IN BRIEF

- Reviews and investigates relationships between functional occlusion and TMD.
- Reviews and investigates relationships between bruxism and TMD.
- Reviews the aetiology of bruxism.
- Highlights the need for clinicians to consider the quality of evidence as TMD is placed in a wider context, including epidemiological aspects of musculoskeletal disease and factors relating to cause and effect.
- Aims to place the problem of TMD in a wider context and illustrate the need for a more coherent explanation of the disease profile actually seen, so that more informed advice can be given to patients and more informed treatment decisions can be made.

TMD and occlusion part II. Damned if we don't? Functional occlusal problems: TMD epidemiology in a wider context

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Objectives To review studies investigating how functional occlusion may relate to TMD and how bruxism may relate to TMD; to review the epidemiology of TMD and relate this to the context of clinical occlusal studies and other aetiological factors. Deficiencies in study design are highlighted and suggestions made to improve future study designs 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: 40 citations). Citation rate was confirmed using the Web of Science. Study designs are reviewed and weaknesses and implications discussed.

Results Evidence is lacking to suggest functional occlusal factors cause TMD. Investigation of other aetiological factors has been relatively neglected.

Conclusions Neither static nor dynamic occlusal factors (including orthodontics) can be said to 'cause' TMD. However, other potential aetiological factors exist which would benefit from more investigation. This, together with improved study designs, would help provide a stronger evidence-base for clinical practice in the future.

INTRODUCTION

Whilst orthodontic treatment treats the static occlusion (ie malocclusion) as discussed in Part I,[1] attention has also focussed on dynamic aspects of the occlusion, ie functional occlusion – which may itself be affected by orthodontic treatment. In part II, studies investigating how functional occlusion may relate to TMD are reviewed, including the relationship between bruxism and TMD. In addition, in order to strengthen the evidence-base for clinical practice, TMD epidemiology will be reviewed and its relevance and relationship to occlusal studies demonstrated including the need to take other aetiological factors in to account.

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LITERATURE SEARCH METHOD

As described in more detail in Part I,[1] literature searches were carried out using MEDLINE (1966-November 2005) and the Cochrane Database of Systematic Reviews and where 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 II: range 1-426 citations; mean 40 citations; inter-quartile range 12-32 citations).

FUNCTIONAL OCCLUSAL PROBLEMS – A CAUSE OF TMD?

Occlusal interferences and TMD

The suggestion that TMD comes about due to unfavourable, dynamic occlusal contacts is inherent in much of the literature relating orthodontics and TMD, but the mechanism is usually unclear. However, the study which probably helped stimulate the suggestion was by Ramfjord.[2] Thirty-four patients were chosen (because they were 'known, severe bruxists') to undergo electromyography (EMG) in a laboratory during various occlusal exercises and then once more following occlusal adjustment. They were then asked about their bruxing habits 30 minutes to several months later. Ramfjord concluded that any type of occlusal interference may, when combined with nervous tension, initiate bruxism. Furthermore, bruxism could be eliminated by occlusal adjustment. In those with bruxism, it was also suggested that adjustment of the occlusion to centric relation in order to achieve muscle balance was necessary. However, the study was severely flawed. For example:

- Patients were not randomly selected; the selection may therefore have been biased
- There was no control group; none of the patients were orthodontic patients and many were not TMD patients
- No information was given to explain how bruxism was diagnosed

- The patients were assessed in an entirely artificial situation
- The assessor was not blinded and this may again lead to bias
- No power calculation was performed, therefore the sample size may have been too small to find a difference (if one existed).

This study presents profound problems associated with the method adopted and it seems strange now that such a study should have had the influence it appears to have done. Nevertheless, various studies have since been undertaken by numerous authors which appear either implicitly or explicitly to be based on Ramfjord's ideas (see Table 1 for some examples).

