

IN BRIEF

- Reviews and investigates evidence relating malocclusion to TMD.
- Reviews and investigates evidence relating orthodontic treatment to TMD.
- Highlights the need not only for clinicians to consider whether evidence exists but also the quality of that evidence.
- Aims to place the problem of TMD in a wider context so that more informed advice can be given to patients and hence more informed treatment decisions can be made.

TMD and occlusion part I. Damned if we do? Occlusion: the interface of dentistry and orthodontics

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Objectives To review how occlusion, facial growth, TM disc position and malocclusion may relate to TMD; to review clinical studies investigating TMD pre- and post-orthodontic treatment as well as other studies linking occlusal features with TMD highlighting their limitations; and to make suggestions for improved study designs in the future in order to provide an evidence-base for clinical practice.

Design Review article.

Methods Electronic databases (MEDLINE and the Cochrane Database of Systematic Reviews) were used to select relevant and frequently cited studies (mean: 28 citations). Citation rate was confirmed using the Web of Science. Study designs are reviewed and weaknesses discussed.

Results Evidence is lacking to suggest static occlusal factors cause TMD.

Conclusions Poor study designs have led to much of the controversy over whether occlusal factors (including orthodontics) 'cause' TMD. In order to provide an evidence-base for future clinical practice, suggestions to improve study designs are made.

INTRODUCTION

TMD and dentistry

TMD (temporomandibular disorder) is a collective term embracing several clinical problems¹ involving muscles, the TMJ or both. TMDs form a cluster of related disorders with common symptoms:¹

- Pain - fairly localised
- Movement - limited/asymmetric
- Noises - clicks, grating etc.

This clustering of symptoms makes the condition/s awkward to diagnose and treat but as Table 1 indicates, TMD has been

found in individuals all over the world. Prevalence varies considerably however: between 7-84%, with an age range within the studies of 3-74 years. Such variations may result from the differing methods of assessing TMD (for example, by questionnaire or clinical examination or both; the type and quality of questionnaire/examination used, etc) and the selection procedures adopted for the population investigated. These are dealt with elsewhere.² Overall, it is estimated that approximately 10% of the population over the age of 18 years have pain in the TM region.³

Dentists have related TMD to occlusion for many years and this has directly impacted on patients and the treatment modalities offered. However, the quality of the studies on which this relationship has been based deserves an up-to-date review in the light of recent findings. Part I of this paper will examine the possibilities in terms of malocclusion, TM disc position, facial growth and orthodontic treatment. Part II⁴ will examine relationships between functional occlusion and bruxism, but will also examine important epidemiological aspects and place TMD in a wider context.

Table 1 Examples of studies demonstrating the worldwide distribution of TMD

Study	Country
Solberg <i>et al.</i> 1979 ⁶⁰	USA
Nilner and Lassing 1981 ⁶¹	Sweden
Nilner 1981 ⁶²	Sweden
Heikinheimo <i>et al.</i> 1990 ⁶³	Finland
Mohlin <i>et al.</i> 1991 ⁶⁴	Wales
De Kanter <i>et al.</i> 1993 ⁶⁵	Netherlands
Deng <i>et al.</i> 1995 ⁶⁶	China
Alamoudi <i>et al.</i> 1998 ⁶⁷	Saudi Arabia
List <i>et al.</i> 1999 ⁶⁸	Sweden
Otuyemi <i>et al.</i> 2000 ⁶⁹	Nigeria
Thilander <i>et al.</i> 2002 ⁷⁰	Colombia

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Literature search method

It was not possible to carry out a systematic review as the subject matter was too wide-ranging and studies had been carried out with marked variations in their methods, making them too heterogeneous. Also, some subjects had received little or no attention in the dental literature but were nevertheless pertinent to the topic being reviewed. Therefore, study selection was based on the following premise where possible and relevant: as an unsophisticated and general rule only, important papers tend to be cited more frequently than others.⁵ However, many papers are never cited⁶ and it has been suggested⁷ that 35% of existing papers are not cited in any given year whilst only about 9% are cited twice and only 1% six or more times.⁷ Furthermore, not all references are counted by citation databases, eg books.⁵ Thus, literature searches were carried out using MEDLINE (1966–November 2005) and the Cochrane Database of Systematic Reviews but so far as was possible, studies were only included if they had been cited at least once in the literature as confirmed by the Web of Science - Science Citation Index expanded, 1900–1914 to 2005 (for part I: range 1–313 citations; mean 28 citations; inter-quartile range 5–30 citations). References to studies earlier than 1966 were accessed by investigation of the reference lists within the studies identified.

