TEMPOROMANDIBULAR DISORDERS IN PRETREATMENT ORTHODONTIC PATIENTS AS RELATED TO MALOCCLUSION

A Thesis Submitted
to the College of Dentistry
University of Baghdad
in Partial Fulfillment of the Requirements
for the Degree of Master of Science
in Orthodontics

By

Akram F. AL-Huwaizi
(B.D.S.)

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بسم الله الرحمن الرحيم

﴿وقل ربي زدني علما﴾

صدق الله العظيم

DECLARATION

This is to certify that the organization and preparation of this thesis have been made by the graduate student "**Akram F. Al-Huwaizi**" under my supervision, in the University of Baghdad as a partial fulfillment for the requirements of the degree of Master of Science in Orthodontics.

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DEDICATION

TO MY

PARENTS
AND
BROTHERS

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First of all, I thank **God** Almighty for granting me the will and strength with which this research was accomplished and I pray that His blessings upon me may continue throughout my life.

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Akram F. Al-Huwaizi

LIST OF ABBREVIATIONS

%	: Percentage.
Ai	: Anamnestic dysfunction index.
Ai0,I,II	: Symptom-free, mild symptoms, severe symptoms.
ant.	: Anterior.
bi.	: Bilateral.
class II 1	: Class II, division 1, malocclusion.
class II 2	: Class II, division 2, malocclusion.
crowd.	: Crowding.
d.f.	: Degrees of freedom.
Di	: Clinical dysfunction index.
Di0,I,II,III	: Sign-free, mild signs, moderate signs, severe signs.
E.	: Examination.
exce.	: Excessive.
F	: Females.
FDI	: Federation Dentaire Internationale.
forw. mand. displ.	: Forward mandibular displacement.
I.	: Interview.
inv. inc.	: Inverted incisors.
M	: Males.
MA	: Medline abstract.
Mi	: Mobility index.
mm	: Millimeters.
n	: Number.
N.S.	: Non-significant.
Q.	: Questionnaire.
spac.	: Spacing.
Symp.	: Symptoms.
T.	: Telephone interview.
TMA	: Translated Medline abstract.
TMD(s)	: Temporomandibular disorder(s).
TMJ(s)	: Temporomandibular joint(s).
uni.	: Unilaterally.
yr.	: Year.

ABSTRACT

Temporomandibular disorders (TMDs) were investigated in 143 pretreatment orthodontic patients (43 males and 102 females) whose age ranged between 10-25 years at the College of Dentistry, University of Baghdad, Iraq. The study was undertaken to elucidate the prevalence and severity of TMDs in malocclusion patients and to define the relationships between malocclusion and TMDs.

The clinical signs and subjective symptoms were recorded according to the principles introduced by *Helkimo (1974b)*. Subjective symptoms were reported by 65.7% of the patients with 22.4% described as severe, and the most common symptoms were TMJ sounds and feeling of fatigue. Clinical signs were observed in 81.8% of the sample with 22.4 and 6.3% described as moderate and severe, respectively, and the most common signs were muscle and TMJ tenderness to palpation. Significant sex differences were few and weak. However, tenderness to palpation decreased with age and dysfunction increased with age.

Recurrent headache was reported by 38.5% of the sample, significantly more by females than males. Oral parafunctions were found in 78.3% of the patients, with females significantly more aware of orofacial parafunctions than males. Dental wear was observed in nearly all the patients increasing in severity significantly with age for all dental regions.

Class II malocclusion, both divisions 1 and 2, were unrelated to TMDs, while an overjet greater than 8 mm and an overbite of 5 mm or more predisposed to TMDs. True class III malocclusion and reversed overjet were associated with TMDs, while postural class III malocclusion, forward mandibular displacement and open bite were not.

Inverted incisors and posterior crossbite were positively associated with TMDs signs, especially bilateral posterior crossbite. Upper anterior crowding appeared to predispose to TMDs, while lower anterior crowding, upper and lower anterior spacing were negatively associated with TMDs.

The results of this study show that TMDs are more prevalent in orthodontic patients than in general population indicating the adverse effect of malocclusion on the function of the masticatory system; and that the incisor relationship is more important than the general occlusion (Angle's classification) in predisposing to TMDs.

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CHAPTER ONE

INTRODUCTION

The term temporomandibular disorders (TMDs) refers to a cluster of disorders characterized by: pain in the preauricular area, TMJ, or the muscles of mastication; limitations or deviations in mandibular range of motion; and noises in the TMJs during mandibular function *(Rugh, 1983; Dworkin et al., 1990)*. It is a multifactorial disorder involving physical, psychological, emotional, social and local factors. Abnormalities of occlusion are included in the local factors *(Ai & Yamashita, 1992)*.

Descriptive surveys have established that TMDs are prevalent in all ages and gender with varying signs and symptoms and varying degrees of pain and/or abnormality *(Nassif & Hilsen, 1992)*. Clinical observations indicate that these disturbances are caused mainly by malocclusion, iatrogenic factors, and increased psycho-emotional tension *(Wigdorowicz-Makowerowa et al., 1979)*.

Analysis of the occlusion and function of the masticatory system have been suggested as a part of a thorough examination of the patient. This is especially important when signs and symptoms of functional disturbances are present *(Nassif & Hilsen, 1992; Nowlin & Nowlin, 1995)*.

Epidemiological studies of TMDs often have not been conducted systematically and have used widely different illness criteria and research designs, making them difficult to compare *(Dworkin et al., 1990)*. In order to assess the severity of the symptoms in a particular patient a numerical quantification is necessary *(Zarb & Speck, 1979)*. *Helkimo (1974b)* designed indices that collect data and classify them according to a numerical system permitting assessment of the prevalence of different symptoms and their severity by applying standardized epidemiological methods to the subjective and objective evaluation of TMDs. This would facilitate comparison between different studies *(Helkimo, 1979)*.

Although it is now generally agreed that the etiology of TMDs is multifactorial, malocclusion has been one of the most frequently cited causes of TMDs *(Moss & Garrett, 1984; Tosa et al, 1990; Motegi et al., 1992)*.

In Iraq, epidemiological studies have revealed the prevalences of TMDs and malocclusion among selected age groups of the population, but the association between the two remains unclear. Worldwide, many investigations on the relationship between malocclusion and TMDs have been carried out in the last two decades among selected population groups or orthodontic patients, but their results were equivocal and often contradictory.

Hence, it was decided to investigate the assumption that malocclusion predisposes to TMDs, by examining the relationships between the different malocclusion variables on one hand and the signs and symptoms of TMDs and some related factors on the other hand, among pre-treatment orthodontic patients were malocclusion is more prominent than in normal population and so correlations with TMDs may be clearer.

CHAPTER TWO

REVIEW OF LITERATURE

2.1 TEMPOROMANDIBULAR DISORDERS:

TMDs are a cluster of related disorders in the masticatory system with many common features. They encompass a wide range of clinical conditions, often overlapping that may involve the TMJ or the neuromuscular system associated with mandibular function *(Zarb & Carlsson, 1988a; Dworkin et al., 1990)*.

2.1.1 TERMINOLOGY:

Since *Costen (1934)* gave his name to the symptoms related to the dysfunction of the TMJ, a plethora of terms has been introduced in the literature. When Costen's theory was proven inaccurate by *Sicher (1948)*, the term "Costen-syndrome" has been most often replaced by "TMJ disturbances" and "TMJ dysfunction syndrome" *(Shore, 1959; Lupton, 1969)* and "functional TMJ disturbances" *(Olsson, 1969; Ramfjord & Ash, 1971)*.

Some stressed pain, such as "pain-dysfunction syndrome" *(Voss, 1964)*, "myofascial pain-dysfunction syndrome" *(Laskin, 1969)*, and "TMJ pain-dysfunction syndrome" *(Schwartz, 1959; Bell, 1969)*.

Since the symptoms are not always isolated to the TMJ, some authors believe that the foregoing are too limited and that a broader more collective term should be used, such as "craniomandibular disorders" *(McNeill et al., 1980)*. *Bell (1982)* suggested the term "temporomandibular disorders", which has gained popularity. It does not merely suggest problems that are isolated to the joints but includes all disturbances associated with the function of the masticatory system.

The wide variety of terms used are still confusing and contribute to the controversy about the etiology, therefore, the American Dental Association *(Laskin et al., 1983)* adopted the term temporomandibular disorders (TMDs), and have since been adopted by several authors.

2.1.2 ETIOLOGY:

Scientific evidence to support a primary etiologic factor in TMDs is absent *(Helkimo, 1979)*. Consequently, there is very little agreement as to the etiology of TMDs, and various theories have been proposed in the dental literature. These theories listed in chronological order are:

A- Mechanical displacement theory:

Prentiss (1918), Monson (1920), Decker (1925), and later *Costen (1934)* stressed the idea that distal condylar displacement after loss of posterior teeth led to condylar impingement on the auriculotemporal nerve, or direct pressure on the ear structures and the eustachian tube.

Zimmerman (1951) and *Sicher (1955)* have pointed out that although direct condylar pressure on the auriculo-temporal nerve itself is not likely, the pain can come from sensitive soft tissue posterior to the condyle due to posterior condylar displacement.

Several clinicians have extended the idea of distal mechanical displacement of the condyle to include both frontal and sagittal changes in condylar position *(Gerber, 1971; Weinberg, 1973; Kundert & Palla, 1977)*. The etiologic factors causing the deviation of the condyles out of their normal centric position have been claimed to be loss of molars and premolars, insufficient occlusal contacts in the molar region, premature contacts and occlusal interferences *(Zarb & Mohl, 1988)*.

B- Neuromuscular theory:

Any type of occlusal interference can cause parafunctions such as grinding or clenching; however, a background of psychic tension, stress or anxiety is an adjunctive necessary etiologic factor. These parafunctions cause muscle spasm and pain in joints and muscles *(Ramfjord, 1961; Olsson & Krogh-Poulsen, 1966)*. Hence, functional disharmony between the dental occlusion and the TMJs is considered by many clinicians as the most acceptable etiologic factor in TMD patients *(DeBoever, 1979)*.

C- Muscle theory:

This theory claims that the primary etiologic factor lies in the muscles of mastication themselves. *Kraus (1963)* described a "hypokinetic disease", which he attributed to the imbalance between a lack of adequate muscle exercise and over stimulation of daily life in this century. He identifies TMDs as only one such disease that can involve the jaws, head and neck. He claimed that under stress, the muscles of the jaw never relax; therefore, tension will increase until a painful spasm occurs. These patients manifest general muscle response in addition to specific ones *(Schwartz, 1959)*.

D- Psychophysiological theory:

It supports an opposite opinion to the one proposed in the neuromuscular theory; that is, it maintains that emotional disturbances lead to grinding of teeth which in turn may lead to occlusal interferences. These interferences may then act as sustaining factors *(Franks, 1964; Laskin, 1969)*. Support for this theory comes from the demonstration of *Yemm (1979)* that hyperactivity can be centrally initiated and provoked by everyday psychological and social difficulties; also patients can benefit from reassurance *(Franks, 1964)*, counseling, placebo drugs and splints *(Green & Laskin, 1972*).

However, the etiology of TMDs is increasingly accepted as multifactorial with both local or peripheral and central factors being considered of importance *(DeBoever, 1979; Mohlin et al., 1980; Wanman & Agerberg, 1986c; Egermark-Eriksson et al., 1987; Ai & Yamashita, 1992; Verdonck et al., 1994; Moss et al., 1995)*.

Recently, *Parker (1990)* proposed a dynamic model of etiology of TMDs; which he claimed was consistent with both the neuromuscular and the psychophysiological theories because it holds muscle hyperfunction to be central to the pathological process, and it identifies stress and occlusion as contributing factors. The model can, also, accommodate a broad spectrum of multifactorial concepts.

2.1.3 SYMPTOMATOLOGY:

Several studies have reported symptoms distribution in populations of TMD patients but the main definitional symptoms which have general agreement are the triad: pain and tenderness of the muscles of mastication and the TMJs, sounds during condylar movement, and limitation of mandibular movements accompanied occasionally with deviation of the mandible from the normal path of closure *(Perry, 1968; Butler et al., 1975; Brooke & Stenn, 1978; Kaye et al., 1979a; Zarb & Speck, 1979; Geissler, 1985; van de Laan et al., 1988; Zarb & Carlsson, 1988b; Greenberg, 1990; McNeill et al., 1990)*.

Some investigators have used broader definitions and have included tooth wear, occlusal stability and centric relation discrepancies as possible related indicants of TMDs *(Heloe & Heloe, 1979; Solberg et al., 1979)*. Recurrent headache, also, has been considered as a definitional symptom *(Magnusson & Carlsson, 1978)*. Symptoms in the ear, tinnitis and vertigo have also been mentioned *(Myrhaug, 1969 a&b; Derksen, 1970; Parker & Chole, 1995)*.

A- Pain:

The most common complaint of patients appears to be pain *(Green et al., 1969; Bell, 1986; Okeson, 1989)*, and is the most disturbing factor *(DeBoever, 1979)* and the most common cause for patient concern and consultation *(Perry & Marsh, 1977)*.It is usually aggravated by chewing or other jaw functions *(McNeill et al., 1990)*.

Clinical descriptions of the reported pain vary considerably ranging from dullache to sharp and acute *(Meklas, 1971; DeBoever, 1979)*, most often reported to be unilateral *(Perry, 1968; Laskin, 1969; Christensen, 1981)* although bilateral pain is very common *(Weinberg, 1980)*. The locations of the pain may range from the back of the head and neck posteriorly, to the temporal area superiorly, and to the angle of the jaw anteriorly *(Moss & Garrett, 1984)* with the most frequently cited pain location being the area in front of the ear *(Bell, 1969; Scott, 1980)*.

Perry (1957) reported that pain is minimal in the morning and progressively intensifies during the course of the day, while *Laskin (1969)* found that for most patients, pain is most intense in the morning (suggesting nocturnal bruxing).

B- Masticatory muscle tenderness:

Tenderness of the muscles of mastication as well as related muscles in the head and neck is one of the most common clinical signs of TMDs which is usually not reported by the patient, and almost always elicited by digital palpation of the examiner *(Bush, 1985; Padamsee et al., 1985a)*.

Tenderness to palpation is mostly attributed to muscle spasm *(Laskin, 1969)*. It is nevertheless not always possible to record electromyographically an increased activity of these muscles *(Yemm, 1971 a&b)*. These tender muscles are recognized as "hot spots" *(Berry & Yemm, 1974)* and may be caused by areas of small hemorrhages or torn fibers.

C- Joint sounds:

These are noises that originate from the joint during various mandibular movements, and are of two general types: clicking and crepitation. Joint sounds are very common complaints cited in every report on TMDs *(Okeson, 1989)*, and are significantly more frequent in TMD patients than in the population *(Dworkin et al., 1990)*.

Clicking is the most frequent reported sound in both patients and population samples *(Green & Laskin, 1988)*. TMJ crepitation is not as common as clicking sounds in population *(Agerberg & Carlsson, 1972)* and is often unilateral, while clicking is often bilateral *(Ingervall et al., 1980; Hansson, 1986)*.

The etiology of clicking is not clearly understood but suggested etiologic factors include uncoordinated muscle function of the lower and upper part of the lateral pterygoid muscles, posterior and anterior position of the articular disk, and irregularities of the components of the joint *(Wanman & Agerberg, 1990)*.

D- Restricted mandibular movements:

This could be classified into two categories: restricted mouth opening (trismus) and limited lateral movement, and deviations during mandibular movements *(Moss & Garrett, 1984)*. Almost all reports agree that limitation and/or deviation of the mandible are very common *(Perry, 1968; Okeson, 1985)*.

The limitations of the movements are due to muscle spasm; however, structural changes as limiting factors can occur but they appear to be less frequent *(DeBoever, 1979)*. Estimation of the limitation of mandibular movements was made from measurement of maximal opening of the mouth, maximal lateral movements and maximal protrusion *(Helkimo, 1974b)*. Decreased lateral movement to one side often reflects a disharmony of the contralateral joint *(Padamsee et al., 1985a)*.

Deviation of the mandible is towards the affected side, and a forced deviation to the non-affected side is painful *(Agerberg, 1974; Padamsee et al., 1985a)*. Deviation of the mandible on opening is usually secondary to muscle spasm or rarely in children or adolescents due to displacement of the meniscus disk *(Pillemer et al., 1987)*.

2.1.4 EPIDEMIOLOGY:

Many epidemiological studies have been carried out either among general population or among certain age groups to detect the prevalence of TMDs, as shown in appendices I to IV.

2.1.4.1 PREVALENCE ACCORDING TO AGE:

A- Studies on general populations:

From the available epidemiological studies (appendix I) it is clear that the signs and symptoms of TMDs are common in general populations; 12-64 % for the symptoms and 20-88 % for the signs among Swedish, Finish, Norwegian, Canadian, Hungarian, Indian, and Iraqi populations.

The symptoms of TMDs have been shown to have no correlation with age *(Swanljung & Rantanen, 1979; Szentpetery et al., 1986; Salih, 1993)*, except for one study which showed that persons aged 44 years and under were more likely to report one or more symptoms than in the younger age groups *(Locker & Slade, 1988)*. Many epidemiological studies have shown an increase of the signs of TMDs with age *(Swanljung & Rantanen, 1979; Szentpetery et al., 1986; Tervonen & Knuuttila, 1988)*, while *Salih (1993)* reported no such increase.

B- Studies on old adults:

The prevalences of subjective symptoms ranged from 60 to 74% and for the clinical signs ranged from 23 to 59% (appendix II).

Heloe and Heloe (1978) showed that the frequency of subjective symptoms tended to increase with age among 65-79 year old adults.

C- Studies on young adults:

Appendix III shows that the prevalence of TMDs in young adults is generally lower than that in elderly subjects, as the prevalence of the symptoms of TMDs was 12-67% and the prevalence of the signs was 28-92%.

Ingervall and Hedegard (1974) and Molin et al. (1976) reported low prevalences of TMDs among Swedish inductees. On the other hand, *Solberg et al. (1979)* reported a higher prevalence of TMDs among American University students and he attributed this variation to sex, geographical and cultural factors.

Military and medical Polish student showed a significantly higher prevalence of TMDs than young soldiers of the same age, with the medical students being more affected than the military students *(Wigdorowicz-Makowerowa et al., 1979)*. They attributed these differences to the influence of environment, particularly the type of work performed and responsibility at work.

Several studies assessed the severity of the signs and symptoms of TMDs according to the anamnestic (Ai) and clinical (Di) dysfunction indices of *Helkimo (1974b)* and reported different findings. Mild and severe symptoms were observed in 17 & 15% *(Droukas et al., 1984)*, 14 & 1% *(Pullinger et al., 1988a)*, 23 & 34% *(Schiffman et al., 1990)*, 36 & 18% *(Abdulla, 1992)*, and 30 & 6%, respectively *(Nourallah & Johansson, 1995)*; whereas mild, moderate and severe signs were recorded in 52, 10 & 2% *(Droukas et al., 1984)*, 41, 17 & 1% *(Pullinger et al., 1988a)*, 34, 33 & 26% *(Schiffman et al., 1990)*, 39, 16 & 6% *(Abdulla, 1992)*, and 33, 3 & 1%, respectively *(Nourallah & Johansson, 1995)*.

D- Studies on adolescents and children:

Epidemiological studies on adolescents and children have shown that TMDs are also common in this age group and their prevalence ranged from 0.6% to 74% for the symptoms, and from 4% to 77% for the signs (appendix IV).

The low prevalence of TMDs among Japanese students documented by *Ogura et al. (1985), Ohno et al. (1988) and Motegi et al. (1992)* if compared with those of Swedish and Polish subjects of corresponding age groups was attributed to cultural factors.

The prevalence of the signs and symptoms of TMDs was shown to increase with age by most investigators *(Grosfeld & Czarnecka, 1977; Egermark-Eriksson et al., 1981; Nilner, 1983c; Gazit et al., 1984; Ogura et al., 1985; Motegi et al., 1992)*, except *Ohno et al. (1988)* who demonstrated a decrease in the prevalence of TMDs symptoms among 10-18 year old Japanese children.

Helkimo's indices (Ai & Di) were used by some investigators to assess the severity of the signs and symptoms of TMDs and they reported that the prevalence of mild and severe symptoms were 13 & 7% *(Wanman & Agerberg, 1986a)*, and 15 & 40%, respectively *(Shereef, 1991)*; and the prevalence of mild, moderate and severe signs was found to be 38, 7 & 1% *(Egermark-Eriksson et al., 1983)*, 49, 17 & 0% among 15 year olds *(Magnusson et al., 1985)*, 42, 14 & 0% *(Wanman & Agerberg, 1986b)*; 34, 22 & 2% *(Kononen et al., 1987)*, and 26, 5 & 1%, respectively *(Shereef, 1991)*.

Longitudinal studies on adolescents and children showed that the signs and symptoms fluctuate with age (appendix IV). A significant increase of the prevalence of TMDs symptoms was registered by *Magnusson et al. (1985)* from 7 to 11 years of age while *Wanman and Agerberg (1986d) and Heikinheimo et al. (1989)* found no such increase. *Magnusson et al.(1985) and Pilley et al.(1992)* found a significant increase of the prevalence of the TMDs signs with age in contrast to *(Wanman & Agerberg, 1986e)*.

In conclusion, the signs and symptoms are relatively uncommon in very young children but increase gradually with age until adolescence where their prevalence approximates that of adults.

2.1.4.2 PREVALENCE ACCORDING TO SEX:

Many clinical studies showed that females outnumbered males several folds in the population seeking TMJ treatment *(Kaye et al., 1979 a&b; Harkins & Marteney, 1985; Al-Hasson et al., 1986; Gross et al., 1988; Lee & Lee, 1989; Parker & Chole, 1995)*. This sex difference was attributed to females being less adaptable to the factors leading to hyperfunction or they are less adaptable to its effects. This decreased adaptability in females may be because of structural differences, females having more stressful life events and having more depression than males, females being more sensitive to pain than males, or because of the presence of oestrogen receptors in the TMJ of females and their absence in male as cited in experiments on baboons *(Parker, 1990)*.

Many epidemiological studies on non-patients have revealed non-significant sex difference in relation to symptoms of TMDs among young adults *(Molin et al., 1976; Solberg et al., 1979; Waltimo & Kononen, 1995)*,and adolescents and children *(Grosfeld & Czarnecka, 1977; Egermark-Eriksson et al., 1981; Nilner & Kopp, 1983; Magnusson et al., 1985; Bernal & Tsamtsouris, 1986; Wanman & Agerberg, 1986a; Kononen et al., 1987; Ohno et al., 1988; Vanderas, 1988 ; Abdulla, 1992)*. Few investigators reported that symptoms were significantly more common in females *(Wanman & Agerberg, 1986d; Heikinheimo et al., 1989; Shereef, 1991; Salih, 1993)*, but the differences were smaller than those reported in clinical studies.

A significant female preponderance in relation to the signs of TMDs was recorded among young adults *(Solberg et al., 1979; Grosfeld et al., 1985; Pullinger et al., 1988a; Abdulla, 1992; Waltimo &* Kononen, *1995)*, and adolescents and children *(Grosfeld et al., 1985; Wanman & Agerberg, 1986 b&e; Shereef, 1991)*. Other investigators documented a non-significant sex difference *(Egermark-Eriksson et al., 1981; Nilner & Kopp, 1983; Gazit et al., 1984; Magnusson et al., 1985; Ogura et al., 1985; Bernal & Tsamtsouris, 1986;*

Kononen et al., 1987; Vanderas, 1988 ; Huber & Hall, 1990; al-Hadi, 1993); whereas *Rao and Rao (1981)* reported that the incidence of TMDs was higher in males than in females.

