arch occlusal discrepancy. They can result in slanting occlusal plane, crossbite, scissors bite and other kinds of malocclusions (Okeson, 1996).

It has been suggested that some occlusal factors that are more prevalent in TMD patient are possibly the result of condylar positional changes following intracapsular alterations as responding to TMD, rather than the etiology of the disorders (Vanderas, 1994).

2.5.1.3. Pathophysiologic factors

2.5.1.3.1. Systemic factors

Systemic pathophysiologic conditions that may be related to TMD would include degenerative, endocrine, infectious, metabolic, neoplastic, neurologic, rheumatologic, and vascular disorders. They can act simultaneously at a central and local level. Generalized joint laxity has been found to be more prevalent in patients with internal derangement than with other TMD or with normal subjects (DeKanter et al., 1993). However, physiologic response of patient appears to be variable and depending on individual (Okeson, 1996).

2.5.1.3.2. Local factors

Local pathophysiologic factors that influence TMD include chewing efficiency, integrity of articular surfaces and the disc, synovial fluid viscosity, and intracapsular pressure (Okeson, 1996).

Chewing efficiency is enhanced when there are greater numbers of chewing units and when the numbers of missing posterior teeth is less than five. The minimum number of posterior chewing units that accounted for the threshold for impaired chewing is three (Leake et al., 1994). Chewing force is also affected by gender, age and pain levels of TMD (Okeson, 1996).

Articular surfaces should be maintained intact to allow for normal functional movement of the joint. The body's physiologic response to disease in the TMJ can be adaptive or pathologic. The thickness and composition of joint cartilage can change in a response to the shearing stresses during functional loading, or to smooth the bony irregularities

through remodelling process. Anyway, fibrous connective tissue covering the articular bone will still remains even if the cartilage is lost (Bibb et al., 1993).

The correct positioning of the disc is needed to maintain joint stability. Displacement of the disc would affect the functional joint movement. Disc displacement without reduction often progresses to osteoarthritic changes over time (Magnusson et al., 1994). Comparatively, Disc displacement with reduction typically shows only few histologic signs of early osteoarthritis. The reasons for progression from disc displacement to osteoarthritis includes failure of the reparative articular chondrocyte response due to metabolic dysfunction, and overloading that lead to articular cartilage biochemical failure (Okeson, 1996).

Disc displacement patterns are highly variable and usually involve all areas of the joint compartments. It involves a combination of restricted disc freedom in the upper joint compartment with an increase of condylar translatory movement in the lower compartment. This results in a lengthening of the anterior recess and a drag on the inferior surface of the disc. This happens may be following prolonged and sustained loading immediately prior to initial movement. Disc displacement without reduction shows an ongoing synovitis in contrast to disc displacement with reduction (Okeson, 1996).

Synovial fluid viscosity and inadequate lubrication may be responsible for clicking and derangement of the TMJ (Toller, 1961). The abnormal concentrations of plasma proteins and neurotransmitters, degradation of enzymes, metabolic by-products are probably related to the pain, inflammation and degeneration of the TMJ. The clenching forces can cause frictional sticking of the disc (Okeson, 1996).

Intracapsular pressure may also affect TMD (Nitzan, 1994). With joint movement, the alternating pressure acts as a pump for joint lubrication, nutrition, blood supply, drug delivery, waste removal, and even help to stimulate condylar growth. Therefore immobilization and prolonged clenching may precipitate TMD. Female hormones may have a role in TMJ disc disease through doubtful presence of estrogen or progesterone receptors within the articular disc (Abubaker et al., 1993). Females are found to have higher intra-articular pressure than men, which may responsible for more ischemia, disc friction or prolongation of chronic inflammatory synovitis (Nitzan, 1994). This may partly explain the higher prevalence of women with TMJ disorders (Okeson, 1996).

2.5.1.4. Psychosocial factors

Psychosocial factors include individual, interpersonal, and situational variables that may affect the patient's ability to function adaptively (DeLeeuw et al., 1994). Individual TMD patients may have personality characteristics or emotional conditions that cause difficulty in managing or coping with life situation. Some TMD patient may experience more anxiety than healthy people do. The pain symptoms may be only part of physical manifestations of emotional distress. Stressful life events may cause higher attention focused on the pain and lead to an increase in the pain level. Depression and anxiety associated with stressful life events can also change the patient's perception and tolerance towards the pain (Magni et al., 1994). TMD patient may have history of other stress-related disorders and they may tend to seek more care. However, some psychological impairment may just be merely due to the presence of the pain. The psychosocial characteristic of patients with chronic TMD is similar to that of patients with other chronic pain such as lower back pain and headache (Okeson, 1996).

TMD patients may also present with mental disorders. TMD patients with pre-existing mental disorders can have anxiety and depression that may initiate or perpetuate the TMD condition (Okeson, 1996).

One's perceptions and responses towards pain and disease can be affected by environmental factor. Some patients may experience reduced level of distress to the extent that psychogenic symptoms resolve pre-existing interpersonal conflict. Once a disorder is present, patient can also gain social benefits, which includes being exempt from ordinary daily responsibilities, being compensated monetarily, avoiding unpleasant tasks for being ill, and gaining attention from others (Okeson, 1996).

The use of pharmaceutical agents such as tranquilizers, narcotics, barbiturates and also alcohol can contribute to the chronicity of TMD. The dependency on this chemical agents and the depression induced, often made the disorders refractory to treatments (Okeson, 1996).

2.5.2. Development of TMD

TMD disorder is not a single problem but represents an umbrella term under which there are multiple disorders. A multitude of conditions can affect masticatory function to cause a variety of disorders according to the structures involved. Okeson (1998) had suggested the following formula to simplify how TMD symptoms arise:

Normal function + An event > Physiologic tolerance → TMD symptoms

Under normal conditions the masticatory system functions without symptom. Occasionally, function of the masticatory system may be interrupted by an event. If the event is significant, it can exceed the physiologic tolerance of the individual to cause a clinical symptom associated with TMD (Okeson, 1998).

2.5.2.1. Interrelationship between muscle disorders and disc derangement

A muscle disorder may induce disc interference by increasing the passive interarticular pressure, or disc interference may induce muscle symptoms by way of protective splinting (Bell, 1990).

In reality, one patient can suffer from more than one subcategory of TM disorders. In many patients one disorder contributes to another. Patient who has a masticatory muscle disorder can have increased tonicity of the elevator muscles, creating an increase in interarticular pressure of the joint to cause disc derangement disorder (Okeson, 1998).

Masticatory muscle disorder → disc derangement disorder

Meanwhile, a patient complaint of an early disc derangement disorder can provoke secondary protective co-contraction. Protracted protective co-contraction would in turn result in local muscle soreness. By this way, a disc derangement disorder has caused a masticatory muscle disorder (Okeson, 1998).

Disc derangement disorder → masticatory muscle disorder

On the other hand, Okeson (1998) also had described a masticatory muscle model that depicts the relationship between different muscle pain disorders (Figure 2.5.2.1(a)). According to Okeson (1998) also, disorders of the TMJ often follow a path of progressive events from the initial signs of dysfunction to osteoarthritis. They are summarized as in Figure 2.5.2.1(b).

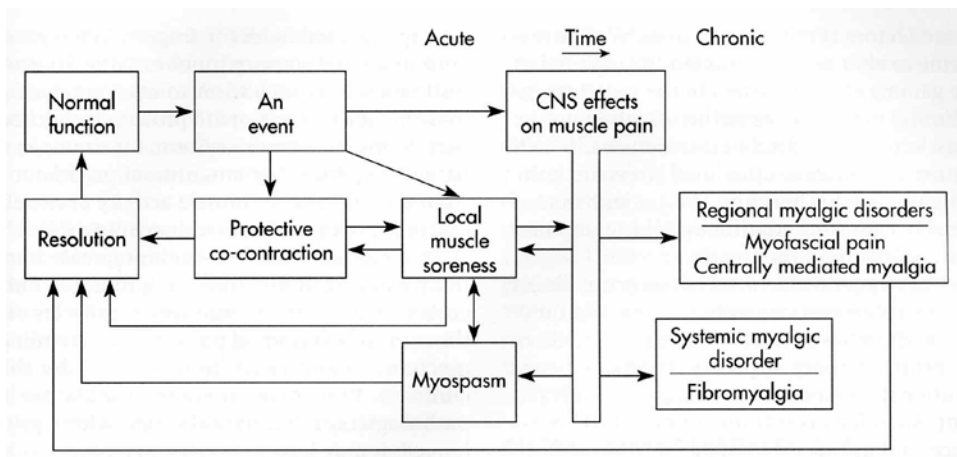


Figure 2.5.2.1(a) Masticatory muscle model (Okeson, 1998).