Lund[3] has summarised this 'vicious cycle' theory of muscle pain (or 'pain-spasm-pain' theory of chronic muscle pain), which is based on the idea that persistence of chronic muscle pain is explained if pain itself causes or maintains muscle hyperactivity. Thus muscle spasm is seen as the main cause of TMD and the main cause of muscle spasm is seen as bruxism: a vicious cycle ensues as abnormal muscle function triggers more bruxism. The mechanism of how 'unfavourable' occlusal contacts actually trigger TMD (or a form of TMD) is seldom spelled out in detail, but often appears to invoke the vicious cycle idea which itself derives from earlier work such as that of Laskin[4] and even earlier workers – Lund[3] cites Schwartz (1959) and Travell (1942). Lund[3] has explained how this approach has led to treatment aimed at reducing muscle activity either directly, eg by biofeedback, or indirectly by correcting 'abnormal' anatomy, eg occlusal adjustment, use of occlusal splints, orthodontics or surgery etc. However, where a disease is to be diagnosed and treated, it is necessary to establish not only the aetiological factors but also how those factors operate. Lund[3,5] has reviewed the evidence to ascertain whether the 'vicious cycle' would stand up to scrutiny, ie that muscle hyperactivity could lead to pain and that pain leads to tonic hyperactivity. Lund concluded:

- Sustained or unaccustomed patterns of exercise can cause tissue damage and pain, yet
- There is little evidence that this is the aetiology of forms of TMD or of headache, fibromyalgia or chronic lower back pain
- It is clear that chronic pain *reduces* the strength of contraction of agonist muscles (ie muscle activity) and...it may *slightly* increase the level of activity in antagonist muscles in the painful body part (the author's italics and parentheses)
- Pain also causes distinctive facial expressions, body postures, and gestures. All these effects of pain appear to be adaptive.

Table 1 Finnish studies using occlusal adjustment

Study	Year	Cohort	n	Effect
Forsell, Kirveskari and Kangasniemi[38]	1986	Headache patients	96	+
Kirveskari *et al.*[39]	1989	Dental students	62	+
Kirveskari *et al.*[40]	1989	Healthy 15 year olds	147	+
Kirveskari *et al.*[41]	1992	Healthy five and 10 year olds	96-106 five year olds 64-74 10 year olds	-/+
Karjalainen *et al.*[42]	1997	Ortho-treated adolescents	123	+
Kirveskari *et al.*[43]	1998	Healthy children/ adolescents	146	+

+ = positive effect; - = negative effect of adjustment.

Posterior crossbites, displacements on closure and TMD

Another form of functional malocclusion can be said to exist when posterior crossbites are considered. To quote from Harrison and Ashby's 2001 Cochrane review[6] on orthodontic treatment for posterior crossbites:

'A posterior crossbite occurs when the top teeth or jaw are narrower than the bottom teeth and can happen on one or both sides of the mouth. When this takes place the bottom jaw may have to move to one side or the other to allow the back teeth to bite together. This abnormal movement of the lower jaw causes the top back teeth to bite inside the bottom back teeth, ie a posterior crossbite. This movement is more likely to occur when the posterior crossbite involves one side of the mouth. Many believe that the abnormal movement of the lower jaw associated with a crossbite can have long term effects on the growth and development of the teeth and jaws. It is unlikely that young children with a posterior crossbite will experience any pain or have problems with chewing. However, the postulated abnormal movement of the lower jaw may put a strain on the jaw muscles and joints which may cause problems in later life – for example pain, clicking or locking of the jaw joints. Such problems have many causes but, studies of teenagers and adults have shown that patients with a crossbite have an increased risk of developing jaw joint problems and show more signs and symptoms of these problems (Mohlin 1984; Riolo 1987; Egermark 1990; Pullinger 1993; Ninou 1994; O'Bryn 1995; McNamara Jr 1997).'

However, the problem remains that of establishing cause and effect. Furthermore, as stated in Part I,[1] unfortunately no studies have actually reported on factors such as the resolution of pain and clicking as a result of treatment for the crossbite.

Overall, it appears that unsubstantiated assumptions have misled both researchers and clinicians and studies which could have investigated cause and effect have not done so. A key question still therefore remains - does bruxism cause TMD?