Heloe and Heloe (1975) attributed the overrepresentation of females in clinical matter to inequalities in demand as women are more prone to seek advice and treatment.

2.1.4.3 THE MOST PREVALENT SYMPTOMS:

TMJ sounds was reported in several studies as the most frequent symptom among young adults *(Molin et al., 1976; Heloe & Heloe, 1979; Solberg et al., 1979; Ogura et al., 1985;Droukas et al., 1984; Schiffman et al., 1990; Abdulla, 1992)*, and adolescents and children *(Nilner, 1981; Nilner & Lassing, 1981; Wanman & Agerberg, 1986a; Heikinheimo et al., 1989; Shereef, 1991)*. Pain or tiredness when chewing was reported as the most common symptom by other investigators *(Egermark-Eriksson et al., 1981; Magnusson et al., 1985; Kononen et al., 1987; Widmalm et al., 1995a)* and fatigue or stiffness in the masticatory muscles was reported by *Pilley et al. (1992)* as the most frequent symptom.

Some studies reported headache as the most prevalent symptom when included with the symptoms of TMDs *(Heloe & Heloe, 1979; Solberg et al., 1979; Nilner & Lassing, 1981; Bush et al., 1982)*.

The skewed sex distribution of various symptoms of TMDs have been shown in several studies. Females complained more often than males of tiredness of the jaws *(Wanman & Agerberg, 1986a)*; TMJ sounds and facial pain *(Wanman & Agerberg, 1986d)*; feeling of fatigue, locking of the jaw, facial and/or jaw pain, pain and/or tiredness on chewing *(Shereef, 1991)*; pain on movement of the mandible *(Abdulla, 1992)*; locking or luxation, clicking and pain around the TMJ

(Pilley et al., 1992); TMJ sounds, difficulty in opening the mouth widely and locking of the jaw *(Salih, 1993)*; TMJ sounds and pain during chewing *(Widmalm et al., 1995a)*.

Other studies showed a non-significant sex difference with regard to different symptoms of TMDs *(Solberg et al., 1979; Egermark-Eriksson et al., 1981; Nilner, 1983c; Kononen et al., 1987; Heikinheimo et al., 1989)*.

2.1.4.4 THE MOST PREVALENT SIGNS:

Many epidemiological studies showed tenderness of the masticatory muscles to palpation was the most prevalent sign among young adults *(Molin et al., 1976; Solberg et al., 1979; Droukas et al., 1984; Grosfeld et al., 1985; Pullinger et al., 1988a; Schiffman et al., 1990; Abdulla, 1992)*, and adolescents and children *(Grosfeld & Czarnecka, 1977; Egermark-Eriksson et al., 1981; Nilner, 1981; Nilner & Lassing, 1981; Wanman & Agerberg, 1986b; Kononen et al., 1987; Nielsen et al., 1989; Pilley et al., 1992)*.

The most common localization of masticatory muscle tenderness was the lateral pterygoid and insertion of temporalis muscles *(Molin et al., 1976; Wanman & Agerberg, 1986b; Kononen et al., 1987; Dworkin et al., 1990; Schiffman et al., 1990; Pilley et al., 1992)*. The lateral pterygoid was found to be the most frequently tender muscle in many other studies *(Solberg et al., 1979; Egermark-Eriksson et al., 1981; Droukas et al., 1984; Grosfeld et al., 1985; Pullinger et al., 1988a; Mohlin et al., 1991; Abdulla, 1992; Wadhwa, 1993). Shereef (1991) and Salih (1993)* found that the anterior temporalis and masseter muscles were the most commonly tender muscles.

TMJ sounds, especially clicking, was reported by many other studies as the most frequent sign *(Ogura et al., 1985; Wanman & Agerberg, 1986e; Shereef, 1991, Motegi et al., 1992; al-Hadi, 1993; Wadhwa, 1993; Verdonck et al., 1994)*.

The skewed sex distribution of masticatory muscle tenderness and TMJ sounds has been shown by several epidemiological studies in which females were overrepresented *(Solberg et al., 1979; Magnusson, 1986; Wanman & Agerberg, 1986 b&e; Pullinger et al., 1988a)*. Whereas, non-significant differences were found between both sexes among subjects examined by other investigators *(Nilner, 1983c; Gazit et al., 1984; Ogura et al., 1985; Kononen et al, 1987; Abdulla, 1992; Keeling et al., 1994)*.

Shereef (1991) and Salih (1993) found no sex difference in relation to TMJ sounds, while muscle tenderness was more frequent among girls, while *Egermark-Eriksson et al. (1981) and Pilley et al. (1992)* found a significant sex difference in relation to TMJ sounds and not muscle tenderness.

The anatomy of the temporomandibular joints and muscles of mastication is shown in appendicies VIII and IX.

2.1.5 RELATED FACTORS:

2.1.5.1 RECURRENT HEADACHE:

Headache is a term which literally describes pain felt any where in the head *(Macleod et al., 1987)*. Headache is a very common subjective symptom that can have various origins *(Magnusson & Carlsson, 1978a; Ash, 1986)*. Muscular contraction or tension headache is, however, considered to be the major type of headache *(Gelb & Tarte, 1975)*. *Diamond and Baltes (1973)* tabulated that 90% of all headaches are the result of muscle contraction directly related to anxiety, depression and stress.

Since TMDs is also attributed to increased muscle tension, one would expect a high incidence of headaches in this population of patients *(Kaye et al., 1979b)*. Indeed, many chronic headaches may well be referred pain from the muscles of mastication and as such symptomatic of TMDs *(Butler et al., 1975; Ash, 1986)*.

Headache was found to be a common complaint in TMJ patients *(Solberg, 1986; Agerberg, 1987)*, both in adults *(Magnusson & Carlsson, 1978 & 1980)* and in adolescents and children *(Padamsee et al., 1985)* and it can be alleviated by treatment of TMDs *(Agerberg & Carlsson, 1974)*. Thus, recently the International Headache Society included TMDs in their classification of headache *(McNeill et al., 1990)*.

Prevelance: Epidemiological studies (appendix V) have revealed prevalences of 5-24% among population and 1-30% among adults, adolescent and children.

The prevalence of recurrent headache among the Iraqi population has been shown to be 33% among adolescents *(Shereef, 1991)*, 41% among University students *(Abdulla, 1992)*, and 38% among workers *(Salih, 1993)*.

Sex distribution: Many epidemiological studies reported recurrent headache to be more common in females than in males *(Heloe & Heloe, 1979; Solberg et al., 1979; Egermark-Eriksson et al., 1981; Nilner & Lassing, 1981; Bush et al., 1982; Magnusson et al., 1985; Wanman & Agerberg, 1986a; Pullinger et al., 1988a; Heikinheimo et al., 1989; Shereef, 1991; Abdulla, 1992; Pilley et al., 1992; Widmalm et al., 1995b).*

Other studies reported a non-significant sex difference in the prevalence of recurrent headache *(Nilner, 1981; Helm et al., 1984; Kononen et al., 1987; Salih, 1993).*

Age distribution: Recurrent headache was found to increase in frequency with age among children *(Nilner & Lassing, 1981; Egermark-Eriksson, 1982; Pilley et al., 1992).*

Oster (1972), among 2000 Danish children observed that the prevalence of headache increased up to the age of 12 and then decreased. *Magnusson et al. (1985),* also, reported a significant increase of recurrent headache from 7 to 11 years of age but not from 11 to 15. However, *Heikinheimo et al. (1989)* found that recurrent headache increased in girls and decreased in boys from 12 to 15 years of age; and *Wanman & Agerberg (1986a)* found no increase in headache from 17 to 19 years of age.

Relation with TMDs: Several epidemiological studies revealed a substantial increase in the frequency of recurrent headache along with increasing severity of the signs and symptoms of TMDs *(Wanman & Agerberg, 1986f; Shereef, 1991; Abdulla, 1992; Salih, 1993).*

A significant association was reported between muscle tenderness to palpation and recurrent headache *(Molin et al., 1976; Solberg et al., 1979; Nilner,* *1983 a&b; Mongini et al., 1988; Shereef, 1991; Abdulla, 1992; Salih, 1993)* which supports the clinical report of *Magnusson and Carlsson (1978)*.

The positive association between recurrent headache and TMDs was also reported by *Geering-Gaerny and Rakosi (1971), Heloe and Heloe (1979), Egermark-Eriksson (1982), Magnusson et al. (1985), and Mohlin et al. (1991).*

Magnusson et al.(1985) reported a correlation between recurrent headache and the subjective symptoms of TMDs, but not with the clinical dysfunction index (Di).

2.1.5.2 ORAL PARAFUNCTIONS:

Masticatory muscles are commonly active during non-functional periods, since this activity is not associated with chewing, swallowing or speech, it is by definition parafunctional *(Okeson, 1989)*.

Oral parafunctions (oral habits) are stereotypic repetitive functions of the masticatory system, often subconscious, differing qualitatively and quantitatively from its physiologic function *(Wigdorowicz-Makowerowa et al., 1979)*. The following types may be distinguished: cheek-, tongue-, and lip-biting, finger and thumb sucking, unusual postural habits, unilateral chewing, many occupational related activities such as biting on pencils, pins, or nails or even holding objects under the chin, and continuous gum chewing *(Scharer, 1974; Okeson, 1989)*.

Although the etiology of parafunctional activities seems to be multifactorial *(Rugh & Solberg, 1979; Ash, 1986; Cash, 1988)*, two factors were said to increase parafunctional activity, malocclusion and emotional stress *(Lindqvist, 1974; Arnold, 1981; Mejias & Mehta, 1982)*.

The signs and symptoms of parafunctions could be seen in adults, adolescents and children. These manifestations depend on: *(1)* frequency of the habit, *(2)* the intensity with which the subject is doing the habit, and *(3)* the age of the subject which may be associated with the duration of the habit *(Ahmad, 1986; Cash, 1988)*.

Another type of parafunction is known as bruxism which is non-functional movements of the mandible with or without audible sounds occurring during the day or night *(Vanderas & Manetas, 1995)*. It is the most problematic parafunctional behaviour *(Rugh & Ohrbach, 1988; Ramer, 1990)*. However, the etiologic theories of nocturnal bruxism have been classified as occlusal, psychological and systemic causing increased muscle tonus leading to non-functional grinding and clenching *(Kydd & Daly, 1985; Rugh & Ohrbach, 1988; Ramer, 1990)*.

Grinding often occurs during sleeping and in the day time clenching of the teeth is much more common than grinding *(Agerberg & Carlsson, 1972; Ramfjord & Ash, 1983)*.

The amount of force in parafunctional activity (nocturnal bruxism) can reach *57,600* lb-second per day, while in functional activity it is *17,200* lb-second per day during chewing and swallowing *(Okeson, 1989)*.

It is difficult to establish whether a patient is bruxing or not since even patients with severe parafunctions are not aware of their habit and become conscious of it only after prolonged self observation *(Graf, 1969; Okeson, 1985)*.

Prevelance: The prevalence of one or more parafunctions was reported to be 26-50% among general population, 31-57% among young adults, 46-78% among adolescents and children (appendix VI) and 40% among 3-5 year old children *(Bernal & Tsamtsouris, 1986)*.

Different methods have been employed to assess the existence of bruxism. Epidemiological studies in which interview or questionnaire were used alone to examine the existence of bruxism have revealed prevalences of 5-39% among adolescents and children, which is quite comparable to 5- 34% among adults (appendix VI); whereas studies using clinical examination of bruxofacets have revealed prevalences up to 69% *(Wigdorowicz-Makowerowa et al., 1979)* and studies using electromyography or study casts have revealed prevalences of 91-100% *(Clark et al., 1981; Rugh et al., 1984; Kydd & Daley, 1985; Seligman et al., 1988)*.

***Sex distribution**:* Some studies reported a non-significant sex difference regarding the prevalence of oral parafunctions *(Egermark-Eriksson et al., 1981; Magnusson et al., 1985; Kononen et al., 1987; Abdulla, 1992; Pilley et al., 1992)*, while *Wanman and Agerberg (1986a), Shereef (1991), Salih (1993), and Widmalm et al. (1995)* showed that females reported oral parafunctions significantly more than males, and other studies reported that male were more aware of bruxism *(Swanljung & Rantanen, 1979; Nilner & Lassing, 1981; Helm et al., 1984)* and nail biting *(Nilner, 1981)* than females.

In Iraqi studies significant sex differences with overrepresentation of females were registered in relation to clenching, object-, lip-, and cheek-biting *(Shereef, 1991)*, gum chewing *(Abdulla, 1992)*, and clenching, nail biting and gum chewing *(Salih, 1993)*. In all the three studies females reported one or more orofacial parafunctions more frequently than males.

***Age distribution**:* It is clear from the results of the epidemiological studies shown in appendix VI that adolescents and children are more aware of orofacial parafunctions than adults.

Wigdorowicz-Makowerowa et al. (1979), Gazit et al. (1984), and Wanman and Agerberg (1986d) found that the prevalence of orofacial parafunctions declined with age, while other studies reported no such difference among the same age range *(Egermark-Eriksson et al., 1980; Nilner & Lassing, 1981; Egermark-Eriksson, 1982; Kononen et al., 1987).*

Orofacial parafunctions were found to increase in prevalence from 7 to 11 years, but not from 11 to 15 years of age, while bruxism did not change significantly with age *(Egermark-Eriksson et al., 1981; Magnusson et al., 1985).*

Reding et al. (1966) and Heikinheimo et al. (1989) documented that the prevalence of bruxism significantly declined with age among children, and *Nilner and Kopp (1983)* attributed the higher frequency of bruxism among the younger age group (7-14 years) of her sample to the mixed dentition stage, whereas, *Wanman and Agerberg (1986d) and Pilley et al. (1992)* found a significant increase of clenching with age.

Relation with TMDs: Some epidemiological studies found a non-significant relationship between oral parafunctions and TMDs (Bernal &, Tsamtsouris, 1986; Wanman & Agerberg, 1986c; Schiffman et al., 1990; Moss et al., 1995; Vanderas & Manetas, 1995; Widmalm et al., 1995b); whereas Nilner (1983 a&b) observed a positive correlation between oral parafunctions and muscle tenderness, and Nilner and Lassing (1981) and Magnusson et al. (1985) found a significant correlation between oral parafunctions and the subjective symptoms of TMDs, whilst no correlation with the clinical signs was registered.

The number of various oral parafunctions was correlated with the anamnestic dysfunction index *(Wanman & Agerberg, 1986c; Shereef, 1991; Abdulla, 1992; Salih, 1993)* and with the clinical dysfunction index *(Kononen et al., 1987; Shereef, 1991; Abdulla, 1992; Salih, 1993).*

Many investigators *(Wigdorowicz-Makowerowa et al., 1979; Solberg et al., 1979; Egermark-Eriksson et al., 1981; Droukas et al., 1984; Shereef, 1991;*

Abdulla, 1992; Salih, 1993) found a significant relationship between bruxism and the signs of TMDs, and *Nilner (1983 a&b)* reported correlations between bruxism and muscle tenderness.

Several investigators documented the role of bruxism in recurrent headache *(Molin et al., 1976; Solberg et al., 1979; Nilner, 1983 a&b; Wanman & Agerberg, 1986f)*. Others reported a significant relationship between oral parafunctions, recurrent headache and muscle tenderness *(Shereef, 1991; Abdulla, 1992)*.

2.1.5.3 DENTAL WEAR:

It is said that extensive tooth wear in modern humans is indicative of parafunctional activities, mainly bruxism *(Carlsson & Ingervall, 1988)*; as the modern diet does not contain enough abrasive foods to wear the teeth *(Okeson, 1989)*, and most tooth wear results from eccentric tooth contacts, i.e. it created by bruxing type of movement *(Zarb & Carlsson, 1988a)*.

Tooth wear can be a very destructive process and eventually lead to functional problems. For the most part, however, it is normally asymptomatic and therefore perhaps the most tolerated form of breakdown in the masticatory system *(Okeson, 1989)*.

Dental wear examination has been recommended for routine TMDs examination *(Zarb & Carlsson, 1988a; Nassif & Hilsen, 1992)*; as occlusal or incisal attrition patterns that do not conform to or coincide with normal masticatory or swallowing wear patterns are perhaps the most significant dental sign of bruxism *(Ramfjord & Ash, 1983; Okeson, 1989)*.

Prevalence and severity*: Okeson (1989)* stated that "tooth wear is by far the most common sign of breakdown in the dentition and is probably seen more often than any other functional disturbance in the masticatory system."

Okeson and Kemper (1982) observed that 95% of 168 dental patients had some form of tooth wear and suggested that nearly all patients experienced some form of parafunctional activity at some time during their lives, and *Mohlin (1983)* found wear facets on the teeth of 60.5% of 202 women that may probably or certainly have been caused by bruxism.

Dental wear was found in nearly all 7-18 year olds examined by *Nilner and Lassing (1981) and Nilner (1981)*, and wear reaching the dentine was common, while extensive wear was found only occasionally. Dental wear in the mixed dentition reflected chiefly the wear of the primary teeth.

Dental wear of one or more incisors was observed in 77% and 90% of 11 and 15 year old children respectively, while only 14% of the 15 year olds had dentine visible on at least one incisor and no significant sex differences were found in any age group *(Egermark-Eriksson et al., 1981)*. In her follow-up examination *Egermark-Eriksson et al. (1987)* reported that dental wear was greater in the frontal region in all age groups, with the incisors being the most worn, but no extensive dental wear was recorded and the only significant sex difference was that upper canines were severely worn more often in males than in females.

Dental wear was on average confined to enamel for most of the teeth of 48 dental students examined by *Droukas et al. (1984)* and wear into dentine was found in 40% of the canines and 15% of other segments.

Age and sex distribution*: Carlsson & Ingervall (1988)* stated that "loss of tooth substance is, in general associated with aging or, more strictly speaking, with the length of time the teeth have been exposed to occlusal functions."

A significant increase of dental wear prevalence with age was reported by *Bernal and Tsamtsouris (1986)* among 3-5 year olds; *Egermark-Eriksson et al. (1981)* between 11 & 15 years of age in all regions of permanent dentition except the molar region; and *Egermark-Eriksson et al. (1987)* in all age groups and for all regions. However, dental wear was found not to increase with age by *Gazit et al. (1984)* among 10- 18 year olds.

Egermark-Eriksson et al. (1981) found no sex difference in all age groups, while on her follow-up examination *(Egermark-Eriksson et al., 1987)* canines were severely worn more often in boys than in girls.

Relation with TMDs: The severity of TMDs symptoms was found to be interrelated with dental wear *(Egermark-Eriksson et al., 1983; Lieberman et al., 1985; DeLaat et al., 1986)*. Dental wear was also correlated to TMJ pain, the severity of TMDs signs *(DeLaat et al., 1986)*, and recurrent headache *(Nilner, 1983a)*.

Egermark-Eriksson et al. (1983) and DeLaat et al. (1986) found muscle tenderness to a significantly higher degree in subjects with dental wear, and they attributed this muscle tenderness to prolonged muscle hyperactivity caused by bruxism.

Egermark-Eriksson et al. (1983) and Gazit et al. (1984) found that dental wear increased the probability of joint sounds; and *Runge et al. (1989)* found a significant association between reciprocal clicking and moderate to severe dental wear, in contrast to *DeLaat et al. (1986)* who found no correlation between dental wear and clicking.

Relation with bruxism: For most patients, periodic nocturnal bruxism will cause only mild tooth wear which is adaptive and may be viewed as an accepted aspect of aging. In extreme cases, bruxism may cause abnormal wear of the teeth. Patients who have a diurnal bruxing habit may acknowledge this, but, unfortunately, nocturnal bruxism often goes unnoticed *(Okeson, 1989)*.

Several studies found no correlation between reported bruxism and dental wear *(Lindqvist, 1971; Kuch et al., 1979; Egermark-Eriksson, 1982; Droukas et al., 1984; Bernal & Tsamtsouris, 1986)*.

Egermark-Eriksson et al. (1987) found a significant correlation between the degree of dental wear and reported bruxism only in the younger age groups, and explained this in two ways: *(1)* the information on bruxism attained from the subjects is not reliable because many individuals are not aware of their parafunctions, and *(2)* dental wear is caused by many factors other than bruxism.

DeLaat et al. (1986) documented that subjects aware of bruxing habit showed more occlusal wear.

2.2 EFFECT OF MALOCCLUSION ON TMDs:

Malocclusion has been implicated as an etiologic factor in patients with TMDs *(Perry, 1969; Thompson, 1972; Roberts, 1974; Roth, 1982)*; however, the results of correlational studies seeking to verify this relationship have been equivocal. Some found a significant correlation between malocclusion and TMDs *(Williamson, 1977; McNamara, 1978; Mohlin et al., 1980; Maruyama et al., 1982; Egermark-Eriksson et al., 1983; Gazit et al., 1984; Lieberman et al., 1985)*; while others have been unable to corroborate these results *(Thompson, 1971; Mohlin & Kopp, 1978; DeBoever & Adriaens, 1983; Droukas et al., 1984; Gazit et al., 1984; Helm et al., 1984; Nesbitt et al., 1985; Wanman & Agerberg, 1986c; de Boever et al., 1987; Gunn et al., 1988; Mongini et al., 1988; Helm & Petersen, 1989)*.

Judging from electromyographic *(Ingervall & Thilander, 1975; Moller & Troelstrup, 1975)* and kinesiologic studies *(Moller, 1981; Hamerling, 1983)*, malocclusions appear to cause neuromuscular dysfunction and reflex mandibular positioning *(Hamerling, 1983)* and contribute to observable disharmonies in chewing patterns *(Ahlgren, 1967; Gibbs & Lundeen, 1982)*. Because these dysfunctions are sufficient to produce ischaemic circulatory effects, malocclusions may be a significant factor predisposing to TMDs *(Moller, 1981)*.

The basis of support for TMDs occurring as a function of malocclusion comes from two areas:

A-The finding that many TMD patients have malocclusion *(Ramfjord, 1961; Krogh-Poulsen & Olsson, 1968)*, and that malocclusion patients have more signs and symptoms of TMDs than control subjects *(Olsson & Lindqvist, 1992; Wadhwa et al., 1993; Miyazaki et al., 1994; Egermark & Ronnerman, 1995)*.