The TMD controversy: medicolegal and professional conduct matters.

The evidence base and its relevance to dentists and orthodontists

Contrasting legal cases from the UK and USA demonstrate the need for all clinicians to be aware of developments regarding TMD. In 1987 in the US⁸ a patient developed TMD symptoms during orthodontic treatment and the orthodontic treatment was held responsible. Evidence to suggest that orthodontics had not caused the problem was lacking. This was because it was based mostly on anecdotal case reports.^{9,10} The case went against the orthodontist, resulting in almost a million dollars being paid out in compensation. Subsequently, the American Association of Orthodontics funded research to investigate the relationship between orthodontics and TMD and an entire issue of the *American Journal of Orthodontics and Dentofacial Orthopaedics* was devoted to the subject in 1992.

In the UK, two cases have very recently come before the Professional Conduct Committee (PCC) of the GDC. In 2002 a dentist was charged with serious professional misconduct and admonished¹¹ as the diagnosis and advice he gave his patient (that she had early TM joint dysfunction and required further orthodontic treatment) was wholly unjustified and without any proper clinical basis. In 2005, another case was reported in the GDC Gazette.¹² A dentist ‘...diagnosed a patient as suffering from internal derangement of the temporomandibular joint (a form of TMJ Disorder) when she did not have sufficient symptoms to justify this diagnosis and did not tell her that the treatment he proposed was not supported by informed, evidence-based opinion within the dental profession.’ He did not have her informed consent and ‘...then persisted with this flawed (orthodontic) treatment for over six years.’ The PCC found the dentist guilty of serious professional misconduct but postponed determination for 18 months.¹² However, the ruling has clear implications for any dentist providing treatment. These contrasting cases demonstrate the dichotomy that exists in the literature.

Claims that orthodontic treatment may cause (or cure) TMD can be worrying for patients who may wish to discuss such

matters with their dentist. However, it is generally accepted that claims in support of a particular treatment should be supported by (preferably) good evidence, which means other possibilities can be discounted. Otherwise, unsubstantiated claims may lead to problems of over or unnecessary treatment.

In Part I therefore, the aims are to review:

- How occlusion, facial growth, TM disc position and malocclusion may relate to TMD
- Clinical studies investigating TMD pre- and post-orthodontic treatment as well as other studies linking occlusal features with TMD, highlighting their limitations.

Suggestions are made to improve study designs in the future in order to provide an evidence-base for clinical practice.

MALOCCLUSION AS A CAUSE OF TMD

Various malocclusions have been associated with TMD signs or symptoms^{13–16} (eg class II and distal molar occlusions; anterior open bites and non-working side contacts; class III; crossbites; and deepbites). Unfortunately, these studies are difficult to perform and have generally not been carried out in an ideal manner and are therefore open to substantial criticism. A further problem when assessing the value of such clinical studies is the method by which TMD was assessed. Few studies in the orthodontic literature have used an appropriate index; the vast majority have used the Helkimo Index or a modification of it, but as highlighted by van der Weele and Dibbets¹⁷ and Luther,² this is not appropriate. Bias is also likely to be present – workers often cannot be blinded and the authors are usually orthodontists. A detailed review of such problems has previously been undertaken.^{2,18}

The correlations that have been reported between TMD and the various malocclusion types are low and unlikely to be of direct clinical significance (even if statistically significant). More importantly, correlation alone does not imply cause, yet the fact that such correlations exist appears to form the basis of statements such as ‘*Malocclusion is one of the most common causes of craniomandibular disorders.*’¹⁹ Furthermore, it is possible that these sorts of misapprehensions have led to orthodontic treatment being undertaken to treat TMD. However, this is not to say that any links with malocclusion are entirely spurious – it may be that more complex interpretation is required, as described below and in Part II.⁴ It is also necessary to investigate facial growth, as this will influence the type of malocclusion seen.

MIGHT TMD CAUSE MALOCCLUSION?

