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# Occlusion, Orthodontic Treatment and Temporomandibular Disorders: Myths and Scientific Evidences

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## 1. Introduction

Temporomandibular joint (TMJ) disorders and related masticatory muscle pain represent the most common chronic orofacial pain condition, and are the main cause of pain of non dental origin in the oro-facial region including head, neck and face (de Leeuw 2008). The etiology of temporomandibular disorders (TMD) is multifactorial. One of historical proposed factors was improper occlusion (Egermark-Eriksson et al 1990, Kirverskari et al 1992, Pullinger 1993).

In the late 1980s the attention of the orthodontic community regarding TMD was awakened following litigation involving orthodontic treatment as the cause of TMD in an orthodontic patient in the US court. The orthodontist at cause lost the case only because at that time there was a lack in evidence based medicine literature (Pollack 1988).

In 1987 the Board of Trustees of the American Association of Orthodontists (AAO) passed a motion "that the AAO immediately initiate a program to conduct documented studies for the purpose of determining the relationship, or lack thereof, between orthodontic treatment and temporomandibular joint disorders." They also moved to form a new task-oriented committee, the Scientific Studies Committee, to conduct the program. Early in 1988, the committee was formed, consisting of persons with recognized knowledge in this area but with differing backgrounds: a prosthodontist, an oral pathologist, a general practitioner, and two orthodontists. Their conclusion was that orthodontic treatment generally is not a primary factor in TMD (Behrents and White 1992).

Since then many important investigations have been conducted, but still the possible association between orthodontic therapy and TMD signs and symptoms is a matter of debate among orthodontists, orthognatic surgeons, dentists and dental patients.

With the development of new aesthetic orthodontic techniques (lingual orthodontics, invisalign etc.) more adults seek orthodontic treatments, and therefore there appears to be an increased likelihood of orthodontic patients having TMD. Orthodontist should be capable to recognize the signs and symptoms of TMD already during the anamnestic

appointment, to inform the patient of the finding, to point it out in the patient file, and if necessary to refer the patient to an Orofacial/TMD specialist.

The objective of this chapter is to discuss the effectiveness of orthodontic intervention in reducing symptoms in people with temporomandibular disorders and to establish if there is any evidence based data that proves that active orthodontic intervention leads to TMD.

In order to fulfill these objectives the following questions should be asked:

1. Does occlusal interferences cause TMD?
2. Does malocclusion cause TMD?
3. Does orthodontic treatment cause TMD?
4. Does orthodontic treatment cure or prevent TMD?

## 2. Temporomandibular disorders

Temporomandibular disorder (TMD) is a collective term that embraces a number of clinical problems that involve the masticatory muscles, the TMJs and its associated structures, or both. TMD is considered a musculo-skeletal disorder. It is the most prevalent clinical entity affecting the masticatory apparatus, and is the main cause of pain of non dental origin in the oro-facial region (de Leeuw 2008). The main TMD symptom is pain in the masticatory muscles, preauricular area and/or TMJ. As usual in all the musculo-skeletal disorders, pain increases during masticatory function. Other common signs or symptoms are limited or altered jaw movements, joint noises (eg. clicks, crepitus, etc), earache, headache, non specific dental tooth pain etc (Carlsson and de Boever 1994; Dworkin and LeResche 1992). For details regarding the guidelines for classification, assessment and management of TMD please refer to de Leeuw 2008.

The prevalence of TMD signs (e.g. abnormal jaw movements, joint noises, and tenderness on palpation) in the general population, as demonstrated by epidemiologic studies ranges up to 75% of the population. Approximately 33% of the population has at least one symptom (e.g. facial pain, joint pain) (Rugh 1985; Schiffman 1988; Friction and Schiffman 1995).

It is important to state that symptoms and signs are not real muscular or articular compound temporomandibular disorders. A single symptom or sign from the masticatory system is not synonymous with TMD, or automatically leads to a TMD diagnosis. In order to diagnose TMD formal diagnostic criteria should be fulfilled. For more details regarding the diagnosis of TMD, please refer to the AAOP guidelines (de Leeuw 2008), or to the Research Diagnostic Criteria for TMD (Dworkin and Le Resche 1992).

