ORIGINAL ARTICLE

Orthodontics and temporomandibular disorder: A meta-analysis

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As the importance of evidence-based health care has grown, meta-analysis has become more widely used in the medical and dental fields. In this meta-analysis, the relationship between traditional orthodontic treatment, including the specific type of appliance used and whether extractions were performed, and the prevalence of temporomandibular disorders (TMD) was investigated. After an exhaustive literature search of 960 articles, we found 31 that met the inclusion criteria (18 cross-sectional studies or surveys and 13 longitudinal studies). We divided and extracted data from the 31 articles according to study designs, symptoms, signs, or indexes. Due to severe heterogeneity, the results were summarized without further statistical analysis. The heterogeneous result might originate from lack of a universal diagnostic system and the variability of TMD. Because of heterogeneity, a definitive conclusion cannot be drawn. The data included in this comprehensive meta-analysis do not indicate that traditional orthodontic treatment increased the prevalence of TMD. It is apparent that a reliable and valid diagnostic classification system for TMD is needed for future research. (Am J Orthod Dentofacial Orthop 2002;121:438–46)

Temporomandibular disorders (TMD) are a collection of pathologic and functional conditions affecting the temporomandibular joint (TMJ) and the muscles of mastication as well as contiguous tissue components.[1] Although epidemiologic data are inadequate, the number of TMD sufferers in the United States is estimated at more than 10 million.[1] Unfortunately, many aspects of the etiology and pathophysiology of TMD are not well known and remain controversial. After a technology assessment conference in 1996 about managing TMD, the National Institutes of Health (NIH) concluded that the natural history and etiology of TMD are not well understood and that most TMD symptoms are self-limiting, can recur, and may fluctuate over time.[1]

Although current evidence suggests that orthodontic treatment has little to do with TMD,[2-12] orthodontists in the United States are occasionally blamed for causing TMD. Epidemiologic studies show that TMD symptoms are most prevalent among patients between 15 and 25 years old; symptoms then level out as patients approach age 35.[13,14] Because some people in this age group receive orthodontic treatment that can last for several years, orthodontists may encounter patients who complain about TMD during or after treatment. As the number of adult orthodontic patients grows, these complaints might increase. Without sound knowledge, some might erroneously conclude that orthodontic treatment causes or contributes to TMD symptoms.

Since the late 1980s, the orthodontic community has become increasingly interested in TMD, and many well-designed studies of the TMD-orthodontic relationship have been published. Because practitioners cannot read every article, they may rely on literature overviews. Many overviews are well designed,[2-12] but others are biased due to lack of formal methodology and inclusion criteria.[15] As evidence-based health care has grown in importance,[16] systematic reviews or meta-analysis studies are appearing more often in medical and dental literature. Meta-analysis is defined as a systematic review that uses statistical methods to combine and summarize the results of several studies.[17]

To perform this meta-analysis, we used evidence from 31 primary studies to evaluate or analyze the relationship between orthodontic treatment and TMD. This meta-analysis was undertaken to answer the following questions: Does traditional orthodontic treat-

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ment change the prevalence of TMD? Does the use of a specific appliance change the prevalence of TMD? Does extraction during orthodontic treatment change the prevalence of TMD?

MATERIAL AND METHODS

To identify all studies that examined the relationship between orthodontic treatment and TMD, we performed a computerized MEDLINE literature search (from 1966 through September 2000). "Orthodontics" was searched in the subject heading, and it was crossed with various combinations of the following terms: "temporomandibular disorder," "craniomandibular disorder," and "temporomandibular joint." We conducted a library search using the references in the review articles,[2–12] and we also referred to a list[18] of published and unpublished articles compiled by Dr Rolf G. Behrents.

