# Orthodontic treatment and temporomandibular disorders

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The relationship between orthodontic treatment and temporomandibular disorders (TMDs) has long been of interest to the practicing orthodontist, but only during the past decade have a significant number of clinical studies been conducted that have investigated this association. This interest in orthodontics and TMD in part was prompted in the late 1980s after litigation that alleged that orthodontic treatment was the proximal cause of TMD in orthodontic patients. This litigious climate resulted in an increased understanding of the need for risk management as well as for methodologically sound clinical studies. The findings of current research investigating the relation of orthodontic treatment and TMD can be summarized as follows: (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) 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. **(Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:107-17)**

The interest of the orthodontic specialty and of other disciplines of dentistry and medicine concerning the association, or lack of association, between orthodontic treatment and temporomandibular disorders (TMDs) has increased dramatically during the past decade. Although long recognized by orthodontists as a clinical problem, the diagnosis and treatment of TMD was not emphasized within the specialty until the mid 1980s. The attention of the orthodontic community regarding TMD, however, was heightened in the late 1980s after litigation involving allegations that orthodontic treatment was the proximal cause of TMD in orthodontic patients, with substantial monetary judgments awarded to several plaintiffs.[1] The outcome of these court cases resulted in a burst of research activity investigating the relation of orthodontic intervention to TMD.

This article represents the evolution of a solicited manuscript first presented at the International Workshop on the TMDs and Related Pain Conditions, sponsored by the National Institutes of Health (Hunt Valley, Md., April 17 to 20, 1994). The original version of this article[2] considered the broad topic of the relation of occlusal factors and orthodontic treatment to TMDs. The current iteration of this article, which focuses on the orthodontic–TMD interface, was presented at the National Institutes of Health Technology Assessment Conference on the Management of Temporomandibular Disorders.

## HISTORICAL PERSPECTIVE

During the past half century, several prominent orthodontic clinicians and researchers have had a keen interest in the diagnosis and treatment of TMDs, primarily as specific clinical entities rather than simply as an aspect of routine orthodontic treatment. One of the earliest pioneer orthodontists in TMD recognition and therapy was Thompson,[3-5] who noted that patients with disturbances in the vertical dimension appeared to be more prone to temporomandibular joint (TMJ) problems. He stressed the establishment of normal vertical dimensions, especially in deep bite patients, and advocated the elimination of all interferences in the "freeway space" envelope of mandibular movement. Graber[6,7] was one of the first researchers to call attention to the multifactorial nature of TMD, with occlusion being only one factor. Graber cited stress and uncontrolled nocturnal parafunction as contributing factors, and he cautioned against treatments using strict gnathologic concepts, stating that the articulator cannot properly emulate TMJ function and condylar position. His treatment recommendations extended beyond the narrow confines of the dentition, with stress control and psychological counseling as part of the therapy.

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One of the largest early patient studies was conducted by Ricketts,[8-11] who developed a cephalometric laminographic technique to evaluate the temporomandibular articulation. He evaluated the TMJ regions of more than 400 persons and used the range of variation found in 50 patients with "satisfactory occlusions" as a basis for comparison of individual pathologic conditions. Ricketts[5] also stressed the role of the musculature in the determination of condylar position relative to the glenoid fossa.

Despite the interest in TMD among a few prominent orthodontic clinicians, before the late 1980s orthodontists typically did not focus on TMD problems in their patients, except in instances of severe clinical problems. Traditionally, scant mention was made of TMD treatment in the curricula of graduate programs in orthodontics, and only cursory examinations of the TMJ region were conducted in routine orthodontic clinical examinations. In addition, before the mid 1980s only a limited number of methodologically sound clinical studies regarding the relation between orthodontic treatment and the TMDs had been published, at least as judged by current standards of clinical research. The past decade, however, has seen an explosion of new information regarding orthodontics and TMD.

## EARLY CLINICAL STUDIES

In a comprehensive review of the literature on this subject published between 1966 and 1988, Reynders[12] divided 91 publications into three categories: viewpoint articles, case reports, and sample studies. The most numerous were viewpoint articles ($n = 55$), publications that usually were anecdotal, stating the opinion of the author regarding the orthodontic–TMD relation. Little (or, more commonly, no) data were presented to support the author's opinion. Furthermore, Reynders[12] notes that 23 of the 55 viewpoint articles were published in *The Functional Orthodontist*, with articles advancing the concept that orthodontic treatment can either cause or cure TMD.