The aetiology and the pathophysiology of TMD are poorly understood. It is generally accepted that it is a multifactorial phenomenon. Contributing factors (central, peripheral, behavioral psychological, physical, etc) may predispose, initiate, or perpetuate temporomandibular disorders. Normally great physiologic and external forces are absorbed in the masticatory system with no consequence. But, if the forces exceed the individual genetic- physiologic tolerance the system may undergo detrimental changes. When the structural tolerance is exceeded breakdown will occur in the weakest structure of the system (teeth, muscles or joints) (Okeson, 2003). In the past occlusion was considered to be the most important contributing factor in TMD, but more recent studies concluded that occlusal factors play no role in the developing of TMD (see below) .

### 3. Occlusion and occlusal adjustment

Occlusion is defined as "the static relationship between the incising or occlusal surfaces of the maxillary or mandibular teeth or tooth analogues. The occlusion should be balanced and as stress free as possible" (The glossary of prosthodontics terms, 2005)

When occlusion was recognized as the main etiologic factors of bruxism and TMD, one of the main therapies used was occlusal adjustment that tried to eliminate all "tooth contacts that inhibit the remaining occluding surfaces from achieving stable and harmonious contacts (occlusal interferences) and may produce pathologic changes in the stomatognathic system (Bakke et al 1992). With time more and more evidenced based data accumulated against this invasive, irreversible technique. We should keep in mind that the prevalence of malocclusion is high: 42 % of the population exhibit Angle class 1, 23 % is class 2 malocclusion and 4% have class 3 malocclusion (Gremillion 1995). In other words, only 31% of the population has a normo-occlusion ("ideal occlusion") according to Angle's classification. Does 69% of the population suffer from TMD, and need to be treated? The answer is definitively NO!

### 4. Temporomandibular disorders & occlusion

The possible relationship between malocclusion and TMD was first reported in 1934 by the otorhinolaryngologist Costen (Costen 1934). After analyzing 11 patients Costen hypothesized that dental changes (e.g. loss of vertical dimension and deep bite) led to anatomical changes in the temporomandibular joints, creating a syndrome composed of impaired hearing, tinnitus, dizziness, burning sensation in the throat and pain of unknown origin on side of face. The treatment proposed by Costen was "correction of the overbite, renewal of molar support to take pressure off the condyle...". The Costen's syndrome converted the temporomandibular disorders into another dental discipline. Dentists started treating patients suffering from "Costen syndrome" with bite raising appliances that augmented the vertical occlusal dimension of the face.

Old myths regarding the relationship between orthodontics treatment and TMD were twofold: In one hand, the myths that orthodontic treatment when done according to specific functional occlusion guidelines (gnathologic principles) reduces the likelihood of subsequently developing temporomandibular disorders, was rebutted. On the other hand the fact that the use of certain traditional orthodontic procedures and/or appliances may increase the likelihood of subsequently developing temporomandibular disorders could not be evidence proved (Rinchuse and Kandasamy , 2009). Many common myths among orthodontists were discussed and declined (Rinchuse and Kandasamy, 2009). The myths were that people with certain types of untreated malocclusion (eg. class II Division 2, deep overbite, and crossbite), excessive incisal guidance or people with gross maxilla - mandibular disharmonies are more likely to develop TMD. Other myths discussed were that pre-treatment radiographs of both TMJs should be taken before starting orthodontic treatment since the position of each condyle in its fossa should be assessed and corrected. The myth rebutted was that adult patients who have some type of occlusal disharmony along with the presence of temporomandibular disorder symptoms will probably require some form of occlusal correction. Finally, they could not find any evidence that retrusion

of the mandible (because of natural causes or after treatment procedures) may cause the articular disc to slip off the front of the condyle and become a major factor in the aetiology of temporomandibular disorders. The assumption that premolar extractions in the upper arch can cause a posterior displacement of the condyle which in turn could be associated with increased risk of joint dysfunction was also refuted (Bonilla et al 1999; Keshvad and Winstanley 2001; Gallo et al 2005). It can be concluded that since none of the above was ever proven, and accordingly cannot stand as evidence based medicine, clinicians should refrain from adopting therapeutic procedures based on it.