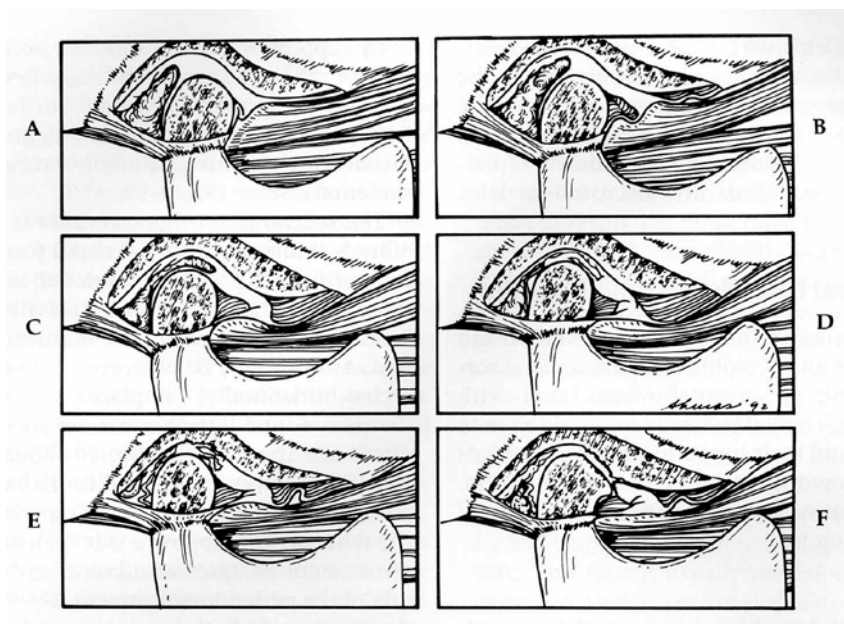


Figure 2.5.2.1(b) Various states of internal derangement of the temporomandibular joint. A, Normal joint. B, Functional displacement of the disc. C, Functional dislocation of the disc. D, Impingement of retrodiscal tissues. E, Retrodiscitis and tissue breakdown. F, Osteoarthritis (Okeson, 1998).

2.6. Examination and diagnosis

2.6.1. Examination

In examining the TMD patient, the articulatory system can be assessed in three components:

1. masticatory muscle
2. temporomandibular joint
3. occlusion

In addition, the records of complaint and history of patient are extremely important.

2.6.1.1. Complaint and history taking

The details of patient's complaints should be recorded. The duration, site and severity of symptoms, and the time when the pain is at its worst are taken. The record of history may include the date of first history of symptoms as well as the duration and frequency of occurrences. The query of parafunctional habits or adverse usage and general stress factors can also be included. Parafunctional habits includes clenching, grinding, cheek biting, object biting, poor sleeping posture and chin resting in hand; while adverse usage includes heavy mastication, gum chewing, wide yawning, and playing some musical instruments. General stress factors include stress from the studies, job, family and friend or major life event changes such as divorce and engagement in litigation.

2.6.1.2. Examination of the masticatory muscle

The most common sign and symptoms of patients with masticatory muscle disorders is muscle tenderness during palpation and pain during function. Digital palpation is a widely accepted method of assessing muscle tenderness.

The temporalis can be palpated by dividing it into anterior, middle and posterior portions. In addition, the tendon of temporalis can be palpated intraorally by running the finger up the anterior border of the ascending ramus. The masseter muscle can be palpated by dividing it into superior, middle and inferior thirds. They are easily palpated extraorally.

2.6.1.3. Examination of TMJ

2.6.1.3.1. Range of movement

The common clinical symptom associated with masticatory muscle disorders is dysfunction. It is usually seen as a decrease in the range of mandibular movement and can be objectively measured. Quantifying the mandibular movement is important as a record of the severity of symptoms and to show the degree of improvement. The reduction in movement can be due to muscular problem or a physical obstruction in the joint (Gray et al., 1995). Range of movement can be measured as:

1. Pain-free incisal opening

This is measured vertically from the upper incisal edge to lower incisal edge by asking the patient to open to the limit of their pain-free. For classification purpose, the overbite is measured and added to obtain actual inter incisal distance.

2. Maximum incisal opening

This is measured as the inter incisal distance with the patient making maximum opening. The overbite is added to this measurement too.

3. Lateral excursions

This is measured horizontally from the upper central incisors mid-line to the lower central incisors mid-line with the patient first moving the mandible to one side then the other, to their maximum extent. The patient tends to move away

from the affected side to a lesser distance. The midline difference at intercuspal position should be subtracted from the above measurement to get actual lateral excursion distance.

In addition, the pathway of opening should be observed from the front for any:

1. transient deviation

The opening pathway is straight in the beginning and then deviated to one side with maximum opening.

2. lasting deviation

The opening pathway is straight in the beginning and followed by deviation to one side in the middle of opening but then returns to a normal midline relationship.

2.6.1.3.2. TMJ tenderness

The TMJ can be palpated at the pre-auricular area by pressing gently over the lateral aspect of the joint. The TMJ can also be examined by intra-auricular palpation through placement of little finger in the external auditory meatus with gentle forward pressure applied. In this way both the lateral and posterior aspects of the joint can be palpated (Gray et al., 1995).

2.6.1.3.3. TMJ sound

Pathological displacement of the disc would result in production of sound during movement. Displacement may occur due to injury to the bilaminar zone, to the disc or its attachments, or to hypertonicity in the superior head of the lateral pterygoid (Gray et al., 1995).

During mouth opening, the condyle translates forward and pushes the body of the disc forwards, which stretch the bilaminar zone. Eventually, the tension in the bilaminar zone exceeds the pressure exerted by the condyle on the disc, which causes the disc to move backward suddenly to produce an opening click sound. Meanwhile, a closing click may also occur due to the sudden forward movement of the posteriorly compressed disc during closing. This may be due to passive release of the compressed disc or/and forward pull exerted on the disc by the hypertonicity of the superior head of the lateral pterygoid. Clicking during both opening and closing phases of mandibular movement is referred as reciprocal clicking (Gray et al., 1995).

Clicks are usually single. Multiple clicks imply a very unstable disc or disc with perforations. Click is not normally painful. Pain may be associated with acute disc displacement or with secondary muscle spasm due to prolonged internal derangement. Sometimes, a prolonged continuous noise may be produced in the joint and is referred to as crepitus. It occurs most commonly in patient with degenerative joint disease such as osteoarthritis. The condyle and fossa surfaces may also display erosions and the disc may be roughened with decreased synovial lubrication. The joint may not be painful at rest but may become tender to pressure and painful on function (Gray et al., 1995).

2.6.1.3.4. Radiographs

Radiographs are indicated only if clinical examination suggests existence of bone pathology such as erosion of condyle or fossa surfaces or presence of crepitus joint sound. All the plain TMJ radiographs have their own limitation and the information provided cannot be regarded as being conclusive (Gray et al., 1995). Advance imaging technique such as magnetic resonance imaging and computed tomographic scan can give better image.

Orthopantomograph gives the view of the condylar outline only. The standard transcranial oblique lateral radiograph at open and closed views of the TMJ usually only provides information on the gross bony morphology and the range of joint movement. The image actually shows only the lateral pole of the condyle and the lateral rim of the fossa, but not the load-bearing area due to the angulations of x-ray centre beam (Gray et al., 1995).

It is estimated that between 40 to 60% decalcification of the head of the condyle must have occurred before any erosions become radiographically visible. Erosion must therefore be fairly large before they are readily seen (Gray et al., 1995).

2.6.1.4. Examination of occlusion

2.6.1.4.1. Incisor classification

Anterior tooth relationships can be classified as

1. Class I

The incisal edges of the mandibular incisors contacting the middle third of the palatal surfaces of the maxillary incisor in intercuspal position.

2. Class II

The incisal edges of the mandibular incisors contacting more palatally than the middle third of the palatal surfaces of the maxillary incisor in intercuspal position. If the maxillary incisors are proclined, it is considered as division 1. If the maxillary incisors are retroclined, it is termed as division 2.

3. Class III

The incisal edges of the mandibular incisors contacting more labially than the middle third of the palatal surfaces of the maxillary incisor in intercuspal position.