The second most frequent type of article ($n = 30$) was the case report, a category of publication that described the influence of certain orthodontic treatment modalities used in one or more patients on the signs and symptoms of temporomandibular dysfunction. The least numerous ($n = 6$) were in the third category, that of sample studies, investigations that reported data from large sample groups. These studies were of variable quality, often having the same methodologic problems and limitations as discussed previously for studies of occlusal factors. Since 1988, however, a substantial number of clinical investigations have considered the association of orthodontics and TMDs.

## RECENT CLINICAL STUDIES

Viewpoint articles, of course, are not suitable for critical evaluation of associations between two entities such as orthodontic treatment and TMD; however, they are useful in identifying questions that may be worthy of scientific investigation. Although the literature is not as extensive on the relation of orthodontics to TMD as it is to the occlusal factors–TMD relation (see McNamara et al.[13] for a review of the literature on this subject), the questions discussed below have been addressed in a substantial number of recent studies. These reports are discussed in detail below, with many of the investigations considering more than one question.

### What is the prevalence of signs and symptoms of TMD in orthodontically treated populations?

Numerous epidemiologic studies have examined the prevalence of signs and symptoms associated with TMD in a wide variety of subject populations. In general, the prevalence has been shown to be of significance, with an average of 32% reporting at least one symptom of TMD and an average of 55% demonstrating at least one clinical sign.[7]

Cross-sectional epidemiologic studies of specific adult nonpatient populations indicate that at any given time, between 40% and 75% have at least one sign and about 33% report at least one symptom of TMD.[14-19] According to Montegi et al.,[20] the point prevalence of symptoms in children and teenagers is lower, about 12% to 20%.

Because of the longitudinal nature of orthodontic treatment (e.g., 2 to 3 years for adolescents and 5 to 7 years for patients starting a two-phase treatment protocol in the early mixed dentition), an understanding of the changes in the signs and symptoms of TMD in a healthy population is essential. Several investigators have reported that, in general, signs and symptoms of TMD increase in frequency and severity, beginning in the second decade of life.[21-23] Wänman and Agerberg[24] have noted that the incidence of joint sounds in young adults in their late teens can be as high as 17.5% over a 2-year period. Therefore the occurrence of joint sounds during orthodontic treatment must be considered within the context of longitudinal changes in a comparable untreated population studied during the same interval.

### Does orthodontic treatment lead to a greater incidence of TMD?

Two of the first investigations sponsored by the National Institutes of Health to consider the relation between orthodontics and TMD were initiated about 15 years ago (Table I). These research efforts consid-

**Table I.** Major studies of relation between orthodontic treatment and signs and symptoms of TMD

| *Authors* | *Sample* | *Appliance* | *Extraction vs. nonextraction* | *Relation* |
|---|---|---|---|---|
| Sadowsky and Begole (1980)[25] | 75 Treated<br>75 Untreated | Fixed | No | No |
| Larsson and Rönnerman (1981)[28] | 23 Treated | Fixed | No | Improvement |
| Janson and Hasund (1981)[44] | 60 Treated<br>30 Untreated | Fixed<br>Functional | Yes | Improvement |
| Sadowsky and Polson (1984)[26] | 207 Treated<br>214 Untreated | Fixed | No | No |
| Pancherz (1985)[45] | 22 Treated | Functional | No | No |
| Dibbets and van der Weele (1987)[41] | 135 Treated | 72 Fixed<br>63 Functional | Yes | No |
| Dahl et al. (1988)[30] | 51 Treated<br>47 Untreated | Fixed<br>Functional | No | No |
| Smith and Freer (1989)[33] | 87 Treated<br>28 Untreated | Fixed | No | No |
| Sadowsky et al. (1991)[53] | 160 Treated | Fixed | Yes | No |
| Dibbets and van der Weele (1991)[42] | 109 Treated | Fixed<br>Functional | Yes | No |
| Kundlinger et al. (1991)[58] | 29 Treated | Fixed | Yes | No |
| Luecke and Johnston (1992)[64] | 42 Patients | Fixed | Yes | No |
| Årtun et al. (1992)[66] | 63 Treated | Fixed | Yes | No |
| Kremenak et al. (1992)[34] | 65 Treated | Fixed | Yes | No |
| Kremenak et al. (1992)[35] | 109 Treated | Fixed | No | No |
| Egermark and Thilander (1992)[81] | 402 Mixed | Fixed<br>Functional | No | Improvement |
| Rendell et al. (1992)[31] | 462 Treated | Fixed | No | No |
| Hirata et al. (1992)[37] | 102 Treated<br>41 Untreated | Fixed | No | No |
| Wadhwa et al. (1993)[32] | 31 Treated<br>71 Untreated | Fixed | No | No |
| O'Reilly et al. (1993)[57] | 60 Treated<br>60 Untreated | Fixed | Yes | No |
| Olsson and Lindqvist (1995)[84] | 210 Treated | Fixed? | No | Improvement |