Micheloti et al (2005) investigated the effect of an acute occlusal interference on habitual muscle activity. Each individual was monitored for 6 weeks in 4 different conditions: 1.interference free at the beginning, 2.active interference, 3.dummy interference, 4. interference free at the end .The activity of the masseter muscle ipsilateral to the interference side was recorded by a portable EMG recorder. The response of the masticatory system to active occlusal interferences was a reduction in daytime habitual activity of the masseter muscle. None of the subjects reported signs and/or symptoms of TMD. It should be kept in mind that this study was performed on healthy subjects (without present or passed history of TMD). It may be possible that patients suffering already from TMD react differently to an experimentally introduced occlusal interference due to a deficiency in their adaptation capacity (Le Bell et al 2002). This hypothesis is also based in the observation that TMD patients do keep their teeth in contact more often during daytime (Chen, 2005) and therefore are more likely to feel the interference as a disturbing factor (Le Bell et al, 2006; Cao et al 2009). To test this assumption Le Bell et al (2002) performed a randomized double-blind clinical set-up that included healthy women without TMD as well as women with an earlier TMD history. Both groups were randomly divided into true and placebo interference groups. The subjects without a TMD history showed fairly good adaptation to the interferences, but the subjects with a TMD history and true interferences showed a significant increase in clinical signs compared to the other groups. The authors suggest that the etiological role of occlusal interferences in TMD may not have been correctly addressed in previous studies with artificial interferences and allow no conclusions as regards TMD etiology. Bell's group further analyzed the subjective reactions of these individuals. They found that the most prominent symptoms were occlusal discomfort and chewing difficulties. The group reached the conclusion that difference in outcome between the groups with and without a TMD history suggests that there are individual differences in vulnerability to occlusal interferences (LeBell 2006). In a third study (Niemi et al, 2006) the group tested the psychological factors and responses to artificial interferences in subjects with and without a history of TMD. They concluded that psychological factors appear significant for the symptom responses to artificial interferences, and they seem to play a different role in responses in subjects with an earlier TMD history compared to those without.

An occlusal interference animal model was conducted on rats (Cao 2009) by directly bonding crowns of different heights on their molars. The rats showed bilateral mechanical hyperalgesia in the masticatory muscles. The induced hyperalgesia remained 6 days after removal of the crowns and was reduced by injecting N-methyl-D-aspartate antagonist, suggesting a central sensitization mechanism. The animal model described mimics clinical masticatory muscle pain and provided a method to further investigate mechanisms of occlusion - related muscle hyperalgesia, and to explore possible pain management strategies.



Christensen and Rassouli (1995) placed a rigid unilateral intercuspatal interference in 12 subjects, and obtained bipolar surface electromyograms from the right and left masseter muscles during brisk and forceful clenching on the interference. On the side opposite the interference, myoelectric clenching activity was significantly reduced. Correlation analyses showed that the interference elicited a non-linear (complex) co-ordination of the amplitude, but not the duration, of bilateral masseteric clenching activity, i.e. frequently there was significant motor facilitation on the side of the interference, and significant motor inhibition on the side opposite the interference. The author further performed theoretical considerations that predicted that the observed contraction patterns would easily lead to frontal plane rotations of the mandible.

This was further supported by Clark et al (1999). The conclusion of their literature review was that experimental occlusal interferences may induce transient local tooth pain, loosening of the tooth, a slight change in postural muscle tension levels, chewing stroke patterns, and sometimes a clicking joint. They were of the opinion that since such findings are present in relatively asymptomatic patients, these data do not prove that occlusal interferences are causally related to a chronic jaw muscle pain or TMD.