2.6.1.4.2. Anterior guidance

The anterior guidance is provided by those teeth that touch during lateral excursion, while disocclude the rest of the teeth. As opposed to anterior guidance, posterior guidance is provided by the TMJ. Anterior guidance can be provided by the canines alone, which is termed canine guidance, or by several teeth, which is termed group function. Group function can be provided either by the anterior teeth or the posterior teeth. However the further back the anterior guidance is, the more likely it is to interfere with condylar movements (Gray et al., 1995).

2.6.1.4.3. Non-working side interferences

The non-working side is the side from which the mandible is moving away. The contact on the non-working side that interferes with anterior guidance is called non-working side interference. The interference at the non-working side is more significant because the condyle on the non-working side is moving more than that on the working side during lateral excursion (Gray et al., 1995).

2.6.1.4.4. Signs of bruxism

The signs of bruxism include attrition, tooth sensitivity, tooth or restoration fracture, scalloping of tongue and ridging of buccal mucosa. Tooth attrition as heavy wear facets and often fracture of the tooth or restoration may suggest the present of bruxism. The patient may also complaint of tooth sensitivity especially at the anterior teeth and more on waking. Scalloping of the lateral border of the tongue and the ridging of the buccal cheek mucosa along the occlusal line are most reliable sign of active bruxism (Gray et al., 1995).

2.6.2. Diagnosis

In management of TMD, a proper diagnosis is extremely important in order for an appropriate treatment to be given. Generally, a diagnosis is made according to a list of criteria, to separate the disorders into common groups of symptoms and etiologies. The advancement in understanding TMD depends on an accepted taxonomy and corresponding diagnostic criteria. These would allow for comparisons and communication between researchers or clinicians (Dworkin & LeResche, 1992).

However, the diagnostic criteria used for research purposes are usually different from that for clinical purposes due to the different requirement in diagnostic sensitivity and specificity. The researcher needs to recruit subject with high possibility of having a certain disease, whereas the clinician needs to identify patients presenting with the entire spectrum of a certain disease. In other words, the diagnostic criteria used in research condition require higher specificity but lower sensitivity, whereas clinical condition requires higher sensitivity but lower specificity (Okeson, 1996). The most popular research diagnostic criteria is the one advocated by Dworkin and LeResche (1992), which has been widely used in research condition.

2.6.2.1. Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD)

According to Dworkin and LeResche, (1992) a critical obstacle to our further understanding of temporomandibular disorders is the lack of standardized diagnostic criteria for defining clinical subtypes of TMD. Therefore, the RDC/TMD project was undertaken to redress the lack. The signs and symptoms are organized into reliable diagnostic classification systems that are described using quantitative criteria.

This dual-axial diagnostic system was developed to gather concurrent physical, mental and social conditions into two axes, which also allow for multiple diagnoses. The diagnoses are made according to physical factors (Axis I) and psychosocial factors (Axis II):

A) Axis I: Clinical TMD conditions.

A set of research diagnostic criteria was used to investigate the masticatory muscle pain, disc displacements, and degenerative diseases of the TMJ.

B) Axis II: Pain-Related Disability and Psychological Status

A set of research diagnostic criteria was also used to assess chronic pain dysfunction, depression, nonspecific physical symptoms, and orofacial disability.

2.6.2.2. Summarized RDC/TMD Axis I criteria list

Group I: Muscle Disorders

The following uncommon conditions should first be ruled out:

- 1) Muscle spasm,
- 2) Myositis, and
- 3) Contracture.

I.a. Myofascial Pain:

1. Report of pain in the jaw, temples, face, preauricular area, or inside the ear at rest or during function; plus
2. Pain reported by the subject in response to palpation of three or more of the 20 muscle sites. At least one of the sites must be on the same side as the

complaint of pain. The 20 muscle sites (right side and left side count as separate sites for each muscle) are:

- i. posterior temporalis
- ii. middle temporalis
- iii. anterior temporalis
- iv. origin of masseter,
- v. body of masseter
- vi. insertion of masseter
- vii. posterior mandibular region
- viii. submandibular region
- ix. lateral pterygoid area
- x. tendon of the temporalis

I.b. Myofascial Pain With limited opening:

1. Myofascial pain as defined in I.a.; plus
2. Pain-free unassisted mandibular opening of less than 40 mm; plus
3. Maximum assisted opening of 5 mm or more, greater than pain-free unassisted opening.

Group II: Disc Displacements

II.a. Disc Displacement With Reduction:

1. Either:
 - a. Reciprocal clicking in TMJ (click on both vertical opening and closing that occurs at a point at least 5mm greater interincisal distance on opening than on closing, and is eliminated on protrusive opening); or

- b. Click in TMJ on both vertical range of motion (either opening or closing), *and* click during lateral excursion or protrusion

II.b. Disc Displacement Without Reduction, With Limited Opening:

1. History of significant limitation in opening; plus
2. Maximum unassisted opening $\leq 35\text{mm}$; plus
3. Passive stretch increases opening by $\leq 4\text{ mm}$ over maximum unassisted opening; plus
4. Contralateral excursion $< 7\text{ mm}$ and/or uncorrected deviation to the ipsilateral side on opening; plus
5. Either:
 - a. Absence of joint sounds, or
 - b. Presence of joint sounds not meeting criteria for disc displacement with reduction as defined in II.a.

III.b. Disc Displacement Without Reduction, Without Limited Opening:

1. History of significant limitation in opening; plus
2. Maximum unassisted opening $> 35\text{mm}$; plus
3. Passive stretch increases opening by $\geq 5\text{ mm}$ over maximum unassisted opening; plus
4. Contralateral excursion $\geq 7\text{ mm}$, plus
5. Presence of joint sounds not meeting criteria for disc displacement with reduction as defined in II.a.
6. (In those studies that allow imaging , the following imaging criteria should also be met)
 - a. Arthrography:

- (1) In intercuspal occlusal position, the anterior compartments appear larger and markedly more filled with contrast medium than in a normal joint;
- (2) On opening, significant contrast medium is retained anteriorly.

b. MRI:

- (1) In intercuspal occlusal position, the posterior band of the disc is located clearly anterior to the 12:00 position, at least at the 11:30 position;
- (2) On full opening, the posterior band remains clearly anterior to the 12:00 position.

Group III: Arthralgia, Arthritis, Arthrosis

The following conditions should first be ruled out:

- 1) Polyarthritides
- 2) Acute traumatic injuries
- 3) Infections in the joint

III.a. Arthralgia:

1. Pain in one or both joint sites (lateral pole and/ or posterior attachments) during palpation; plus
2. One or more of the following self-reports of pain:
 - a. Pain in the region of the joint
 - b. Pain in the joint during maximum unassisted opening
 - c. Pain in the joint during assisted opening

- d. Pain in the joint during lateral excursion
- 3. For a diagnosis of simple arthralgia, coarse crepitus must be absent
- III.b. Osteoarthritis of the TMJ:
 - 1. Arthralgia as defined in III.a.; plus
 - 2. Either a or b, or both:
 - a. Coarse crepitus in the joint
 - b. Imaging-Tomograms show one or more of the following:
 - (1) Erosion of normal cortical delineation
 - (2) Sclerosis of parts or all of the condyle and articular eminence
flattening of the condyle and articular eminence
 - (3) Flattening of joint surfaces
 - (4) Osteophyte formation
- III.c. Osteoarthrosis of the TMJ:
 - 1. Absence of all signs of arthralgia as defined in III.a.; plus
 - 2. Either a or b, or both:
 - a. Coarse crepitus in the joint
 - b. Imaging-Tomograms show one or more of the following:
 - (1) Erosion of normal cortical delineation
 - (2) Sclerosis of parts or all of the condyle and articular eminence
flattening of the condyle and articular eminence
 - (3) Flattening of joint surfaces
 - (4) Osteophyte formation

2.7. Treatment of TMD

The treatment modalities for TMD that have been suggested vary enormously over a great spectrum. Okeson (1998) mentioned that the list of treatment options with at least some scientific support have grown to 49. Therefore a great confusion still exists in TMD management. Frequently, the treatment methods were geographically regionalized or correlated strongly to the specialty of the clinicians. An appropriate treatment should be based on adequate scientific evidence.

2.7.1. Objective of treatment

The treatment objectives for TMD include decreased pain, decreased adverse loading, restoration of function, and resumption of normal daily activities.