ered the prevalence of TMD and the status of the "functional occlusion" (to be discussed later) in large groups of subjects who had undergone orthodontic treatment at least 10 years previously.

Sadowsky and Begole[25] reported on the findings from a University of Illinois study of 75 adult subjects who at least 10 years previously had been treated with full orthodontic appliances as adolescents. The treated group was compared with a group of 75 adults with untreated malocclusions. In a subsequent article by Sadowsky and Polson,[26] the sample from the Illinois study (increased to 96 treated and 103 control subjects) was compared with a treatment group of 111 subjects who had been treated at least 10 years previously at the Eastman Dental Center and a control group of 111 persons with untreated malocclusions. In the two studies, 15% to 21% of the subjects had at least one sign of TMD and 29% to 42% had at least one symptom of TMD, usually joint sounds. There were no statistically significant differences between the treated and untreated groups.[27] The results of these two studies provide evidence in support of the concept that, in general, orthodontic treatment performed during adolescence does not increase or decrease the risk of development of TMD later in life.

Another study of the long-term effects of orthodontic treatment was conducted by Larsson and Rönnerman.[28] They examined 23 adolescent patients who had been treated orthodontically at least 10 years earlier. Eighteen of the patients had been treated with fixed appliances, and five patients had received activator treatment. With use of the Helkimo[29] index as an evaluative tool, mild dysfunction was found in eight patients and severe dysfunction in one patient. Comparing their results with published epidemiologic studies, Larsson and Rönnerman[28] stated that comprehensive orthodontic treatment can be undertaken without fear of creating TMD problems.

Dahl et al.[30] examined 51 subjects 5 years after the completion of orthodontic treatment. Signs and symptoms of TMD were noted and compared with the findings from a similar group of 47 untreated persons.

According to the authors, "Nobody really had craniomandibular disorders" in either group. Severe symptoms (e.g., difficulties in wide opening, locking, or pain on mandibular movement) typically were not observed; however, mild symptoms (e.g., joint sounds, muscle fatigue, or stiffness of the lower jaw) were observed more frequently in the untreated group than in the treated group, a statistically significant difference. Dahl et al.[30] noted that the number of subjects in both groups who had at least one mild symptom was relatively high (70% in the treated group and 90% in the untreated group), especially in comparison with the previously mentioned investigation of Larsson and Rönnerman,[28] who reported a 27% occurrence of mild dysfunction in their treated patients. The latter group reported that differences between samples may result as much from measuring differences (e.g., lack of factor definition or differences in the interpretation of the criteria of the Helkimo[29] index) as from a true reflection of differences between groups.

Rendell et al.,[31] using a modification of the Helkimo[29] index, examined 462 patients receiving treatment in an orthodontic graduate clinic (90% adolescents and 10% adults). Eleven of the patients had TMD signs or symptoms before treatment. During the 18-month study period, none of the patients who had been sign- or symptom-free at the beginning of treatment developed signs or symptoms of TMD. No clear or consistent changes in the levels of pain and dysfunction occurred during the treatment period in the patients with preexisting signs or symptoms. Rendell et al.[31] concluded that a relation could not be established in their patient population between orthodontic treatment and either the onset or the change in severity of TMD signs and symptoms.