Facial growth, malocclusion and TMD

In a longitudinal cephalometric growth study, Dibbets *et al.*²⁰ found that those teenagers who developed symptoms of TMD had a different facial form or appearance to those who did not. Those with symptoms tended to have longer faces and were more skeletally class II. Similar findings have been reported in more recent cephalometric studies, which also assessed disc displacement by magnetic resonance imaging (MRI) as shown in Table 2.

In contrast it has also been suggested by Schellhas and co-workers²¹ that if orthodontics was undertaken for some Class II division 1 patients, the mechanics could exacerbate a pre-existing internal derangement by applying forces to the joint

possibly causing condylar necrosis. However, this suggestion has never been substantiated and the study itself was severely flawed, for example by being based on a pre-selected and thus biased sample. (For a critique of this study, see the 14 pages of letters subsequently printed in an issue of the same journal).²²

The problem of distinguishing causality from correlation is returned to in Part II,⁴ but the possibility that TMD causes malocclusion rather than malocclusion causing TMD should at least be considered.

TM DISC POSITION, MALOCCLUSION AND TMD

The possibility that malocclusion could be a side-effect of TMD had not been much investigated but as MRI has become more widely available, recent studies have begun to suggest that further investigation could be justified. The best way of assessing disc position is using MRI scans.^{2,23,24} Several studies²⁵⁻²⁸ have attempted to quantify the strength of links between malocclusion and TMD and/or disc displacement (DD) and a recurring theme is apparent: degree of skeletal class 'I-ness', class III malocclusions, crossbites and asymmetry seem to be frequently related to increased prevalence of TMD and/or disc displacement.

The role of 'abnormal' disc position and the relationship with malocclusion is being actively investigated in the literature. Using methods other than MRI, several studies have reported associations between DD and some forms of untreated malocclusion, eg class III malocclusions and asymmetry,^{29,30} and similar associations have apparently been confirmed by others using MRI (see Table 2). For example Nebbe *et al.*³¹ reported that adolescent females with bilateral, total DD exhibited significant cephalometric differences as compared with those with normal disc position. Such studies suggest relationships may exist between facial characteristics and DD, but serious methodological problems prevent them from being clinically useful, despite their potential importance (see Table 2). Nevertheless, it is interesting that the types of malocclusion which appear to show associations with DD are similar to those which have been said to 'cause' TMD. For example, DD appears to be related to patients with class II skeletal patterns and increased vertical proportions, ie long faces as shown in Table 2. In addition, case reports exist³²⁻³⁴ linking condylar resorption following osteotomy with (once again) severe, long face ('high angle') class II skeletally-based malocclusions, and very recent work using biomolecular techniques has shown that these same types of

Table 2 MRI Studies and malocclusion

Study	Malocclusion	Patient number	Findings
Schellhas <i>et al.</i> ²¹	II/1	128 Sample biased – all referred for suspected DD (letters pages ¹⁰)	112/128 had internal derangement of TMJ
Brand <i>et al.</i> ⁷¹	TMD patients vs controls	MRI + ceph study TMD patients n = 24; controls n = 23 Ages: 18-63 years TMD cases had mix of unilat & bi DD No statement re: blinding No power calculation	TMD cases had smaller mandibles and maxillae
Fernández San Román <i>et al.</i> ⁷²	Class I vs Class II vs Class III patients	MRI, CT + ceph study Class I n = 9; Class II/1 n = 8; Class II/2 n = 6; Class III n = 25 Patients with facial disproportion (n = 48) vs controls (n = 10, confirmed no DD by MRI) Ages: 15-45 years No statement re: blinding	56% of Class II cases had DD vs 10% Class I cases or Class III cases
Nebbe <i>et al.</i> ³¹	Females + Bi-DD cases vs normals	MRI + ceph study 17 females/grp Not blinded Ages: 10-17years	Bi-DD cases had high-angle, class II characteristics
Bósio <i>et al.</i> ⁷³	M + F symps + Bi-DD vs symps no DD vs asymp no DD	MRI + ceph study Matched age, sex, malocclusion. 96 people divided into 3 equal-sized groups: 32 asymp with no DD; 32 symp + DD; 32 asymp (7M: 25F/grp) Not blinded	Symptomatic Bi-DDs had retrognathic (ie. class II) mandible NB. Started with larger sample but many eliminated.
Gidakou <i>et al.</i> ⁷⁶	Asymp vols vs symp patients + Bi-DD	MRI + ceph study 46 normal F vs 42 symp F No statement re: blinding + no clear selection criteria Selection bias	Sig craniofacial diffs incl. SNA/SNB smaller in symp grp.
Gidakou <i>et al.</i> ⁷⁵	Asymp vols vs symp patients + UDDN	MRI + ceph study 46 normal F vs 12 symp F Blinded but no clear selection criteria Selection bias	Craniofacial diffs found eg steeper mandibular plane angle in symps.
Gidakou <i>et al.</i> ⁷⁴	Asymp vols vs symp pts + NDP	MRI + ceph study 46 normal F vs 42 symp F Blinded but no clear selection criteria Selection bias	No sig craniofacial diffs found except greater LLS retroclination in asymp

