European Journal of Orthodontics 19 (1997) 249–258

The aetiology of temporomandibular disorders: a philosophical overview

John R. C. Mew
Purley, UK

SUMMARY Over the last 50 years many theories have been put forward to explain the syndrome currently called 'temporomandibular disorders'. However, it is doubtful whether any single theory fits all the complex features of this condition. The 'multifactorial' explanation also appears flawed. On the assumption that a valid theory should fit the whole evidence, this paper starts by separating the 'predisposing' from the 'initiating' factors, and notes that most existing theories appear to identify the former rather than the latter. The research material is examined in an effort to formulate a theory that is both logical and fits all the known clinical findings.

Introduction

Many diseases do not have a clear-cut aetiology and this is especially true of temporomandibular disorders (TMD). Not only are there many competing theories to be considered, but a clear definition of the condition itself has yet to be agreed between the many disciplines involved in diagnosis and treatment. Despite this lack of consensus, clinicians are expected to provide care for TMD sufferers, especially if they are in pain, and a number of medical and dental treatment regimes have been developed. Each of these tend to have a loyal group of disciples following set clinical procedures, most of which have been developed on an empirical basis. Many of them have similar success ratios of around 75 per cent, which might suggest that they are influencing the same factor or factors. The percentage of patients who fail to respond are often passed from one specialist to another, until they refuse further treatment or learn to accept their condition.

The underlying problem has been a failure to establish the aetiology of TMD. There is an abundance of research material available, but unfortunately too little effort has been made to integrate and rationalize the facts. Some would seek to classify TMD on clinical grounds, but this creates innumerable subdivisions which confuse the allocation of cause. As a result, a number of contrasting theories have evolved, some of which appear to be tailored to fit the treatment administered rather than the evidence. An article of this size can only review a small proportion of the literature and so references will only be given where differences of opinion might exist.

Assessment

In order to construct a tenable theory for the cause of TMD, the circumstances that lead to or are associated with failure of the joint need to be analysed. Rationally, each living species represents the end result of an evolutionary sequence stretching back to the origins of life. As such, their organs and structures might be expected to function satisfactorily for an average lifespan and indeed there is little to suggest that our ancestors had any problems with their joints. Why then, in current times, do between 35 and 72 per cent of human temporomandibular joints appear to suffer from pathological signs or symptoms? (Dibbets *et al.*, 1985; Egermark-Ericsson *et al.*, 1987; Schiffman and Fricton, 1988; Mohlin *et al.*, 1991).

The pattern of failure of all functioning systems, whether mechanical or biological, obeys scientific laws. These can be considered under the following headings:

(A) Structural. An intrinsic fault in the structure

of the machine or organism. Mechanical faults can often be corrected; however, biological faults tend to result in the extinction of the species, thus removing the fault.

(B) Precipitating. The factor that triggers the failure.

(C) Predisposing. A number of factors may weaken a system so that it becomes vulnerable to failure.

(D) Resultant. These arise directly from the failure itself.

(E) Associated. These accompany the failure, but play no part in its cause, although they are sometimes portrayed as such.

(F) Unrelated. These would be random variations unrelated to any of those listed above.

Normally, a failure is triggered by a single 'precipitating' factor, but on occasions two factors may be involved coincidentally. However, this is extremely unlikely. On the other hand, it is common for several 'predisposing' factors to be involved simultaneously. Obviously, a similar failure on a separate occasion could be due to a different 'precipitating' factor, but here again mathematical probabilities come into play. For example, an automobile bearing could fail for one of a number of reasons, such as shortage of oil, overloading, over-revving, incorrect assembly, overheating, etc., most of which would be 'predisposing' factors. However, if the same bearing failed in a number of cars of one particular model, a trained engineer would look for a common 'precipitating' cause, and try to separate this from the range of 'predisposing' factors.

On an actuarial basis, repeated similar failures of one type of machine or biological structure are due to the same single 'precipitating' factor, regardless of how many or how varied are the 'predisposing' factors.

It is necessary to emphasize this because TMD, in company with other human pathologies, is sometimes labelled 'multifactorial'. Whilst this may be true in the sense that there could be many predisposing and associated factors, it tends to divert attention from the need to identify the single most likely 'initiating' factor. For this, a rational assessment is just as essential for the clinician as it is for the motor mechanic. As Garn (1961) concluded 'When the unitary explanations were exhausted the multifactorial hypothesis was advanced ... this yielded an equation with an unknown number of unknowns instead of just one'.

Establishing a sound theory

Some years ago, the following definition was put forward. A sound theory should fit all the available evidence rather than rest on part of it. It needs to be both logical and specific. It is additionally convincing if it is compatible with evidence other than that upon which it was based, especially if no additional corollaries are required. The final test is if new prognostications can be drawn from it which when tested are found to correlate with both existing and future research results.