B-The finding that some TMD patients can be treated successfully by orthodontics *(Egermark-Eriksson et al., 1975; Ingervall, 1978; Bakke, 1981; Aoshima & Satoh,* *1994),* and that treatment of certain malocclusions can reduce the signs and symptoms of TMDs in orthodontic patients *(Wisth, 1983; Oku et al., 1990; Sadowsky et al., 1991; Egermark-Eriksson & Thilander, 1992; Krenemak et al., 1992; Egermark & Ronnerman, 1995; Olsson & Lindqvist, 1995).*

On the other hand, the prevalence of TMDs in malocclusion patients was found to be slightly lower than has been presented in studies of randomly selected children of approximately the same age in one reported study *(Mohlin et al., 1991).*

Malocclusion evaluated in autopsy specimens was weakly associated with morphologic changes in the TMJs (not necessarily degenerative), particularly when considered with age. This evidence supports the belief that longer exposure to malocclusion may be associated with more extensive TMJ change *(Solberg et al., 1986).*

Others claim that malocclusion, per se, does not give rise to TMDs. However, certain types of morphological malocclusion predispose to occlusal interferences and those, according to some, may contribute to their etiology *(Ramfjord & Ash, 1983; Mongini & Schmid, 1989).*

There are divergent opinions about the impact of specific features of occlusion on TMDs and hence will be reviewed separately:

2.2.1 ANGLE'S CLASSIFICATION:

Rugh (1983) listed Angle class of occlusion among other factors that may affect adaptability. Angle Class II and III have been associated with TMDs *(DeLaat et al., 1986; Solberg et al., 1986; Egermark-Eriksson et al., 1990)*, but not consistently *(Helm et al., 1984; Bush, 1985; Lieberman et al., 1985; Helm & Petersen, 1989; Keeling et al., 1994).* These structural features may reduce the adaptive capacity of patients predisposing them to TMDs *(Parker, 1990).*

Some investigators *(Stringert & Worms, 1986; Dworkin et al., 1990)* found no statistically significant differences in the proportions of subjects in specific Angle's classification groups between TMD patients and control subjects. Other investigators found class II malocclusion to be heavily represented in TMDs populations *(Perry, 1969;. Lioselle, 1969; Rey et al., 1981).*

Lieberman et al. (1985) and Runge et al. (1989) found no correlation between Angle's classification and the signs and symptoms of TMDs. Several other studies also found no correlation between class II and III molar relationship and dental wear *(Lindqvist, 1971; Wigdorowicz-Makowerowa et al., 1979; Gunn et al., 1988)* in contrast to the findings of *Nilner (1983a), Brandt (1985) and Egermark-Eriksson et al. (1990).*

A- Class II division 1 malocclusion:

Subjects with class II, division 1, malocclusion have been reported to be more conscious of TMDs symptoms *(Nilner, 1983b)* and more vulnerable to develop TMDs than other classes *(Riolo et al., 1987; Mohlin et al., 1991; Egermark-Eriksson et al., 1990; al-Hadi, 1993).* Other investigators found no correlation between class II malocclusion and TMDs *(Mohlin, 1983; Pullinger et al., 1988 a&b);* and *Egermark-Eriksson et al. (1983)* found a negative association with TMDs symptoms and headache.

Angle class II subjects averaged significantly less maximal mouth opening *(Riolo et al., 1987)* and more clinical protrusion and lateral movement capacities than class I subjects *(Zimmer et al., 1991).*

Electromyographically, *Moller (1966)* reported an increased activity in the posterior temporal muscle in class II, division 1, children; while *Ahlgren (1966) and Ahlgren et al. (1973)* found no significant differences in muscle activity with a tendency for hypofunction in class II children, which was also observed by *Pancherz (1980)*.

More recently, *Mongini and Schmid (1985)* found that children with class II, division 1, malocclusion showed the tendency to chew in a position more protruded than the intercuspal position (centric occlusion).

Non-concentric condylar position was a feature of class II malocclusion with significantly more anterior positions in class II, division 1, than class I subjects *(Solberg & Seligman, 1985; Pullinger et al., 1987)*.

Forward posture of the mandible is also described in class II, division 1, persons in rest position *(Ricketts, 1953)*, intercuspation, and cosmetic profile *(Heloe et al., 1980)*.

B- Class II division 2 malocclusion:

Functionally, the class II, division 2, malocclusion is characterized by a very steep incisal guidance during gliding movements due to the deep overbite and retroclined position of the maxillary incisors *(Carlsson & Ingervall, 1988)*.

Berry and Watkinson (1978) suggested that clicking could be the result of lingually tipped maxillary incisors, which was confirmed by *Runge et al. (1989)* who found a significant association between large interincisal angle and TMJ sounds. Also, class II, division 2, subjects have more posteriorly positioned condyles than the other classes *(Pullinger et al., 1987)*, which may predispose to clicking *(Artun et al., 1992)*.

Patients with a class II, division 2, malocclusion have a tendency to more frequently show TMJ tenderness *(Pullinger et al., 1988a)*, generalized muscle tenderness *(Pullinger et al., 1988b)*, and TMDs symptoms *(Schupp, 1992)*. This may be because of the greater disk rotation movement *(Berry & Watkinson, 1978;*

Bell, 1982) and the greater TMJ compressive loading that occurs in this type of malocclusion *(O'Ryan & Epker, 1984)*.

Class II, division 2, patients have also been described as less vulnerable to develope TMDs than other classes of malocclusion *(al- Hadi, 1993)*, and this was attributed to the reduction of the overjet and the dominance of canine protected eccentric occlusion typical of this class of Angle's classification. *Pullinger et al. (1988a)* documented the absence of condyle luxation in this class and attributed it to be the result of more vertical chewing characteristics.

C- Class III malocclusion:

The class III malocclusion is usually combined with crossbite of the anterior teeth and often also with anterior open bite, the overjet and overbite are then negative. Besides possible occlusal interferences, this situation may often lead to lack of sufficient occlusal support *(Carlsson & Ingervall, 1988)*. Thus, *Ingervall et al. (1979)* found that, on average, only 11 teeth were in antagonistic contact in a sample of class III adults, compared with 16 teeth after surgical correction of the malocclusion.

Wisth (1983) showed that surgical correction of mandibular prognathism reduces the signs and symptoms of TMDs, decreases pain on movement, and increases the maximal mouth opening and protrusion capacities; indicating that mandibular setback had created a better functional environment. *Ingervall et al. (1979)* reported an increased efficiency of the patients masticatory function postoperatively, characterized by a normalization of the muscle activity during maximal bite and chewing, and a reduction in the number of chewing cycles and duration of the act of chewing. *Janson (1982)*, also, found that correcting class III malocclusion reduces symptoms.

Class III patients have a different muscle coordination in biting and chewing compared to normal occlusion and, also, reduced average electromyographic potential *(Ahlgren, 1967 & 1970)*. Children with class III malocclusion have been shown to have a tendency to chew posteriorly with respect to intercuspal position, opposing to class II malocclusion *(Mongini & Schmid, 1985)*.

In a recent computerized tomographic study *(Seren et al., 1994)*, class III subjects had a relative mediolateral elongation of the condyle within a relatively small glenoid fossa and a relative condylar protrusion which was also observed by *Pullinger et al. (1987)*.

Angle class III malocclusion was associated with the severity of clinical signs of TMDs assessed by Helkimo's dysfunction index *(Mohlin et al., 1980; Mohlin, 1983; Egermark-Eriksson et al., 1983)*. *Pullinger et al. (1988a) and Egermark-Eriksson et al. (1990)*, also, found a correlation between class III malocclusion and TMDs; while *Bush (1985)* mentioned that class III subjects tended to have less muscle tenderness than class I or II subjects.

Class III subjects averaged significantly less clinical protrusive and lateral movement capacities than class I subjects *(Mohlin et al., 1980; Zimmer et al., 1991)*.

2.2.2 OVERJET:

It has been suggested that excessive protrusive movement on a chronic basis for both esthetic and incising purposes in subjects with excessive overjet, is harmful to the joint structures, as it may lead to hyperactivity of the muscles and can convey the forces of hyperfunction adversely towards weaknesses in the masticatory system and induce symptoms *(Ricketts, 1953; Gawley, 1982; Parker, 1990; al-Hadi, 1993)*.

Overjet, thought to predispose to TMDs, has not been shown to be significantly associated with TMDs among children and adolescents *(Hultgren et al., 1980; Egermark-Eriksson et al., 1983; Helm et al., 1984; Lieberman et al., 1985; Keeling et al., 1994)*, adults *(Mohlin, 1983)*, orthodontic patients *(Runge et al., 1989)*,and TMD patients *(Mohlin & Kopp, 1978; Droukas et al., 1985; Dworkin et al, 1990)*. Condylar position was, also, unrelated to the degree of overjet *(Pullinger et al., 1987)*.

Heloe et al. (1980) and Stringert and Worms (1986) reported higher overjet values in TMD patients than among control subjects, and *Solberg et al. (1986)* related overjet to internal derangements in cadavers.

Riolo et al. (1987) found subjects with more than 5 mm of overjet averaged significantly more maximal mouth opening than those with less overjet, and those with reversed or excessive overjet were more likely to have joint tenderness. Excessive overjet was associated with TMJ sounds *(Riolo et al., 1987; Kritsineli et al., 1992)*, and the clinical dysfunction index *(Egermark-Eriksson et al., 1990)*.

In a study on University students, *al-Hadi (1993)* reported no significant increase in the incidence of TMDs with a 0 to 2 mm overjet, whereas a 2.5 to 3.5 mm overjet was associated with a reduction of TMDs; however, TMDs appeared to increase sharply when the overjet exceeded 6 mm.

2.2.3 OVERBITE:

The relationship between overbite and TMDs is controversial. For example, *Droukas et al. (1985)* found that overbite was significantly negatively correlated with the severity of the signs of TMDs and reported bruxism; and Stringert and Worms (1986) reported a non-significant difference between internal derangement patients and control subjects in relation to the average of overbite, while *DeLaat et al. (1986), Runge et al. (1989), and Kritsineli et al. (1992)* found a significant correlation between overbite and TMJ clicking. Moreover, condylar position was unrelated to the degree of overbite *(Pullinger et al., 1987).*

A- Open bite:

Due to the negative overbite in anterior open bite their is no anterior guidance during gliding movements. This often leads to a non-working side occlusal interference *(Mohlin & Kopp, 1978; Egermark-Eriksson, 1982; Mohlin, 1983)* especially when combined with crossbite of the posterior teeth. This emphasizes the importance of anterior guidance of disclusion of posterior teeth during gliding movements *(Carlsson & Ingervall, 1988)..*

Open bite has been associated with low electromyographic threshold values for the genioglossus and masseter muscles *(Lowe, 1980)*, true condylar distractions and mandibular fulcruming of the condyles *(Solberg & Seligman, 1985).*

Recent review articles continue to suggest open bite as a predisposing factor for TMDs *(Seligman & Pullinger, 1991; Vanderas, 1993; Bales & Epstein, 1994; McNamara et al., 1995)* and several published studies have documented this association among children and adolescents *(Egermark-Eriksson et al., 1983 & 1990; Brandt, 1985; Riolo et al., 1987; Kritsineli & Shim, 1992; Miyazaki et al., 1994),* orthodontic patients *(Williamson, 1977),* and TMD patients *(Mohlin & Kopp, 1978; Seligman & Pullinger, 1989).* Open bite subjects, also, averaged a significantly larger maximmal mouth opening capacity then subjects with positive *(Riolo et al., 1987).*

Lieberman et al. (1985) and Runge et al. (1989) found no association between open bite and the signs or symptoms of TMDs.

B- Deep bite:

Deep bite is common in class II, division 2, malocclusion, and in dental malocclusion with shortened arch length and insufficient space for canine eruption where the incisors may be intruding into the free way space. The deeper the overbite the more distally the condyles may be displaced. As the overbite becomes excessive the lower teeth and mandible may retreat posteriorly to avoid anterior interferences with possible clicking of the joint *(Thompson, 1994)*. When the overbite is corrected, the mandible is free to reposition itself anteriorly *(Owen, 1984)*.

The distally displaced condyle approximates the petrotympanic fissure that is relatively open in children. Pressure is created through the fissure to the middle ear giving rise to symptoms such as fullness, earaches, roaring, tinitus and vertigo, therefore, potential TMDs can be misdiagnosed in the pre-teens on certain occasions due to the presence of only earaches and headaches *(Padamsee et al., 1985b)*.

Deep overbite has been associated with strong retrusive activity of the posterior temporalis muscles *(Moller, 1966)*. Other studies have shown that overbite is associated with posterior disclusion *(Egermark-Eriksson, 1982)*, which reduces rather than aggravates jaw elevator muscle activity *(Williamson and Lindqvist, 1983; Belser & Hannam, 1985)*. Generalized muscle tenderness was more common in subjects with deep bite than in those with less overbite *(DeLaat et al., 1986; Pullinger et al., 1988b)*.

A significant association was found between deep bite and TMDs recorded in general population of children and teenagers *(Brandt, 1985; Lieberman et al., 1985; Keeling et al., 1994)*, orthodontic patients *(Williamson, 1977)*. Other studies attributed no significance to overbite *(Hultgren et al., 1980; Mohlin et al., 1980; Riolo et al., 1987; Glaros et al., 1992)*.

Deep bite is, also, cited as heavily represented in TMJ patient populations with deep bite as a cause of posterior condylar displacement, disk laxity, TMJ clicking and pain *(Gerber, 1971; Berry & Watkinson, 1978; Heloe et al., 1980;*

Bell, 1982; Owen, 1984). However, not all studies attributed significance to deep overbite *(Mohlin & Kopp, 1978)*.

Egermark-Eriksson et al. (1981) found less dental wear in deep bite children than in those without this type of malocclusion and concluded that tooth grinding is not common in children with deep bite. While, *Nilner (1983b)* found that deep bite was correlated to clenching and frontal dental wear.

In a computerized electromyographic study *(Alexander et al., 1984)*, no significant disturbance of the smoothness and regularity of chewing was demonstrated with deep bite malocclusion, and no greater smoothness or regularity was evident following its correction orthodontically.

2.2.4 CROSSBITE:

Among various morphologic types of malocclusion, crossbite occlusion shows cuspal interferences most frequently *(Ahlgren & Posslet, 1963)*, hence, TMJ clicking has been observed in children with anterior or posterior crossbites as young as 3-5 years of age *(Perry & Marsh, 1977; Bernal & Tsamtsouris, 1986)*.

Crossbite can produce mandibular displacement from centric relation *(Chate, 1994)*, and if left untreated, excessive tension can be placed on the TMJ causing marked deviations of the mandible on opening and closing *(Payne et al., 1981)* and may result in slight facial asymmetry and cant of the occlusal plane *(Padamsee et al., 1985b)*; therefore, the early correction of crossbites has been advised to permit normal jaw growth, normal eruption and improve condyle position *(Myres et al., 1980)*.

Unilateral crossbites, in children and teenagers, may induce asymmetric muscle activity *(Haralabakis & Loutfy, 1964; Troelstrup & Moller, 1970; Ingervall & Thilander, 1975)* and geometrically alter condyle fossa relationships *(Myres et al., 1980; Pullinger et al., 1985; Iijima, 1990; O'Bryn et al., 1995)*; but condylar displacement in adulthood was less clear and probably modified through skeletal adaptation with asymmetric muscle activity no longer present *(Pullinger et al., 1985 & 1988b). Solberg et al. (1986)*, also, identified greater condylar osseous adaptive changes in individuals with crossbites.

Posterior crossbites have been significantly positively correlated with TMDs signs and symptoms *(Mohlin et al., 1980: Egermark-Eriksson, 1982; Brandt, 1985; Fushima et al., 1989; Egermark-Eriksson et al., 1990; Kritsineli et al., 1992; Miyazaki et al., 1994)*. Others found posterior crossbites to be not significant factors in the development of TMDs *(Mohlin, 1983; Lieberman et al., 1985; Runge et al., 1989; Seligman & Pullinger, 1991)*.

No significant difference in the influence between unilateral and bilateral posterior crossbites was documented *(Mohlin et al., 1980; Egermark-Eriksson & Ingervall, 1982). While, Egermark-Eriksson et al. (1983)* revealed that subjects with bilateral posterior crossbites had more severe TMDs signs than those with unilateral crossbites as judged by the clinical dysfunction index (Di).

A relationship between posterior crossbite and hyperactivity in the temporal muscle in rest position has been mentioned *(Haralabakis & Loutfy, 1964; Troelstrup & Moller, 1970; Ingervall & Thilander, 1975)* and a different chewing pattern was observed in children with anterior and posterior crossbites when compared with those with normal occlusion *(Ahlgren, 1967)*.

Posterior crossbites have, also, been positively associated with muscle tenderness *(Egermark-Eriksson et al., 1983)*, locking of the mandible *(Helm et al., 1984)*, TMJ sounds *(Brandt, 1985; Riolo et al., 1987; Egermark-Eriksson et al., 1990; Kritsineli et al., 1992)*, luxation of the condyle *(Pullinger et al., 1988a), and* TMJ tenderness *(Egermark-Eriksson et al., 1990)*; but negatively with reported tenderness or fatigue of the cheeks *(Helm et al., 1984)*.

Crossbite was significantly more in those with TMDs signs and symptoms than in those without it, only among 19-22 year olds and not among 15-18 year olds which suggests the role of time in the damaging influence of crossbite occlusion on the TMJ structures *(Grosfeld et al., 1985)*. While, *Dworkin et al.(1990)* showed that TMD patients did not differ from controls with regard to posterior crossbite in contrast to the finding of *Mohlin and Kopp(1978)*.

Inversion of incisors was shown to have no association with TMDs *(Mohlin et al., 1980; Egermark-Eriksson et al, 1983; Mohlin, 1983)*, but *Egermark-Eriksson et al. (1990)* reported a correlation between inversion of incisors and TMJ tenderness.

Anterior crossbite, also, was reported to have no significant association with TMDs *(Helm et al., 1984);* but *Fushima et al. (1989)* found that symmetrical anterior crossbite showed low incidence of TMDs. While, *Dworkin et al. (1990)*, surprisingly, reported that asymptommatic controls had more anterior crossbite than TMD patients. One study, among cleft palate children reported a correlation between anterior crossbite and definitional symptoms of TMDs *(Vanderas, 1989)*.

Functional shift was negatively associated with TMJ and muscle tenderness *(Riolo et al., 1987)*. However, the results of the study of *Fushima et al. (1989)* suggested that mandibular lateral displacement may be related with the appearance of TMDs.

2.2.5 CROWDING/SPACING:

In crowding the teeth are forced to take up positions buccal or lingual to their normal positions in the dental arch, or in a rotated position in the arch, due to lack of space. Occlusal interferences are thus often found in crowded dentitions. The other type of space anomaly, spacing, is less apt to give rise to occlusal problems *(Carlsson & Ingervall, 1988)*.

A significant association was found between anterior crowding and the signs and symptoms of TMDs *(Lieberman et al., 1985; Keeling et al, 1994; Verdonck et al., 1994)*. Others found no relationship between crowding or spacing of the dental arches and TMDs *(Mohlin, 1983; Helm et al., 1984; Runge et al., 1989)*. However, *Mohlin et al. (1980)* found that maxillary crowding had as strong negative association with 'subjective dysfunction symptoms'; and they attributed this to that crowding is an expression of that few teeth had been lost.

In a recent electromyographic study *(Wang, 1994)*, malocclusion of the third molar was suggested as a cause for TMJ clicking.

AIMS OF THE STUDY

1- To detect the prevalence of the signs and symptoms of TMDs; and some possible related factors (recurrent headache, oral parafunctions, and dental wear) in pretreatment orthodontic patients.

2- To estimate the degree of severity of the signs and symptoms of TMDs.

3- To study the relation of sex and age with the signs and symptoms of TMDs and their severity, and related factors.

4-To examine the association between TMDs and malocclusion variables and estimate possible etiologic relations.

5- To compare the results of the present investigation with others reported in the literature, to draw meaningful conclusions about the effect of malocclusion, per se, on TMDs.

6-An attempt to determine priority of treatment of malocclusion as judged by its effect on the function of the masticatory system.

7-To provide a baseline data for a longitudinal study on orthodontic patients to determine the effect of orthodontic treatment on TMDs, in the future.

CHAPTER THREE

MATERIALS AND METHODS

3.1 MATERIALS:

3.1.1 THE SAMPLE:

The sample consisted of 143 patients attending the Postgraduate Orthodontic Clinic at the College of Dentistry in Baghdad, from October 1995 to January 1996. Their age ranged from 10 to 25 with a mean age of 17.8 ± 5.2 years. Male to female ratio was 1:2.5. The age and sex distribution is presented in table 3.1.

Age group (years)	Males	Females	Total
10-14	10	29	43(30%)
15-19	14	34	44(31%)
20-25	17	39	56(39%)
Total	41(28.7%)	102(71.3%)	143

Table 3.1: Sample distribution according to age and sex.

All patients had come to the clinic for orthodontic treatment and not for relief of symptoms that might be indicative of TMDs. Patients with history of orthodontic treatment were excluded from the sample.

3.1.2 INSTRUMENTS AND SUPPLIES:

The following instruments and supplies were used:

1- Millimeter graded vernier (Inox, Zurcher Modell, Dentaurum 042-751).

2- Metric ruler.

3- Indelible pencil.

4- Plain mouth mirror.

5- Stethoscope.

6- Kidney dish.

7- Air syringe.

8- Cotton wool.

3.2 METHODS:

The sample was investigated by an interview and a clinical examination; and the data were recorded on a special form (Appendix VII) for each patient. The method of examination of the masticatory system was based on the principles of *Helkimo (1974b)* and in accordance with previous Iraqi studies *(Shereef, 1991; Abdulla, 1992; Salih, 1993)*.

3.2.1 THE INTERVIEW:

While the patient was seated, general information including serial number, date of the examination and sex were recorded. The interview included the following information:

A- The name and age.

B- The subjects were asked about the subjective symptoms of TMDs that he or she might have felt. After explanation of each symptom, when needed, the answers were recorded as either yes (1) or no (0). The questions were as follows:

1- Do you notice TMJ sounds?

2- Do you suffer from feeling of fatigue of the jaws?

3- Do you suffer from feeling of stiffness on awakening or on movements of the lower jaw?

4- Do you notice difficulty in opening your mouth wide?

5- Do you have locking in your jaw on mandibular movement?

6- Do you have luxation in your jaw on mandibular movement?

7- Do you feel pain on movement of the mandible?

8-Do you feel pain in the region of the TMJ or of the masticatory musculature (cheeks)?

Then these symptoms were graded according to the anamnestic dysfunction index *(Helkimo, 1974b)* to determine the severity of dysfunction of the masticatory system based on the patients account of the condition. This index was classified into three grades:

AiO denotes complete absence of symptoms of dysfunction of the masticatory system i.e. symptoms mentioned under AiI and AiII.

AiI denotes mild symptoms of dysfunction; one or more of the following symptoms were reported in anamnesis: joint sounds, feeling of fatigue of the jaws, feeling of stiffness of the jaws on awakening or on movement of the lower jaw. None of the symptoms given under AiII were reported.

AiII denotes severe symptoms of dysfunction; one or more of the following symptoms were reported in anamnesis: difficulties in opening the mouth wide, locking, luxation, pain on movement of the mandible, facial and jaw pain.

C- Information about the related factors to TMDs were obtained through asking the patient about:

- Recurrent headache (once a week or more),
- Oral parafunctions: the patients were asked if he or she frequently did one or more of the following oral habits: nocturnal tooth grinding, diurnal tooth clenching, nail biting, biting on foreign objects, lip- and cheek- biting, digit sucking and gum chewing.

3.2.2 CLINICAL EXAMINATION:

The patient was seated on a dental chair with the back tilted slightly backwards. The clinical examination included the following:

3.2.2.1 Examination of the masticatory system:

The functional status of the masticatory system was examined in a systemic way according to the clinical dysfunction index *(Helkimo, 1974b)*, which is designed on the basis of five common clinical signs:

A- Examination of impaired range of movement:

Three types of movements were examined:

1-Maximal opening of the mouth:

It is the summation of interincisal distance on maximal mouth opening and the overbite. In open bite cases the amount of negative overbite was subtracted from the maximal interincisal distance to give the maximal opening capacity of the mouth; in accordance with *Nilner and Lassing (1981) and Schiffman et al. (1990).*

The interincisal distance was measured by a millimeter graded vernier. The patient was encouraged to open his mouth as wide as possible. Then one end of the vernier was placed in the median plane against the incisal edge of one of the lower incisors and the distance to the incisal edge of the opposing upper incisor was measured to the nearest half of a millimeter, giving the interincisal distance on maximal opening.