Finally it could be hypothesized that subjects who are occlusally hypervigilant and or predisposed to suffer from TMD may be disturbed by occlusal interferences and increase the activity of the masticatory muscles which leads to pain and dysfunction as demonstrated by McDermid et al (1996); Raphael et al (2000); Hollins et al (2009). In some cases a very serious intractable disorder may be induced by occlusal changes. This disorder was term by Clark (2003) occlusal dysesthesia and is defined as "a persistent uncomfortable sense of maximum intercuspation after all pulpal, periodontal, muscle and TMJ pathologies have been ruled out and a physically obvious bite discrepancy cannot be observed". This serious disorder is was previously termed by Marbach et al (1983) "phantom bite syndrome".

## **5. Temporomandibular disorders & orthodontics**

An article by McNamara et al (1995) represents the evolution of a solicited manuscript first presented at the International Workshop on the TMDs and Related Pain Conditions, sponsored by the National Institute of Health (Hunt Valley, Md., April 17 to 20, 1994).

Its conclusions were: "(1) signs and symptoms of TMD may occur in healthy persons; (2) signs and symptoms of TMD increase with age, particularly during adolescence, until menopause, and therefore TMDs that originate during orthodontic treatment may not be related to the treatment; (3) in general, orthodontic treatment performed during adolescence does not increase or decrease the chances of development of TMD later in life; (4) the extraction of teeth as part of an orthodontic treatment plan does not increase the risk of TMD; (5) there is no increased risk of TMD associated with any particular type of orthodontic mechanics; (6) although a stable occlusion is a reasonable orthodontic treatment goal, not achieving a specific gnathologic ideal occlusion does not result in signs and symptoms of TMD; and (7) there is little evidence that orthodontic treatment prevents TMD, although the role of unilateral posterior crossbite correction in children may warrant further investigation." (McNamara et al, 1995; McNamara and Turp 1997).

Pullinger et al (1993) used a multiple logistic regression analysis to compute the odds ratios for 11 common occlusal features for asymptomatic controls vs. five temporomandibular disorder groups. They found that the following features did not increase the odds to develop TMD: retruded contact position (RCP) to intercuspal position (ICP) occlusal slides < 2 mm, slide asymmetry, unilateral RCP contacts, deep overbite, minimal overjet, dental midline discrepancies, < 4 missing teeth, and maxillo-mandibular first molar relationship or cross-arch asymmetry. They found that groupings of a minimum of two to at most five occlusal variables contributed to the TMD patient groups. On the other hand, significant increases in risk occurred selectively with anterior open bite, unilateral maxillary lingual crossbite overjets > 6-7 mm > 5-6 missing posterior, and RCP-ICP slides > 2 mm. The authors were of the opinion that certain features such as anterior open bite in osteoarthritis patients were considered to be a consequence of rather than etiological factors for the disorder. They concluded "that occlusion cannot be considered the unique or dominant factor in defining TMD populations".

The hypothesis that different orthodontic techniques such as functional appliances class I/II elastics, chin-cup, headgear, fixed or removable appliances as aetiological factors for TMD has been tested in many studies. Dibbets and Van der Weele (1992) compared children treated with different procedures. Patients were monitored for a 20 year period after the start of orthodontic treatment. Although signs and symptoms of TMD increased with age, after 20 years neither orthodontic treatment showed a causal relationship with signs and symptoms of TMD. Henrikson and Nilner (2000) compared class II division 1 treated and untreated females with normal occlusion (11-15 years old) monitored for 2 years. They reported individual fluctuations of TMD symptoms in all 3 groups. Orthodontic treatment did not increase the risk for aggravating pre-treatment signs of TMD. On the contrary subjects with class II and TMD of muscular origin seemed to improve. Rey et al (2008) compared a sample of class III patients treated with mandibular cervical headgear and class I patients treated orthodontically and no treated subjects. No difference in TMD prevalence was found between the 3 groups after 2-3 years. Regarding orthognathic surgery, Farella et al (2007), reported that bi-maxillary osteotomy did not initiate or aggravate signs and symptoms of TMD. A 20 year cohort longitudinal study by MacFariane et al (2009) investigated the relationship between orthodontic treatment and TMD concluded that orthodontic treatment neither causes nor prevents TMD and that participants with a history of orthodontic treatment did not have higher risk of new or persistent TMD.