IS BRUXISM A MAJOR CAUSE OF TMD?

The earlier discussion was based on the assumption that bruxism is a significant cause of TMD. The mechanism (put forward by Laskin[4] as the psychophysiologic theory of TMD) – if true – is suggested to be via muscle spasm, which is actually the primary factor responsible for signs and symptoms of TMD since bruxism causes muscle spasm via muscle fatigue.[3,4] It was also suggested that the condition became self-perpetuating (the vicious cycle once more)[3,4] and could lead to organic disease including not only TMD but also degenerative arthritis. As a consequence of this one might therefore anticipate that patients bruxing at night, would have their worst pain the morning after. This whole question has also been extensively reviewed by Lobbezoo and Lavigne[7] who also concluded that the supporting evidence for this vicious cycle was weak. For example, the association of pain with bruxism is not a universal finding – three studies are cited which show that many

nightly bruxers fail to have any pain. Dao *et al.*[8] even found that whilst those bruxers who reported pain experienced worse pain than the patients with myofascial pain, pain was not their chief complaint. Furthermore, the fact that some studies suggest that the prevalence of bruxism is highest in children but decreases into adulthood[9] is at odds with the known prevalence of TMD. Such findings reduce the probability of an absolute cause and effect relationship.[7] However, the problems of establishing who bruxes (and how much) and establishing cause and effect are discussed later, but are compounded when examining sleep and bruxism, as such studies require volunteers to take part in sleep lab studies.

Assessment and quantification of bruxism

The problems of quantifying and assessing bruxism have only relatively recently been considered. Study model assessment or patient recollection[7] are insufficient: the former being only a snapshot (is the problem ongoing? Is bruxism the only cause of any wear seen?) and the latter unreliable. Instead, it is suggested that sleep lab data (complete with video, sound and EMG recordings) should be used together with patient reports and attrition assessments (by observation of serial study models and monitoring appliances). Unfortunately, few such studies have been carried out – particularly on a large scale basis – probably because they require extensive and expensive specialist facilities, making it difficult to accommodate large numbers of volunteers. The true prevalence of sleep bruxism may therefore be underestimated.[9]

Does occlusal adjustment stop bruxism?

In order to address this question one would ideally undertake a randomised clinical trial in which one group of bruxists receives occlusal adjustment whilst the control group of bruxists does not, and the effects are assessed by clinicians with no knowledge (blind) of the purpose or indeed even the existence of the study. However, a recent review[10] has confirmed that no such ideal clinical trials have assessed whether occlusal adjustment influences bruxism. Bailey *et al.*[11] did attempt an investigation, albeit with only nine patients who sought treatment for bruxism. They underwent EMG recordings before, during and after occlusal adjustment (at two weeks apart). Six patients experienced no effect, one improved and two experienced worsening of their bruxism. The value of occlusal adjustment has also been examined by others.[10,12,13] Tsuykiyama *et al.*[10] specifically examined studies investigating the effects of occlusal adjustment on TMD. Eleven studies were reviewed and their numerous shortcomings highlighted. These included lack of operator blinding and calibration, and lack of valid TMD assessments.

In addition, mixtures of TMD diagnostic types and treatment combinations rather than comparisons of single types of treatment were used. It is clear that such studies are not methodologically sound and Tsuykiyama *et al.*[10] concluded that available research evidence in this area is minimal.

An alternative approach has been adopted by a Finnish group and simply involves assessing the effect of occlusal adjustment on TMD symptoms and signs in a population. In a series of so-called randomly controlled clinical trials (see Table 1), two cohorts (usually) of individuals were followed up for periods of up to four years. No examination for bruxism was undertaken. The individuals varied but were generally healthy children or teenagers; they did not complain of TMD symptoms nor were they TMD patients, except for one group who had attended for treatment of headaches. The cohorts were split 'randomly' into two groups and were assigned to either a true occlusal adjustment group or a mock procedure. Adjustments took place either in one episode or at roughly six monthly intervals. In one study, adjustment was 'sporadic.'