Only articles that satisfied the following criteria were included:

- Orthodontic treatment was completed in each patient. Studies dealing with orthodontic therapy or orthognathic surgery to treat TMD were excluded.
- Clinical TMD evaluation was performed in each patient (including at least 1 clinical evaluation after treatment). Imaging evaluations (cephalometric radiographs, tomograms, magnetic resonance imaging), occlusal interference evaluations, and electromyogram studies were excluded.
- Studies were case series, surveys, retrospective studies including only posttreatment evaluation with or without controls, nonrandomized prospective studies without controls, case-control studies, cohort studies, and randomized clinical trials. Case reports and opinion papers were excluded.
- Articles were written in English.
- No multiple-publication bias existed. To avoid multiple-publication bias (in which the same study is reported by different authors, under different titles, or in different journals), we chose 1 representative article from the independent reports.

Data were extracted using a standardized form. First, the 31 primary studies were divided into 2 groups according to study design (cross-sectional studies and surveys or longitudinal studies). Then, the data were divided and extracted based on symptoms, signs, or indexes. If several TMD evaluations after orthodontic treatment were performed, the latest evaluation was used.

To test whether all primary studies attempted to estimate or observe the same true effect, and whether variability between results of the studies was due to random error only (intrastudy variability), a statistical test for the hypothesis of parametric homogeneity (H) was conducted.[19]

We constructed probabilities of homogeneity (binomial parameters) based on the number of patients who had TMD signs or symptoms. We used the data to determine the posterior point estimates and the central credibility intervals from the corresponding β-probability density functions with respect to uniform prior specification for the binomial parameter. The point estimates corresponded to the mean of the posterior density, and the central credibility intervals were obtained from the relationship between the β and F distributions. The posterior probability of the hypothesis of a common underlying binomial parameter is denoted by $P(H)$ and was derived from the Bayes factor: the ratio of averaged likelihoods for the hypothesis and its alternative. With the appropriate data, the posterior odds on H, computed by $P(H)/(1-P(H))$, is the product of the Bayes factor and the prior odds on H.

In this analysis, we set the prior odds at 1, indicating a 50:50 prior belief in the hypothesis. In general, the posterior probability of a hypothesis quantifies its credibility given the observed data. The value of $P(H)$ ranges from 0 to 1, with values near 0 indicating strong evidence against the hypothesis, and values near 1 indicating strong evidence for the hypothesis. There is no evidence to support or reject H when $P(H)$ is near .5.

In this study, when the value of $P(H)$ was greater than .8, the corresponding binomial parameters were judged to be homogeneous. In addition, when the value of $P(H)$ was less than .2, the parameters were considered heterogeneous.

To identify the potential cause of the heterogeneity, we assessed the effect of sequentially removing 1 study at a time. The sensitivity of each $P(H)$ of κ-independent studies was tested, based on κ-posterior probabilities associated with homogeneity of κ-1 studies (removing 1 study at a time).

RESULTS

The MEDLINE search identified 960 articles. These articles, plus references cited in the review articles and in Dr Behrents' list, were reviewed. Before considering multiple publications, we found 38 studies that met our inclusion criteria.[20-57] After eliminating overlapping reports, 31 articles met the inclusion criteria, of which 18 were cross-sectional studies or surveys, and 13 were longitudinal studies.

The data for TMD symptoms, signs, or indexes showed an extremely heterogeneous situation. Even when 1 study at a time was sequentially removed, the data remained extremely heterogeneous. The $P(H)$

Table I. Results of test for homogeneity

	Cross-sectional/survey		*Longitudinal*	
	P(H) in control	*P(H) in case*	*P(H) before tx*	*P(H) after tx*
TMJ sound	.999*	.000	.000	.003
Muscle tenderness	.008	.000	.000	.005
TMJ pain	1.000*	.101	.204	.787
Pain on movement	.000	.000	.000	.000
Limitation of opening	.319	.343	.000	.000
Di (Helkimo index)	.000	.000	.963*	.994*
Ai (Helkimo index)	.000	.000	NR	NR

*Homogenous ($P(H) > .8$).
tx, Treatment; *Di*, dysfunction index; *Ai*, anamnestic index; *NR*, no report.

outcome is summarized in Table I. Because of the severe heterogeneity, we decided not to pool the results but to summarize them in tabular form without further statistical analysis. This analysis still has value for the clinician and is the most complete one of the literature at this time.