Wadhwa et al.,[32] also using the Helkimo[29] index, compared the status of signs and symptoms of TMDs in three groups of adolescents and young North Indian adults. The groups consisted of 30 persons with normal occlusion, 41 with untreated malocclusions, and 31 with treated malocclusions. The results showed that the group with normal occlusion had the maximum number of persons free from any dysfunction, but the differences among the groups in the distribution of persons according to the anamnestic and clinical dysfunction indexes were not significant. The only statistically significant finding was the difference in the clinical dysfunction index scores of the persons with normal occlusions and untreated malocclusions. According to the anamnestic portion of their study, the most frequently reported symptoms were related to periods of stress. Among the clinical signs and symptoms, the most commonly occurring were crepitations on palpation and sounds on auscultation of the joints in all three groups.

One of the few clinical studies to report positive findings is the investigation of Smith and Freer,[33] who examined 87 patients treated with full orthodontic appliances during adolescence. About two thirds of the patients had permanent teeth removed as part of the treatment protocol. The treated group was compared with an untreated control group of 28 persons. Four years after the end of retention, symptoms were found in 21% of the treated group and 14% of the control subjects, a difference that was not significant statistically. The investigators, however, did note that a single sign was statistically significant, the exception being the association between what they termed "soft clicks" and previous treatment. Soft clicks were found in 64% of the treatment group and 36% of the untreated group. They did not find any difference in joint sounds (i.e., crepitus as determined by stethoscopic examination) between the two groups. Of note, the authors concluded the article by stating, "The null hypothesis that there is a significant association between orthodontic treatment and occlusal or joint dysfunction has been rejected by nearly all previously reported studies and continues to be rejected by the present study."

Relatively few prospective studies have examined the relation of orthodontics to TMD. The two major investigations have been conducted at the University of Groningen, The Netherlands (to be discussed below), and at the University of Iowa.[34-36] In the latter ongoing study, 30 new orthodontic patients have been enrolled annually since 1983. The method of Helkimo[29] was used to collect TMD data before orthodontic treatment and at yearly intervals after the completion of treatment. Patients were treated with comprehensive edgewise appliances with and without extractions. No longitudinal data on a comparable untreated population were obtained.

Kremenak et al.[35] have reported data from pretreatment and posttreatment examinations from 109 patients. Data on follow-up examinations from 1 to 6 years after treatment were available on declining sample sizes of 92, 56, 33, 19, 11, and 7 persons. No significant differences were noted between mean pretreatment and posttreatment Helkimo[29] scores for any of the various groupings. Ninety percent of the patients had Helkimo scores that remained the same or improved, and 10% had scores that worsened (an increase of two to five Helkimo points). Kremenak et al.[34, 35] concluded that the orthodontic treatment experienced by their patients was not an important etiologic factor for TMD.

Hirata et al.[37] examined 102 patients before and after orthodontic treatment for signs of TMD. Findings from this group were compared with findings from 41 untreated subjects matched for age. The incidence of temporomandibular signs for the treatment and control groups was not significantly different. Pocock et al.,[38] using an anamnestic questionnaire, similarly found no differences between an orthodontically treated population when compared with various normative populations. Furthermore, they noted no significant differences between scale scores of various combinations of malocclusions or treatment subgroups of the treated patients.

### Does the type of appliance (e.g., fixed vs functional or orthodontic vs orthopedic) make a difference?

In the other major longitudinal study of this subject, Dibbets et al.[39-43] conducted a long-term study of 171 patients, 75 of whom were treated by the Begg technique (most patients had extractions as part of their treatment protocol). Sixty-six patients were treated with activator therapy, and 30 patients were treated with chin cups. The pretreatment documentation revealed a strong dependence of the prevalence of signs and symptoms on age: from 10% at age 10 years, signs increased to 30%, and symptoms increased to more than 40% at age 15 years. They also noted that at the end of treatment, the fixed-appliance group had a higher percentage of objective symptoms than did the functional group, but no differences existed at the 20-year follow-up evaluation.[37]