Overall, these authors considered that occlusal adjustment had a protective effect against TMD. However, before accepting their conclusions, it is worth considering at least the following:

- Are all randomly controlled clinical trials good randomly controlled clinical trials?
- The individuals involved were mostly 'disease'-free. Clinical trials are appropriate when it appears that a new therapy may give better results than an established therapy.[14] Is it therefore ethical to undertake an irreversible procedure which is not guaranteed to be preventive or beneficial (or indeed more beneficial than some other, reversible treatment) even where a real rather than – as in this case – a largely non-existent disease entity is considered?
- The method of symptoms/signs assessment of TMD was inadequate
- No power calculations were undertaken so the studies may lack power
- The means of randomisation is mostly unstated/unclear and may therefore be open to bias
- Assessors were said to be blinded. However, this may be impossible to achieve practically if researchers are aware of the study and its aims: irreversible procedures undertaken intra-orally may have been apparent to patients and operators alike
- Assessors were not independent of the study aims
- Possible complications such as sensitivity are poorly reported
- In the 1998 study,[40] the drop outs may have had a significant effect on the results; a small change may have reversed the results; there may also have been significant volunteer bias (only 170 took part out of 1,100 invitations), especially when the information given out to volunteers is considered
- This type of study has not been undertaken by others. How 'generalisable' are the results?

Overall, Tsuykiyama *et al.*[10] agreed with Forsell's[12] findings and concluded that the experimental evidence was not sufficiently convincing to support the performance of occlusal therapy as a general method for treating non-acute TMD, bruxism or headache. Similar conclusions have been reached by Stohler,[15] Clarke *et al.*[16] and by Koh and Robinson's Cochrane Review[13] which investigated the value of occlusal adjustment for both treatment and prevention of TMD. Other problems associated with occlusal adjustment have also tended to be disregarded, such as:

- The need to repeat occlusal adjustments
- The lack of information regarding the longevity and long-term stability of 'functional occlusions'[17]
- The variation in what is considered to be an appropriate occlusal adjustment scheme.[17]

Stohler[15] has also pointed out that pain in or around the TM joint may in any case cause the occlusion to change, resulting in an occlusal contact.

The aetiology of bruxism – what does cause bruxism?

It appears that bruxism does not always cause TMD and indeed, the cause or causes of bruxism remain to be established. Lobbezoo and Naeije[18] suggested that daytime bruxism may differ from night time bruxism and have noted the relevance of the sleep arousal response. An arousal response is a sudden change in the depth of sleep during which the sleeper either arrives in a lighter sleep stage or wakes up. This change is accompanied by gross body movements such as:

- Turning over
- EEG changes
- Increased heart rate
- Respiratory changes
- Peripheral vasoconstriction
- Increased muscle activities.

Sleep lab studies have found that rhythmic masticatory muscle activity at night (RMMA) is normal and affects 60% of adults. Consequently, and as a result of RMMA, people not infrequently grind their teeth together but this occurs far more frequently in those with sleep bruxism.[19] RMMA can be increased by various factors such as a central chemical imbalance, drugs, alcohol and psychological factors.

In summary, the available evidence fails to support the assumption that occlusal interferences cause bruxism. The role of bruxism itself as an aetiological factor in TMD requires further investigation, but it is doubtful that bruxism always causes TMD. In addition:

- Occlusal adjustment has not been shown to prevent bruxism
- Occlusal adjustment has not been shown to prevent TMD
- Occlusal adjustment has not been shown to be more effective than other, non-invasive treatments.

CAUSE AND EFFECT: HOW CAN THEY BE ESTABLISHED?

This is difficult. Elwood[14] suggested a causal factor was one *'whose operation increases the frequency of an event.'* However, other factors also need to be in place. Lobbezoo and Lavigne[7] cited Spilker (1991) who suggested that the following factors at least should apply before a factor could be considered as possibly causal:

- Bias, chance, confounders should be absent
- The association should be consistent and reproducible
- The potential cause must precede the effect
- A dose response gradient should be present
- The association should make epidemiologic sense and be specific.