The overbite was measured with a metric ruler, while the patient was in centric occlusion with his occlusal plane horizontal using the same incisors used for measuring the interincisal distance. The amount of vertical overlap of the upper incisors on the labial surface of the lower incisors was marked with an indelible pencil using the incisal edge of the opposing upper incisor to guide the pencil. The upper conical plane of the sharpened part of the pencil itself was placed parallel to the patients occlusal plane. The distance between the marked line and the lower incisal edge was measured to the nearest half of a millimeter. In open bite cases the negative overbite was measured directly by the vernier *(FDI, 1973)*.

2- Maximal lateral movements:

While the patient was in centric occlusion with his occlusal plane horizontal, a vertical line was marked with an indelible pencil in the midline from the upper incisors down to the opposing lower incisor. Then the patient was asked to move his mandible to the right as far as he could and the distance between the pencil markings on the upper and lower incisors in the horizontal plane was measured by a metric ruler to give the maximal lateral movement capacity to the right. In a similar manner, the maximal lateral movement capacity to the left was measured *(Padamsee et al., 1985; Okeson, 1989)*.

3- Maximal protrusion:

It is the distance between the labial surfaces of the upper and lower central incisors on maximal protrusion of the mandible plus the overjet *(Ingervall et al., 1980; Nielsen et al., 1989)*. In reversed overjet cases the negative overjet was subtracted from the distance between the labial surfaces of the incisors to give the maximal protrusion capacity.

One end of the metric ruler was placed on the labial surface of an upper incisor and the horizontal distance to the labial surface of the lower incisor was measured to the nearest half of a millimeter, while the patient protruded his or her mandible as much as he or she could.

The overjet was measured with a metric ruler, while the patient was in centric occlusion with his occlusal plane horizontal, using the same incisors used for measuring the maximal protrusion distance.

All movements were performed twice and the highest value was recorded *(Ingervall et al., 1980; Egermark-Eriksson et al., 1981)*. The four different movements of the jaw were judged separately and the scores awarded were added using the mobility index (table 3.2). The following code was used for the mobility index before it was entered in the clinical dysfunction index (Di):

Mi0 = 0 points = clinically normal mobility.

MiI = 1-4 points = slightly impaired mobility.

MiII = 5-20 points = severely impaired mobility.

B- Examination of impaired TMJ function:

This includes the investigation for the presence of the following:

1- ***TMJ sounds:*** This was assessed with the use of a stethoscope in accordance with *Nilner and Lassing (1981), Pilley et al. (1992)*. The stethoscope was placed over the joint area and the patient was asked to open and close his mouth and the absence or presence of joint sounds was noted..

2- ***Deviation on maximal mouth opening:*** While the patient was in centric occlusion and his occlusal plane horizontal, a vertical line was marked with an indelible pencil on the midline from the upper incisors down to the opposing lower incisor. Then the patient was asked to open his or her mouth and with the aid of a ruler held vertically in the midline by the examiner as a guide, any

	Maximal mandibular movement	Criteria	Score
A	Maximal mouth opening.	Normal range of movement (40 mm or more).	0
		Slightly impaired range of movement (30-39 mm).	1
		Severely impaired range of movement (less than 30 mm).	5
B	Maximal lateral movement to the right.	Normal range of movement (7 mm or more).	0
		Slightly impaired range of movement (4-6 mm).	1
		Severely impaired range of movement (less than 4 mm).	5
C	Maximal lateral movement to the left.	Normal range of movement (7 mm or more).	0
		Slightly impaired range of movement (4-6 mm).	1
		Severely impaired range of movement (less than 4 mm).	5
D	Maximal mandibular protrusion.	Normal range of movement (7 mm or more).	0
		Slightly impaired range of movement (4-6 mm).	1
		Severely impaired range of movement (less than 4 mm).	5

sum A+B+C+D= Mobility index (0-20).

*** Reproduced from Helkimo (1976).**

Table 3.2: Mandibular mobility index (Mi).

horizontal deviation of 2mm or more, estimated by the naked eye, between the two labeled lines was recorded. The direction towards which the mandible shifted was also registered as right or left *(Swanljung & Rantanen, 1979; Padamsee et al., 1985)*.

3- ***Locking:*** It is an occasional blocking of short duration (fixation) of a mandibular movement *(Helkimo, 1974b)*.

4- ***Luxation:*** It is a forward dislocation of the condylar head out of the glenoid fossa combined with fixation in that position *(Helkimo, 1974b)*.

C- Muscle palpation:

The masticatory muscles were examined for tenderness and spasm using digital palpation. Both right and left sites were palpated extraorally and simultaneously except for the lateral pterygoid and the insertion of the temporalis muscles which were palpated intraorally and individually. Palpation was performed mainly by the palmer surface of the middle finger, with the index finger and forefinger testing the adjacent areas. A single soft but firm pressure of 1 to 2 seconds duration was applied to the designated muscles, the fingers compressing the adjacent tissues in a small circular motion *(Padamsee et al., 1985; Okeson, 1989)*.

Only if palpation produced a clear reaction of the patient (such as a palpebral reflex) or if the patient stated that the site palpated was clearly more tender to palpation than the surrounding structures or corresponding structures on the other side; this was assessed as muscle tenderness *(Helkimo, 1974b)*.

The method of examination of these muscles is as follows *(Okeson , 1989) :*

-Posterior part of the temporalis muscle: palpated on the side of the head above and behind the ears.

-Anterior part of the temporalis muscle: palpated above the zygomatic arch and anterior to the TMJ.

-Insertion of the temporalis muscle in the coronoid process: palpated by placing the finger of one hand intraorally on the anterior border of the ramus of the mandible and the finger of the other hand extraorally on the same area. The fingers are moved simultaneously up the anterior border of the ramus until the coronoid process and the tendon are palpated.

-Profound masseter muscle (deep masseter): palpated at its superior attachment; the fingers are placed on the zygomatic arches just anterior to the TMJ.

-Superficial masseter muscle: palpated at its inferior attachment on the inferior borders of the rami.

-Medial pterygoid muscle: palpated extraorally at its insertion on the medial surface of the mandibular angles.

-Lateral pterygoid muscle: palpated by placing the index finger in the maxillary buccal vestibule, and the patient was instructed to move the mandible towards the side being palpated to gain better access through the shifting of the coronoid process. The palmer surface of the index finger is moved posteriorly, superiorly and medially into the area of the infratemporal fossa, posterior to the maxillary tuberosity.

D- TMJ palpation:

Tenderness of the posterior aspect of the TMJs was determined by placing the small finger of each hand into the external auditory meatus and applying forward pressure while the patient kept his teeth in centric occlusion. Lateral palpation of the joints was accomplished by pressing against and encircling both condylar heads simultaneously with the middle finger tips as the patient opened and closed his or her mouth *(Bush, 1985)*.Only clear reactions of the patient were registered as positive results *(Helkimo, 1974b)*.

E- Examination of pain on mandibular movements:

The patient was asked to systematically perform different movements of the mandible and report any pain felt during these movements i.e. opening, closing, protrusion, lateral movements to the right and left. In doubtful cases the movements were repeated against resistance of the examiner's hand *(Helkimo, 1974b)*.

TMJ sounds, muscle and TMJ tenderness were recorded separately for the right and left sides. Then the results were combined and treated as either affirmative or negative to be used in calculating the clinical dysfunction index *(Helkimo, 1974b)*.

The previously mentioned five objective signs were graded according to the index (Di) to determine the severity of dysfunction of the masticatory system. Each sign was judged according to a three- grade scale of severity: absence of the sign was awarded 0, mild sign 1, and a severe sign 5 points (table 3.3). Then the scores awarded for the five signs were added together. Thus, each patient had a total dysfunction score ranging from 0 to 25 points. The severity of clinical signs according to this score was classified into four groups:

Di0 = Dysfunction group 0 = 0 points = Clinically sign-free.

DiI = Dysfunction group 1 = 1-4 points = Mild clinical dysfunction.

DiII = Dysfunction group 2 = 5-9 points = Moderate clinical dysfunction.

DiIII = Dysfunction group 3 = 10-25 points = Severe clinical dysfunction.

	Clinical signs	Criteria	Score
A	Impaired range of movement/Mobility index (Mi)	Normal range of movement (Mi0).	0
		Slighly impaired mobility (MiI).	1
		Severely impaired mobility (MiII).	5
B	Impaired TMJ function	Smooth movement without TMJ sounds and deviation < 2 mm on maximal opening.	0
		TMJ sounds in one or both joints and/or deviation ≥ 2 mm on maximal opening.	1
		Locking and/or luxation of the TMJs.	5
C	Muscle tenderness	No tenderness to palpation in masticatory muscles.	0
		Tenderness to palpation in 1-3 palpation sites.	1
		Tenderness to palpation in 4 or more palpation sites.	5
D	TMJ tenderness	No tenderness to palpation.	0
		Tenderness to palpation laterally.	1
		Tenderness to palpation posteriorly.	5
E	Pain on movement of the mandible	No pain on movement.	0
		Pain on one movement.	1
		Pain on two or more movements.	5

A+B+C+D+E = Dysfunction score (0-25 points)

*** Reproduced from Helkimo (1976)**

Table 3.3: Clinical dysfunction index.

3.2.2.2 EXAMINATION OF OCCLUSION:

The following types of occlusal anomalies were examined with the mandible in centric occlusion:

A- Antero-posterior occlusion:

1- ***Angle classification***: The class of malocclusion was assigned according to molar *(Angle, 1899)* or canine relationships *(Foster, 1982)* with due consideration to the effect of tooth migration, if any; in accordance with *Nilner and Lassing (1981) and Egermark-Eriksson (1981)*. An intermaxillary discrepancy of greater than one half of the cusp width was used as the criterion for the determination of class II and III relationships.

2- ***Overjet:*** It is the horizontal distance from the most prominent point on the incisal edge of the maxillary central incisors to the most prominent point on the labial surfaces of the mandibular incisors, measured parallel to the occlusal plane when the dental arches are in centric occlusion *(Hoffding & Kisling, 1978)*. It was measured to the nearest half of a millimeter in accordance with *Pullinger et al. (1988a)*. The value of excessive overjet was calculated later.

B- Vertical occlusion (Overbite):

Overbite is the vertical distance from the incisal edge of a maxillary central incisor to the incisal edge of the corresponding mandibular central incisor, when the dental arches are in centric occlusion *(Smith & Bailit, 1979)*. It was measured to the nearest half of a millimeter in accordance with *Pullinger et al. (1988a)*.

An overbite of 5 mm or more was regarded as deep bite while negative values were recorded as open bite *(Bjork et al., 1964; Pullinger et al., 1988a)*.

C- Transverse occlusion:

1- ***Anterior crossbite:*** Inversion of incisors was recorded when one, two or three maxillary incisors occluded lingually to the mandibular incisors and the number of involved pairs of teeth was recorded. Reversed overjet was only registered when all four incisors were involved *(Bjork et al., 1964; Egermark-Eriksson et al., 1981).*

2- ***Posterior crosssbite:*** It was recorded if the buccal cusps of the maxillary teeth occluded lingually to the buccal cusps of the corresponding mandibular teeth and was recorded as either unilateral or bilateral *(Bjork et al., 1964).*

3- ***Mandibular displacement:*** It is a sagittal or lateral displacement of the mandible as a result of a premature contact *(Houston, 1983).* It was examined when anterior or posterior crossbite were recognized *(Padamsee et al., 1985b).*

4- ***Midline shift:*** The horizontal distance between themidlines of the maxillary and the mandibular dentitions was measured by a metric ruler to the nearest half of a millimeter. The side towards which the mandibular teeth shifted was recorded as right or left *(FDI, 1973).*

D- Intra -arch discrepancies:

1- ***Crowding/spacing:*** It was assessed separately for the maxillary and mandibular dentitions, and for the anterior and posterior teeth. Crowding or spacing was registered when a deviation of at least 2mm per segment was diagnosed *(Bjork et al., 1964; FDI, 1973).*

2- ***Dental wear (Attrition):*** It was registered in four regions; incisor , canine , premolar and molar; according to a five- point scale *(Hansson & Nilner, 1975; Nilner & Lassing, 1981)*:

0 = no wear,

1= wear of enamel only,

2= one or more teeth worn into dentine,

3= one or more teeth worn up to one third of the crown, and

4= extensive wear of one or more teeth more than one third of the crown.

3.2.3 PILOT STUDY:

After training on the method of clinical examination over a period of time, a pilot study was carried out. To ensure a valid application of the diagnostic criteria, inter- and intra-examiner calibration procedures were carried out on twenty patients. The patients were re-examined after 3 hours to minimize the influence of time in accordance with *Kopp and Wenneberg (1983)*. The patients were examined in a random order to minimize the risk of memorizing the individual findings.

The Student t-test was used to examined the differences between the two sets of observations concerning parametric variables (interincisal distance on maximal opening, overbite, maximal lateral movements to the right and left, maximal protrusion, overjet, and dental midline shift). The results showed no significant differences between the first and second examinations ($p>0.05$) in both inter-and intra-calibration procedures (table 3.4).

On the other hand, percentage agreement was used to examine the differences between the sets of observations concerning non-parametric variables (Mi, impaired TMJ function, muscle tenderness, TMJ tenderness, pain on movement, Di, Angle's class of occlusion, presence of crowding or spacing, crossbites, inversion of incisors and dental wear). The results showed a high agreement in both inter- and intra-calibration procedures for all tested variables ranging between 85% to 100% (table 3.5).

Variable	Inter-calibration	Intra-calibration
Maximal interincisal distance	t=0.431 N.S.	t=0.371 N.S.
Overbite	t=-0.102 N.S.	t=0.035 N.S.
Maximal opening capacity	t=0.217 N.S.	t=0.174 N.S.
Maximal right movement	t=0.456 N.S.	t=0.323 N.S.
Maximal left movement	t=0.497 N.S.	t=0.317 N.S.
Protrusion movement	t=0.257 N.S.	t=0.204 N.S.
Overjet	t=0.701 N.S.	t=-0.044 N.S.
Maximal protrusion capacity	t=0.413 N.S.	t=0.109 N.S.
Midline shift	t=0.105 N.S.	t=0.091 N.S.

Table 3.4: Calibration of parametric variables.

Variable	Inter-calibration agreement	Intra-calibratioon agreement
Mobility index (MI)	90%	100%
Impaired TMJ function	90%	100%
Muscle tenderness	85%	90%
TMJ tenderness	95%	95%
Pain of movement	95%	100%
Dysfunction index (Di)	85%	90%
Angle's classification	100%	100%
Posterior crossbite	95%	100%
Inversion of incisors	100%	100%
Crowding	95%	95%
Spacing	95%	100%
Dental wear	100%	95%

Table 3.5: Calibration of non-parametric variables.

3.2.4 STATISTICAL ANALYSES:

1- Descriptive statistics including prevalence and percentage for non-parametric variables; mean and standard deviation for parametric variables.

2- Correlations between variables including age and sex were calculated by means of Spearman's rank correlation test in accordance with *Egermark-Eriksson et al. (1987)*.

3- The relationship between mandibular movement capacities and sex was tested by Students t-test, while its relation with age was examined by "one way analysis of variance test."

4- Excessive overjet was defined as the threshold necessary to yield a statistically significant increase in the signs and symptoms of TMDs as judged by Helkimo's indices (Ai & Di); in accordance with *Riolo et al. (1987)*. Initially the threshold was chosen as 1mm. A Student t-test was then performed to determine whether this classification of overjet was significantly associated with signs or symptoms of TMDs. When no significance resulted, then the test was repeated with greater thresholds until statistical significance was achieved.

For all the foregoing statistical methods p values exceeding 0.05 were considered to be non-significant.

5- Stepwise multiple regression analyses were performed to assess the influence of different factors taken together on the development of TMDs signs and symptoms and some related factors; in accordance with *Mohlin (1983) and Keeling et al. (1994)*. Significance levels of the whole model was regarded as 0.1% ($P < 0.001$).

Sex, age and all the malocclusion variables were included as independent variables in the regression equations. This procedure provided a ranking of the independent variables according to the predictive value and excluded the independent factors that have been explained by others.

All calculations were performed using Microstat software (Ecosoft, 1984).

CHAPTER FOUR

RESULTS

The sample consisted of 143 orthodontic patients (43 10-14 year olds, 44 15-19 year olds, and 56 20-25 year olds), 29% males and 71% females. When chi square test was applied to the age and sex distribution of the sample no significant differences were found ($p>0.05$) indicating the homogenicity of the sample.

4.1 PREVALENCE AND SEVERITY OF TMDs:

4.1.1 PREVALENCE AND SEVERITY OF TMDs SYMPTOMS:

It was found that 65.7% of the interviewed orthodontic patients complained of one or more symptoms of TMDs (table 4.1). The most common were TMJ sounds (43.4%) and feeling of fatigue of the jaws (39.9%); followed by feeling of stiffness (21.7%), pain on movement of the mandible (18.6%) and difficulty in opening the mouth wide (16.7%). Other symptoms were found to be less frequent as facial pain (9.1%) and locking of the jaw (7.7%). None of the interviewed patients were aware of luxation of the jaw.

Above one third (34.3%) of the sample were completely symptom-free, more frequent patients (43.4%) had mild symptoms and the remaining (22.4%) had severe symptoms as judged by the anamnestic index (Ai).

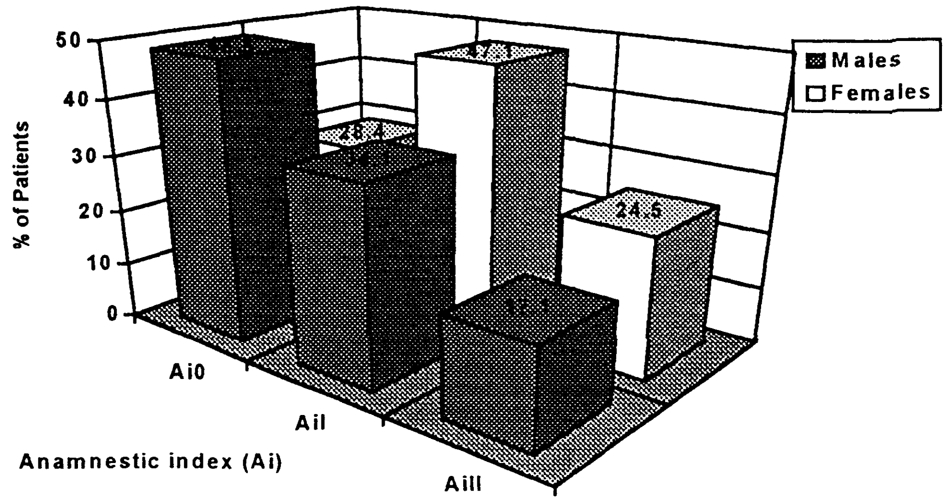
The sex distribution of the symptoms of TMDs is demonstrated in tables 4.1 and figure 1. Table 4.1 shows that the percentage of females who complained of one or more symptoms (70.6%) was higher than that of males (53.7%), but the difference did not reach a statistically significant level. All the symptoms except locking of the jaw were reported more by females, but feeling of fatigue of the jaws and facial pain were the only symptoms that reached the significance level $p<0.05$.

Figure 1 demonstrates the distribution of males and females according to the anamnestic dysfunction index (Ai). The percentage of males with no symptoms (Ai0) was higher (48.8%) than that of females (28.4%), while the prevalence of mild (AiI) and severe symptoms (AiII) in females was higher (47.1 & 24.5%) than that of males (34.1 & 17.1%, respectively). However, the difference between the two sexes was statistically non-significant.

TMDs Symptoms	Total	Sex difference			Age difference			
		Males	Females	p level	10-14 years	15-19 years	20-25 years	p level
	n=143	n=41	n=102		n=43	n=44	n=56	
TMJ sounds	43.4	31.7	48.0	N.S.	39.5	47.7	42.9	N.S.
Feeling of fatigue	39.9	26.8	45.1	P<0.05	44.2	27.3	46.4	N.S.
Feeling of stiffness	21.7	19.5	22.5	N.S.	16.3	15.9	30.4	N.S.
Pain on movement	18.6	14.6	20.6	N.S.	16.3	18.3	21.4	N.S.
Difficult wide mouth opening	16.7	9.8	19.6	N.S.	14.0	18.2	17.9	N.S.
Facial pain	9.1	0.0	12.7	P<0.05	7.0	6.8	12.5	N.S.
Locking	7.7	9.8	6.9	N.S.	2.3	6.8	12.5	P<0.025
Luxation	0.0	0.0	0.0	N.S.	0.0	0.0	0.0	N.S.
One or more symptoms	65.7	53.7	70.6	N.S.	65.1	65.9	66.1	N.S.

*** Figures represent percentages of the respective samples.**

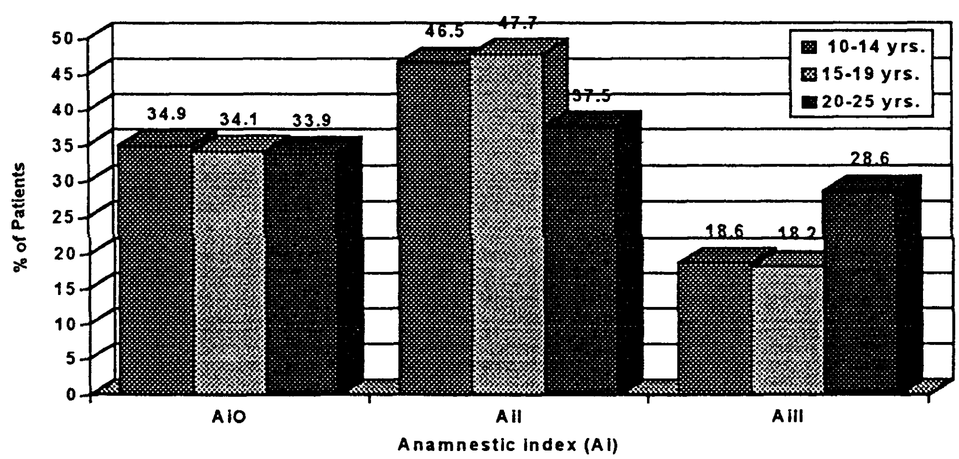
Table 4.1 : Frequency and relative distribution of the symptoms of TMDs according to sex and age.



N.S. P>0.05

Figure1: Frequency and relative distribution of the patients according to sex and the severity of TMDs symptoms.

The age distribution of the symptoms of TMDs is demonstrated in tables 4.1 and figure 2. Both tables show no significant differences between the three age groups in relation to the prevalence of symptoms and the anamnestic dysfunction index except for a significant increase ($p<0.025$) in the prevalence of locking of the jaw with age.



N.S. P>0.05

Figure2: Frequency and relative distribution of the patients according to age and the severity of TMDs symptoms.

4.1.2. PREVALENCE AND SEVERITY OF TMDs SIGNS:

It was found that 81.8% of the examined patients had one or more clinical signs of TMDs (table 4.2). The most common signs were muscle tenderness (68.5%) and TMJ tenderness to palpation (43.4%); followed by impaired TMJ function (25.9%) and restricted mandibular movement (25.2%); while pain on mandibular movement was the least frequently found (11.9%).

The most common location of masticatory muscle tenderness to palpation was in the lateral pterygoid (55.9%), insertion of the temporalis (53.8%) and medial pterygoid muscles (40.6%); followed by profound masseter (18.9%) and anterior temporalis muscles (12.6%); while superficial masseter (5.6%) and posterior temporalis muscles (2.1%) were found to be tender only occasionally (table 4.2).