In many patients with disc displacement, painless jaw function is still possible. The internal derangement of the TMJ often exhibits a natural progression of compensatory adaptation and remodelling. Muscle disorders more frequently require recurrent treatment than TMJ disorders (Okeson, 1996). Joint noise, moderate restriction of mandibular movement, unconscious deviation of the incisal path, and isolated instances of discrete TMD symptoms may just need to be observed over time to see if progressiveness is evident (Bell, 1990).

2.7.2. Principles of TMD management

Clark (1987) had stated three principles of TMD management as follows:

1. Treatment should be based on a specific diagnosis
2. Treatment should address the cause of the symptoms
3. Treatment should reflect the relative urgency of the symptoms

Okeson (1998) has reviewed 30 long-term studies and found that conservative and nonconservative therapies seem to report similar success rates of 70% to 85% on a long-term basis. In many TMD, the signs and symptoms over time may be transient and self-limiting, resolving without serious long-term effects. It would appear therefore that a logical approach to patient management is to first attempt conservative treatment such as patient education and self-care, behavioural modification, physical therapy, medications, and orthopaedic appliances. Early use of aggressive, irreversible treatments such as complex occlusal therapy or surgery should be avoided (Okeson, 1996).

Precipitating and perpetuating factors must be identified with the history and clinical examination such as bruxism and other parafunctional habits, trauma, adverse anatomic relationships, and pathophysiologic and psychosocial conditions. All these relevant contributing factors should be placed into a prioritized problems list to facilitate the treatment planning and sequencing. Single etiology, single diagnosis and single treatment approach should be avoided. Management goals are best achieved by using the optimal treatment combination with prioritized sequences. All treatment programmes should be time-limited and not left open-ended.

The American Academy of Orofacial Pain (AAOP) had given a comprehensive account on treatment options which include patient education and self-care, cognitive intervention, pharmacotherapy, physical therapy, occlusal appliances therapy, occlusal therapy and surgery. Besides, psychological factors also need to be considered in TMD management (Okeson, 1996).

2.7.3. Treatment modalities

Treatment modalities can be systematically categorized into five broad groups which include patient education and self-care, cognitive behavioural intervention, pharmacologic therapy, physical therapy, occlusal therapy, and surgery (Okeson, 1996; Kaplan & Assael, 1991).

2.7.3.1. Patient education and self-care

Patient education and self-care comprises the most basic treatment modality for TMD. The clinician need to take time to explain the clinical findings, diagnostic data, treatment options, and prognosis to the patient. This is important to promote patient motivation, cooperation and compliance which can determine the success of a self-care programme. Reassurance and education is a significant factor in treatment planning. Self-care programme should allow healing and prevents further injury to the masticatory system and would include the following:

1. Resting of the masticatory system through voluntary limitation of mandibular function. Patient need to avoid heavy mastication, gum chewing, wide yawning and singing, and playing some musical instruments.
2. Habit awareness and modification which include reversal of parafunctional habit such as clenching, bruxing, tongue thrusting, cheek biting, poor sleeping posture and object biting. Parafunctional habits can be modified with habit awareness, motivation to change, and knowledge of how to change. Stickers can be used to remind the patient in creating habit awareness.
3. Home physiotherapy programme includes application of heat or cold to the affected areas, massage of the affected muscles, and gentle exercises can decrease tenderness and pain and increase range of mandibular movement. Heat can be applied with warm towel or hot packs to stimulate muscle relaxation and

vascular perfusion. While cold can be applied using ice as analgesic and anti-inflammatory agent in muscle and joint tissues. However, heat should not be used for acute injury of about less than 72 hours from onset, acute inflammation or infection (Okeson, 1996).

2.7.3.2. Cognitive behavioural intervention

Cognitive therapy is any of a variety of techniques in psychotherapy that utilizes guided self-discovery, imaging, self-instruction, symbolic modelling, and related forms of explicitly elicited cognitions as the principal mode of treatment. Cognition refers to that operation of the mind by which one becomes aware of objects of thought or perception; it includes all aspects of perceiving, thinking, and remembering (Pugh, 2000).

2.7.3.3. Pharmacologic therapy

Pharmacologic agents have widely been used for pain management and functional rehabilitation in TMD. However, no one drug has been proven effective for the entire spectrum of TMD. In pharmacologic management of TMD, the treatment effect of variety of drugs should be maximized, and at the meantime, avoiding adverse drug interaction and complications. Besides, drug misuse and abuse are also of concern. The most effective pharmacologic agents for the management of TMD include analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, anxiolytic, muscle relaxants, and low-dose antidepressants. The analgesics, corticosteroids, anxiolytics are indicated for acute TMD pain; the NSAIDs and muscle relaxants may be used for both acute and chronic conditions; and the tricyclic antidepressants are primarily indicated for chronic orofacial pain (Okeson, 1996). The use of anxiolytics, tricyclic antidepressants and also hypnotics in the management of TMD is controversial (Gray et al, 1995).

2.7.3.3.1. Analgesics

The analgesics are used to reduce pain and are generally divided into opioid and non-opioid preparation. Opioid analgesics are used for moderate to severe pain. However they can cause central nervous systems depression and addiction (Way et al, 1998). They are most useful in short-term, acute pain condition. Opioid analgesics such as codeine are usually used in combination with non-opioid analgesic such as aspirin or paracetamol (Okeson, 1996).

Non-opioid analgesics are basically comprised of nonsteroidal anti-inflammatory drugs and other analgesic such as paracetamol (Way et al, 1998).

2.7.3.3.2. Nonsteroidal anti-inflammatory drugs (NSAIDs)

All nonsteroidal anti-inflammatory drugs are antipyretic, analgesic, and anti-inflammatory but with differences in their effects. They are effective for mild to moderate inflammatory conditions and acute pain. They are useful in the management of musculoskeletal pain such as pain dysfunction and osteoarthritis. These drugs provide only symptomatic relief but with possible arrestment of the progression of active inflammatory joint disease. Ibuprofen and mefenamic acid are among the most commonly prescribed NSAIDs in TMD. They should be avoided in peptic ulceration, asthma, pregnancy or kidney and liver disorders (Katzung & Furst, 1998).

2.7.3.4. Physical therapy

Physical therapy helps to relieve musculoskeletal pain, restore normal function and promote repair of tissues. It is usually used as an adjunct to other treatments. It is a conservative and effective method in treatment of TMD (Okeson, 1996). Physical therapy for TMD had been discussed in relationship with craniocervical disorders

(Dunn, 1991). Physical therapy comprised posture training, exercise, mobilization, and physical agents or modalities. Physical agents or modalities would include electrotherapy, ultrasound, short wave diathermy, soft laser, iontophoresis, anesthetic agents and acupuncture.

2.7.3.5. Occlusal therapy

Occlusal therapy referred to treatment that alters the occlusal condition of the patient. It is divided into two types which are the reversible occlusal therapy and irreversible occlusal therapy.

2.7.3.5.1. Reversible occlusal therapy

Reversible occlusal therapy only alters the patient's occlusion temporarily without permanent changes. This can be accomplished with the use of occlusal appliance. Occlusal appliance therapy has traditionally been considered reversible, but unexpected mandibular shifting may be reported especially in long-term full-time usage. Repositioning splint therapy can be considered in the early stages as reversible occlusal therapy but can rapidly cause changes in tooth and jaw relationships in prolonged usage (Kaplan & Goldman, 1991).

2.7.3.5.1.1. Occlusal appliance therapy

Occlusal appliance is most commonly called splint. It was also referred as occlusal or interocclusal splint, orthotic, orthosis, bite guard, bite plate, nightguard, bruxism appliances or orthopaedic appliance. A splint is a removable device that covers the teeth to alter the occlusal relationship, to change the structural relationships of the TMJ, and to redistribute occlusal forces. The functions of the splints are to provide optimum functional occlusion, to establish a more orthopedically stable joint position, and to

prevent tooth wear and mobility. It can also reduce bruxism or parafunctional habits, and to treat masticatory muscle pain and dysfunction; by reorganizing the neuromuscular reflex activity which in turn reduces abnormal muscle activity, and encouraging more normal muscle function (Okeson, 1996).

The reduction of painful symptoms by using splint has been well documented. Clark (1984) reviewed the effectiveness of the splint and found success rate of 70% to 90%. Splint therapy is basically a conservative treatment. However excessive and improper use of the splint can cause complication such as caries, gingival inflammation, mouth odours, speech difficulties, occlusal changes, and psychological dependence on the appliance. Long-term and full-time use of splint especially with the partial-arch coverage design can cause major irreversible changes to the occlusion and jaws relationship. The most common splints used in the treatment of TMD are stabilization splint, anterior repositioning splint and soft splint.