Henrikson and Nilnerl (2000), prospectively and longitudinally studied signs of TMD and occlusal changes in girls with Class II malocclusion receiving treatment, compared to subjects with untreated Class II malocclusion and with normal occlusion subjects. They concluded that orthodontic treatment does not increase the risk for TMD or for worsen pre-treatment signs. On the contrary, they found that subjects with Class II malocclusion and signs of muscular TMD seem to benefit from the orthodontics treatment.

## 6. Conclusions & clinical aspects

A recent Cochrane systematic review was published (Luther et al 2010). Its objective was to establish the effectiveness of orthodontic intervention in reducing symptoms in patients

with TMD (compared with any control group receiving no treatment, placebo treatment or reassurance) and to establish if active orthodontic intervention leads to TMD. The authors identified 284 records from all databases, but only four demonstrated any data that might be of value with respect to TMD and orthodontics. After further analysis of the full texts of the four studies identified, none of the retrieved studies met the inclusion criteria and all were excluded from this review. The authors' conclusions were: "1. There is insufficient research data on which to base our clinical practice on the relationship of active orthodontic intervention and TMD; 2. There is an urgent need for high quality randomized controlled trials in this area of orthodontic practice; 3. When considering consent for patients it is essential to reflect the seemingly random development/alleviation of TMD signs and symptoms.

## 7. Summary and conclusions

The main articles reviewed in this chapter are summarized in table 1.

Study Reference*	Study design	Conclusions & Comments
Al-Riyami et al (part 2) 2009	Systematic Review	Although orthognatic surgery should not be advocated solely for treating TMD, patients having orthognatic treatment for correction of their dento-facial deformities and who are also suffering from TMD appear more likely to see improvement in their signs and symptoms than deterioration
Behrents & White 1992	Viewpoint intended to recount a research program initiated by the American Association of Orthodontists	(1) Consistently significant associations between structure (dental and osseous) and TMD have not been demonstrated. (2) The development of TMD cannot be predicted. (3) No method of TMD prevention has been demonstrated. (4) The prevalence of TMD symptoms increases with age; thus TMD may originate during orthodontic treatment, but not be related to the treatment. (5) Orthodontic treatments per se do not initiate TMD. (6) Evidence favors the beneficial nature of orthodontic treatment; orthodontics, as a part of the regimen of care, may assist in the lessening of symptoms. (7) Once TMD is present, TMD cures cannot be assumed or assured.
Dibbets & Van der Weele 1992	Prospective- longitudinal	Based upon the finding of similar prevalences after 20 years of observation, it appears that neither orthodontic treatment nor extraction has a causal relationship with the signs and symptoms of TMD



Study Reference*	Study design	Conclusions & Comments
Egermark-Eriksson et al 1990	Longitudinal	No differences in prevalences of occlusal interferences, or in signs or symptoms of TMD were found between subjects that had corrective orthodontic treatment and those without such treatment. The associations between TMD and different morphological malocclusions were low. Nevertheless, in a long-term perspective cross-bite, both uni- and bilateral, anterior open bite, post-, and prenatal occlusion had some association with the development of CMD.
Farella et al 2007	Longitudinal	Pressure pain thresholds of the masseter and temporalis muscles did not change significantly from baseline values throughout the whole study period. The occurrence of signs and symptoms of TMD fluctuates with an unpredictable pattern after orthognathic surgery for class III malocclusions.
Gremillion 2006	Review article	Scientific literature has not convincingly demonstrated a definitive relationship between static occlusal factors and TMD.
Henrikson et al 2000	Prospective-longitudinal	Orthodontic treatment do not increase the risk for TMD or for worsen pre-treatment signs. On the contrary, they found that subjects with Class II malocclusion and signs of muscular TMD seem to benefit from the orthodontics treatment.
Le Bell et al 2002	Randomized double-blind clinical set-up	Since subjects with a TMD history and true interferences showed a significant increase in clinical signs compared to the other groups. The authors suggest that the etiological role of occlusal interferences in TMD may not have been correctly addressed in previous studies with artificial interferences and allow no conclusions as regards TMD etiology
Le Bell et al 2006	Randomized double-blind clinical set-up	The most prominent symptoms following the introduction of artificial occlusal interferences were occlusal discomfort and chewing difficulties. The difference in outcome between the groups with and without a TMD history suggests that there are individual differences in vulnerability to occlusal interferences.