Tenderness to lateral palpation of the TMJ was found in 33.6% of the sample, and tenderness to posterior palpation was found in 9.8%

Regarding impaired TMJ function, TMJ sounds were found in 16.8% and deviation on maximal opening in 11.9% of the sample. Locking of the jaw was only found in one patient (0.7%), while luxation was not detected in any patient. Clicking was found more frequently (22 patients, 15.4%) than crepitation (2 patients, 1.4%).

Restricted lateral mandibular movements (less than 7mm) to the right or left were detected in 19.6% of the sample, restricted protrusive movement (less than 7mm) in 11.8%, while restricted maximal mouth opening capacity (less than 40mm) was only found in three patients (2.1%).

According to the clinical dysfunction index (Di), 18.2% of the sample were completely free of signs of TMDs (Di0). Mild signs (DiI) were more frequent (53.1%) than moderate signs (DiII, 22.4%), whereas severe signs (DiIII) were less frequently found (6.3%).

TMDs Signs	Total n=143	Sex difference Males n=41	Females n=102	p level	Age difference 10-14 years n=43	15-19 years n=44	20-25 years n=56	p level
Muscle tenderness:	68.5	58.5	72.5	p<0.05	65.1	72.7	67.9	N.S.
Temoralis-posterior	2.1	0.0	2.9	N.S.	0.0	2.3	3.6	N.S.
-anterior	12.6	9.8	13.7	N.S.	11.6	11.4	14.3	N.S.
-insertion	53.8	43.9	57.8	N.S.	53.5	65.9	44.6	N.S.
Masseter-profound	18.9	9.8	22.5	N.S.	30.2	18.2	10.7	P<0.025
-superficial	5.6	2.4	6.9	N.S.	4.7	6.8	5.4	N.S.
Medial pterygoid	40.6	29.3	45.1	N.S.	51.2	43.2	30.4	P<0.05
Lateral pterygoid	55.9	53.7	56.9	N.S.	55.8	68.2	46.4	N.S.
TMJ tenderness:	43.4	31.7	48.0	N.S.	55.8	36.4	39.3	P<0.025
-laterally	33.6	26.8	36.3	N.S.	32.6	29.5	37.5	N.S.
-posterioly	9.8	4.9	11.8	N.S.	23.3	6.8	1.8	P<0.01
Impaired TMJ function:	25.9	14.6	30.4	N.S.	20.9	22.7	32.1	P<0.05
TMJ sounds	16.8	7.3	20.6	P<0.05	9.3	13.6	25.0	P<0.025
Deviation on opening	11.9	7.3	13.7	N.S.	9.3	20.5	7.1	N.S.
Resticted movement	25.2	34.1	21.6	N.S.	16.3	22.7	33.9	P<0.01
Pain on movement	11.9	7.3	13.7	N.S.	9.3	13.6	12.5	N.S.
One or more signs	81.8	73.2	85.3	N.S.	65.1	79.5	87.5	N.S.

*** Figures represent percentages of the respective samples.**

Table 4.2 : Frequency and relative distribution of the signs of TMDs according to sex and age.

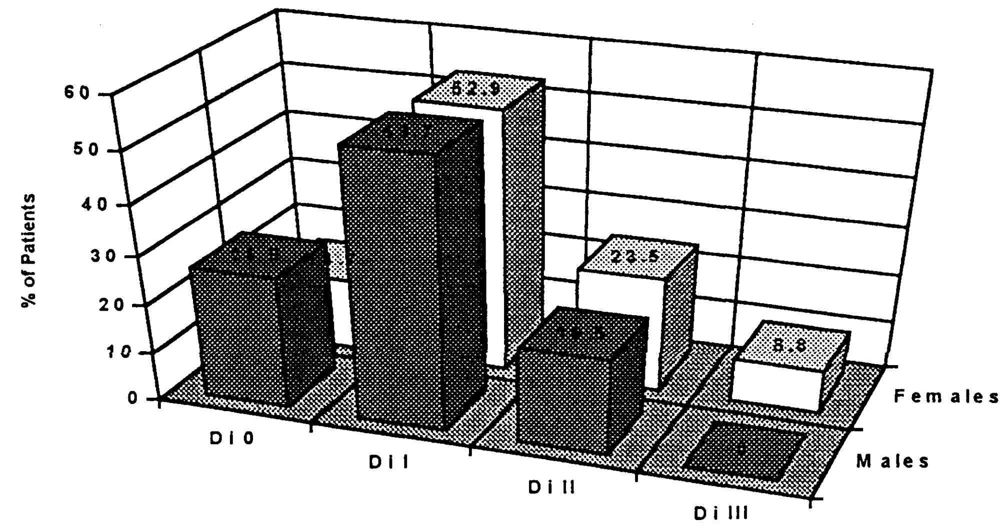
The sex distribution of the clinical signs of TMDs is demonstrated in tables 4.2 and figure 3. Table 4.2 shows that all the TMDs signs except for restricted mandibular movement were more frequently found in females than in males, but only muscle tenderness and TMJ sounds reached a significant level ($p<0.05$).

The percentage of males with no signs (Di0) was higher (26.8%) than that of females (14.7%), and the frequency of mild signs (DiI) was comparable in both sexes, while the prevalence of moderate signs (DiII) in females was higher (23.5%) than in males (19.5%). None of the male patients had severe signs (DiIII) which was found in 8.8% of the female patients (figure 3). Hence, females had significantly ($P<0.05$) more severe signs than males did.

The age distribution of the signs of TMDs is demonstrated in tables 4.2 and figure 4. Tenderness of the profound masseter and medial pterygoid muscles significantly decreased with age, while tenderness of the lateral pterygoid and insertion of the temporalis muscles was found to be more frequent among 15 to 19 year olds than among the younger and older age groups (table 4.2).

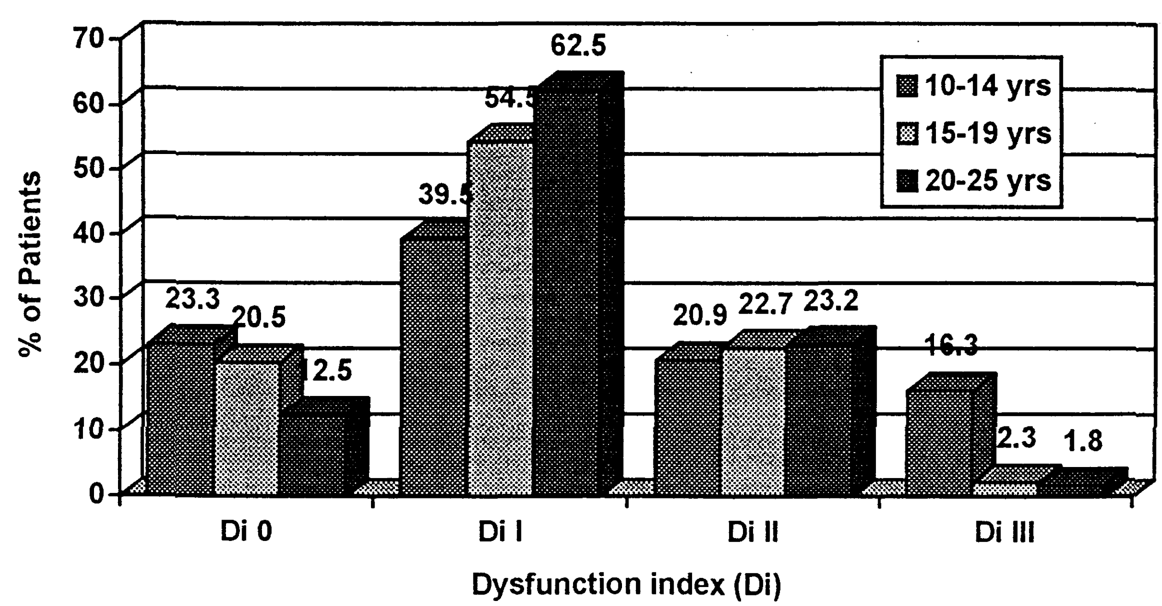
Tenderness of the TMJ to posterior palpation significantly decreased with age, while TMJ sounds and restricted mandibular movement significantly increased with age. Pain on movement and deviation on maximal opening were found to be more frequent among 15-19 year old patients (table 4.2).

Figure 4 displays the distribution of the patients according to age and clinical dysfunction index (Di). The youngest group (10-14 year olds) had more sign-free (Di0) and severe signs (DiIII) patients than the older two age groups, while the oldest group (20-25 year olds) had more mild (DiI) and moderate signs (DiII) patients than the younger two age groups. However, the difference between the three age groups was found to be statistically non-significant.



P<0.05

Figure 3: Frequency and relative distribution of the patients according to sex and the severity of TMDs signs.



N.S. P>0.05

Figure 4: Frequency and relative distribution of the patients according to age and the severity of TMDs signs.

Maximal mouth opening had a highly statistically significant ($p<0.001$) relationship with sex, where males had a higher mean maximal mouth opening capacity (54.21 ± 6.49 mm) than females (49.90 ± 6.29 mm). The other three movements showed no significant sex differences. The relationship between all four mandibular movements examined by 'one way analysis of variance test' (ANOVA) showed non-significant relationships with age (table 4.3).

Maximal Mandibular Movements Capacities		Total	Sex difference			Age difference			
			Males	Females	p level	10-14 years	15-19 years	20-25 years	p level
		n=143	n=41	n=102		n=43	n=44	n=56	
Mouth opening	mean	51.13	54.21	49.90	p<0.001	50.95	51.82	50.73	N.S.
	S.D.	6.67	6.49	6.29		5.35	5.95	5.79	
Right lateral	mean	9.27	9.36	9.24	N.S.	9.44	9.68	8.82	N.S.
	S.D.	1.86	1.78	1.90		1.59	1.86	2.01	
Left lateral	mean	8.95	8.98	8.94	N.S.	9.05	9.18	8.69	N.S.
	S.D.	1.92	1.74	2.00		1.36	1.84	2.34	
Protrusive	mean	8.53	8.16	8.68	N.S.	8.26	8.56	8.72	N.S.
	S.D.	2.04	2.01	2.04		1.58	2.29	2.16	

*** Figures are presented in millimeters.**

Table 4.3 : Maximal mandibular movement capacities according to sex and age.

4.2 RELATED FACTORS:

4.2.1 RECURRENT HEADACHE:

Recurrent headache was reported by 38.5% of the sample. Figure 5 displays the distribution of recurrent headache according to sex and age. Females complained of recurrent headache (45.1%) significantly ($p<0.025$) more often than males (22.0%).

Although recurrent headache was reported by the oldest group (20-25 year olds) more frequently (46.4%) than by the younger two groups (34.9%of 10-14 year olds and 31.8% of 15-19 year olds), this difference did not reach a significant level.

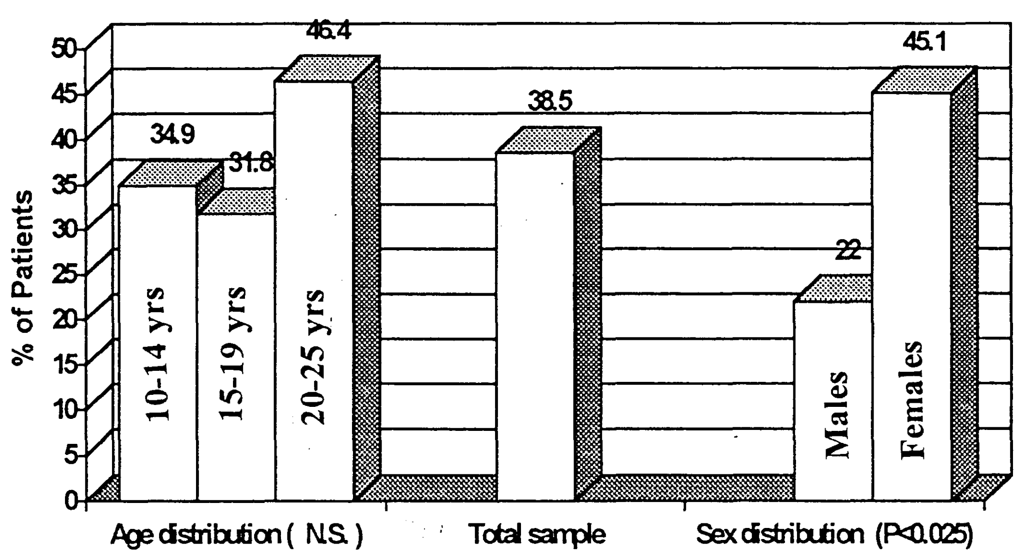


Figure 5: Frequency and relative distribution of recurrent headache according to sex and age.

4.2.2 ORAL PARAFUNCTIONS:

The prevalence of one or more oral parafunctions among the investigated patients was rather high (78.3%). The prevalence of each oral parafunction is given in table 4.4. The prevalence of orofacial parafunctions (68.5%) was higher than the prevalence of bruxism (44.1%). Only two subjects were at the time of examination digit suckers, while sixteen patients reported a history of digit sucking giving a total prevalence of past and present digit sucking of 12.6%.

The sex distribution of oral parafunctions is demonstrated in table 4.4. All parafunctions except digit sucking were more frequently reported by females than males. The sex difference of orofacial parafunctions (nail biting, object biting and gum chewing) reached a significant level ($p<0.05$, $p<0.001$ and $p<0.025$), while bruxism did not reach a statistically significant level.

Oral Parafunctions	Total	Sex difference			Age difference			
		Males	Females	p level	10-14 years	15-19 years	20-25 years	p level
	n=143	n=41	n=102		n=43	n=44	n=56	
Grinding	25.2	19.5	27.5	N.S.	32.6	25.0	19.6	P<0.05
Clenching	27.3	19.5	30.5	N.S.	18.6	31.8	30.4	N.S.
Bruxism (clenching &/or grinding)	44.1	34.1	48.0	N.S.	44.2	54.5	35.7	N.S.
Nail biting	22.4	14.6	25.5	P<0.05	25.6	22.7	19.6	N.S.
Object biting	26.6	4.9	35.3	P<0.001	32.6	31.8	17.9	N.S.
Lip and cheek biting	37.1	26.8	41.2	N.S.	44.2	31.8	35.7	N.S.
Gum chewing	31.5	17.1	37.3	P<0.025	25.6	45.5	25.0	N.S.
Digit sucking (past & present)	12.6	17.1	10.8	N.S.	9.3	15.9	12.5	N.S.
Orofacial parafunctions (Nail-, object-, lip- and cheek-biting and gum chewing)	68.5	48.8	76.5	P<0.005	72.1	75.0	60.7	N.S.
One or more parafunctions	78.3	63.4	84.3	P<0.01	76.7	84.1	75.0	N.S.

*** Figures represent percentages of the respective samples.**

Table 4.4 : Frequency and relative distribution of oral parafunctions according to sex and age.

The age distribution of oral parafunctions is, also, demonstrated in table 4.4. Grinding significantly ($p<0.05$) decreased with age while clenching tended to increase with age. Nail and object biting, also, tended to decrease with age. Patients, 15-19 year olds, reported parafunctions (especially gum chewing) more frequently than both the older and younger age groups.

4.2.4 DENTAL WEAR:

Only the first four grades of the five grade scale used to assess dental wear came in use in this investigation as grade 4 dental wear (extensive wear more than one third of the crown) was not noticed in any of the examined patients.

Dental wear was noticed in most of the patients, 93% in the incisor region, 73.4% in the canine region, 79.7% in the premolar region and 69.2% in the molar region. Most of the dental wear recorded was of a mild character (grade 1; wear into enamel) as shown in table 4.5.

Grade 2 dental wear (reaching the dentine) was noticed most frequently in the incisor region (39.9%), followed by the canine region (24.5%), molar region (17.5%) and the premolar region (9.1%). Grade 3 dental wear (till one third of the crown) was only noticed in three patients (2.1% of the sample) and only confined to the incisor region.

The sex and age distribution of dental wear according to its location is presented in table 4.5, which shows no significant sex differences in all four dental regions, however, a significant increase in the prevalence of dental wear with age was seen in all four dental regions.

Dental wear		Total	Sex difference			Age difference			
Location	Grade	n=143	Males n=41	Females n=102	p level	10-14 years n=43	15-19 years n=44	20-25 years n=56	p level
Incisor	0	7.0	7.3	6.9	N.S.	7.0	9.1	5.4	P<0.005
	1	53.1	51.2	53.9		58.1	59.1	44.6	
	2	37.8	39.0	37.3		34.9	31.8	44.6	
	3	2.1	2.4	2.0		0.0	0.0	5.4	
Canine	0	26.6	31.7	24.5	N.S.	39.5	31.8	12.5	P<0.005
	1	49.0	43.9	51.5		46.5	50.0	50.0	
	2	24.5	24.4	24.5		14.0	18.2	37.5	
Premolar	0	20.3	31.7	15.7	N.S.	27.9	25.0	10.7	P<0.005
	1	70.6	61.0	74.5		62.8	70.5	76.8	
	2	9.1	7.3	9.8		9.3	4.5	12.5	
Molar	0	30.8	24.4	33.3	N.S.	34.9	43.2	17.9	P<0.05
	1	51.7	65.9	46.1		51.2	38.6	62.5	
	2	17.5	9.8	20.6		14.0	18.2	19.6	

*** Figures represent percentages of the respective sample.**

Table 4.5 : Frequency and relative distribution of dental wear according to location, severity, sex and age.

4.3 PREVALENCE OF MALOCCLUSION:

Concerning Angle's classification, 54.5% had class I relationship, 34.3% had class II relationship (28.7% division 1 and 5.6% division 2), and 11.2% had class III relationship (5.6% postural and 5.6% true).

Excessive overjet greater than 8mm was found in 9.8% of the sample, while reversed overjet was observed in 11.2% of the patients. A deep bite of 5mm or more was found in 11.2%, whereas 14.7% of the sample were found to have anterior open bite.

Fourteen percent of the examined patients had inverted incisors; 15% of which were associated with anterior mandibular displacement of the mandible. Posterior crossbite was found in 11.2% of the sample (4.2% unilateral, 6.5% bilateral, and 0.7% scissors bite).

Anterior mandibular displacement was found in 7.7% of the sample; 5.6 % associated with postural class III and 2.1% associated with inversion of incisors. Lateral mandibular displacement associated with unilateral posterior crossbite was found in only three patients (2.1%); two to the right and one to the left.

Regarding crowding and spacing of the dentition, 41.3% had crowding (35% maxillary and 16.8% mandibular), and 10.5% of the sample had spacing (9.8% maxillary and 4.2% mandibular).

A midline shift of 1mm or more was noticed in 64.4% of the examined patients, being equal in frequency to either side (32.2%).

4.4 THE EFFECT OF MALOCCLUSION ON TMDs:

The regression analyses identified the independent variables that were most strongly correlated with the various dependent variables, and these are shown in tables 4.6 to 4.10.

The severity of the signs and symptoms of TMDs (Ai & Di) varied across the various magnitudes of overjet and was not statistically associated with overjet except when greater than 8 mm and hence, excessive overjet was regarded as any overjet exceeding 8 mm.

4.4.1 TMDs SYMPTOMS:

The severity of TMDs symptoms as assessed by the anamnestic index (Ai) was negatively associated with lower anterior crowding and upper anterior spacing. However, each individual symptom was explained by a different set of independent variables as shown in table 4.6.

For the presence of TMJ sounds six malocclusion variables were selected, as it was negatively associated with class II (both divisions 1 and 2) malocclusion and lower anterior crowding, but significantly increased with the presence of excessive overjet, deep bite and upper anterior crowding. However, the association between reported TMJ sounds and deep bite was on the borderline of significance ($p=0.0506$).

Feeling of fatigue was associated negatively with upper anterior spacing and together with feeling of stiffness were weakly associated with true class III malocclusion ($p=0.0763$ and $p=0.055$, respectively).

The dependent variables pain on movement and difficulty in opening the mouth widely were explained by true class III malocclusion, while facial pain was best explained by excessive overjet.

Reported locking of the jaw significantly increased with age and was associated with lower anterior crowding (negatively) and with the amount of midline shift (positively), but both relationships were weak (p=0.0829 and p=0.0679, respectively).

The sex differences presented by Spearman's rank correlation test regarding feeling of fatigue and facial pain (table 4.1) did not appear in the regression equation (table 4.6). Furthermore, open bite and crossbites were found to have no association with the symptoms of TMDs.

TMDs symptoms	Age	class II$_1$	class II$_2$	true class III	exce. over-jet	deep bite	upper ant. crowd.	lower ant. crowd.	upper ant. spac.	mid-line shift
TMJ sound		-0.77	-1.52		+0.09	+5.06	+0.06	-1.26		
Feeling of fatigue				+7.63					-1.38	
Feeling of stiffness				+5.50						
Pain on movement				+1.46						
Difficult wide mouth opening				+0.49						
Facial pain					+3.57					
Locking	+3.45							-8.29		+6.79
Anamnestic index (Ai)								-2.62	-1.14	

*** Figures represent levels of significance of the regression coefficient in percentages.**
*** (+) means a positive association, and (-) means a negative association.**

Table 4.6 : Independent variables for explaining the variation of the dependent variables 'subjective TMDs symptoms'.

4.4.2 TMDs SIGNS:

The variables in the regression analysis that were found to have significant influence on the variable 'clinical dysfunction index' (Di) were excessive overjet and bilateral posterior crossbite (table 4.7).

Muscle tenderness was found more common among females than males (not significantly, p=0.0863). It, together with tenderness of the lateral pterygoid, superficial and profound masseter muscles, were not associated with any malocclusion variable.

For the presence of tenderness of the anterior temporalis muscle to digital palpation four factors were selected: unilateral posterior crossbite, deep bite, reversed and excessive overjet, while lower anterior crowding was associated with tenderness of both the posterior part (positively) and insertion of the temporalis muscle (negatively).

Tenderness of the profound masseter muscle decreased significantly with age. Medial pterygoid tenderness to palpation was negatively associated with unilateral posterior crossbite, but not significantly (p=0.0769).

TMJ tenderness to lateral palpation was weakly associated with excessive overjet (p=0.0812), while tenderness to posterior palpation decreased in prevalence significantly with age, was negatively associated with lower anterior crowding, and positively associated with excessive overjet, deep bite and bilateral posterior crossbite.

TMJ sounds increased significantly with age, was negatively associated with lower anterior crowding, and positively associated with excessive overjet, unilateral posterior crossbite and upper anterior crowding. However, the latter two associations were weak (p=0.0643 and p=0.0628).

Deviation of the mandible on maximal mouth opening was associated with open bite, and pain on mandibular movement was associated with excessive overjet (weak association, p=0.0688) and inversion of incisors.

Restricted mandibular movement increased significantly with age and was highly significantly associated with reversed overjet.

The sex differences in TMJ sound and the clinical dysfunction index (Di), and the significant association between tenderness of the medial pterygoid muscle and age presented by Spearman's rank correlation test (table 4.2) did not appear in the regression equation (table 4.7). However, Angle's classification of occlusion, spacing and midline shift were observed to have no association with the signs of TMDs.

TMDs signs	sex	age	overjet		overbite		inv. inc.	posterior crossbite		anterior crowding	
			reversed	excessive	open bite	deep bite		uni.	bi.	upper	lower
Muscle tenderness:	+8.63										
Temporalis-posterior											+3.07
-anterior			+0.62	+1.66		+3.62		+3.36			
-insertion											-4.86
Masseter-profound		-2.73									
Medial pterygoid								-7.69			
TMJ tenderness -laterally				+8.12							
-posteriorly		-0.32		+0.05		+3.68			+0.00		-1.05
TMJ sounds		+0.73		+1.68				+6.43		+6.28	-0.11
Deviation on opening					+0.47						
Restricted movement		+0.61	+0.05								
Pain on movement				+6.88			+0.82				
Dysfunction index (Di)				+0.11					+0.78		

*** Figures represent levels of significance of the regression coefficient in percentages.**
*** (+) means a positive association, while (-) means a negative association.**

Table 4.7 : Independent variables for explaining the variation of the dependent variables 'clinical TMDs signs'.