2.7.3.5.1.1.1. Stabilization splint (hard splint)

Stabilization splint, which commonly known as hard splint, is usually made of hard acrylic and designed to cover all the occlusal and incisal surface of the maxillary or mandibular teeth, to provide precise occlusal contact with the teeth of the opposing arch (Okeson, 1998). It was also called flat plane, gnathologic or muscle relaxation splint, Tanner appliance, Fox appliance and Michigan splint or the centric relation appliance (Gray et al, 1995). The functions of the splint are to provide joint stability, to protect the teeth, to relax the elevator muscle and to decrease parafunctional habit include bruxism. The mechanisms of the treatment effect are not well understood. The splint helps to increase the awareness of the patient and provide a stable physiologic mandibular position. To fabricate a stabilization splint, the occlusal surface of the splint

should be adjusted to provide bilateral, even posterior occlusal contacts for all the opposing teeth on closure. The stabilization splint should also have an anterior guidance that usually provided by acrylic guide ramps in the canine or anterior region of the appliance, to separate the opposing posterior teeth from the appliance in all excursive movements of the mandible. The occlusal surface of the splint needs to be adjusted periodically to compensate for changes in the maxillomandibular relationship as level of pain, inflammation, oedema and muscle activity change. A good occlusal stabilization splint should have the following eight criteria recommended by Okeson (1998):

1. Accurately fit the maxillary teeth, with total stability and retention.
2. In centric relation, all posterior mandibular buccal cusps must contact on flat surfaces with even force.
3. During protrusive movement, the mandibular canine must contact the appliance with even force. The mandibular incisors may also contact it but not with more force than the canines.
4. In any lateral movement, only the mandibular canine should exhibit laterotrusive contact on the appliance.
5. The mandibular posterior teeth must contact the appliance only in the centric relation closure.
6. In the alert feeding position, the posterior teeth must contact the appliance more prominently than the anterior teeth.
7. The occlusal surface of the appliance should be as flat as possible with no imprints for mandibular cusps.
8. The occlusal appliance is polished so it will not irritate any adjacent soft tissues.

The stabilization splint may be worn full-time especially in acute cases of TMD but should be monitored with care. Eventually, use only at night as symptom reduced, or

intermittent use at night during periods of increased stressful life events may be indicated. For patient with nocturnal bruxism, appliance wear was only considered at night. However, effect of splint on reduction of bruxism activity is variable. Patient with articular disorders may benefit from full-time wear (Wilkinson et al., 1992). However, full-time use of splint should be avoided whenever possible.

Patient who does not showing a positive response within 3 to 4 weeks should be reevaluated for factors such as chronic pain behaviour, non-compliance, misdiagnosis, or degree of TMJ pathology. Combination of splint therapy with other kind of treatment may be more beneficial.

2.7.3.5.1.1.2. Soft splint

Soft splint is usually made of soft resilient material like polyvinyl to cover all the teeth of the maxilla or mandible but no adjustment is made to balance it to the opposing teeth. It is quick, easy and economical to fabricate. The soft splint is of uniform thickness, made by a vacuum forming technique onto the model. Night time wear is usually recommended. It may absorb occlusal forces by virtue of its soft nature, or act as a habit breaker by increasing the patient awareness. Soft splint provide a mixed results in reducing nocturnal bruxism and signs and symptoms of TMD. Occlusal changes following the use of soft occlusal splints should be of concern (Singh & Berry, 1985). Wright et al. (1995) suggest that the efficacy of soft splint may be related to the stability of the appliance occlusion and that with stable contacts, occlusal changes do not occur. However the use of soft splint is best suited for short-term treatment. It can also be used in treatment for children with mixed dentition as the soft splint seems to have minimal effect on dental development while increasing wear compliance (Ingerslev, 1983).

2.7.3.5.1.1.3. Soft splint versus hard splint

Although hard splint was more widely used in the treatment of TMD, its effectiveness was still not confirmed. A systematic review of randomized controlled trial reported that the use of occlusal splints may be of some benefit in the treatment of TMD but the evidence is scarce (Forssell et al, 1999). Another systematic review of randomized or quasi-randomised controlled trial also revealed that there is insufficient evidence either to support or against the use of stabilization splint therapy for the treatment of temporomandibular pain dysfunction syndrome (Al-Ani et al, 2004).

Okeson (1987) in his study of the effect of hard and soft splints on nocturnal bruxism reported that the hard splint significantly reduced nighttime muscle activity while the soft splint significantly increased the muscle activity. However, Wright et al. (1995) had pointed out three observations on this Okeson's study. First, the treatment effect of soft splint may not be a result of reduced nocturnal EMG activity. A reduction in symptoms may be unrelated to reduced nocturnal EMG activity. Second, the response of TMD patients towards the splint may be different from that of healthy subjects in the study by Okeson. Third, the acknowledged differences in the occlusal adjustment methods for the soft and hard splints in Okeson's study may have affected the outcome. Therefore, Okeson's study still could not confirm that the hard splint was more superior to the soft splint in the TMD treatments. Another study of the effect of hard and soft splints on the EMG activity in healthy subjects by Al-Quran and Lyons (1999), reported that the EMG activity of the masseter muscle was non-significantly decreased with hard splint but significantly increased with soft splint. Since the decrease in EMG activity of masseter muscle with hard splint was not significant, it was inconclusive to claim that the hard splint was superior to soft splint. Moreover, the study was also carried out on healthy

subjects. Thus, the hard splint was not confirmed to be more effective than the soft splint in treating the TMD patients.

Pettengill (1998) suggested that the soft and hard stabilization appliances may be equally useful in reducing masticatory muscle pain in short-term appliance therapy. Wright et al. (1995) suggested that the soft splint is an effective short-term treatment for reducing the signs and symptoms of masticatory muscle pain in TMD patients, in comparison with palliative treatment and no treatment after 4 to 11 weeks of treatment. They also advocated that the soft splint does not cause occlusal changes, which was opposed to the studies by Singh and Berry (1985). Patient also seemed to have better compliance with the soft splint than the hard splint (Wright et al., 1995).

2.7.3.5.1.1.4. Mechanism of action of the splint

2.7.3.5.1.1.4.1. Sensory changes in masticatory system

The effect of the splint to the masticatory activities could be due to changes in sensory information from peripheral receptors. The peripheral receptors likely to be involved would include those in the TMJ (due to altered condylar position), from the muscles (due to longer working length), from the periodontal ligaments (due to altered tooth contacts), and from the tongue, lips and the oral mucosa due to the presence of a foreign object (i.e. the splint). When a splint is being inserted into the mouth, it actually invades the freeway space. As a result, the teeth will usually bite onto the splint. However the tooth contact onto the splint is taken as though the teeth are biting into foreign body. This proprioceptive input from the periodontium structures will then be conveyed to the central nervous system which later modifies the muscle activity (Al-Quran & Lyons, 1999).

2.7.3.5.1.1.4.2. Increased vertical dimension

One of the ways the splint helped in reducing the muscle activity was by increasing the vertical dimension between the maxilla and the mandible. Majewski and Gale (1984) in their study (n=11) reported that with increased vertical mandibular opening from centric occlusion, postural EMG activities of the anterior temporal and masseter decreased. At mandibular opening about 3 mm, the postural EMG activity of anterior temporal muscle would decrease to a minimum level, which the EMG activity did not change significantly with further opening. At mandibular opening about 3 mm also, the postural EMG activity of masseter muscle would decrease to a minimum level, but the EMG activity would increase with further opening.

The increase of the intermaxillary vertical dimension by means of soft splint can reduce the EMG activity especially during clenching. Manns et al. (1983) and Manns et al. (1985) had reported that elongation of elevator muscle by means of occlusal splint produced neuromuscular relaxation. Emshoff and Bertram (1998) had suggested that the efficacy of splint may be associated with the reduction of the local muscle thickness. This mechanism of action may partly explain the reduction in the EMG activity of the muscles.

2.7.3.5.1.1.4.3. Reduction of parafunctional activity

Solberg et al. (1975) found that nocturnal masseter muscle activity was reduced immediately after the insertion of stabilization splint. Sheikholeslam et al., (1986) reported that occlusal splint can offer an optimum occlusal condition that reorganizes the neuromuscular reflex activity, which in turn reduces abnormal muscle activity. Thus, it can decrease parafunctional activity that often accompanies periods of stress.