Janson and Hasund[44] conducted a similar study of adolescent patients with class II, division 1 malocclusion who were examined 5 years out of retention. Thirty patients underwent a two-phase treatment regimen (headgear-activator therapy followed by fixed appliances) without the removal of teeth, and 30 patients were treated with fixed appliances after the removal of four premolars. An additional 30 untreated persons served as control subjects. One or more symptoms were reported in about 42% of the subjects overall (treated and untreated), with similar findings for the clinical dysfunction index.[29]

One prospective study examined the effect of functional mandibular advancement in patients with class II, division 1 malocclusions. Pancherz[45] used the banded Herbst appliance alone in 22 adolescent patients with class II, division 1 malocclusions during a treatment period of 6 months. After an initial incisal edge-to-edge bite registration, several patients reported muscle tenderness during the first 3 months of treatment. At 12 months after treatment, however, the number of subjects with symptoms was the same as that before treatment.

### Does the removal of teeth as part of an orthodontic protocol lead to a greater incidence of TMD?

Viewpoint articles and texts, publications that primarily present opinions rather than data, have strongly associated the extraction of premolars with the occurrence of TMD in orthodontic treatment.[46-52]

The clinical studies that have dealt with this issue have not shown a relation between premolar extraction and TMD. For example, Sadowsky et al.[53] reported findings from 160 patients, 54% of whom were treated using extraction treatment strategies. Joint sounds were monitored before and after treatment in 87 orthodontic patients undergoing extraction and 68 not undergoing extraction. Before treatment, 25% of patients had joint sounds whereas 17% had sounds after treatment. Similarly, 14% of patients had reciprocal clicking; only 8% had clicking after treatment. The investigators concluded that their findings did not indicate a progression of signs and symptoms to more serious problems during treatment. They also reported no increase in the risk of development of joint sounds regardless of whether teeth were removed.

The long-term effect of extraction and nonextraction edgewise treatments were compared in 63 patients from St. Louis University with Class II, division 1 malocclusions who were identified by discriminant analysis as being equally susceptible to the two treatment strategies.[54, 55] In terms of a menu of 62 signs and symptoms (e.g., muscle palpation, joint function) that commonly are thought to be characteristic of TMD, there were no differences between extraction and nonextraction samples. A follow-up study by Luppanapornlarp and Johnston[56] that examined an additional 62 ''clear-cut'' patients (those in the tails of the distribution) also noted that both extraction and nonextraction samples demonstrated similar findings.

The longitudinal studies at Iowa also have addressed this question. Kremenak et al.[34] studied three groups of patients: 26 patients treated without extraction, 25 patients with four premolars extracted, and 14 patients with two upper premolars extracted. No significant intergroup differences between mean pretreatment or posttreatment Helkimo scores were noted. A small but statistically significant improvement in Helkimo scores was observed posttreatment in both the nonextraction group and the four premolar extraction group.

Dibbets and van der Weele[42] studied 111 of the original 172 orthodontic patients in the Groningen

study over a 15-year follow-up period. In this group, a nonextraction approach was used in 34% of the patients; four premolars were extracted in 29%; and other extraction patterns were used in the remaining 37%. Functional appliances were used in 39%; fixed appliances (Begg) were used in 44%; and chin cups were used in 17% of the patients. Symptoms increased from 20% to 62%; signs of clicking and crepitus increased from 23% to 36% after 4 years and then stabilized. In contrast to the finding from the first 10 years,[41] during which there was no difference between the three treatment groups with regard to clicking, after 15 years[42] this symptom was seen more often in the premolar extraction group. The authors noted, however, that clicking was observed more frequently in the premolar extraction group before treatment was started and concluded that the original growth pattern, rather than the extraction protocol, was the most likely factor responsible for the TMD complaints seen many years after treatment. These investigators also noted that for a substantial number of patients, symptoms of TMD appeared and disappeared during the course of study. At the 20-year follow-up evaluation,[43] however, the difference between groups had disappeared completely. They also noted that even though the overall incidence of symptoms increased with time, many previously symptomatic children were found to have become asymptomatic at the time of subsequent evaluations.

O'Reilly et al.[57] examined 60 treated patients and 60 untreated subjects who were the treated subjects' nearest-age siblings. The treated patients underwent fixed orthodontic treatment that included extraction and the wearing of Class II intermaxillary elastics. No difference were seen between the treated and untreated groups. Kundlinger et al.[58] compared 29 extraction-treated and 29 untreated subjects with regard to condylar position using tomograms and electromyography of some of the muscles of mastication. No differences were observed between groups.