Table II shows the characteristics for all 38 studies identified in this meta-analysis. Some authors reported the same study under different titles or in different journals; when we found overlapping studies, we used well-described ones. If the control groups and the experimental groups were matched according to gender and age, they were considered to be matched. Table II also shows the various study designs, the number of dropouts in longitudinal studies, the gender ratios, and the types of appliances.

The outcome of each study, along with different types of assessments conducted over various lengths of time, is summarized in Table III. No study indicated that traditional orthodontic treatment or the use of a specific appliance increased the prevalence of TMD, except for mild or transient signs, and only 1 article[23] showed that extraction during orthodontic treatment changed the prevalence of TMD.

DISCUSSION

Relationships between traditional orthodontic treatment, orthodontic appliances, extraction during orthodontic treatment, and TMD

Since a well-publicized lawsuit[58] in 1987, interest in the relationship between orthodontic treatment and TMD has grown, and many studies have been conducted. Although most studies show little or no relationship between orthodontic treatment and TMD, some orthodontists still suffer from anecdotal testimonials.[59] In this meta-analysis, an exhaustive literature search attempted to find every study that evaluated the relationship between orthodontic treatment and TMD, including case series.[27,34,51] Case reports (fewer than 10 subjects in the sample) and opinion papers were excluded in this study.

Although we did not statistically combine the data due to severe heterogeneity, we found consistent results among the 38 primary studies. No study indicated that traditional orthodontic treatment increased the prevalence of TMD except for mild signs (soft click,[32] tenderness on palpation[33]). It is well accepted that TMJ sounds without pain or functional limitation are common and that most are normal variants, not pathologic.[60–61] Furthermore, the technique used to evaluate TMJ sounds and masticatory muscle tenderness has been reported to have low reliability.[62] Only 1 article[23] showed that extraction during orthodontic treatment changed the prevalence of TMD.

Many researchers have investigated the effects of specific appliances on TMD. Studies with Begg appliance and chincup,[29,36,38] Herbst appliance,[27,34] Class II elastics and extraction,[45] bionator and headgear,[50] facial mask,[51] and chincup[53] showed that traditional orthodontic appliances did not increase the prevalence of TMD. Some studies claimed that certain appliances (ie, bionator and Herbst)[27,34,50] reduced the symptoms.

Explanation of heterogeneous result and critique of primary studies

Due to severe heterogeneity, we could not perform a true meta-analysis. Although the sensitivity analysis did not indicate the cause of heterogeneity, we can discern the reasons. TMD does not represent a single entity; it has multifactorial origins.[63] As the NIH report[1] concluded, there are also significant problems with present diagnostic classifications of TMD, because these are based on signs and symptoms rather than on etiology. Carlsson and LeResche[64] reviewed 18 epidemiologic studies and reported that the prevalence rates of TMD were high and extremely variable: 16% to 59% for reported symptoms and 33% to 86% for clinical signs. They also noted that the wide variation is mainly