In the study by Okeson (1987), the nocturnal masseter EMG activity was increased in subjects with soft splint but significantly reduced in subjects with hard stabilization splint. However the Okeson's study was carried out on subjects without TMD symptoms. The effect of splint on normal subjects might be different from TMD patient as it was found in this study. Besides, the relationship between the nocturnal muscles activities might not be in a linear regression with the severity of TMD symptoms among TMD patients.

2.7.3.5.1.1.4.4. The pivoting effect of the splint

According to Mansour and Reynik (1975), the mandible functions as a class II lever during the biting along the second molar. This would cause a distracting force on the condyle since the condyle is free to move to certain extent. This was disagreed by Ito et al. (1986) as their studies showed that the pivoting appliance that fulcrum around the second molar would actually load the condyle. Moncayo (1994) however found that the joint was distracted with biting on a bilateral pivoting appliance as revealed by tomograms.

The temporomandibular system is considered acting as a level system Class II in simplified analysis. Since the thickness of soft splint is quite even, it is thus expected that the posterior teeth will start to occlude onto the soft splint earlier than the anterior teeth. Due to the soft nature of the soft splint, it will be compressed as the posterior teeth were biting further. This will allow the subsequent contact making by the front teeth. From the biomechanics viewpoint, this may be beneficial as the biting force is greater at the posterior teeth than the anterior teeth due to the shorter lever arm. It was suggested the pivoting effect of splint may even be greater in the presence of third molar.

The benefit in the individual with the present of third molars compared to the absent of third molars in this context however needs further investigation.

2.7.3.5.2. Irreversible occlusal therapy

Irreversible occlusal therapies referred to treatment modalities that alter the occlusal condition of the patient permanently, which include occlusal adjustment, restorative therapy and orthodontic-orthognathic therapy. Although numerous studies had been carried out to investigate TMD in relationship with occlusal discrepancies, it is still difficult to establish any significant cause-and-effect relationships due to the many variables involved, which is difficult to exclude clinically (Okeson, 1996).

Occlusal therapy should be used cautiously, since there is lack of evidence that occlusal discrepancy is a common cause of TMD (Pullinger et al, 1993). Malocclusion may be a sequelae of the TMD rather than a cause. There is also no evidence that anterior teeth guidance is superior to posterior teeth guidance. Generally, there is lack of evidence to ensure the use of complex occlusal therapy to provide an ideal occlusion in routine TMD management (Rugh et al.,1989; Minagi et al., 1990).

2.7.3.6. Surgery

Surgery may be recommended in the treatment of TMJ disorders, which include arthrocentesis, arthroscopy and arthrotomy. However, the complexity of available techniques, potential complications, prevalence of psychosocial contributing factors, and availability of many nonsurgical treatment modalities suggested that surgery should only be used in selected cases. The decision to treat a patient surgically depends on the degree of anatomic derangement present within the joint, the potential for repairing the condition, the severity of impairment the problem brought to the patient and the outcome of appropriate nonsurgical treatment. American Association of Oral and Maxillofacial Surgeons' position paper on TMJ surgery has suggested a criteria list that should be met by the patient before attempting TMJ surgery (Okeson, 1996).

Preoperative and postoperative nonsurgical management is needed as part of the surgical treatment plan to decrease the functional joint loading, to eliminate or modify contributing factors such as parafunctional habits, and to provide psychological and medical support (Okeson, 1996).

2.8. Electromyography (EMG)

Electromyography is a technique of recording the intrinsic electrical activity of a muscle. It carries the acronym of EMG. It is useful in the study of the muscle and neuromuscular system. The study of the electrical activity of contracting muscle provides information regarding the structure and functioning of the motor units. Whenever the muscle fiber contracts, the surface membrane undergoes depolarisation so that an action potential can be recorded from the fiber. EMG involves the studying of the characteristics of the different potentials that have been recorded. There are five

characteristics which can be described for any EMG potential as following (Echternach, 1994):

1. Voltage

Voltage refers to the amplitude of the response. This involves the measurement of the size of the potential from peak to peak. This measurement is in microvolt (μV) or millivolt (mV).

2. Duration

Duration is the time it takes for the individual potential to complete itself; from the beginning of the first of its deviations from the baseline to the last deviation and back to the base line. This measurement is expressed in millisecond (ms).

3. Wave forms

Wave forms are EMG potentials that may be monophasic, biphasic and etc. This describes the numbers of deviations from the baseline that the potential makes.

4. Frequency

Frequency is a description of how often the potential fires within a certain time frame. It is usually expressed as some number per second.

5. Sound

EMG potentials are displayed not only visually but can also be heard on a speaker and their individual sound characteristics can be used in determining what type of EMG potential is being displayed.

2.8.1. Device

The basic device of EMG includes the following:

1. Electrode

Electrode is a simple device that is capable of conducting electricity. It can be made of metal or fabric, in plate or needle forms. The plate or disc forms electrodes are used in surface EMG to be placed over the muscle to record an electrical potential difference. The needle form electrodes are used in clinical EMG to be inserted into the muscle to also record an electrical potential difference or to apply an electrical current.

There are three electrode components that are necessary for EMG testing, which are the recording electrode, reference electrode and ground electrode. The ground electrode helps to reduce extraneous noise and interference. The recording electrode is also known as active electrode and is labelled as negative electrode. The reference electrode is also known as the passive electrode and labelled as positive electrode. These two electrodes must work in pairs to pick up the potential difference between them. However, in surface EMG, there are usually no true differences in these two electrodes but more of a convention in practice.

The potential difference that has been picked up is then conducted to amplifier through the connecting wire.

2. Amplifier

The amplifier magnified the potential difference from the electrodes and then sends the signal to computer or other compatible combination of devices.

3. Computer or other compatible combination of devices.

The computer process the signal coming from the amplifier and then either display it on the monitor, hear it on the speaker, store it in the storage disk or print it in the printer.

2.8.2. Types of EMG

EMG can generally be divided into two types, which are the surface electromyography and the intramuscular electromyography.

Intramuscular electromyography is an investigation test that usually measures muscle response to nerve stimulation. It is used to evaluate muscle weakness and to determine if the weakness is related to the muscles themselves or a problem with the nerves that supply the muscles. Needle electrode is used in intramuscular electromyography which is being inserted into the muscle. Intramuscular EMG gives a more localized view of the muscle condition when the whole muscle response is not required. However it is more expensive and invasive compared to surface EMG.

Surface electromyography records the summation of electrical signals or action potentials generated by muscle fibers contraction. The summation can be regarded as the total contractive force exerted by the muscle. However, it does not differentiate between isotonic and isometric contractive force. In surface electromyography, surface

electrodes are placed on the skin overlaying the muscle. Unlike intramuscular EMG, surface EMG allows the study of the whole muscle response. Surface EMG records the summated compound potential of many motor units instead of individual ones.

2.8.3. Application

EMG has been used widely in kinesiological analysis of the muscle to allow for the study of the muscle movements. Recording the electrical activity of the masticatory muscle has also been advocated by many as a measure to discover the resting and maximal activities of the muscle.

EMG examination of masticatory muscles may have more value in clinical diagnosis of myospasm, which characteristically shows a marked increase in EMG activity when the muscle is at rest (Travell & Simons, 1999). It may be used also for identifying asymmetry of muscle action and particularly for assessing the results of therapy and normally gross differences are only detected.

EMG can be used to study the muscle weakness and to determine if the weakness is related to the muscles themselves or related to the nerves that supply the muscles. It is also used in the study of the muscle silent period. Silent period is the time during which there is no electrical activity in a muscle following its rapid unloading or which there is any pause in an otherwise continuous series of electrophysiological events. The masseteric EMG silent period has been found to be prolonged in patients with myofascial pain-dysfunction symptoms. However, the practical application for it in the clinical management of TMD is limited due to its marked variability and inconsistency of silent periods.

EMG has also been used in a variety of biofeedback applications which have used surface electrodes. These applications have been used as adjuncts to treatment programmes aimed at increasing or decreasing muscle activity pertaining patients to gain greater control over their muscle activity.

A lot of studies had been carried out to facilitate the use of EMG recording in the diagnosis and treatment of TMD. However, EMG was generally not been supported to be used as a routine diagnostic tool for TMD. To date, EMG is more frequently been used under research conditions to provided useful information on muscle function.