Study Reference*	Study design	Conclusions & Comments
Luther et al 2010	Systematic Review (COCHRANE)	(1) There are insufficient research data on which to base the clinical practice on the relationship of active orthodontic intervention and TMD. (2) There is an urgent need for high quality randomized controlled trials in this area of orthodontic practice. (3) When considering consent for patients it is essential to reflect the seemingly random development/alleviation of TMD signs and symptoms.
Macfariane et al 2009	Prospective	Orthodontic treatment neither causes nor prevents TMD. Female sex and TMD in adolescence were the only predictors of TMD in young adulthood.
McNamara, Jr 1997	Review Article	(1) signs and symptoms of TMD may occur in healthy persons; (2) signs and symptoms of TMD increase with age, particularly during adolescence, until menopause, and therefore TMDs that originate during orthodontic treatment may not be related to the treatment; (3) orthodontic treatment performed during adolescence does not increase or decrease the chances of development of TMD later in life; (4) the extraction of teeth as part of an orthodontic treatment plan does not increase the risk of TMD; (5) there is no increased risk of TMD associated with any particular type of orthodontic mechanics; (6) although a stable occlusion is a reasonable orthodontic treatment goal, not achieving a specific gnathologic ideal occlusion does not result in signs and symptoms of TMD; and (7) thus far, there is little evidence that orthodontic treatment prevents TMD, although the role of unilateral posterior crossbite correction in children may warrant further investigation
Michelotti & Iodice. 2010	Review Article	(1) TMD is a multifactorial pathology, and it is difficult to demonstrate a direct correlation between one of the causes, such as occlusion, and TMD. (2) Dysfunctional patients have a lower adaptive capability to occlusal changes because they seem to be more vigilant on their occlusion and are easily disturbed by occlusal instability. (3) When severe pain is present, occlusal treatments (such as orthodontics and prosthodontics) have to be postponed until symptoms are improved.

Study Reference*	Study design	Conclusions & Comments
Niemi et al 2006	Randomized double-blind clinical set-up	Psychological factors appeared significant for the symptom responses to artificial interferences, and they seem to play a different role in responses in subjects with an earlier TMD history compared to those without.
Pullinger et al 1993	A multiple logistic regression analysis to compute the odds ratios for 11 common occlusal features for asymptomatic controls (n = 147) vs. five temporomandibular disorder groups (n=413).	Occlusion cannot be considered the unique or dominant factor in defining TMD populations.
Rey et al 2008	Retrospective Comparative	Subjects with Class III malocclusions treated with mandibular cervical headgear and fixed appliances do not have greater prevalence of TMD symptoms than do Class I subjects treated with fixed appliances or untreated subjects.
Rinchuse & Kandasamy 2009	Special Review Article	(1) Orthodontic gnathologists have proved no health benefit to justify the many perfunctory exercises of the philosophy. (2) The view that occlusion and condyle position are the primary causes of TMD, and that diagnoses and treatments should be based on these notions, has been discredited. (3) There is little to no evidence that treating subjects with TMJ ID will prevent or mitigate future TMD.

\* References has been arranged alphabetically according to the first author

Table 1. Summary of main articles reviewed.