4.4.3 RECURRENT HEADACHE AND ORAL PARAFUNCTIONS:

Recurrent headache was positively associated with three independent variables: sex (being more in females), true class III malocclusion (weakly associated, p=0.0655), and deep bite (table 4.8).

Negative associations were observed between grinding of the teeth and open bite and inverted incisors, and between clenching of the teeth and forward mandibular displacement, but the latter two associations were weak (p=0.0613 & p=0.0711, respectively).

Lip and cheek biting was best explained by upper anterior crowding (positively but weakly, p=0.0526) and the amount of midline shift (negatively).

For gum chewing three independent variables were selected: sex, excessive overjet (negatively) and deep bite; while, object biting was only associated with sex (more in females).

Nail biting was positively associated with postural class III malocclusion, but negatively associated with deep bite. However, digit sucking was best explained by open bite and upper anterior spacing.

The significant sex difference in nail biting and the significant association between grinding and age presented by Spearman's rank correlation test (table 4.3) did not appear in the regression equation (table 4.8). However, the independent variables age, reversed overjet, crossbites did not fit in the models explaining the variation in recurrent headache and oral parafunctions.

4.4.4 DENTAL WEAR:

Dental wear in the four dental regions (incisor, canine, premolar and molar) were positively associated with age (table 4.9); and the significance of this association was strongest in the canine region and then in the premolar region, while in the molar region it did not reach a statistically significant level (p=0.0573).

Dental wear in the incisor, canine and molar regions was positively associated with inversion of incisors; and the significance of this association was strongest in the incisor region (p=0.0254) and weakest in the canine region (p=0.0573).

	sex	post. class III	true class III	exces over-jet	open bite	deep bite	inver incis	forw. mand. displ.	up. ant. crowd.	upper ant. spac.	mid-line shift
Recurrent headache	+2.65		+6.55			+3.83					
Grinding					-4.81		-6.13				
Clenching								-7.11			
Lip and cheek biting									+5.26		-0.56
Gum chewing	+0.23			-3.24		+2.30					
Object biting	+0.07										
Nail biting		+0.14				-0.57					
Digit sucking					+1.07					+2.93	

*** Figures represent levels of significance of the regression coefficient in percentages.**
*** (+) means a positive association, while (-) means a negative association.**

Table 4.8 : Independent variables for explaining the variation of the dependent variables 'recurrent headache and oral parafunctions'.

Dental wear Location	age	inverted incisors
Incisor	+0.35	+2.54
Canine	+0.00	+6.83
Premolar	+0.11	
Molar	+5.73	+3.26

*** Figures represent levels of significance of the regression coefficient in percentages.**
*** (+) means a positive association.**

Table 4.9 : Independent variables for explaining the variation of the dependent variables 'dental wear' according to location.

4.4.5 MANDIBULAR MOVEMENT CAPACITIES:

Maximal mouth opening was highly significantly associated with sex, being higher in males. Patients with class II, division 1, malocclusion averaged significantly more maximal mouth opening capacity, while open bite and lower anterior spacing were negatively associated with it (table 4.10).

Maximal lateral mandibular movement capacity was only explained by age and reversed overjet being negatively associated with both dependent variables.

Maximal protrusion capacity was highly significantly associated with overjet (p=0.00000) as it was negatively associated with reversed overjet and positively associated with excessive overjet.

Maximal mandibular movement capacity	sex	age	class II 1	overjet		open bite	lower ant. spac.
				reve rsed	exce-ssive		
Mouth opening	-0.09		+7.91			-3.93	-2.05
Lateral movement		-3.34		-0.07			
Protrusive				-0.57	+0.10		

*** Figures represent levels of significance of the regression coefficient in percentages.**
*** (+) means a positive association, while (-) means a negative association.**

Table 4.10 : Independent variable for explaining the variation of the dependent variables 'maximal mandibular movement capacities'.

CHAPTER FIVE

DISCUSSION

The present study examined the prevalence, severity, sex and age distribution of TMDs signs and symptoms and some related factors in a sample of pretreatment orthodontic patients. The relation of several malocclusion variables with various TMDs variables was also assessed.

No attempt was made to select the patients according to the type of malocclusion, sex or age; therefore, the sample investigated can be considered as a representative of the patients attending the orthodontic clinic. Their age ranged from 10 to 25 years, starting from the mixed dentition ending with full permanent dentition. The selection of the age grouping (10-14, 15-19, and 20-25 years) was made to facilitate comparison with other Iraqi epidemiological studies *(Shereef, 1991; Abdulla, 1992)*.

Compared with other investigations in other countries, the present subjects represent a different population from a geographical and cultural points of view, and differ from most of the previously investigated subjects by distribution according to sex and age. Also, comparison with other studies is not always possible because of the differences in definitions and in evaluation of the signs and symptoms, varying examination techniques and different indices used. Thus, comparison will only be made with studies carried out on a similar age range using a similar methodology especially those performed on Iraqi samples.

5.1 PREVALENCE OF TMDs AND RELATED FACTORS:

5.1.1 SYMPTOMS OF TMDs:

In this study, 65.7% of the interviewed patients complained of one or more symptoms (table 4.1).This percentage falls within the range (10-74%) reported by previous investigators as shown in appendices III and IV. This prevalence is consistent with those reported by *Grosfeld et al. (1985), Magnusson et al. (1985) and Heikinheimo et al. (1989)*, but is lower than that reported by *Egermark-Eriksson et al. (1981)* among 15 year olds (74%) and much higher than those reported by *Ingervall and Hedegard (1974),Molin et al. (1976), Grosfeld and Czarnecka (1977), Solberg et al. (1979), Droukas et al. (1984), Wanman and Agerberg (1986a), Pullinger et al. (1988a) and Ohno et al. (1988)*. This remarkable difference may have resulted from differences in questionnaire form, wording of the questions by the investigator, meaning and number of questions used or due to differences in the composition of the material.

On the other hand, this finding was higher than that reported by epidemiological studies carried out on the Iraqi population *(Shereef, 1991*, 55%; *Abdulla, 1992*, 55%; *Salih, 1993*, 42.1%), which reflects the adverse effect of malocclusion on the function of the masticatory system, hence increasing the prevalence of subjective TMDs symptoms.

The most common TMDs symptom in this study was TMJ sounds (table 4.1). This symptom was reported as the most frequent in several studies *(Molin et al., 1976; Solberg et al., 1979; Heloe & Heloe, 1979; Nilner, 1981; Nilner & Lassing, 1981; Wisth, 1983; Droukas et al., 1984; Wanman & Agerberg, 1986a; Heikinheimo et al., 1989; Schiffman et al., 1990; Shereef, 1991; Abdulla, 1992; Salih, 1993)*, however, some disagreed *(Egermark-Eriksson et al., 1981;*

Magnusson et al., 1985; Kononen et al., 1987; Pilley et al., 1992; Widmalm et al., 1995a).

None of the interviewed patients complained from locking of the jaw, hence, it was the least frequent symptom which is in conformity with the findings of all previous investigations except *Abdulla (1992)* who reported luxation of the jaw in 4.8% of 19-24 year old University students.

Regarding the distribution of patients according to the anamnestic dysfunction index (Ai) (figure 1), this study revealed that mild symptoms were more frequent than severe symptoms which is consistent with that reported by *Droukas et al. (1984), Wanman and Agerberg (1986a), Pullinger et al. (1988a), Abdulla (1992), Salih (1993), and Nourallah and Johansson (1995).* A contradictory finding was reported by *Schiffman et al. (1990) and Shereef (1991)* who documented the predominance of severe symptoms among their sample.

5.1.2 SIGNS OF TMDs:

In this study, the presence of one or more signs was 81.8% (table 4.2). This percentage falls within the range(6-93%)reported by other investigators (appendices III & IV). This result complies with those of *Solberg et al. (1979) and Nilner (1981)*, but is lower than that reported by *Schiffman et al. (1990)* and much higher than those reported by *Molin et al. (1976), Wigdorowicz-Mackowerowa et al.(1979), Nielsen et al. (1989), Ogura et al. (1985), Kononen et al. (1987) and Pullinger et al. (1988a).* Differences in methods of examination and/or different composition of the material probably acted to increase the variability in the prevalence of the signs of TMDs reported by various investigators.

On the other hand, our finding was higher than that reported by epidemiological studies on the Iraqi population by *Shereef (1991), Abdulla (1992),* *Salih (1993), and al-Hadi (1993)* which were 32, 60, 67 and 50%, respectively, supporting the previous statement that malocclusion appears to have an adverse effect on the function of the masticatory system.

The most prevalent sign in this investigation was masticatory muscle tenderness to palpation (table 4.2). This finding agrees with that reported by several investigators *(Molin et al., 1976; Grosfeld & Czarnecka, 1977; Solberg et al., 1979; Egermark-Eriksson et al., 1981; Nilner, 1981; Nilner & Lassing, 1981; Wisth, 1983; Droukas et al., 1984; Grosfeld et al., 1985; Wanman & Agerberg, 1986b; Kononen et al., 1987; Pullinger et al, 1988a; Nielsen et al., 1989; Schiffman et al., 1990; Abdulla, 1992; Pilley et al., 1992)*, while other investigators reported the predominance of TMJ sounds *(Ogura et al., 1985; Wanman & Agerberg, 1986e; Shereef, 1991; Motegi et al., 1992; al-Hadi, 1993; Salih, 1993; Wadhwa et al., 1993; Verdonck et al., 1994).*

A certain over-recording of muscle and TMJ tenderness is not unlikely because some patients may have not differentiated pain from discomfort.

The most common localization of muscle tenderness in this study was the lateral pterygoid and insertion of the temporalis muscles (table 4.2). This result complies with the result of other studies *(Molin et al., 1976; Wanman & Agerberg, 1986b; Kononen et al., 1987; Schiffman, 1990; Dworkin et al., 1990; Pilley et al., 1992)*, but contradicts those of others *(Shereef, 1991; Salih, 1993)* who reported that the anterior temporalis and masseter muscles were most commonly tender.

Regarding the distribution of patients according to the clinical dysfunction index (figure 3), this study showed that mild signs were more frequent than moderate signs, while severe signs were the least frequent. This agrees with the findings reported by other investigators *(Egermark-Eriksson et al., 1983; Droukas et al., 1984; Magnusson et al., 1985; Wanman & Agerberg, 1986b; Kononen et al., 1987; Pullinger et al., 1988a; Schiffman et al., 1990; Abdulla, 1992; Salih,* *1993; Nourallah & Johansson, 1995)*, but the latter found that the percentage of those with severe signs was relatively high, while *Magnusson et al. (1985) and Wanman and Agerberg (1986b)* did not observe any severe signs in their samples.

5.1.3 RECURRENT HEADACHE:

In the present study, 38.5% of the orthodontic patients complained of recurrent headache (figure 5). This figure is remarkably higher than the prevalence figures (8- 28%) reported among children, adolescents and young adults other than Iraqis (appendix V). This wide range of prevalence may be attributed to different interviewing techniques, different definitions of recurrent headache and different populations.

On the other hand, the prevalence or recurrent headache was comparable to that reported among the Iraqi population: 33% of adolescents *(Shereef, 1991)*, 41% of University students *(Abdulla, 1992)*; and 44% of 16-24 year old workers (Salih, 1993). The results of this study and the former three studies demonstrated that Iraqis suffer from more headache than Americans and Europeans probably because of stress induced by the embargo forced on the Iraqi population.

5.1.4 ORAL PARAFUNCTIONS:

The prevalence of one or more parafunctions in this study was 78.3% (table 4.4). This result is consistent with that reported by *Egermark-Eriksson et al. (1981), Nilner (1981), Nilner and Lassing (1981), and Kononen et al. (1987)* which were 78, 77, 74, and 75%, respectively, but is higher than those of the remaining studies (appendix VI) including the three Iraqi studies by *Shereef*

(1991), Abdulla (1992) and Salih (1993) who reported prevalences of 70, 57 and 37%.

The high prevalence of oral parafunctions in pretreatment orthodontic patients may be responsible for the high prevalence of masticatory muscle tenderness. Regarding different oral habits, in the present study bruxism was found in 44.1% of the sample. This finding is lower than that reported by *Wigdorowicz-Makowerowa et al. (1979)*, but is higher than that reported by all the remaining studies carried out on a similar age group (appendix VI) which supports the neuromuscular theory.

Orofacial parafunctions were reported by 68.5% of the present sample, which is higher than that reported by all the previous investigators (appendix VI)with the exception of *Egermark-Eriksson et al. (1981)*. The preponderance of these stress relieving oral habits among these patients supports the previous statement that they are under stress.

When comparing the prevalence of each oral parafunction found in this study with that of three Iraqi epidemiological studies on similar age groups *(Shereef, 1991; Abdulla, 1992; Salih, 1993)*; a few observations can be seen:

1-Clenching was more common than grinding in the present study and the three forgoing studies. This is consistent with the finding of previous investigations on similar age groups *(Ingervall & Hedegard, 1974; Molin et al., 1976; Egermark-Eriksson et al., 1981; Nilner, 1981; Nilner & Lassing, 1981; Wanman & Agerberg, 1986 a&d)* but contradicts that of *Lindqvist (1971) and Kononen et al. (1987)*.

2-Orofacial parafunctions were more common than bruxism in the four studies. This is in agreement with the finding of other investigators *(Wigdorowicz-Makowerowa et al., 1979; Egermark-Eriksson et al., 1981; Nilner, 1981; Nilner* *and Lassing, 1981; Magnusson et al., 1985; Wanman and Agerberg, 1986 a&d; and Kononen et al., 1987).*

3-The prevalence of each oral parafunction except clenching of the teeth was higher among the malocclusion patient (present sample) than in normal population in the other three studies.

The fact that the prevalence of clenching of teeth was less common among malocclusion patients than normal population may explain why the most common site of muscle tenderness in the malocclusion patients was the lateral pterygoid while in normal population the anterior temporalis and masseter muscles were the most common sites for muscle tenderness *(Shereef, 1991; Abdulla, 1992; Salih, 1993)*.This is probably because of the significant relationship between clenching habit and tenderness in the anterior temporalis and masseter muscles *(Ahmad, 1986; Shereef, 1991)*.

The prevalence figures of oral parafunctions are not quite comparable with other studies because of different environmental factors, different methods used to investigate the oral habit, different definitions for bruxism, or different types and number of oral habits included in the orofacial habits.

The oral habits in the present study were recorded on the basis of the subject's own acknowledgment. Patients were often unaware or uncertain of their oral habits (especially bruxism), therefore, the occurrence of oral parafunctions was undoubtedly higher than that reported in answers to the interview as stated by *Agerberg and Carlsson (1972) and Egermark-Eriksson et al. (1981)*.

5.1.5 DENTAL WEAR:

Dental wear was observed in 93% of the present sample being more common in the incisor region (table 4.5). This is consistent with the results of *Nilner (1981), Nilner and Lassing (1981), Egermark-Eriksson et al. (1981 & 1987), and Droukas et al. (1984).*

In this study most of the diagnosed dental wear was of a mild character (not reaching dentine) and no extensive wear was recorded, in agreement with the findings of *Egermark-Eriksson et al. (1981 & 1987);* while *Nilner (1981) and Nilner and Lassing (1981)* among 7- 18 year olds reported wear into the dentine to be common which may be attributed to the inclusion of deciduous teeth in their examination, however, they observed extensive dental wear only occasionally.

5.2 DISTRIBUTION OF TMDs ACCORDING TO SEX:

5.2.1 SYMPTOMS OF TMDs:

In this study, no sex difference was found concerning one or more symptoms (table 4.1). This result agrees with that of other studies *(Molin et al., 1976; Grosfeld & Czarnecka, 1977; Solberg et al., 1979; Egermark-Eriksson et al., 1981; Nilner, 1983c; Magnusson et al., 1985; Wanman & Agerberg, 1986a; Kononen et al., 1987; Ohno et al., 1988; Vanderas, 1988 ; Abdulla, 1992; Waltimo & Kononen, 1995)*; but disagrees with that of *Wanman and Agerberg (1986d), Heikinheimo et al. (1989), Shereef (1991), and Salih (1993)* who reported that females complained of one or more symptoms more than males.

Feeling of fatigue of the jaws and reported facial pain were significantly - reported more frequently by females than males in the current study. This agrees with *Bush et al. (1982); Wanman and Agerberg (1986 a&d), Shereef (1991), and Pilley et al. (1992);* but disagrees with *Abdulla (1992), Salih (1993) and Widmalm et al. (1995a)* who reported a non-significant sex difference.

5.2.2 SIGNS OF TMDs:

Females were observed to have more severe signs of TMDs than males as judged by the clinical dysfunction index (figure 3). This result agrees with the findings of *Solberg et al. (1979); Grosfeld et al. (1985), Wanman and Agerberg (1986 b & e), Pullinger et al. (1988a), Shereef (1991), Abdulla (1992), and Waltimo and Kononen (1995)*; but it disagrees with that of other studies *(Egermark-Eriksson et al., 1981; Nilner, 1983c; Gazit et al., 1984; Ogura et al.,* *1985; Magnusson et al., 1985; Kononen et al., 1987; Vanderas, 1988 ; Huber & Hall, 1990; al-Hadi, 1993)* who reported a non-significant sex difference.

Muscle tenderness and TMJ sounds were significantly observed more frequently by females than males in the current study. This agrees with *Solberg et al. (1979), Wanman and Agerberg (1986b & e), Magnusson (1986), Pullinger et al. (1988a), Shereef (1990), and Salih (1993)*; but disagrees with the findings of others *(Nilner, 1983c; Gazit et al., 1984; Ogura et al., 1985; Kononen et al., 1987; Abdulla, 1992; Keeling et al., 1994)* who reported a non-significant sex difference.

Maximal mouth opening capacity was significantly higher in females than in males (table 4.3) which is in conformity with the findings of *(Solberg et al, 1979; Gazit et al., 1984; Wanman & Agerberg, 1986e; Shereef, 1991; Abdulla, 1992; Salih, 1993; Pilley et al., 1992)*; whereas, other investigators reported no such difference *(Agerberg, 1974; Wanman & Agerberg, 1986e; Kononen et al., 1987; Nielsen et al., 1989; Dworkin et al., 1990).*

On the other hand, no significant sex differences were found concerning the lateral and protrusive mandibular movements, in agreement with the result of *Agerberg (1974), Kononen et al. (1987), Nielsen et al.(1989), and Dworkin et al. (1990)*; but disagrees with those of *Egermark-Eriksson et al. (1981), Wanman and Agerberg (1986b & e), and Salih (1993).*

5.2.3 RECURRENT HEADACHE:

The significant preponderance of recurrent headache among females,in the present study (figure 5),is in consistent with the findings of several studies *(Solberg et al., 1979; Heloe & Heloe, 1979; Egermark-Eriksson et al., 1981;*

Bush, 1982; Magnusson et al., 1985; Wanman & Agerberg, 1986a; Pullinger et al., 1988a; Heikinheimo et al., 1989; Shereef, 1991; Abdulla, 1992; Pilley et al., 1992; Widmalm et al., 1995b); however *Nilner (1981),Helm et al.(1984),Kononen et al.(1987), and Salih (1993)* reported no such sex differences.

5.2.4 ORAL PARAFUNCTIONS AND DENTAL WEAR:

The skewed sex distribution of one or more oral parafunctions observed in this study (table 4.4), is consistent with the findings of *Wanman and Agerberg (1986a), Shereef (1991), and Salih (1993)*; but contradicts those of others *(Egermark-Eriksson, 1981; Magnusson et al., 1985; Kononen et al., 1987; Pilley et al., 1992; Abdulla, 1992)*. This can be attributed to the fact that females in this study reported orofacial parafunctions (especially gum chewing, nail and object biting) significantly more than males which complies with the findings of *Shereef (1991), Abdulla (1992), and Salih (1993)*.

The remarkable awareness of tension relieving oral habits among females, in this study, gives additional explanation to the significant preponderance of TMDs among them. Parafunctions induce muscle spasm which in turn causes feeling of fatigue, facial pain, headache, and TMJ sounds which were significantly more frequent among females. However, no significant sex differences were found in relation to the awareness of bruxism and presence of dental wear (table 4.5). This is in agreement with the results of *Egermark-Eriksson et al. (1981) and Heikinheimo et al. (1989)*; while *Egermark-Eriksson et al. (1987)* reported dental wear of the canines to be more common in males than females. Moreover, other studies reported the preponderance of bruxism among males *(Nilner & Lassing, 1981; Helm et al., 1984)*, or females *(Shereef, 1991)*.

5.2.5 MULTIPLE REGRESSION ANALYSES:

The results of the multiple regression analyses have shown that the contribution of sex in explaining the variance of the signs and symptoms of TMDs (feeling of fatigue, facial pain, TMJ sounds, and the severity of clinical signs) became non-significant when malocclusion variables were introduced in the model (tables 4.6 and 4.7). This means that these sex differences were caused by unequal distribution of malocclusion variables in both sexes.

However, muscle tenderness to palpation was unrelated to malocclusion, but when parafunctions were included in the test as possible causes of muscle tenderness (independent variables), the inclusion of gum chewing and clenching in the model made the contribution of sex non-significant. This means that the sex difference was caused by females having more parafunctions (especially gum chewing) than males.

On the other hand, the participation of sex in the model was unaffected by the introduction of malocclusion variables for the dependent variables: maximal mouth opening capacity, recurrent headache, object biting and gum chewing.

5.3 DISTRIBUTION OF TMDs ACCORDING TO AGE:

There are wide differences in the results of the various studies in the literature concerning the age distribution of TMDs, which is probably because of the varying age range, sex distribution and race of the sample of each study.

5.3.1 SYMPTOMS OF TMDs:

The lack of increase in the prevalence of one or more symptoms in the present material (table 4.1) is in agreement with the results presented by *Egermark-Eriksson (1981), Nilner (1983c), Wanman and Agerberg (1986c), Ohno et al. (1988); Heikinheimo et al. (1989), and Keeling et al. (1994)*; but disagrees with that of *Magnusson (1986)*.

Locking of the jaw was found to increase in prevalence with age which is in agreement with the findings of *Helkimo (1974d) and Heikinheimo et al. (1989)*, and may be because of the increased risk to occupational trauma with age as stated by *Helkimo (1974d)*.

5.3.2 SIGNS OF TMDs:

Although non-significant the prevalence of one or more signs, in this study, was found to increase with age (table 4.2), which is consistent with the findings of other investigators *(Egermark-Eriksson, 1981; Nilner, 1983c; Gazit et al., 1984; Grosfeld et al., 1985; Ogura et al., 1985)*. However, *Verdonck (1994)* reported that TMDs signs decreased with age.

Tenderness to palpation of the posterior aspect of the TMJ, profound masseter and medial pterygoid muscles were found to decrease with age which is in agreement with *Grosfeld et al. (1985) and Verdonck (1994)* but disagrees with

Egermark-Eriksson et al. (1981), Magnusson et al.(1985), and Wanman and Agerberg (1986e) who reported a significant increase of these signs with age. This finding may be attributed to ear and tonsil infections being more common in children *(Ballantyne & Groves, 1971)*. Furthermore, the preponderance of grinding in the youngest group (table 4.3) may explain the decrease in the prevalence of the masseter muscle tenderness with age.