Another specific concern expressed in viewpoint articles is that orthodontic treatment involving the extraction of first premolars causes a decrease in the vertical dimension of occlusion. Staggers[59] examined the records of 45 class I patients not undergoing extraction and 38 class I patients undergoing extraction of the first premolar. The pretreatment and posttreatment cephalograms were analyzed to evaluate the vertical changes occurring as a result of orthodontic treatment. Statistical analysis of the data revealed no significant differences between the vertical changes occurring in the extraction and nonextraction groups. On average, the vertical dimension increased in both the extraction and the nonextraction treatment groups.

Although not a study that specifically concerned orthodontic patients, Pullinger et al.[60] also investigated the association of missing teeth to signs and symptoms of TMD. They used a blinded multifactorial analysis to determine the "weighted influence" of various occlusal factors (e.g., anterior open bite, maxillary lingual posterior crossbite, overbite, overjet, midline discrepancy, and missing posterior teeth) acting in combination with other occlusal factors. The interaction of 11 occlusal factors, including missing teeth, was considered in five randomly collected but strictly defined diagnostic groups (i.e., disk displacement with reduction, disk displacement without reduction, TMJ osteoarthrosis with a history of disk displacement, primary osteoarthrosis, and myalgia only) compared with asymptomatic normal subjects. The asymptomatic control subjects were considered the "gold standard," in that the subjects in this group were without signs and symptoms and had no history of TMD. The groups were demographically representative, and the occlusal factors studied were collected blindly and strictly defined. A multiple logistic regression model was used by Pullinger et al.[60] for simultaneous assessment of the relative odds of each potential occlusal factor. The outcome always was the disease classification compared with the asymptomatic controls. On the basis of this analysis, Pullinger et al.[60] reported that the contribution of the extraction of two to four teeth per se, for example as part of an orthodontic treatment protocol, was negligible in most patients when other variables were controlled.

### Can orthodontic treatment lead to a posterior displacement of the mandibular condyle?

Several viewpoint articles have asserted that a wide variety of traditional orthodontic procedures (e.g., premolar extraction, extraoral traction, and retraction of upper anterior teeth) cause TMD signs and symptoms by producing a distal displacement of the condyle.[46, 51, 61, 62] This allegation is contradicted by the gnathologist's view of condylar position, a topic that is considered in the next section.

Gianelly et al.[63] used corrected tomograms to evaluate condylar position before beginning orthodontic treatment in 37 consecutive patients aged 10 to 18 years and compared them with tomograms from 30 consecutively treated patients treated with fixed appliances (edgewise or Begg) and the removal of four premolars. No differences in condylar position were noted between groups. The position of the condyle tended to be centered within the glenoid fossa, but wide variation in condylar position was noted in both groups.

Luecke and Johnston[64] evaluated the pretreatment

and posttreatment cephalograms of 42 patients treated with fixed appliances in conjunction with the removal of two upper premolars. The results of this study indicated that the majority of patients (about 70%) undergo a forward mandibular displacement and a slight opening rotation of the mandible. The remainder of the sample had distal movement of the condyle. Incisor changes were essentially unrelated to condylar displacement during treatment. Luecke and Johnston[64] stated that a change in the spatial position of the mandible is a function of changes in the anteroposterior position of the occluding buccal segments rather than the relatively nonoccluding incisors. These observations also are supported by the findings of Tallents et al.[65]

The recall studies of Beattie et al.[55] and Luppanapornlarp and Johnston[56] reported no differences between groups with regard to TMD signs and symptoms. They also noted that both extraction and nonextraction treatments produced a mean anterior displacement of the mandible.

Årtun et al.[66] also investigated the relation of orthodontic treatment to posterior condylar displacement. Sixty-three female patients were evaluated after routine fixed appliance treatment (29 with extraction and 34 without extraction). Condylar position was measured in percentage anterior and posterior displacement from absolute concentricity on the basis of sagittally corrected tomograms. The investigators did note a mean difference in condylar position between the two treatment groups, but the difference was due mainly to the occurrence of presumed anteriorly displaced condyles in the nonextraction group (data on the pretreatment position of the condyles were not obtained). They did note that the condyles in patients with clicking were in a more posterior position, although there was a wide variation of condylar position in all samples, and this variation also extended to different tomographic sections within the same condyle. These researchers concluded that posterior condyle position was not a result of orthodontic treatment.