Key: DD = disc displacements; UDDN = unilateral DD without reduction; NDP = normal disc position; Symp = symptomatic; Asymp = asymptomatic; Vols = volunteers; Grp = group; Unilat = unilateral; Bi = bilateral; M = male/s; F = females; Ceph = cephalometric; sig = significant; diffs = differences; LLS = lower labial segment.

patients have a different muscle make up to those with short faces.³⁵ Thus, malocclusion - rather than causing TMD - may actually be a signal, indicative of changes occurring elsewhere about which we as yet know very little.

Indeed, whilst Paesani *et al.*³⁶ in a rather unusual study were unable (using MRI) to find DD in children and babies ranging from two months old to five years old, others³⁷ using (mostly) arthrography and (some) MRI have reported sexual dimorphism in symptomatic DD. Teenage females had three times the risk of DD compared with teenage boys, whilst the age of peak risk for DD showed no sex difference. A possible link with hormonal changes was suggested. Others have also reported DD in asymptomatic juveniles with prevalence varying with age (mean age 11 years, prevalence 8%³⁸ rising to 34% in a sample ranging from 6-25 years of age³⁹).

However, whilst MRI studies may provide a basis for future research, study designs still require improvement and if links between DD and malocclusion are confirmed, then the relationship is unlikely to be simple. Not only is DD common in asymptomatic volunteers (34%) but the prevalence of DD is even greater in symptomatic patients.^{39,40} Furthermore, of patients followed up prospectively for longer periods,^{40,41} only about 30% show no improvement in their symptoms. MRI evidence of osteoarthritis and advanced stages of internal derangement are associated with a poor prognosis. Familial aggregation of joint problems also occurs.⁴²

Whilst coincidence cannot be eliminated, the associations already described could form part of a bigger picture in which the TMJ and associated tissues are themselves affected by an underlying condition. For example, hormone (oestrogen) level reductions can be related to musculoskeletal problems. In the ovariectomised and hence osteoporotic rat (the female rat being the accepted animal model for human, postmenopausal osteoporosis), bone shape, quantity and quality is detrimentally affected by hormonally provoked atrophy and osteoporosis.⁴³ On the other hand, healthy rats given plenty of exercise show improvements in muscle/tendon structure and thickness, bone quantity, quality and form.⁴⁴ These muscle and tendon insertions comprise collagen type III (Sharpey) fibres - the same type of collagen which is affected in the genetic disorder Ehlers-Danlos syndrome. Genetic factors can influence tissue response and are known to influence human susceptibility to osteoporosis.⁴⁵ Furthermore, a genetic disorder known as generalised joint hypermobility (GJH) may be related to some forms of TMD and GJH is itself associated with some forms of Ehlers-Danlos syndrome. Unfortunately, we still await definitive evidence of whether such links definitely exist in connection with human TMD: a recent systematic review⁴⁶ concluded that it is not clear whether GJH is associated with TMD because of the methodological flaws present in the various studies reviewed.

Having said all of this, a recent study⁴⁷ assessed 3,033 subjects to investigate the relationship between self-reported TMD symptoms, overjet and overbite but found no association. There could be various explanations for this (including that there is no association), but the findings of all of these studies provide more questions than answers and at this stage highlight the need for studies to have sound methods - avoiding bias and the other problems so prevalent in much of the previous literature.

In summary at this stage, the possibility that TMD affects facial growth (hence malocclusion) cannot yet be dismissed. There is, however, another way of looking at the problem: to investigate the effects of orthodontic treatment on TMD. If malocclusion caused TMD, presumably TMD sufferers would be cured by orthodontics or the prevalence of TMD would at least be much reduced. These types of studies will be reviewed next.