Table II. The characteristics of studies

Author(s) and reference no.	Year of publication	Overlapped sample (reference no.)	Sample	Control	Matched control	Study design	Male : female ratio	Appliance type	Dropouts
Bucci[20]	1979	N	115 tx	Y	N	C	30:85	F	
			50 no tx (malocclusion)				5:45		
			50 no tx (normal)				22:28		
Sadowsky & BeGole[21]	1980	Y (25)	75 tx	Y	Y	C	29:46	F	
			75 no tx				28:47		
Gold[22]	1980	N	170 tx	Y	Y	S	49:121	F, FA	
			201 no tx				75:126		
Janson & Hasund[23]	1981	N	60 tx	Y	N	C	30:30	F	
			30 no tx				12:18		
Larsson & Ronnerman[24]	1981	N	23 tx	N	N	C	11:12	F, FA	
Sadowsky & Polson[25]	1984	Y (21)	96 tx	Y	Y	C	33:63	F	
			103 no tx				36:67		
			111 tx	Y	Y	C	47:64	F	
			111 no tx				49:62		
Melcher[26]	1984	Y (39, 40)	30 tx	Y	Y	C	14:16	F	
			30 no tx				13:17		
Pancherz[27]	1985	N	22 tx	N	N	L	NR	FA	
Sadowsky et al[28]	1985	Y (35)	98 pre-tx	N	N	C	NR	F	
			176 tx						
			73 post-tx						
Dibbets & van der Weele[29]	1987	Y (36, 38)	172 tx	N	N	P, L	61:74	F, FA, CC	69
Loft et al[30]	1988	N	568 dental students	NR	NR	S	474:94	NR	
Dahl et al[31]	1988	N	51 tx	Y	N	C	23:28	NR	
			47 no tx				28:19		
Smith & Freer[32]	1989	N	87 tx	Y	N	C	27:60	F	
			28 no tx				12:16		
Nielsen et al[33]	1990	N	295 tx	Y	N	C	NR	F, FA	
Hansen et al[34]	1990	N	19 tx	N	N	C	19:00	FA	
Sadowsky et al[35]	1991	Y (28)	160 tx	Y	N	L	68:92	F	
			90 no tx						
Dibbets & van der Weele[36]	1991	Y (29, 38)	172 tx	N	N	P, L	78:94	F, FA, CC	63
Kess et al[37]	1991	N	54 tx	Y	N	C	NR	F, FA	
			52 no tx						
Dibbets & van der Weele[38]	1992	Y (29, 36)	172 tx	N	N	P, L	78:94	F, FA, CC	80
Kremenak et al[39]	1992	Y (26, 40)	65 tx	N	N	P, L	21:44	F	23
Kremenak et al[40]	1992	Y (26, 39)	109 tx	N	N	P, L	40:69	F	17-102
Egermark & Thilander[41]	1992	N	402 mixed	Y	Y	P, L	NR	F, FA	109
Hirata et al[42]	1992	N	102 tx	Y	N	P, L	43:59	F	62
			41 no tx				21:20		
Rendell et al[43]	1992	N	462 tx	N	N	S	NR	F	
Wadhwa et al[44]	1993	N	31 tx	Y	N	C	3:28	F	
			71 no tx				30:41		
O'Reilly[45] et al	1993	N	60 tx	Y	Y	P, L	30:30	F	
			60 no tx				25:35		
Luppanapornlarp & Johnston[46]	1993	N	62 tx	N	N	C	26:36	F	
Beattie et al[47]	1994	N	63 tx	N	N	C	32:31	F	
Egermark & Ronnerman[48]	1995	N	50 tx	Y	epidemiologic sample	L	23:27	F, FA	
			135 no tx						

Table II, cont'd. The characteristics of studies

Author(s) and reference no.	*Year of publication*	*Overlapped sample (reference no.)*	*Sample*	*Control*	*Matched control*	*Study design*	*Male : female ratio*	*Appliance type*	*Dropouts*
Olsson & Lindqvist[49]	1995	N	210 tx	N	N	P, L	94:116	F	
Keeling et al[50]	1995	N	60 tx	Y	Y	RCT	69:62	FA	
			71 tx					H	
			60 no tx						
Ngan et al[51]	1997	N	10 tx	N	N	L	NR	PH	
Lagerstorm et al[52]	1998	N	260 tx	Y	epidemiologic sample	C	123:137	F, FA	
			121 no tx						
Deguchi et al[53]	1998	N	86 tx	N	N	S	NR	CC	
Henrikson et al[54]	1999	Y (55, 56)	65 tx	N	N	P, L	0:65	F	4
Henrikson et al[55]	2000	Y (54, 56)	65 tx	Y	Y	P, L	0:65	F	1
			58 no tx CII				0:58		1
			60 no tx normal				0:60		0
Henrikson & Nilner[56]	2000	Y (54, 55)	65 tx	Y	Y	P, L	0:65	F	1
			58 no tx CII				0:58		1
			60 no tx normal				0:60		0
Imai et al[57]	2000	N	18 tx after splint	Y	N	P, L	4:14	F	
			27 tx without splint				3:24		
			13 no tx after splint				4:9		