### Should the occlusions of orthodontic patients be treated to specific gnathologic standards?

Several viewpoint articles, including those by Roth et al.[67-70] and Williamson,[71] have maintained that TMDs may result from a failure to treat orthodontic patients to gnathologic standards that include the establishment of a "mutually protected occlusion"[67-70] and proper seating of the mandibular condyle within the glenoid fossa (in contrast to the more anterior position of the condyle advocated by the so-called "functional orthodontists"). The gnathologists claim that nonfunctional occlusal contacts, when introduced through orthodontic treatment, can lead to signs and symptoms of TMD.

The discussion of the relation of occlusion and malocclusion to TMD presented earlier in this article illustrates the lack of association between most occlusal factors and TMD. Pullinger et al.[60] reported that small occlusal slides, most less than 1 mm, are common in asymptomatic subjects as well as patients with TMD. Only when a slide between retruded cuspal position and intercuspal position becomes extreme ($\geq$5 mm) does the odds ratio (i.e., chance) for disease increase. Thus a modest slide after orthodontic treatment typically is within the adaptive capabilities of most patients.

Sadowsky and Begole[25] and Sadowsky and Polson[26] evaluated the prevalence of nonfunctional occlusal contacts in patients at least 10 years after orthodontic treatment. They noted a high incidence of such occlusal contacts in both orthodontic and control groups. Similar findings have been reported by Cohen[72] and Rinchuse and Sassouni,[73] among others.

Hwang and Behrents[74] investigated the effects of orthodontic treatment on centric discrepancy. Thirty-six persons who had received orthodontic treatment were compared with 30 subjects who had received no treatment. After a leaf gauge was used to record centric position, centric slide and centric prematurity were recorded using an articulator and a mandibular position indicator. No differences were noted in the amount or direction of centric slide between the orthodontic and control groups, and the authors concluded that in general, orthodontic treatment does not result in an increase in centric discrepancy.

It probably is prudent to establish morphologic treatment goals that mimic what is observed in untreated occlusions that have been judged normal or ideal, such as the "six keys of ideal occlusion" advocated by Andrews,[75, 76] and to treat a patient so that there is a minimal (<2 mm) slide between retruded cuspal position and intercuspal position. The establishment of an occlusion that meets gnathologic ideals, however, probably is unnecessary, particularly in adolescent patients, and sometimes the attainment of a gnathologic ideal may be impossible in some adult patients.

### Does orthodontic treatment prevent TMD?

This topic of whether orthodontic treatment prevents TMD is the most difficult to investigate, given the prevalence of signs and symptoms of TMD in healthy persons and the many types of orthodontic treatment philosophies, goals, and techniques in existence today. The question of whether orthodontic

treatment can prevent TMD is complicated further by many of the unsubstantiated viewpoint articles that claim preventive capabilities of nonextraction treatment, functional appliances, and some of the more nontraditional orthodontic treatment protocols (e.g., extraction of the second molar extraction and replacement of the third molar) that have been advocated vigorously.[49-51, 77-79]

As previously discussed, most studies that have compared treated and untreated populations have found no differences between groups in the occurrence of TMD signs and symptoms. One of the few investigations that found improved TMD health in a treated group was the sample studied by Magnusson et al.[80] and Egermark and Thilander.[81] These investigators reevaluated at 5 and 10 years, respectively, a group of 402 children and adolescents who originally had been evaluated cross-sectionally by Egermark-Eriksson et al.[82, 83] The sample originally was divided into three groups according to age (7, 11, and 15 years). About one third of the sample had received orthodontic treatment at the end of the final examination period. Bruxism awareness and subjective symptoms of TMD increased in all age groups, with symptoms slightly more pronounced in untreated persons. The investigators also noted that clicking recorded at the first examination sometimes disappeared at subsequent examinations and that clicking sometimes appeared at subsequent intervals, regardless of whether the subject underwent orthodontic treatment. As in many previous studies, the Helkimo[29] index was used to measure clinical signs of TMD in the oldest age group (25 years). The clinical dysfunction index outcome was lower in those who had undergone orthodontic treatment than in those who had not undergone such treatment.