2.8.4. Reliability and validity

EMG measurements were found to be technically sensitive. It has been demonstrated that relatively small variations in electrode placement can significantly change EMG recordings (Rugh, 1988). Therefore, recording taken during multiple visits cannot be compared accurately. Extreme care may be taken to place the electrode in the exact same location for each recording, but this is not always possible.

Some studies do show higher EMG activity in the TMD patients than the control subjects. However, most of these EMG differences are very small and are often less than the variations that occur between patients, such as between male and female, and between thin face and fat face (Oltjen et al, 1990). With such slight differences and such great variations, EMG recordings should not be used without care such as for diagnosis or monitoring treatment of TMD. Some studies (Shi & Wang, 1989; Pinho et al., 2000; Glaros et al., 1997) have reported that TMD is associated with a slight increase in muscle basal tone. However, Glaros et al. (1997) have objected the use of a cut-off

score based on EMG values to accurately distinguish TMD patients with myofascial pain from healthy subjects.

The issues of reliability and validity of EMG in diagnosis and treatment of TMD had been discussed by Mohl et al. (1990) and Widmer et al. (1990). They concluded that there is no evidence to support the use of surface EMG for the evaluation or diagnosis of TMD. EMG differences among different facial types, age and sex, thickness of subcutaneous fat, and history of bruxism were suggested as factors that affect diagnostic validity.

2.8.5. Uses of EMG in TMD

It was originally felt that painful muscle may be in spasm or associate with increased EMG activity. However, studies now demonstrate that muscle pain is often not associated with any significant increase in EMG activity (Majewski & Gale, 1984). Moreover, most muscle pain seems to be not a result of myospasms but more of local muscle soreness, trigger point myalgia, or myositis. These conditions are not directly associated with muscle contraction (Okeson, 1993). It is widely accepted in dentistry that the mandibular position and occlusal contact patterns of the teeth can influence the amount of muscle hyperactivity that takes place. Since alteration of occlusal condition can affect muscle function (Riise & Sheikholeslam, 1984), EMG study can be done to assess the effect of occlusal splint on muscle function (Kawazoe et al, 1980).

2.8.6. EMG acquisition setup

One of the most significant variables in the acquisition setup was the setting of low-pass filter and high-pass filter. The low-pass filter setting of 20 Hz are able to suppress the movement artifacts especially during the EMG recording of maximum clenching. The

movement artefact were seen as irregular spikes on the EMG waveforms displayed on the monitor (Soderberg, 1992).

The high-pass filter could remove the thermal noise which was generated due to the resistance to current flow by the conductor in the circuit. The thermal noise is generated in the electrodes, in the wire leads connecting the electrodes to the amplifier, and in the host of electronic components internal to the EMG instrumentation (Soderberg, 1992). The high-pass filter was also said it could reduce noise due to the synchronous activity of the motor units (Brinkworth & Turker, 2003). The surface EMG spectrum normally ranges from 10 Hz to about 500 Hz. The optimal high-pass filter setting should best remove most of the noise and at the meantime preserved the entire interested EMG signal. The high setting of high-pass filter such as 10000 Hz would result in incorporation of much interference into the EMG signal. The high-pass filter was conservatively set as twice the highest interested EMG signal frequency (Basmajian & DeLuca, 1985). The high-pass filter setting of 1000 Hz preserved more raw EMG signals than the setting of 500 Hz (Donegan et al., 1990; Cram et al., 1998).

2.9. Splint and electromyography in TMD

2.9.1. The effect of the splint on muscles activities

2.9.1.1. The effect of the soft splint on muscles activities

Soft splint could alter the masseter and anterior temporal muscles activities. There were relatively less studies carried out to investigate the effect of soft splint on EMG activity of masticatory muscle. Two EMG studies that involve soft splint were actually intended to compare the effect of the hard splint with the soft splint which were the study by Okeson (1987) and another by Al-Quran and Lyons (1999). However, these two studies were carried out on healthy subjects but not on TMD patients. Moreover, these two

studies also did not report data for the effect of splint on the postural activity of the masticatory muscle.

To date, no EMG study has investigated the effect of soft splint on TMD patients. There is also no EMG study that has compared the effect of soft splint on TMD patients with healthy subjects. There is also no EMG study that has investigated the effect of soft splint on the postural activity of the masticatory muscle.

Okeson (1987), in his study (n=10) of the effect of hard and soft splints on nocturnal bruxism in healthy subjects, reported that the hard splint significantly reduced while the soft splint significantly increased the masseter muscle activity. His study actually investigated the dynamic muscle activity, which is the nocturnal muscle activity; but not the static muscle activities, which were the postural and maximum clenching muscle activities. His study also did not report data for anterior temporal muscle.

Al-Quran and Lyons (1999), in their study (n=10) of the effect of hard and soft splints on the EMG activity in healthy subjects, reported that the maximum clenching activity of the masseter muscle was non-significantly decreased with hard splint but significantly increased with soft splint. According to them, the increase in activity of the masseter muscles when clenching maximally on a soft splint could simply be because the soft material was more comfortable to bite on compared to hard splint. They also reported that both the hard and soft splint caused a non-significant decrease in the maximum clenching activity of anterior temporal muscle. Their study did not report data for postural muscle activity.

As a summary, there was no conclusive report on the effect of the soft splint on the postural and maximum clenching activity of the masticatory muscle except the soft splint could increase the maximum clenching activity of the masseter muscle of healthy subjects.

2.9.1.2. The effect of the hard splint on muscles activities

Numerous studies had been carried out to study the effect of the hard splint to the masticatory muscles activities. However, some studies used TMD patients as experimental subject where else some other studies used healthy subjects. As note also by Wright et al. (1995), the response of TMD patients towards the splint may be different from that of healthy subjects. Thus,

The literature review here was done by first differentiating these studies into those that involved TMD patients or those that involved healthy subjects. It was found that the hard splint would produce different effect on TMD patients compared to healthy subjects. For this reason, it was suggested that the EMG study done on healthy subjects could not be regarded as identical to EMG study done on TMD patient. Any result reported from the study done on healthy subjects should not be used boldly to make inference on TMD patients.

2.9.1.2.1. The effect of the hard splint on TMD patients

The study (n=60) by Shi and Wang (1989) showed that the EMG activity of the anterior temporal and masseter muscles of TMD patients with masticatory muscle pain, at the postural position and during maximum clenching, was significantly reduced after insertion of the hard occlusal splint.

Holmgren et al. (1985) in the study of TMD patients (n=31) with a habit of nocturnal bruxism, reported that fifteen minutes after insertion of hard occlusal splint, the postural EMG activity of the anterior temporal muscles in TMD patients decreased significantly in 52%, increased in 22% and remained unchanged in 26% of the patients. These TMD patients presented with nocturnal bruxism, muscle pain and with or without joint pain. In the masseter muscle, the postural EMG activity decreased in 19%, increased in 10% and remained unchanged in 71% of the patients. As a whole, the reduction was highly significant in the anterior temporal muscle. Data on maximum clenching activity was published in another paper (Holmgren et al., 1990).

Ferrario et al. (2002) in their study (n=14) observed that the hard occlusal splint significantly reduced the EMG activity of the anterior temporal and masseter muscles during maximum clenching among TMD patients with disc displacement with reduction. It was not mentioned that whether the TMD patients presented with pain or not. This study did not report data on postural activity.

Kawazoe et al. (1980) in their study (n=7) found that the masseter muscle activity during maximum clenching in myofascial pain patients was significantly lower with hard occlusal splints than without splints. However in healthy subjects, such a significant difference could not be observed. This study did not report data on postural activity.

In contrary, Holmgren et al. (1990) in their study (n=31), reported that the hard occlusal splint generally cause no significant difference in the maximum clenching EMG activity of the anterior temporal and masseter muscles. Data on postural activity was reported in previous paper (Holmgren et al., 1985).

Another study (n=8) on nocturnal EMG activity of bruxism patients by Solberg et al. (1975) reported that the hard occlusal splint significantly reduced the nocturnal masseter muscle activity, which was in line with the Okeson (1987)'s study.

As a summary, the hard splint generally could result in a decrease of both the postural and maximum clenching activity in both the masseter and anterior temporal muscles of the TMD patients.

2.9.1.2.2. The effect of the hard splint on healthy subjects

Wood and Tobias (1984) in their study (n=7) showed that the maximum clenching activity in healthy subjects was significantly increased with hard occlusal splint. The anterior temporal muscle activity was increased by 15 % while the masseter muscle by 27 %.