ORTHODONTICS AND TMD

Does orthodontics cause TMD?

Clinical studies examining orthodontic treatment have investigated possible links between orthodontics and TMD by comparing symptoms and/or signs of TMD in:

- Treated *versus* untreated patients
- Patients treated by different means
- Longitudinal studies - the same patients are compared before and after treatment.

Other methods have also been adopted, eg comparing joint spaces cephalometrically before and after treatment, but the assumptions and methods on which such studies are based are in any case flawed² and will not be discussed here. Many of the weaknesses associated with orthodontic studies have been reviewed previously^{2,18} and the problems of how TMD is assessed should be remembered, as few studies in the orthodontic literature have used an appropriate index; the vast majority have tended to use the Helkimo Index or a modification of this but this - as has been highlighted previously^{2,17} - is not appropriate.

Studies comparing treated *versus* untreated patients

Many such studies have been undertaken (see Table 3) and most have used patients with a mixture of malocclusions. Only those asterisked in Table 3 have undertaken studies of patients with specific types of malocclusion.⁵⁰⁻⁵³ This is relevant, as it has been argued that failure to segregate samples sufficiently causes dilution of the very effects one is looking for.⁵⁴

Henrikson and Nilner⁵³ compared 11-15 year old, treated and untreated female subjects with class II division 1 malocclusions - defined as having uni- or bilateral class II relations of at least half a cusp ($n = 65$ and $n = 58$ respectively). These subjects were compared with females with normal occlusions ($n = 60$). Patients were recruited prospectively and consecutively from a treatment waiting list; they were treated with fixed appliances (some with headgear, class II elastics and 50% with extractions). All other individuals, ie untreated subjects, were recruited from school screening programmes; drop outs were also accounted for. Assessments for these prospective studies were undertaken 'blind' to an extent; Henrikson also adopted some principles advocated in one of the more valid and reproducible TMD assessment indices - the Research Diagnostic Criteria (RDC).⁵⁵

As a result of such improvements in study design, Henrikson's work may provide more insight into the true association (or otherwise) between orthodontics and TMD. He found that subjects with normal occlusions had the lowest prevalence of TMD signs compared with those with treated or untreated class II division 1 malocclusions, who always had a higher prevalence of TMD signs/symptoms. Treated class II division 1

patients had a lower prevalence of TMD signs/symptoms than untreated subjects with a similar malocclusion. Henrikson and Nilner⁵⁶ suggested that this may be due to improvements in dental occlusion and occlusal stability, but these studies were not designed to confirm this nor do they exclude other explanatory factors.

Studies comparing patients treated by different means

These studies compare and contrast treatment mechanics that maximise differences in condyle position, testing the assumption that treatment mechanics trap the mandible posteriorly, forcing the TM disc anteriorly which has been said to 'cause' TMD.^{2,9} Such specific, longitudinal studies have been undertaken infrequently for obvious reasons: large sample sizes are likely to be necessary to achieve sufficient statistical power (to ensure that sufficient numbers of patients are included to find a clinically significant difference should one exist) and patient drop out may be high over the long periods required. However, Dibbets and van der Weele⁵⁷ compared children who had either been treated with four premolar extractions and Begg fixed appliances (n = 72) or non-extraction with activator functional appliances (n = 63). Patients were assessed before treatment, four years post-retention and finally at 10 years after the start of orthodontic treatment. Signs and symptoms of TMD increased with age but 10 years after the study had started, the initial differences in symptoms and signs between the two groups no longer existed. This suggested that age was a confounding factor: the prevalence of TMD increases with age - children treated with functional appliances start their treatment earlier and thus have a lower prevalence of TMD initially.

Longitudinal studies

A number of such studies have been undertaken (see Table 4) and again require especially good, long-term follow up. Recently, a meta-analysis⁵⁸ concluded that the data did not indicate that traditional orthodontic treatment increased the prevalence of TMD.

Orthodontics and functional occlusal factors

It has been suggested that an internal derangement may be caused by the mandible being retracted during some forms of orthodontic treatment.⁵⁹ Premolar extractions in the upper arch (as might be needed for overjet reduction) have been said to cause the mandible to be trapped, forcing the TM disc anteriorly due to the condyle having been forced posteriorly.⁹ However, as reported previously² there is no evidence to support this idea and for example, the work of Wyatt⁵⁹ was based only on personal opinion.