Olsson and Lindqvist[84] conducted a longitudinal study of 245 consecutive prospective orthodontic patients before and after the orthodontic treatment. Of the 245 referred patients, eight declined treatment and 27 moved before the treatment was completed, leaving a sample of 210 patients. Symptoms of TMDs were found in 17% of the patients before treatment and in 7% after treatment. The number of subjects without signs or symptoms of TMD increased from 27% before treatment to 46% afterward. According to the Helkimo[29] index, 32% of the patients had a moderate mandibular dysfunction and 14% had a severe mandibular dysfunction before the start of orthodontic treatment. After treatment, the corresponding figures were 14% and 6%, respectively. The authors concluded that "orthodontic treatment can to some extent prevent further development of and cure temporomandibular disorders."

As previously mentioned, a trend toward decreased prevalence of TMD signs and symptoms in treated patients also was noted by Sadowsky and Polson[26] and Dahl et al.[30] The signs and symptoms of TMD in the previously treated orthodontic patients very seldom were so severe that it could be said that these patients suffered from TMD (even if they had signs and symptoms).

Finally, one clinical condition that may be worthy of further investigation is unilateral posterior crossbite in growing children. The relation of unilateral posterior crossbite to TMD has been studied from several perspectives. As mentioned earlier, Pullinger et al.[60] examined five strictly defined patient groups in comparison with asymptomatic control subjects (the "gold standard"). For a clinically perceptible influence to be significant, Pullinger et al.[60] stated that an occlusal feature would need at least to double the risk of disease (at least a 2:1 mean odds ratio). Only five occlusal conditions reached this threshold, including unilateral posterior crossbite. This occlusal feature, occurring in about 10% of the adult population, resulted in a greater chance of assignment to the TMJ-derangement groups. Nearly 25% of the patients with nonreducing disk displacement had this feature, and the odds ratio that a person with this type of crossbite also would have TMJ disk displacement with reduction was more than 3:1. Similar odds ratios were seen for the disk displacement group without reduction (2.6:1) and also in the patients with osteoarthrosis with a history of disk displacement (1.96:1).

Pullinger et al.[60] noted that the persistence of an odds ratio for disease association into adulthood indicates that the adaptive response in a small percentage of subjects may be less than optimal and leads to the suggestion that functional adaptation to a unilateral posterior crossbite in childhood may occur at the expense of the articular disk through the development of internal derangement, eventually progressing to arthrosis in a small number of patients. These investigators believe that a case can be made for the treatment of children with unilateral crossbites to reduce the adaptive demands on the masticatory system. Conversely, the orthodontic correction of unilateral crossbite in adults to prevent development of TMJ derangement probably is not warranted, because skeletal adaptation already has occurred.

Thilander[85-87] has recommended the treatment of posterior crossbite at a young age to prevent not only asymmetrical facial growth, but also to prevent unilateral posterior condylar displacement. She hypothesizes that muscular hyperactivity on the crossbite side[88, 89] may unfavorably influence craniofacial and

TMJ growth, the latter region readily affected by changes in the functional environment at a young age. Thilander et al.,[90] on the basis of a longitudinal study of early interceptive treatment in children with unilateral crossbite, recommend selective equilibration of the deflecting supracontacts in the deciduous dentition or aggressive expansion treatment in patients with more severe maxillary transverse discrepancies. Although there appears to be some rationale for early correction of unilateral posterior crossbites in growing children, no prospective clinical trial of this type of treatment efficacy has been conducted to date.

## SUMMARY AND CONCLUSIONS

This article reviews the current literature regarding the relation of orthodontic treatment to the TMDs. Although the orthodontic community has had a persistent interest in the association between orthodontics and TMD, this association became a focus of conversation within the dental and legal communities in the late 1980s, resulting in a burst of research activity during the past decade.

The findings of current research on the relation of orthodontic treatment to the TMDs can be summarized as follows:

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. 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 odds of developing 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 evidence of an elevated risk for 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 the development TMD signs and symptoms.
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.

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