Y, yes; *N*, no; *tx*, treated; *C*, cross-sectional; *L*, longitudinal; *P*, prospective; *S*, surgery; *RCT*, randomized clinical trial; *F*, fixed appliance; *FA*, functional appliance; *H*, headgear; *CC*, chin-cup; *PH*, protraction headgear; *NR*, no report.

due to the lack of generally accepted standards of definitions, methods of investigation, and presentation of results.

We found that symptoms, signs, or indexes were used to diagnose or classify TMD in all primary studies that we identified in this meta-analysis. However, the presence of clinical signs (TMJ sounds, or tenderness of the masticatory muscles or the TMJ) or elevated Helkimo index scores do not necessarily represent disease or treatment need. Even though the Helkimo index continues to be widely used for epidemiologic studies, it cannot be used to evaluate treatment need.[65,66] Van der Weele and Dibbets[67] also questioned the internal and external validity, and general applicability of the Helkimo index.

The limitations of TMD studies are evident from this meta-analysis. Although many have been offered, the ideal classification scheme, which provides both research and diagnostic advantages, has not been developed.[68] For future studies, development and evaluation of a reliable and valid diagnostic classification system for TMD is necessary.

For ethical and practical reasons, it is difficult to conduct randomized clinical trials to investigate the relationship between orthodontic treatment and TMD. Fortunately, many of the strengths of randomized clinical trials can be imitated,[47] and carefully designed studies were found. However, highly variable study designs and qualities among the 38 primary studies are reported here. Some studies had no control groups, and some used epidemiologic controls. Even when studies had control groups, most were not strictly matched to the experimental groups. Most authors did not mention how (or if) they controlled bias in their studies (randomization, blinding during assessments, or selecting a proper sample). Many reports were of cross-sectional studies. In general, we cannot determine a cause-and-effect relationship with cross-sectional studies. Some longitudinal studies[29,36,38-42] lost much data during the follow-up periods, raising questions about study validity. Few studies were conducted to investigate the prevalence or incidence of TMD in adult orthodontic patients after treatment.

CONCLUSIONS

We conducted this meta-analysis of the literature to elucidate the relationship between orthodontic treatment and TMD. Because of the unknown cause of TMD, methodologic shortcomings, and lack of a widely accepted classification scheme, definitive conclusions cannot be drawn. The data in this meta-analysis do not indicate that traditional orthodontic treatment increases the prevalence of TMD.

In addition, it is clear that a reliable and valid diagnostic classification system for TMD is needed for future research.