The main conclusions are the following:

1. TMD is a collective term embracing a number of clinical problems that involve the masticatory muscles and the TMJs.
2. The pathogenesis of TMD is not dental – related but rather is a part of a wider family of orofacial pain disorders which account for the need to consider neurologic, endocrine and psychosocial factors during the diagnostic process. Occlusion, condyle position, and lack of canine guidance are not the primary causes of TMD (Manfredini and Nardini 2010).
3. TMD treatments are no longer dental, but are based on biopsychosocial approach (Rinchuse and Kandasamy 2009). Treatment options are: patient education , cognitive behavior therapy (CBT) (Turk et al 1996; Turk 1997), bio feedback, physiotherapy (Stholer 1999) , acupuncture (List et al 1993), transcutaneous nerve stimulation (TENS), low intensity laser , splint therapy (Greco et al 1997; List and Axelsson 2010), drug therapy, surgical intervention (Al-Riyami et al 2009), but not occlusal definitive;

4. TMD signs and symptoms are often resolved by conservative and reversible therapies.
5. No scientific evidence exists that orthodontic treatment will prevent or mitigate the development of future TMD, or cure an existing disorder.
6. Orthodontic treatment performed during adolescence does not increase or decrease the risk of developing TMD in later life.

The authors' clinical recommendations are the following:

1. An attentive orthodontist should always identify and document findings of the TMJ and related structures. TMD signs and symptoms may occur before, during and after orthodontic treatment even though these findings may not necessarily lead to treatment.
2. Inform the patient of his/her temporomandibular situation and discuss the prognosis. Ask a signed informed consent.
3. Inform the patient that his/her occlusion will undergo changes and that it is essential to avoid parafunctional, constant auto-checking of the bite in order to prevent the possible development of occlusal dysesthesia.
4. If the patient presents TMD symptoms BEFORE treatment:
  - a. Insignificant symptoms such as painless clicking or movement limitation due to prolonged periods of gum chewing or deviations in opening closing pattern should not delay the beginning of orthodontic treatment.
  - b. If pain and severe dysfunction are present the patient should be referred to a TMD specialist before orthodontic therapy is initiated .
5. If the patient develops symptoms DURING treatment :
  - a. Temporarily stop active orthodontics treatment.
  - b. Perform basic pain management and supportive therapy in order to reduce the symptoms, after which orthodontic treatment may continue.
  - c. If the symptoms persist, the treatment plan should be reconsidered because the patient might become hypervigilant and of poor adaptation capability. An alternative treatment plan should be considered.
6. If the patient develops symptoms AFTER treatment :
  - a. If the patient was informed before treatment about a possible development of TMD, there should not be a problem explaining that TMD was probably not a result of the orthodontics.
  - b. As TMD sign and symptoms tend to be observed between 20 to 30 years old (De Kanter et al 1993;Mohlin et al 2004) there is a possibility of an orthodontic patient developing symptoms after treatment based only on his/her age.

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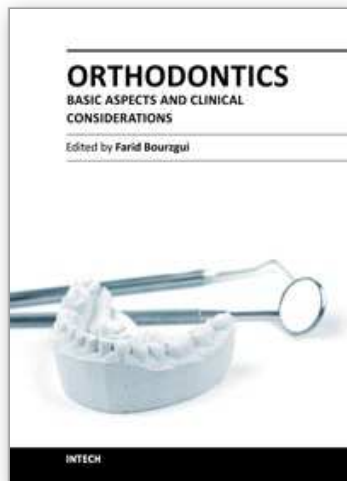
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The book reflects the ideas of nineteen academic and research experts from different countries. The different sections of this book deal with epidemiological and preventive concepts, a demystification of cranio-mandibular dysfunction, clinical considerations and risk assessment of orthodontic treatment. It provides an overview of the state-of-the-art, outlines the experts' knowledge and their efforts to provide readers with quality content explaining new directions and emerging trends in Orthodontics. The book should be of great value to both orthodontic practitioners and to students in orthodontics, who will find learning resources in connection with their fields of study. This will help them acquire valid knowledge and excellent clinical skills.

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