On the other hand, TMJ sounds and restricted mandibular movement were found to increase with age in the present sample. This is consistent with the results of previous studies *(Egermark-Eriksson et al., 1981; Nilner & Kopp, 1983; Gazit et al., 1984; Magnusson et al., 1985; Runge et al., 1989; Pilley et al., 1992)*, but contradicts the findings of others *(Grosfeld et al., 1985; Wanman & Agerberg, 1986e; Motegi et al., 1992; Verdonck, 1994; Keeling et al., 1994)*.

In the present study, the capacities of mandibular movement were not associated with age (table 4.3). This is consistent with the results of *Grosfeld et al. (1985), Heikinheimo et al. (1990), and Salih (1993)*; but contradicts those of *Egermark-Eriksson et al. (1981), Gazit et al. (1984), Magnusson et al. (1985), and Wanman and Agerberg (1986e)*.

5.3.3 RECURRENT HEADACHE:

Recurrent headache was not associated with age in this study (figure 5), which supports the findings of *Magnusson et al. (1985), Wanman and Agerberg (1986a)*, but contradicts those of others *(Nilner & Lassing, 1981; Egermark-Eriksson, 1982; Pilley et al., 1992)*.

5.3.4 ORAL PARAFUNCTIONS AND DENTAL WEAR:

The prevalence of one or more orofacial parafunctions was not associated with age in the current study (table 4.4), in agreement with the results of previous investigators *(Egermark-Eriksson, 1982; Nilner & Lassing, 1981; Kononen et al., 1987)*; but disagrees with those of others *(Wigdorowicz-Makowerowa et al., 1979; Gazit et al., 1984; Wanman & Agerberg, 1986d).*

On the other hand, grinding significantly decreased with age, similar to the results of *Reding et al. (1966), Nilner and Kopp (1983) and Heikinheimo et al., 1989)*, while there was a non-significant tendency of clenching to increase with age, which is in agreement with the findings of *Wanman & Agerberg (1986d) and Pilley et al. (1992).*

However, the severity of dental wear was found to increase with age in the current material (table 4.5), which is in agreement with *Egermark-Eriksson et al. (1981 & 1987)*; but disagrees with *Gazit et al. (1984)* who found no such increase. This increase of dental wear with age was least pronounced in the molar region, in agreement with *Egermark-Eriksson et al. (1981)*, which may be attributed to:

1- By 10 years of age (the youngest in the present sample) the first molars have fully erupted and mild dental wear may have taken place and so further dental wear may be minimal, whereas at that age premolars and canines have not erupted and so score 0 dental wear was registered, but after eruption in older patients dental wear begins.

2- Oral habits like nail and object biting were very common in the sample, and these may contribute to dental wear of the incisors.

3- Many of the patients had anterior crowding making the anterior teeth more susceptible for dental wear.

Gum chewing was reported more frequently by 15-19 year olds than both the younger and older age groups. This may explain the preponderance of pain on chewing, tenderness of the lateral pterygoid and insertion of the temporalis muscles among this age group. Moreover, the beginning of the eruption of the wisdom teeth at this age range may induce these symptoms.

5.3.5 MULTIPLE REGRESSION ANALYSES:

The contribution of age in explaining the variance of the dependent variables (tenderness of the medial pterygoid muscle and tooth grinding) became non-significant when malocclusion variables were introduced in the model. This means that these sex differences which were weakly significant to start with, were caused by the malocclusion variables (table 4.6 and 4.9).

On the other hand, the participation of age in the model was unaffected by the introduction of malocclusion variables for the remaining dependent variables.

5.4 THE EFFECT OF MALOCCLUSION ON TMDs:

The impact of closely correlated specific features of malocclusion on TMDs will be discussed together because the stepwise multiple regression analysis only enters the most significant independent variables and omits those explained by other variables from the model. These associations are presented in tables 4.6 to 4.10.

5.4.1 CLASS II DIVISION 1 AND EXCESSIVE OVERJET:

Class II, division 1, malocclusion was shown to be negatively associated with TMJ sounds reported by the patient. Other than that, no correlations were found between it and the signs and symptoms of TMDs. This complies with the findings of several studies *(Egermark-Eriksson et al., 1983; Mohlin, 1983; Pullinger et al., 1988 a&b)*, but contradicts those of *(Nilner, 1983b; Riolo et al., 1987; Mohlin et al., 1990; Egermark-Eriksson et al., 1990; al-Hadi, 1993)*. This variation in results may be because the latter investigators did not use the stepwise multiple regression analysis which can isolate the effect of excessive overjet from the effect of class II, division 1, malocclusion.

On the other hand, excessive overjet was positively associated with several signs and symptoms of TMDs (tables 4.6 and 4.7) which is consistent with those reported by *Riolo et al. (1987), Egermark-Eriksson et al. (1990), Kritsineli et al. (1992), and al-Hadi (1993)*; but contradicts those of others *(Egermark-Eriksson et al, 1983; Helm et al., 1984; Lieberman et al., 1985; Keeling et al., 1994)* who reported a non-significant association between overjet and TMDs. This may be attributed to the use of the latter investigators of 6 mm as an indication for excessive overjet, whereas, in this study overjet of greater than 8 mm was defined as excessive.

It appears from these results that excessive overjet is a more important factor in predisposing to TMDs than merely having a class II, division 1, occlusion i.e. class II patients with excessive overjet are at a greater risk for developing TMDs than mild class II, division 1, malocclusion patients. This support the suggestion that excessive protrusive movement on a chronic basis for both esthetic and incising purposes, in subjects with excessive overjet, may lead to hyperactivity of the muscles and can convey the forces of hyperfunction adversely towards weaknesses in the masticatory system *(Gawley, 1982; Parker, 1990)*.

Class II, division 1, malocclusion was weakly associated with maximal mouth opening capacity which complies with the finding of *Riolo et al. (1987)* who reported that subjects with more than 5 mm of overjet averaged significantly more maximal mouth opening than those with less overjet. This supports the previous statement of the independence between this type of malocclusion and TMDs as reduced mouth opening capacity has been suggested as an indicant of TMDs *(Shereef, 1991)*.

Excessive overjet was positively associated with mandibular protrusive capacity, in agreement with *Zimmer et al.(1991)*.

However, the negative association found in this study between excessive overjet and gum chewing may be the result of decreased number of occluding pairs of opposing teeth caused by the excessive overjet, leading to difficulties in chewing.

5.4.2 CLASS II DIVISION 2 AND DEEP BITE:

Class II, division 2, malocclusion was negatively associated with TMJ sounds reported by the patient, but not with any other sign or symptom of TMDs. This is in agreement with the finding of *al-Hadi (1993)*, but disagrees with those of other investigators *(Pullinger et al., 1988 a&b; Runge et al., 1989; Schupp et al., 1992).*

However, deep bite was associated with reported TMJ sounds, recurrent headache, tenderness of the anterior temporalis muscle and posterior aspect of the TMJ. This is consistent with the findings of several investigators *(Williamson, 1977; Brandt, 1985; Lieberman et al., 1985; DeLaat et al., 1986; Pullinger et al., 1988b; Kritsineli et al., 1992; Keeling et al., 1994).*

In this study, class II, division 2, malocclusion was positively associated with deep bite ($p<0.01$), as also, reported by several other authors *(Foster, 1982; Houston, 1982 & 1983; Owen, 1984; Carlsson & Ingervall, 1988).*

Hence, it seems that class II, division 2, malocclusion when isolated as a factor does not make a subject susceptible to TMDs, rather than that, the deep bite accompanying that type of malocclusion is the causative factor of the signs and symptoms of TMDs recorded in many studies in patients with this class of malocclusion.

The positive association between deep bite and gum chewing may contribute to its potential to develop TMDs. However, deep bite was negatively associated with nail biting which may be because they need more effort to obtain the edge to edge occlusion necessary for biting their nails; or opposingly, in nail biters who were susceptible to developing a deep bite, the frequent intrusive forces applied on the incisors during nail biting may have decreased their eruption power, and hence decreased the overbite.

5.4.3 CLASS III AND REVERSED OVERJET:

In the present study, true class III malocclusion was associated with several subjective symptoms of TMDs including: feeling of fatigue and stiffness, pain on movement, difficult wide mouth opening, and recurrent headache. This finding agrees with those of *Pullinger et al. (1988a) and Egermark-Eriksson et al. (1990)*. However, postural class III malocclusion was not associated with TMDs probably because the forward mandibular displacement, accompanying this class of malocclusion, was negatively associated with clenching of the teeth which was found to be strongly correlated with the signs and symptoms of TMDs in the present study and in several previous studies *(Droukas et al., 1984; Wanman & Agerberg, 1986f; Shereef, 1991; Abdulla, 1992; Salih, 1993)*.

The previous findings, therefore, suggest the priority of treatment of postural class III over true class III should be reconsidered based on their effect on the function of the masticatory system.

The positive association between postural class III malocclusion and nail biting may be explained in one of two ways: either the continuous bringing of the mandible forward in susceptible class I subjects made them postural class III, or the continuous bringing the mandible backward in postural class III patients to obtain the edge to edge occlusion necessary to bite their nails prevented them from transferring into true class III.

Reversed overjet was positively associated with tenderness of the anterior temporalis muscle which may explain the association with recurrent headache as the former two have been found to be correlated by *Shereef (1991)* and is approved by the results of this study.

Reversed overjet was strongly associated with restricted mandibular movement including decreased lateral and protrusive movement capacities as also found by *Mohlin et al. (1980) and Zimmer et al. (1991)*. However, it is has been considered as a normal functional feature of class III patients resulting from the reversed incisal guidance and not symptomatic of TMDs *(Mohlin et al., 1980)*.

5.4.4 OPEN BITE:

No association was found between open bite and the signs and symptoms of TMDs except for deviation on maximal opening. The latter association may be due to the lack of anterior guidance during gliding movements *(Mohlin , 1983)*. However, the scantiness of the associations with open bite complies with the results of *Lieberman et al.(1985) and Runge et al. (1989)*, but contradicts those of others *(Williamson, 1977; Egermark-Eriksson et al., 1983 & 1990; Brandt, 1985; Riolo et al., 1987; Kritsineli et al., 1992; Miyazaki et al., 1994)*; and may be the result of the negative association between open bite and tooth grinding as tooth grinding has been suggested as a predisposing factor of TMDs *(Shereef, 1991; Salih, 1993)*.

The reason why many investigators continue to suggest open bite as a major etiologic factor of TMDs may be because of its association with true class III malocclusion, which was confirmed by the results of this study. However, the effect of true class III malocclusion was isolated by the stepwise multiple regression analysis in this study, and hence, the effect of open bite was revealed to be only on the development of deviation on maximal mouth opening.

Digit sucking was explained by open bite and upper anterior spacing in the model and was directly correlated with posterior crossbite ($p<0.025$). These results are consistent with the reports of several authors *(Foster, 1982; Houston, 1982 & 1983; Nilner & Kopp, 1983)*.

Open bite was associated with decreased maximal mouth opening capacity which may be due to less need for opening the mouth widely because of the negative overbite, while deep bite patients require to open several more millimeters to overcome the overbite. Hence, when mouth opening capacity was assessed as the maximal interincisal distance only, with no regard to the overbite, *Riolo et al. (1987)* found that open bite subjects had larger mouth opening capacities than subjects with positive overbites.

5.4.5 CROSSBITES:

Inversion of incisors was only associated with pain on mandibular movement which may be due to the occlusal interference caused by this crossbite. This scantiness of associations with the signs and symptoms of TMDs may be because of the negative association with grinding and complies with the findings of *Mohlin et al. (1980), Mohlin (1983), and Egermark-Eriksson et al. (1983)*; but disagrees with that of *Egermark-Eriksson et al. (1990)*. This negative association with grinding may be because of the mandible being not free to perform lateral movements because of the inverted incisors.

Moreover, inversion of incisors was the only malocclusion variable to be associated with dental wear which reflects the necessity for urgent correction of inverted incisors. In the literature, various malocclusion variables have been related to dental wear, but most of the relations were weak and no general agreement is found *(Wigdorowicz-Makowerowa et al., 1979; Egermark-Eriksson, 1982; Nilner, 1983 a&b; Brandt, 1985; Gunn et al., 1988; Egermark-Eriksson et al., 1990)*.

Unilateral posterior crossbite was positively associated with tenderness of the anterior temporalis muscle which was also mentioned by electromyographic studies *(Haralabakis & Loutfy, 1964; Troelstrup & Moller, 1970; Ingervall & Thilander, 1975)* and supports the finding of the epidemiological study of

Egermark-Eriksson et al. (1983), but it was weakly negatively associated with tenderness of the medial pterygoid muscle which reflects the findings of *Ahlgren (1967)* that subjects with posterior crossbites have a different chewing pattern.

Unilateral posterior crossbite was also associated with TMJ sounds, in agreement with several investigators *(Brandt, 1985; Riolo et al., 1987; Egermark-Eriksson et al., 1990; Kritsineli et al., 1992)* and may be the result of asymmetrical condylar position in the glenoid fossa *(Iijima, 1990; O'Bryn et al., 1995)*.

A Highly significant association was detected between bilateral posterior crossbite and tenderness of the TMJ to posterior palpation and may reflect the great tension placed on the TMJ by cuspal interferences common in this type of malocclusion *(Ahlgren & Posselt, 1963)* and complies with the results of *Egermark-Eriksson et al. (1990)*.

Bilateral posterior crossbite was associated with the clinical dysfunction index (Di), and it was also found that subjects with bilateral posterior crossbites had more severe signs of TMDs than those with unilateral crossbites, which is in agreement with *Egermark-Eriksson et al. (1983)* and suggest a reconsideration of treatment priority commonly suggested for unilateral posterior crossbite over bilateral ones.

5.4.6 CROWDING/SPACING:

Upper anterior crowding was positively associated with TMJ sounds, both subjective and clinical, which is consistent with the findings of previous studies *(Lieberman et al., 1985; Keeling et al., 1994; Verdonck et al., 1994)*, but contradicts those of *Mohlin (1983), Helm et al. (1984), and Runge et al. (1989)* who found no relationship between crowding of the dental arches and TMDs. This association may be the result of premature contacts of the crowded teeth initiating clicking of the TMJ *(Egermark-Eriksson et al., 1983)*.

Lower anterior crowding was negatively associated with several signs and symptoms of TMDs (tables 4.6 & 4.7), which may be attribute to the negative influence of class II, division 2, malocclusion which is predominant among the patients with lower anterior crowding in the current sample.

However, lower anterior crowding was positively associated with tenderness of the posterior temporalis muscle which, also, may be the result of class II, division 2, malocclusion and deep bite which have been associated with strong retrusive activity of the posterior temporalis muscle *(Moller, 1966)*.

The negative association between upper anterior spacing and feeling of fatigue and the severity of symptoms (Ai), and absence of association with the clinical signs of TMDs, supports the statement of *Carlsson and Ingervall (1988)* that spacing is less likely to give rise to occlusal problems than crowding.

CHAPTER SIX

CONCLUSIONS AND SUGGESTIONS

6.1 CONCLUSIONS:

1- Subjective symptoms of TMDs were reported by 65.7% of the patients, while clinical signs of TMDs were observed in 81.8% of them, accordingly malocclusion patients were at greater risk for TMDs than normal population.

2- Only a few signs and symptoms of TMDs showed weak significant sex differences being more frequent in females than males.

3- The signs and symptoms of TMDs fluctuated with age, but generally, pain decreased with age, while dysfunction increased with age.

4- Recurrent headache and parafunctions were reported by 38.5% and 78.3% of the patients, significantly more by females than males.

5- Dental wear was diagnosed in nearly all the patients, increasing in severity significantly with age for all dental regions.

6- Class II malocclusion (both divisions) was unrelated to TMDs, while an overjet greater than 8mm and an overbite of 5mm or more predisposed to TMDs.

7- True class III malocclusion and reversed overjet were associated with TMDs, while postural class III malocclusion, forward mandibular displacement, open bite and midline shift were not.

8- Inverted incisors and posterior crossbite were positively associated with TMDs signs, especially bilateral posterior crossbite.

9- Upper anterior crowding appeared to predispose to TMDs, while lower anterior crowding, and anterior spacing were negatively associated with TMDs.

10- The incisor relationship seems to be more important than the general occlusion (Angle's classification) in the development of TMDs.

6.2 SUGGESTIONS:

1- Future studies using similar standardized methods on population samples of specific age groups are recommended to evaluate the role of malocclusion and other risk factors such as stress, socioeconomic and general somatic factors in TMDs.

2- A longitudinal study on TMDs among orthodontic patients, before, during and after treatment to observe the response of different types of malocclusion and evaluate the effect of different types of treatment methods.

3- It is suggested to follow up the TMD status of malocclusion patients who do not undergo orthodontic treatment in a longitudinal epidemiological study.

4- To include a screening for the signs and symptoms of TMDs in routine orthodontic examination seems justified to identify patients who should be observed more closely.

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Appendices

	Author	Sample		Age	Country	Prevalence%		Method
		F	M	(Years)		Symp.	Signs	
1	Agerberg & Carlsson (1973)	575	531	15-74	Sweden	57	-	Q
2	Helkimo (1974a)	165	156	15-65	Finland	57	88	I/E
3	Hansson & Nilner (1975)	82	987	20-65	Sweden	>23	79	Q/E
4	Norheim & Dahl (1978)	163	169	20-69	Norway	14	-	Q
5	Swanljung & Rantanen (1979)	339	253	18-64	Finland	58	41	I/E
6	Ingervall et al. (1980)	-	389	21-54	Sweden	12	60	Q/E
7	Rao & Rao (1981)	561	626	>16	India	-	20	E
8	Mohlin (1983)	272	-	20-45	Sweden	28	66	Q/E
9	Szentpetery et al. (1986)	315	285	12-85	Hungary	21	80	Q/E
10	Tervonen & Knuuttila (1988)	691	584	25-65	Finland	39	4	I/E
11	Locker & Slade (1988)	377	300	>18	Canada	48	-	T
12	Locker & Slade (1989)	91	57	18-82	Canada	64	82	T/E
13	Salih (1993)	465	467	16-54	Iraq	38	58	I/E

Appendix I: Prevalence of TMDS in different populations.

	Author	Sample		Age	Country	Prevalence%		Method
		F	M	(Years)		Symp	Signs	
1	Hansson & Oberg (1971)	26	37	67	Sweden	-	73	E
2	Agerberg & Osterberg (1974)	108	86	70	Sweden	23	74	I/E
3	Heloe & Heloe (1978)	128	113	65-79	Norway	15	27	I/E
4	Osterberg & Carlsson (1979)	198	186	70	Sweden	59	86	Q/E

Appendix II: Prevalence of TMDS in different old adult populations.

	Author	Sample		Age	Population	Prevalence %		Method
		F	M	(Year)		Symp	Sign	
1	Ingervall & Hedegard(1974)	-	287	17-20	Swedish inducties	12	-	Q
2	Molin et al.(1976)	-	253	18-25	Swedish inducties	12	28	Q/E
3	Wigdorowicz-	-	429	-	Polish medical students	-	57*	E
	Mackowerowa et al.(1979)	-	400	-	Polish military students	-	52*	
		-	1000	20-23	Polish young soldiers	-	36*	
		-	1000	39-45	Polish middle-age soldiers	-	45*	
4	Solberg et al.(1979)	370	369	19-25	American University students	26	76	Q/E
5	Heloe & Heloe(1979)	136	110	25	Norwegian population	24	-	
6	Helkimo et al. (unpublished data)	58	-	18-28	Swedish dental training nurses	45	60	Q/E
7	Droukas et al.(1984)	23	25	20-38	Swedish dental students	32	65	Q/E
8	Grosfeld et al.(1985)	192	208	19-22	Polish students	67		I/E
9	Pullinger et al.(1988a)	102	120	19-40	American dental students	35	41	Q/E
10	Schiffman et al.(1990)	250	-	22-25	American nursing students	57	93	Q/E
11	Abdulla(1992)	450	450	19-25	Mosul University students	54	60	I/E
12	Al-Hadi(1993)	311	289	18-22	Mosul University students	-	50	E
13	Nourallah & Johansson (1995)	-	105	20-29	Saudi dental students	36	37	I/E

*** Not including bruxism.**

Appendix III: Prevalence of TMDS in different young adult samples.

	Author	Sample		Age (year)	Country	Prevalence%		Method
		F	M			Symp.	Signs	
A	Cross-sectional studies:							
1	Grosfeld & Czarnecka (1977)	136 133	114 117	6-8 13-15	Poland	6 10	56 68	Q/E
2	Wigdorowicz-Makowerowa et al.(1979)	2100		10-15	Poland	-	27	E
3	Egermark-Erikson et al.(1981)	74 61 59	62 70 76	7 11 15	Sweden	39 67 74	33 46 61	Q/E
4	Nilner & Lassing(1981)	218	222	7-14	Sweden	36	72	I/E
5	Nilner(1981)	162	147	15-18	Sweden	41	77	I/E
6	DeVis et al.(1984)	225	226	3-6	Belgium	0.6	4	Q/E
7	Gazit et al.(1984)	181	188	10-18	-	-	56	Q/E
8	Grosfeld et al.(1985)	203	197	15-18	Poland	68	68	I/E
9	Ogura et al.(1985) Ohno et al.(1988)	387 716	396 699	10-15 15-18	Japan	14 10	6 12	E Q
10	Wanman & Agerberg(1986a,b)	139	146	17	Sweden	20	56	Q/E
11	Bernal & Tsamtsouris(1986)	70	79	3-5	America	38	36	Q/E
12	Kononen et al.(1987)	88	78	10-16	Finland	52	40	I/E
13	Vanderas(1988)	142	108	6-10	America	36	54	Q/E
14	Nielsen et al.(1989)	706		14-16	Denemark	-	30	E
15	Shereef(1991)	436	414	15-19	Iraq	55	32	I/E
16	Motegi et al.(1992)	7337		6-18	Japan	12		Q/E
17	Verdonk et al.(1994)	1182		12&15	Japan	23		Q/E
B	Longitudinal studies:							
1	Magnusson et al. (1985)	66 53		7→11 11→15	Sweden	35→ 62 60→ 66	32→ 66 46→ 66	Q/E
2	Wanman & Agerberg(1986d,e)	285→258		17→19	Sweden	20→ 20	56→ 50	Q/E
3	Heikinheimo et al.(1989)	84	83	12→15	Sweden	64→ 67	-	Q/E
4	Pilley et al.(1992)	508→ 398	507→ 393	12→15	Sweden	-	38→ 55	Q/E

Appendix IV: Prevalence of TMDS in different samples of adolescents and children.