Table III. Summary of outcome

Study reference no.	Age at first assessment (y)	Time of assessment	Type of assessment	Extraction : nonextraction	Relationship between orthodontics and TMD	Relationship between extraction and TMD
20	17.09 ± 2.46 16.23 ± 3.90 22.89 ± 3.45	90% at retention check	TMJ sound	51:64	No	No
21	25-55	10-35 y after retention	Pain, TMJ sound, parafunctional habits	NR	No	NI
22	NR	4 y after debanding	Helkimo index (Di, Ai)	NR	No	NI
23	14-27 18-36	Average 5 y after retention	Helkimo index (Di, Ai)	30:30	Improved	Worsened
24	24-28	About 10 y after tx	Helkimo index (Di, Ai)	6:17	Improved	No
25	38.7 ± 8.4 37.7 ± 9.2 29.3 ± 4.2 32.9 ± 6.5	At least 10 y after retention At least 10 y after retention	Pain, TMJ sound Pain, TMJ sound	28:68 39:72	No No	NI NI
26	18.6 ± 2.3 19.5 ± 3.2	2-3 y after tx	Helkimo index (Di, Ai)	NR	Improved	NI
27	NR	F/U 1 y	TMJ sound, tenderness	NR	No	NI
28	NR	NR	TMJ sound	NR	NI	
29	12.5	F/U 10 y	Subjective symptom, objective symptom	NR	No	NI
30	20-43	NR	Questionnaire	NR	No	NI
31	19 19	Average 5 y after tx	Helkimo index (Di, Ai)	NR	No	NI
32	21.1 19.7	72 mo after retention	Interview, tenderness, TMJ sound	26:61	No (except soft click)	NI
33	14-16 14-16	NR	TMJ sound, deviation, irregular movement, pain tenderness, movement capacity	NR	No (except palpatory finding)	NI
34	20.4 ± 1.0	F/U 7.5 y	Questionnaire, movement, TMJ sound, tenderness	NR	No	NI
35	14.6	After tx	TMJ sound	87:68	No	No
36	12.5	F/U 15 y	Subjective symptom, objective symptom	114:58	No	No
37	20-30	"Finished for many years"	Helkimo index (Di), TMJ sound, limitation, tenderness, pain	NR	Improved	NI
38	12.5	F/U 20 y	Subjective symptom, objective symptom	114:58	No	No
39	16-25	F/U 2 y	Helkimo index (Di)	26:39	NI	No
40	19.7 ± 3.4	1-6 y	Helkimo index (Di)	76:33	No	NI
41	7, 11, 15	10 y	Questionnaire, Helkimo index (Di)	NR	Improved	NI
42	15.5 ± 0.7 16.2 ± 0.4	1.2 y during tx	Questionnaire, maximum opening, TMJ sound, deviation	NR	No	NI
43	NR	During tx	Helkimo index (Di, Ai)	NR	No	NI
44	15-24 13-25	Minimum 6 mo after tx	Helkimo index (Di, Ai)	25:6	No	NI
45	15.3 NR	During, just after tx	Lateral movement, TMJ sound, tenderness	60:0	No	No
46	NR	tx 1969-1980	Craniomandibular index	33:29	NI	No
47	28	tx 1969-1980	Craniomandibular index	33:30	NI	No
48	12.9 15	Before, during, after tx	Questionnaire, Helkimo index (Di)	32:18	Improved	No
49	12.8	After tx	Questionnaire, Helkimo index (Di)	NR	Improved	NI
50	9.80 ± 1.10 9.93 ± 0.82	F/U 2 y	TMJ sound, TMJ pain, muscle pain	NR	No	NI
51	8-14	Before, during, after tx	Masticatory muscle pain on palpation	NR	No	NI

Table III, cont'd. Summary of outcome

Study reference no.	*Age at first assessment (y)*	*Time of assessment*	*Type of assessment*	*Extraction : nonextraction*	*Relationship between orthodontics and TMD*	*Relationship between extraction and TMD*
52	19 20	NR	Questionnaire, Helkimo index (Di)	NR	No	NI
53	10.1	NR	Questionnaire, pain, TMJ sound, mouth opening	NR	Little	NI
54	12.8 ± 1.1	Before, during, after tx, 1 y	Symptoms, signs	35:30	Improved	No
55	12.8 ± 1.1 12.9 ± 1.0 12.7 ± 0.7	2 y after 1st evaluation	Signs (mandibular mobility, pain, TMJ sound)	NR	Improved	NI
56	12.8 ± 1.1 12.9 ± 1.0 12.7 ± 0.7	2 y after 1st evaluation	Symptoms (headache, TMJ sound, pain)	NR	Improved	NI
57	18.6 ± 4.7 18.2 ± 4.6 17.9 ± 3.6	Initial, after splint, after tx, 1 y	TMJ sound, pain, restriction	NR	No	NI

Y, year(s); *mo*, months; *F/U*, follow-up; *tx*, treatment; *Di*, dysfunction index; *Ai*, anamnestic index; *NR*, no report; *NI*, not investigated.

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