A further possibility - often implied rather than explicitly stated in the literature⁶⁰ - is that some occlusal contacts (such as non-working side contacts or interferences) may trigger clenching or grinding which causes TMD. Such contacts may occur normally or could be introduced during orthodontic treatment. Studies investigating these possibilities will be discussed in Part II.⁴ Displacements on closure (for example as associated with crossbites) have also been implicated and have been the subject of a Cochrane Systematic Review.⁶¹ However, whilst the primary outcome measures assessed included patient orientated outcomes such as resolution of pain and clicking as

a result of treatment, the studies that could finally be included rarely actually reported such outcomes. In fact only two trials reported any patient orientated outcomes and these related to treatment times.

Orthodontics as a 'cure' for TMD

Various possibilities have been suggested, but a popular approach adopted by several authors has been to use orthodontics to help 'recapture' the TM disc and thus help treat TMD. However, these suggestions are based on case reports, case series or anecdotal evidence and, as Okeson¹ has stated, management of TMD has been primarily based on treatments selected by the clinician's personal biases. These aspects will therefore not be reviewed further here.

Table 3 Studies comparing treated versus untreated patients

Study	Country
Sadowsky and Begole 1980 ⁷⁷	USA
Sadowsky and Polson 1984 ⁷⁸	USA
Larsson and Rönnerman 1981 ⁷⁹	Sweden
*Janson and Hasund 1981 ⁴⁸	Norway
Dahl <i>et al.</i> 1988 ⁸⁰	Norway
*Keß <i>et al.</i> 1991 ⁴⁹	Germany
Egermark and Thilander 1992 ⁸¹	Sweden
Hirata <i>et al.</i> 1992 ⁸²	USA
Wadhwa <i>et al.</i> 1993 ⁸³	India
Lagerstrom <i>et al.</i> 1998 ⁸⁴	Sweden
*Henrikson 1999 ⁵⁰	Sweden
*Henrikson and Nilner 2003 ⁵¹	Sweden

*Studies assessing patients with a specific malocclusion type

Table 4 Longitudinal clinical studies comparing TMD prevalence pre- and post-orthodontic treatment

Study	Country
Pancherz 1985 ⁸⁵	Sweden
Sadowsky <i>et al.</i> 1991 ⁸⁶	USA
Dibbets and van der Weele 1992 ⁸⁷	Netherlands
*Rendell <i>et al.</i> 1992 ⁸⁸	USA
Kremenak <i>et al.</i> 1992 parts I and II ^{89,90}	USA
Egermark and Thilander 1992 ⁸¹	Sweden
Hirata <i>et al.</i> 1992 ⁸²	USA
O'Reilly <i>et al.</i> 1993 ⁹¹	USA
Egermark and Rönnerman 1995 ⁹²	Sweden
Olsson and Lindqvist 1995 ⁹³	Sweden
Keeling <i>et al.</i> 1995 ⁹⁴	USA
Ngan <i>et al.</i> 1997 ⁹⁵	Hong Kong
Henrikson <i>et al.</i> 1999 ⁹⁶	Sweden
Henrikson and Nilner 2000 ⁵⁴	Sweden
Henrikson <i>et al.</i> 2000 ⁹⁷	Sweden
Imai <i>et al.</i> 2000 ⁹⁸	Japan
Egermark <i>et al.</i> 2003 ²⁸	Sweden

*Studies assessing patients with a specific malocclusion type

The overall findings of orthodontic clinical studies

Currently, and based on the available evidence, the widely accepted conclusion is that orthodontic treatment neither causes nor cures TMD.^{2,58} If further studies are planned, they should (so far as is possible):

- Be prospective
- Be undertaken by clinicians using a reproducible and validated index
- Be undertaken by clinicians blind to which group the patients belong to
- Have patients randomly allocated to treatment/control groups
- Be appropriately powered
- Include patients of similar backgrounds with similar malocclusions and who are compared with appropriate controls.

However, whilst many people undergo orthodontic treatment, even more people do not and since TMD appears to be a common condition, such 'orthodontics only' studies will do little to investigate the major causes of TMD or the best treatments. It would therefore be useful to review other features and factors which may have a more significant role to play in the aetiology of TMD. Such aspects are reviewed in Part II.⁴

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