Kawazoe et al. (1980) in their study (n=7) reported that there was no significant difference in masseter muscle activity during maximum clenching in healthy subjects after insertion of hard occlusal splints. However, significant difference was found in myofascial pain patients.

Christensen (1980) reported in his study (n=6) that the hard occlusal splint caused no significant difference in the maximum clenching EMG activity of the masseter muscle in healthy subjects.

The above three study only reported data on the maximum clenching activity but not the postural activity. The results reported by Kawazoe et al. (1980) and Christensen (1980)

were in line with that reported by Al-Quran and Lyons (1999), which there was no significant difference in the maximum clenching activity of masseter and anterior temporal muscles after insertion of hard occlusal splint in healthy subjects (n=10). Their results were in contrary to that of Wood and Tobias (1984). According to Wood and Tobias, the increase in muscle activity was due to changes in the nature and number of tooth contacts provided by the equilibrated occlusal splint. However, all these study similarly had small sample size which ranged from 6-10. Therefore, study with bigger sample size was needed to provide more powerful result.

Carlsson et al. (1979), who used partial splint that covered mandibular caninies, premolars and molars, which was cemented onto the teeth; reported that the splint significantly reduced the postural activity of anterior temporal muscle but not the masseter muscle. The splint caused no significant difference in the maximum clenching activity of the anterior temporal and masseter muscles.

As a summary, the hard splint generally might cause no difference in the maximum clenching activity of the masseter and anterior temporal muscles in healthy subjects. It still remained unclear for the effect of the hard occlusal splint on the postural activity of healthy subjects.

2.9.1.2.3. The effect of the hard splint on muscles activity after long-term hard splint therapy

There are only one published paper had reported on the effect of the splint to the EMG activity of elevator muscles after long-term (3-6months) occlusal splint therapy and improvement of the signs and symptoms of TMD, which was the study by Holmgren et al. (1990) (n=31). They reported that after long-term hard occlusal splint therapy and improvement of the signs and symptoms of TMD, the number of patients who had an

identical level of EMG activity of the anterior temporal and masseter muscles during maximum clenching in the intercuspal position and on the occlusal splint tended to increase. In other words, after long-term occlusal splint therapy, more patients showed no significant difference in maximum clenching activity before and after insertion of splint. This study did not report data on postural activity.

2.9.1.2.4. EMG activity of muscles after long-term hard splint therapy

Relatively less EMG study had followed up the short term or long term effect of the splint. The studies by Shi and Wang (1989) and Sheikholeslam et al. (1986) actually compared directly the absolute EMG activity of the subjects before and after the treatment.

Shi and Wang (1989) (n=60) reported that 23 patients that had resolved pain in the muscles and TMJ after 3 months of splint treatment showed a significant decrease in postural activity of the anterior temporal muscle but no significant difference for the masseter muscle, compared to that of before treatment. There was a significant increase in the maximum clenching activity of the anterior temporal muscle but again no significant difference for the masseter muscle.

Sheikholeslam et al. (1986) (n=31) reported that 27 patients whose signs and symptoms improved after 3 to 6 months of splint therapy showed a significant decrease in postural activity of both side of anterior temporal muscle and the right masseter muscle, compared to that of before treatment. This study did not report data on maximum clenching activity.

As a summary, the postural activity would be reduced after the splint treatment, compared to that of before the treatment. Meanwhile the maximum clenching activity of the anterior temporal muscle would be increased after the splint treatment. It seems like the postural activity and the maximum clenching activity of the TMD patients were moving closer to that of the healthy subjects.

2.9.2. TMD patients versus healthy subjects

Since the response of TMD patients towards the splint might be different from that of healthy subjects, a literature review was carried out here to get some insight into the EMG activity difference that existed between these two groups of people.

2.9.2.1. Reliability of EMG study comparing TMD patients and healthy subjects

According to many studies, (Rugh, 1988; Frame et al., 1973; Nouri et al., 1976; Soderberg, 1992) relatively small variations in electrode placement can significantly change the readings of EMG activity recording. Therefore readings taken from different electrode setting cannot be compared accurately. This would also make the comparison of the EMG activity between individuals unreliable. However some studies (Visser et al., 1992; Pancherz & Winneberg, 1981) still claimed that there was no significant influence of electrode placement upon the EMG readings recorded over different electrode setting. Nevertheless, studies that had been carried out to compare the EMG activity of TMD patients with healthy subjects might be able to give some clue to the EMG activity differences existed among these two groups of people, provided that extreme care had been taken to reduce the effect due to different electrode placements.

2.9.2.2. EMG activity of TMD patients versus healthy subjects

The studies by Shi and Wang (1989) on TMD patients showed that the EMG activity of the anterior temporal and masseter muscles of the patients was higher in the postural position and lower during maximum clenching, as compared to the healthy subjects. There were 60 patients and 30 controls chosen for their study. They also found that the maximum clenching activity for the TMD patient without muscle tenderness were slightly lower but the postural activity was quite similar compared to healthy subjects. In other words, the TMD patients without muscle tenderness were quite similar to healthy subjects in terms of the EMG activity, differentiating them from TMD patients with muscle tenderness. Glaros et al. (1997) also reported that the postural EMG activity of TMD patients were higher than healthy subjects. However, they objected the use of a cut-off score based on EMG values to accurately distinguish TMD patients with myofascial pain from healthy subjects.

Pinho et al., (2000) in their study (n=40) indicated that TMD patients was associated with a slight increase in postural activity and reduced clenching activity by about half compared to healthy subjects. However, they drawn this conclusion by comparing directly their EMG activity readings from their study with that from others studies, which deemed to be very unreliable. It was because the settings were normally quite different from one study to another. Besides, the variation would exist among the different investigators from different studies. Their sample group consisted of TMD patients that might presented with clicks, pain in TMJ area, headache, TMJ pain during mastication, limited mouth opening or other symptoms.

In contrary, Majewski and Gale (1984) reported in their study (n=11) that the EMG activity of anterior temporal muscle was not significantly different between pain and non-pain sides, of unilateral pain patients. The EMG activity of anterior temporal and

masseter muscles also did not differ significantly between the patients and healthy subjects. The method acquired in obtaining the EMG activity value was also different. The postural activity was divided by the maximum activity and then multiplied by 100 to obtain the EMG percentage values. If one applied the previous results reported by Shi and Wang (1989), and Pinho et al. (2000) into this formula, one would expect that the EMG percentage value between the healthy subjects and the TMD patients would be highly different. However, this was not the case. Anyway it was important to point out that their EMG measurements on patients were actually done during the pain-free periods of the patients, while others studies did the EMG measurements during the pain periods. In others words, their report may be best interpreted as that the EMG activity of the patients during pain-free period was not significantly different from healthy subjects.

As a summary, TMD patients generally might have higher postural activity but lower maximum clenching activity in the anterior temporal and masseter muscles compared to healthy subjects. This difference was expected to cause different effect when the splint was inserted on TMD patients compared to healthy subject.

2.9.3. Anterior temporal muscle versus masseter muscle

It appeared in the literature reviews of this study that the effect of the splint might be difference on the anterior temporal muscle compared to the masseter muscle.

In the study by Al-Quran and Lyons (1999), it was found that apparently there was a more pronounced decrease in the maximum clenching activity by the hard splint on anterior temporal muscle as compared to the masseter muscle. It had been observed that the temporal muscle appeared to be more responsive to occlusal irregularities and changes in occlusal vertical dimension (Ramfjord & Ash, 1995).

Naeije et al. (1989) reported at low clenching levels the temporal muscle tended to dominate, at high levels the masseter muscle was stronger. The anterior temporal muscle is generally more active during natural function than the masseter muscle. The temporal muscle controls mandibular posture and contracts vigorously during chewing, while the masseter muscle contributes little to posture but more to chewing.

It was reported that the temporal muscle predominates in maintaining the postural position of the mandible during rest while the masseter muscle predominates in the maximum clenching (Moller 1966; Lund et al. 1970). Donegan (1990) also reported that the temporal muscle predominated over the masseter muscle in maintaining the postural position of the mandible; however the masseter and anterior temporal muscles showed about equal activity in generating maximum clenching activity.

As a summary, it seemed that the anterior temporal muscle play an important role in both of the postural and clenching activity, while masseter muscle contribute more to clenching activity only. The anterior temporal muscle might also be more responsive to occlusal changes compared to masseter muscle.