Furthermore, LeResche[20] has emphasised that whilst epidemiology is defined as the study of the natural history of disease in populations, disease distribution, and determinants, it is important that pain conditions are studied in the entire population – not just those seeking treatment since otherwise, important aetiological factors may be missed. For example, factors influencing pain may vary with age.

In many studies, these requirements are often not met and therefore the role of occlusal interferences in the causation of TMD seems questionable – especially when viewed in the wider context of TMD epidemiology. Several long term studies exist which suggest that most (85-90%) TMD patients do well with conservative treatment, ie experience relief of symptoms.[21] In relation to disc displacement (DD, internal derangement) the outcome of this condition is typically also benign, with natural compensation and compensatory remodelling. Indeed, DD is common in asymptomatic volunteers (34%) but comparatively recent MRI studies comparing asymptomatic with symptomatic patients have found not only that the prevalence of DD was much greater in symptomatic patients but also that there was a strong association between DD and TMD.[22,23]

OCCLUSION, TMD AND TMD EPIDEMIOLOGY – A WIDER CONTEXT?

Many diseases have characteristic age and sex profiles which relate in some way to that particular disease aetiology. For example:

- Osteoporosis: affects 33% females and 10% males >50 years. Aetiology: physiological bone loss; changes in hormone balance; genetic factors; sedentary lifestyle. Also secondary causes (alcoholism, glucocorticosteroids etc).[24]
- Osteoarthritis: affects females > males but most people over 60 years show radiographic signs although only a third have symptoms. Aetiology: no single known cause; risk factors include age; also obesity, sports injuries, overuse, genetics.[25]
- Rheumatoid arthritis: onset usually in middle age but it is a common disease which affects females > males; also affects young adults and children. Aetiology: not fully known but involves an autoimmune and genetic mechanism.[25]
- Low back pain: commonest in 30-50 year olds; affects females just as much as males. Aetiology: age changes to the bone affecting bone strength and muscle elasticity; back injury or trauma.[26]

It is not surprising that a similar profile exists for TMD. It has been stated[21,27] that cross sectional epidemiologic studies of a specific, non-patient population show that up to 75% have at least one sign and 33% have at least one symptom. However, simply possessing a sign or symptom does not mean that treatment is essential or necessary. In fact, if more than one symptom is considered, LeResche[20] has suggested that only 10% of the population (over 18 years) are likely to have TMD symptoms, whilst Okeson[21] and McNeill[27] – citing other authors' studies – estimate that only 3.6-7% are actually estimated to need treatment (only 3-4% according to others[28]). It therefore seems inappropriate or at least unnecessary to treat all subjects irrespective of whether they complain of symptoms and who – in fact – are quite likely not to experience significant (or possibly even any) symptoms – especially when that treatment outcome is unpredictable. Furthermore, it is important to be aware of the natural history and progression of TMD. In a 30 year, longitudinal study, de Leeuw *et al.*[29] concluded that most TMD articular disorders followed a natural course, irrespective of the treatment approach.

For TMD, the sex ratio (for symptoms) is at least 2:1 females: males. The condition is uncommon in children, prevalence increasing in the late teens and peak prevalence occurring in 35-45 year olds. It has been suggested[30] that sex predilections are due to one of three factors:

- Physiological or anatomical differences
- Behavioural differences
- Genetic differences.

Postmenopausal osteoporosis is an excellent example of a disease which preferentially (but not exclusively) affects females and occurs (at least in part) due to changes in female hormone balance with age. Other factors include differences in macro- and microanatomy, activity levels and genetic susceptibility. A review of evidence associated with TMD and gender[30] has highlighted the dearth of information; the authors suggested that much further work is needed so that the basis of the sex predilection for TMD can be established. For example, the role of hormones (both endogenous and exogenous) in relation to TM bone and joint extracellular matrix changes, the effects of sex on pain perception and its possible modulation by hormones, and the relationship between menstruation and menopause timing and TMD would also benefit from thorough investigation, as would genetic factors.