	Author	Sample		Age (Year)	Country	%	Definition of headache
		F	M				
A	General population:						
1	Agerberg & Carlsson(1972)	575	531	15-74	Sweden	18	*
2	Helkimo(1974a)	165	156	15-65	Finland	21	**
3	Ingervall et al(1980)	-	389	21-54	Sweden	5	*
4	Mohlin(1983)	272	-	20-45	Sweden	9.6	*
5	Szentpetery et al.(1986)	315	285	12-85	Hungary	23.7	*
6	Tervonen & Knuuttila(1988)	691	584	25-65	Finland	10	*
7	Locker & Slade(1988)	377	300	>18	Canada	17.4	**
8	Salih(1993)	465	467	16-54	Iraq	37.7	*
B	Young adults:						
1	Ingervall & Hedegard(1974)	-	287	17-20	Sweden	7.5	often
2	Molin et al.(1976)	-	253	18-25	Sweden	11	*
3	Solberg et al.(1979)	370	369	19-25	America	12.5	-
4	Heloe & Heloe(1979)	136	110	25	Norway	28	some or much
5	Droukas et al.(1984)	23	25	20-38	Sweden	8	*
6	Helm et al.(1984)	428	330	28-34	Denemark	10	*
7	Pullinger et al.(1988a)	102	120	19-40	America	11	moderate & severe
8	Abdulla(1992)	450	450	19-25	Iraq	41.4	*
C	Adolescents and children:						
1	Geering-Gaerny & Rakosi(1971)	281		8-14	-	12	-
2	Nilner & Lassing(1981)	218	222	7-14	Sweden	14	*
3	Nilner(1981)	162	147	15-18	Sweden	16	*
4	Egermark-Eriksson(1982)	194	208	7,11,15	Sweden	23	recurrent
5	Wanman & Agerberg(1986a)	139	146	17	Sweden	12	*
6	Bernal & Tsamtsouris(1986)	70	79	3-5	America	1	frequent
7	Kononen et al.(1987)	88	78	10-16	Finland	13	*
8	Shereef(1991)	436	414	15-19	Iraq	33.7	*
9	Widmalm et al.(1995a)	203		4-6	America	17	*
D	Longitudinal studies:						
1	Magnusson et al.(1985)	66		7→11	Sweden	8→30	1-2 times a month
		53		11→15		17→30	
2	Wanman & Agerberg(1986d)	126	138	17→19	Sweden	12→9	*
3	Heikinheimo et al.(1989)	84	83	12→15	Sweden	7→9	*

* Once a week or more
** Twice a week or more

Appendix V: Prevalence of recurrent headache in different population samples.

	Author	Age	Grinding	Clenching	*	Nail biting	Object biting	lip- (c), cheek-(d), togue-biting(e) gum chewing(f)	finger sucking	one or more
A	<u>General population:</u>									
1	Agerberg & Carlsson (1972)	15-74	10	20		15	5	8(c), (d), 5 (e)		50
2	Helkimo (1974c)	15-65			21		9	12(c-e)		41
3	Ingervall et al. (1980)	21-54	13	5	16		7.5			26
4	Mohlin (1983)	20-45	9.5	11						
5	Locker & Slade (1988)	18-65	18	16						
6	Salih (1993)	16-54	10	9	17	10	2	2(c&d), 5(f)		28
B	<u>Young adults:</u>									
1	Reding et al. (1966)	16-36			5					
2	Helkimo (1974d)	15-24			15		23	19(c-e)		31
3	Ingervall & Hedegard (1974)	18-25	4	7						
4	Molin et al. (1976)	18-25	4	10	13					
5	Droukas et al. (1984)	20-38			25					
6	Pullinger et al. (1988a)	19-40	25	23						
7	Abdulla (1992)	19-25	7	29	34	10	11	2(c&d), 24(f)		57
C	<u>Children and Adolescents:</u>									
1	Reding et al. (1966)	3-17			5					
2	Lindqvist (1971)	10-13	15	1						
3	Kuch et al. (1979)	5-6			15					
4	Wigdorowicz-Makowerowa et al. (1979)	10-15			5			40(a-e)		46
5	Egermark-Eriksson et al. (1981)	7 11 15	20 10 11	9 11 11	25 20 19			51(a-e) 72(a-e) 77(a-e)	20 3 0	60 78 78
6	Nilner & Lassing (1981)	7-14	16	20		50		44(d&e)	5	77
7	Nilner (1981)	15-18	7	13		45		47(d&e)	0	74
8	Gazit et al. (1984)	10-18	6	7						
9	Bernal & Tsamtsouris (1986)	3-5	17			10	3	1(c)	17	40
10	Kononen et al. (1987)	10-16	14	11		44	35	29(c&d)	2	75
11	Shereef (1991)	15-19	18	30	39	20	25	29(c&d), 24(f)		70
D	<u>Longitudinal studies:</u>									
1	Magnusson et al. (1985)	7→11			26→27			44→65(a-e)		55→74
		11→15			19→29			60→61(a-e)		70→72
2	Wanman & Agerberg (1986a,d)	17→19	8→12	11→18	14→25	43→33		38→27(c-e)		68→59
3	Heikinheimo et al. (1989)	12→15			36→30					

* Grinding and\or clenching.

Appendix VI: Prevalence of oral parafunctions in different population samples.

CASE SHEET

INTERVIEW

A-GENERAL INFORMATION:

Name : ______ **Date :** / /199

Age ____ **Year** **Sex** *(1=Female, 2=Male)* ☐ **Serial number** ☐

(1=yes, 0=no)

1- Have you been orthodontically treated ? ☐

2- Have you been treated for TMDs ? ☐

B- ANAMNESTIC INDEX (Ai):

1- Do you notice TMJ sounds? ☐

2- Do you suffer from feeling of fatigue of the jaws? ☐

3- Do you suffer from feeling of stiffness on awakening or on movements of the lower jaw? ☐

4- Do you notice difficulty in opening your mouth wide? ☐

5- Do you have locking in your jaw on mandibular movement? ☐

6- Do you have luxation in your jaw on mandibular movement? ☐

7- Do you feel pain on movement of the mandible?

8- Do you feel pain in the region of the TMJ or of the masticatory musculature (cheeks)?

Ai Score ☐

C- RELATED FACTORS:

1- Do you suffer from recurrent headache (Once a week or more) ? ☐

2- Do you frequently do:- Tooth grinding ☐

- Tooth clenching ☐
- Nail biting ☐
- Biting on foriegn objects ☐
- Lip - and cheek biting ☐
- Digit sucking (present & recent past) ☐
- Gum chewing ☐

CLINICAL EXAMINATION

A-CLINICAL DYSFUNCTION INDEX (Di):

1- Mandibular motility Score ☐

☐ mm + ☐ mm = ☐ mm
overjet protrusion maximal protrusion

☐ mm + ☐ mm = ☐ mm
overbite inter-incisal distance maximal mouth opening

☐ mm mm
right left

2-Pain on movement score ☐

☐ opening ☐ closing ☐ protrusion ☐ right ☐ left

3-Impaired TMJ function score ☐

☐ click ☐ crepitation ☐ devation>mm ☐ lock ☐ luxation

4- TMJ pain(*1=right,2=left,3=both sides)* score ☐

☐ lateral ☐ posterior

5- Muscle palpation score ☐

☐ posterior temporalis ☐ anterior temporalis ☐ profound masseter ☐ superficial masseter ☐ medial pterygoid ☐ lateral pterygoid ☐ temporalis insertion

Total score ☐

Di index ☐

B-EXAMINATION OF OCCLUSION:

1-Angles cassification
(1=Class I, 2=Class II, division1, 3=Class II, division2, 4=Postural class III, 5=True class III) ☐

2-Overjet= ☐ mm, reversed overjet *(1=yes, 0=no)*

3-Overbite= ☐ mm, open bite(<0mm) ☐ , deep bite (> 5mm) ☐

4- Inversion of incisors *(number of involved teeth 1,2 or 3)* ☐

5-Anterior crossbite *(1=yes, 0=no)* ☐

6-Posterior crossbite *(1=unilateral, 2=bilateral, 3=scissors bite)* ☐

7-Mandibular displacement (1=forward, 2=right, 3=left) ☐

8-Crowding *(1=anterior, 2=posterior, 3=both)* ☐

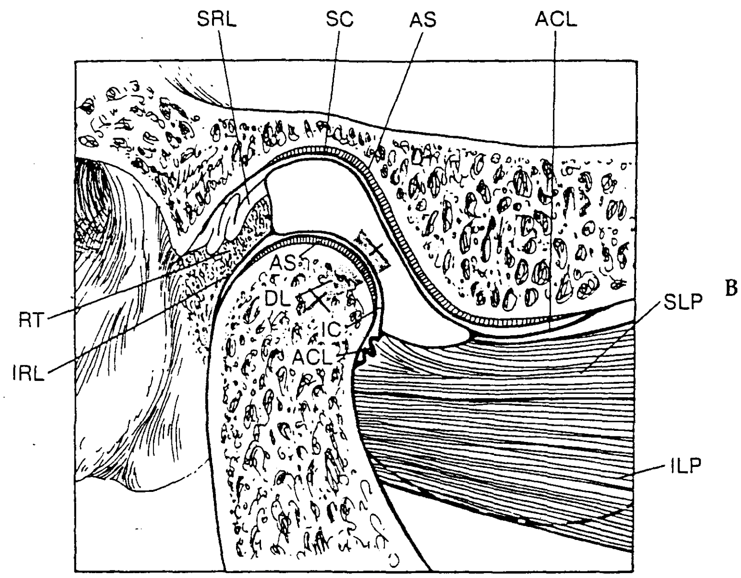
9-Spacing *(1=anterior, 2=posterior, 3=both)* ☐

10-Midline shift ☐ mm *(1=right, 2=left)* ☐

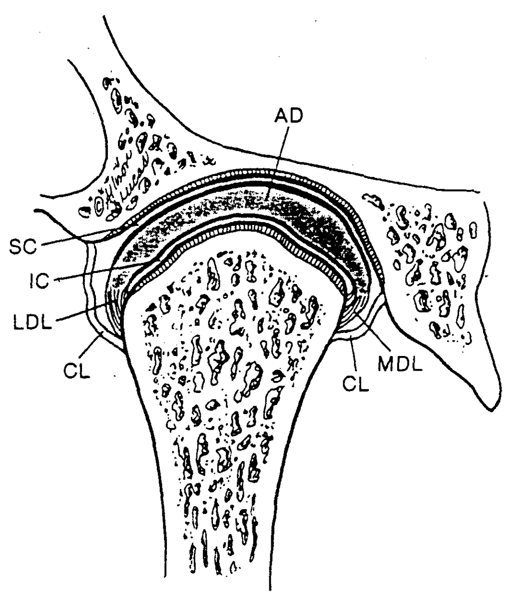
11-Dental wear *(0=none, 1=into enamel, 2=into dentine, 3=till 1/3 of the crown, 4=More than 1/3 of the crown)*

☐	☐	☐	☐
incisors	canines	premolars	molars

Appendix VIII: Anatomy of the TMJ



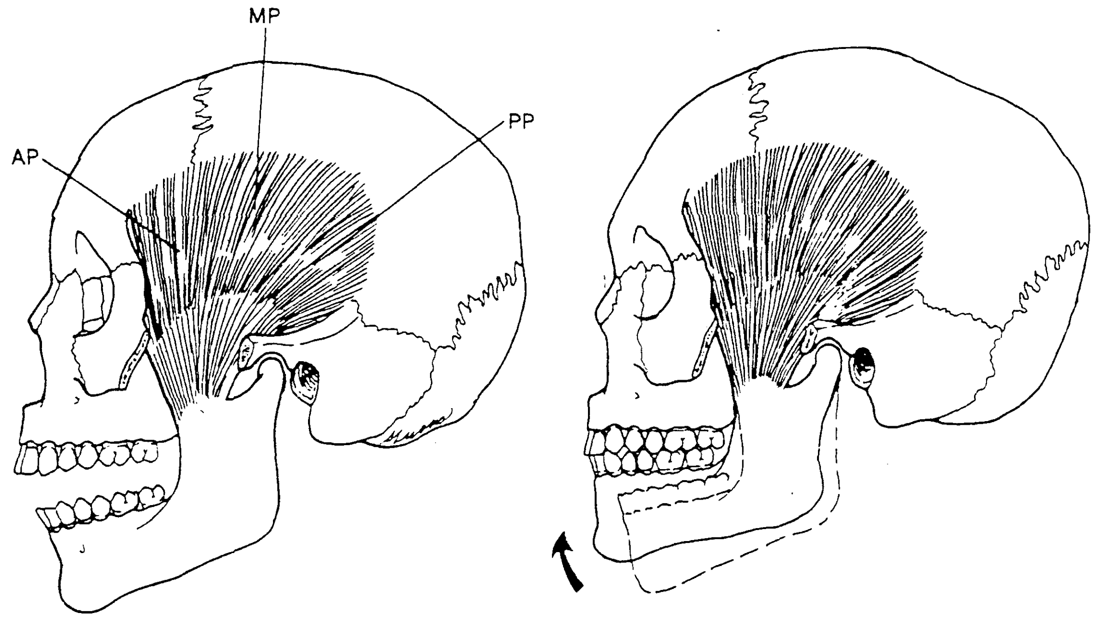
. Temporomandibular joint. **A**, Lateral view and, **B**, diagram showing the anatomic components: *RT*, retrodiscal tissues; *SRL*, superior retrodiscal lamina (elastic); *IRL*, inferior retrodiscal lamina (collagenous); *ACL*, anterior capsular ligament (collagenous); *SLP* and *ILP*, superior and inferior lateral pterygoid muscles; *AS*, articular surface; *SC* and *IC*, superior and inferior joint cavity; *DL*, discal (collateral) ligament.



Temporomandibular joint (anterior view). The following are identified: *AD*, articular disc; *CL*, capsular ligament; *LDL*, lateral discal ligament; *MDL*, medial discal ligament; *SC*, superior joint cavity; *IC*, inferior joint cavity.

Appendix IX: Anatomy of the muscles of mastication.

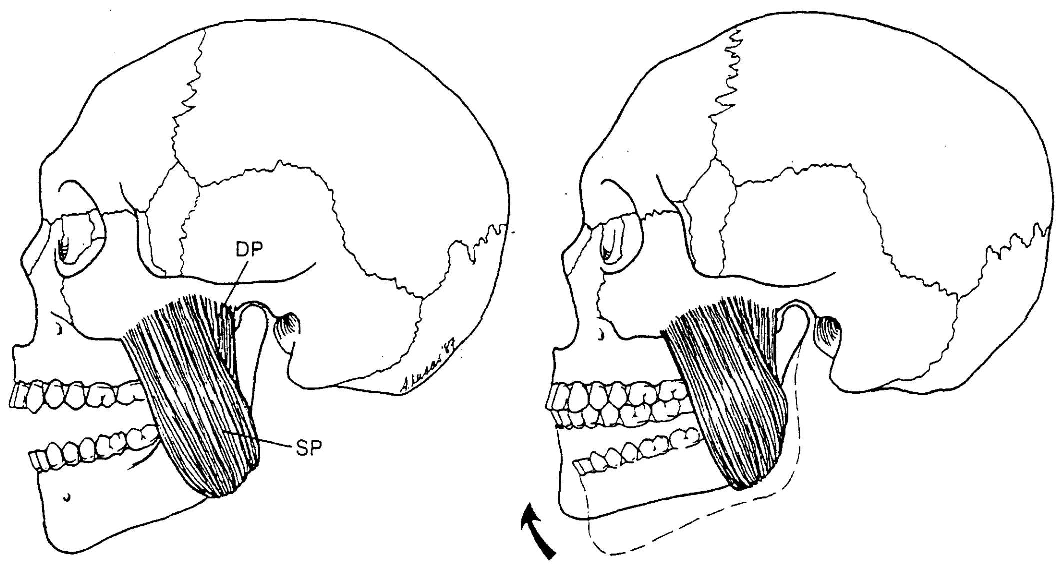
A



B

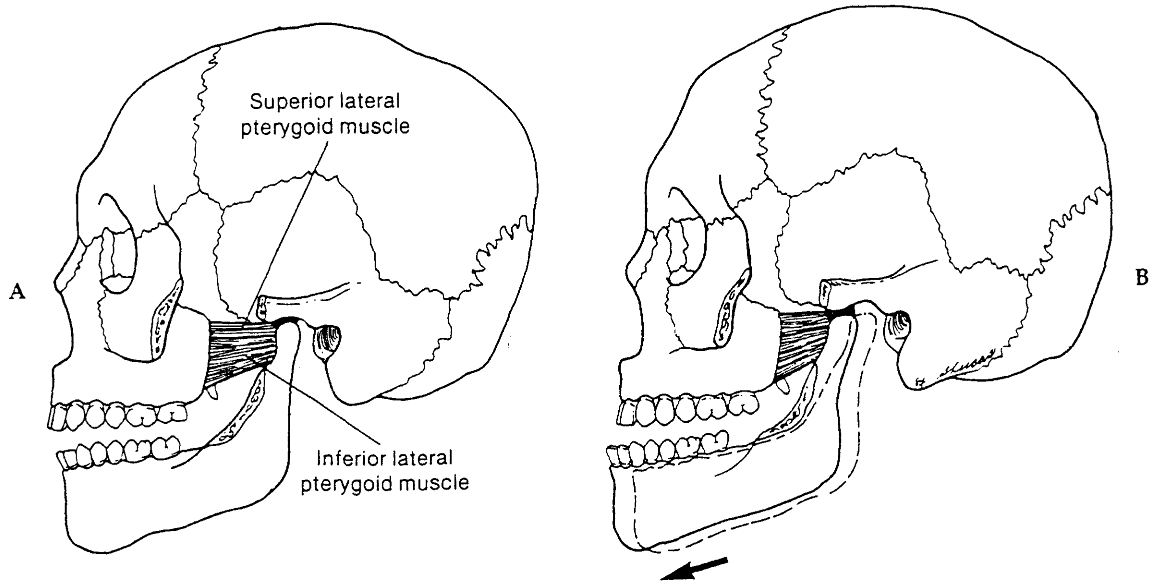
A, Temporal muscle. Note the following: *AP*, anterior portion; *MP*, middle portion; *PP*, posterior portion. B, Function: elevation of the mandible. The exact movement is determined by the location of the fibers being activated.

A

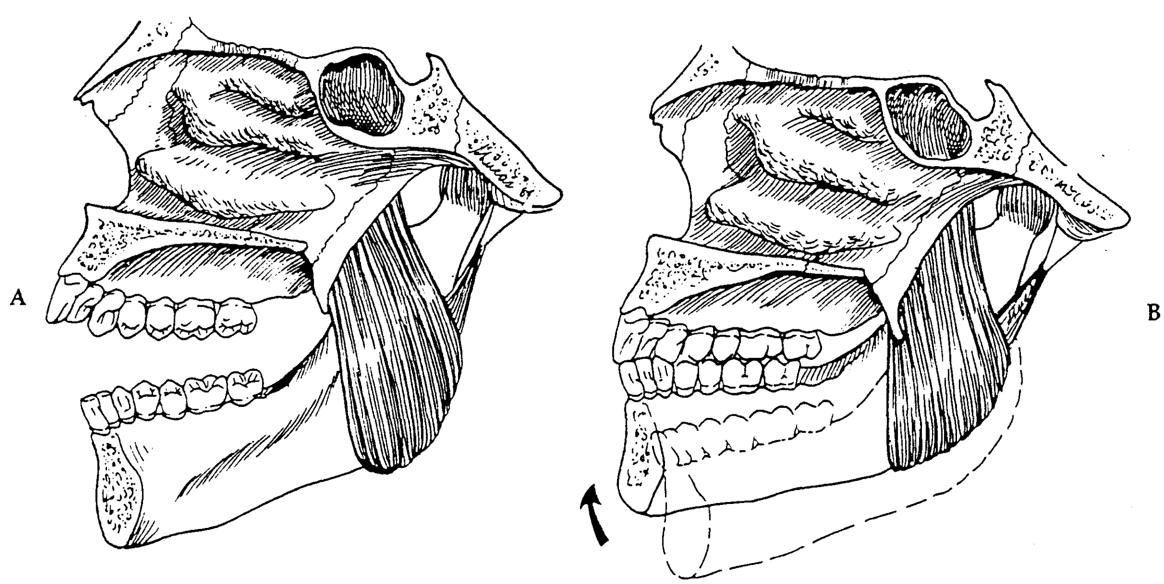


B

A, Masseter muscle. *SP*, Superficial portion; *DP*, deep portion. B, Function: elevation of the mandible.



A, Inferior and superior lateral pterygoid muscles. B, Function of the inferior lateral pterygoid: protrusion of the mandible.



A, Medial pterygoid muscle. B, Function: elevation of the mandible.

الخلاصة

بحثت اضطرابات الفك السفلي الصدغي (TMDS) في عينة ضمت ١٤٣ مريضا عن تقويم الاسنان قبل العلاج (٤١ ذكر و ١٠٢ انثى) وتتراوح اعمارهم بين ١٠ و ٢٥ سنة في كلية طب الاسنان/جامعة بغداد/العراق. كان هدف الدراسة تحديد شدة وانتشار هذه الاضطرابات عند المرضى المصابين بسوء الأطباق وتوضيح العلاقة بين سوء الأطباق واضطرابات الفك السفلي الصدغي.

سجلت العلامات والاعراض السريرية حسب المبادىء المقدمة من قبل هلكيمو عام ١٩٧٤. أظهرت الدراسة أن ٦٥,٧٪ من المرضى ذكروا أعراض هذه الاضطرابات و ٢٢,٤٪ سجلوا كحالات حادة. كانت الاعراض الشائعة هي الاصوات المفصلية والشعور بالارهاق. اما العلامات فلوحظت عند ٨١,٨٪ من العينة ووصف ٢٢,٤٪ كمعتدل و ٦,٣٪ كحاد. كانت العلامات الشائعة هي حساسية المفصل والعضلات تجاه الجس. وقد وجد أن الاختلافات الموضوعية بين الاجناس كانت قليلة وضعيفة. الحساسية تجاه الجس تناقصت مع العمر بينما أضطرابات الوظيفة زادت من العمر.

سجل الصداع المتكرر في ٣٨,٥٪ من العينة، مميزا في الاناث اكثر مما في الذكور. العادات الفموية سجلت في ٧٨,٣٪ من العينة وكانت العادات الفموجهية مميزة في الاناث أكثر منها في الذكور. أما انسحال الاسنان فلوحظ في كل المرضى تقريبا، ويزداد بشكل ملحوظ مع العمر.

الصنف الثاني من سوء الاطباق بأنموذجيه الاول والثاني لا علاقة له بهذه الاضطرابات، بينما بروز الاسنان الامامية أكثر من ٨ ملم والتغطية المساوية لـ ٥ملم أو أكثر كانت تؤهل لهذه الاضطرابات. الصنف الثالث الحقيقي من سوء الاطباق والبروز المعكوس ترافقا مع هذه الاضطرابات بينما لا توجد علاقة مع الصنف الثالث الكاذب من سوء الاطباق والانزياح الامامي للفك السفلي والعضة المفتوحة.

وقد لوحظ وجود علاقة طردية بين العضة الامامية المعكوسة جزئيا والعضة الخلفية المعكوسة مع هذه الاضطرابات وبخاصة العضة المعكوسة الخلفية ثنائية الجانب. كان التزاحم الامامي العلوي أحد العوامل المؤهلة لهذه الاضطرابات بينما كان للتزاحم الامامي السفلي والفراغات الامامية العلوية والسفلية علاقة عكسية مع هذه الاضطرابات.

اظهرت نتائج الدراسة أن علامات واعراض أضطرابات الفك السفلي الصدغي أوسع أنتشارا في مرضى تقويم الاسنان مما في المجتمع مشير الى تاثير سوء الاطباق السلبي على وظيفة الجهاز المضغي. وكذلك كانت العلاقة بين القواطع أهم من حالة الاطباق عموما في التاهيل لهذه الاضطرابات.

أضطرابات الفك السفلي الصدغي
لدى عينة من مرضى تقويم الأسنان قبل العلاج
وعلاقته بسوء الأطباق

رسالة مقدمة الى
كلية طب الأسنان / جامعة بغداد
كجزء من متطلبات نيل درجة الماجستير
في تقويم الأسنان

من قِبل
أكرم فيصل حسين
بكالوريوس طب وجراحة الفم والأسنان

ربيع الثاني ١٤١٧ آب١٩٩٦