The complex nature of TMD is further highlighted by considering that TMD may be only part of the overall pain condition or picture a patient may have. Tertiary care patients exhibit 'comorbid conditions' much more frequently in areas of the body other than the face and their condition is relatively seldom limited only to the face. Indeed, patients with more than one condition are more likely to be referred than patients with only one condition.[31] Furthermore, whilst some conditions might be trivial, others may include depressive illness, sleep disturbances and widespread pain, all of which could affect overall illness severity.[31] Okeson[32] has cited evidence suggesting that the presence of existing severe headache, back, abdominal or chest pain is a better predictor of TMD than is depression; consequently, dentists are likely to be unaware of the other ('comorbid') conditions. That being the case, establishing 'causal factors' may be doomed to failure if all the relevant factors are not taken into account.

If we use osteoporosis as an example, were we only to examine the jaws yet attempt to generalise our findings, we might conclude that osteoporosis does not affect the skeleton much (the effects on the jaws usually being somewhat milder). This would of course be completely wrong. We would simply have looked in the wrong place – perhaps because of lack of awareness or knowledge.

TMD is clearly a complex group of diseases and we still know little about the aetiology/ies. The possibility exists that aspects of facial growth (hence malocclusion) and some TMD problems may in some way be related. For example, it may be that more severe forms of malocclusion may represent a form of proxy for TMD – an indication that some patients might be at greater risk of TMD. Indeed, whilst all of this is clearly speculative, this might explain why TMD can show a correlation with malocclusion yet 'occlusionist' theories do not satisfactorily deal with the known epidemiology TMD.

If viewed in this wider context and in a similar way to other (musculoskeletal) diseases and conditions, it becomes difficult to see why or how (for example) an instanding lateral incisor, a buccal crossbite or an increased overjet could, in themselves, 'cause' the disease profile seen. The TMJ and its associated structures are not so different to other joints that no similarities exist – for example, Gidarakou *et al.*[33] cite evidence that other joints (knees, spine) also commonly have disc displacements and clicking which of themselves do not require treatment.

It therefore seems unlikely that treatment of malocclusion will 'cure' TMD, since any underlying cause/s would still be present. For example:

- Inflammatory processes
- Effects of age changes on muscle, tendon and bone composition – bone density increases up to the fourth decade but significant changes in bone mass and density occur during puberty and the pubertal growth spurt. Age and other physiological changes are known to affect many tissues including muscle[34-36]
- Sex and/or genetic factors – possibly involving hormone level changes and/or pain receptors/perception and/or central factors
- Bruxism and probably other factors.

Ultimately, more coherent explanations for the disease profile and natural history we actually see are required. Allowance needs to be made for the likelihood that there are probably different sub-types of TMD (eg not all TMD involves disc displacement), as has been emphasised in a study by Huang *et al.*[37] Risk factors for TMD were investigated after classification of subjects into diagnostic subgroups of painful TMD, while controlling for age, sex and other potential confounders. Risk factors included trauma, clenching, third molar removal, somatisation and female sex. The need to establish the temporal sequence and the mechanism by which pain is associated with female sex, together with investigations involving larger groups of TMD sub-type are acknowledged, but provide useful clues to other avenues which would merit investigation.

The fact that multiple treatment interventions have the same or similar outcomes to a 'no treatment intervention' suggests (as stated previously by Stohler and Zarb[31]), that we should use a low tech approach.

CONCLUSIONS

- There is no conclusive evidence to show that occlusal factors (including orthodontics) 'cause' TMD
- Orthodontic studies involving the assumption that orthodontic treatment causes TMD have restricted the possibility of more useful studies being done to investigate the aetiology of TMD
- Treating symptoms by using treatments X or Y does not demonstrate cause and effect and the known epidemiology of TMD must be taken into account when investigating the aetiology of TMD
- Investigating the aetiology of TMD requires an evidence-based approach using sound methods; unsubstantiated hypotheses should no longer be pursued.

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