How do the existing theories fit this definition?

Occlusal disharmony precipitates TMD

In the past, many authorities believed (Dawson, 1974) that irregular contacts of the teeth, sliding contacts, crossbites, deviations of the jaw, opposite side contacts, etc., throw unfair strains on the joint. As Sicher (1949) explained, if an individual cusp clashes during mastication, the geometry of the joint inevitably places its two sliding surfaces under torsional stress and damage may result. Treatment on the basis of this theory has continued for many years, involving the equilibration of the occlusion, a procedure which has ranged in complexity from local grinding of the teeth, to detailed adjustments using articulators which record and replicate the movements of the jaw (Dawson, 1974). While satisfactory results are claimed, a ratio of cases fail to respond. The flaw in this theory seems to be that it fails to provide a clear reason for the development of the occlusal disharmony in the first place. Also, the presence or absence of interferences appears to have little relationship to the incidence of TMD (Magnusson and Enborn, 1984), even non-working side contacts, and canine rises appear

unrelated. Finally, regardless of whether or not the TMD responds to occlusal adjustment, the irregular contacts often return, necessitating further equilibration. For these reasons, this must be considered an unlikely theory.

Certain malocclusions precipitate TMD

While covered in part under the last heading, it has also been suggested (Riolo *et al.*, 1987) that certain specific malocclusions, such as lateral open bites, increased overjets, etc., tend to damage the joint. It is the opinion of some clinicians (Witzig and Spahl, 1991) that dental interferences drive the mandible distally and cause damage to the posterior attachment of the disc. However, Stringert and Worms (1986) noted that patients with Angle's Class II division 2 malocclusions (who presumably have their mandibles driven back more than most) have less TMD than average, although this viewpoint is disputed by others. Schellhas *et al.* (1993) noted that internal derangement was more common in Class II malocclusions (56 out of 60), and reversed all the established hypotheses by suggesting that 'internal derangement of the TMJ(s) disc(s) either retards or arrests condylar growth'. In other words, TMD causes the malocclusion. However, they do not appear to have used controls to establish the global incidence of TMD in patients with Class I malocclusion or the proportion of patients suffering TMD who do or do not have Class II occlusions.

Mohlin *et al.*'s (1991) findings suggest that most TMD is associated with crossbites (30 per cent) and increased overjets (20 per cent), while the least is found with deep bites (5 per cent) and prenormal bites (2 per cent). To some extent, these ratios reflect the distribution of these malocclusions themselves, in which case, with the possible exception of crossbites, the type of malocclusion cannot have a great influence. Of more significance, many patients have severe malocclusion of all classifications without any TMD symptoms, making it unlikely that malocclusion itself is the causal factor.

Malposition or malformation of the condyle causes TMD

Some clinicians (Solberg, 1986) consider this an important aspect of TMD and techniques of magnetic resonance imaging (MRI) now enable it to be assessed directly (Dixon, 1994). However, it is still not known whether the variations in shape are the cause or the result of the problem. Surgery to the joint is sometimes recommended, but has its critics (Moffett, 1980).

The subject aroused my interest in 1954 when 23 patients who had previously had a single condyle removed for TMD reasons between 3 and 5 years previously were recalled (unpublished data). Of special interest was the observation that three of the patients whose condyle and disc had been completely removed had subsequently developed a replacement condyle. One of these condyles looked almost normal with a joint space suggesting that the capsule had also regenerated. This material was never published, but similar observations were made by Lund (1974) in his classic work on the repair of condylar fractures. In a study of 38 young patients, he found that the displaced head was initially removed by resorption (Figure 1). Following this, there appeared an 'outgrowth of a bony process on the ramus completely resembling a normal condyle. ... The remodelling consisted of a combination of appositional and resorptive processes.' Aldef (1981) reported similar findings in adults. It would seem that the condyle has unique powers of regeneration. This evidence is hardly compatible with the theory that TMD problems are caused by the malformation or malpositioning of the condyle itself. Why, if it can regenerate entirely, should it be unable to adapt a few millimetres?

Abnormal form or position of the glenoid fossa will precipitate TMD

It has been suggested that the fossa itself may be at fault and certainly there is evidence to suggest that the temporal bone, together with the glenoid fossa, may be displaced during orthodontic treatment (Agronin and Kokich, 1987). Joints in general display remarkable adaptability; for instance, if the neck of the femur is dislocated in infancy and left uncorrected, it slides several centimetres up the innonimate bone where an entirely new socket may be formed around it (Figure 2). In other words, the head of the femur appears to have the ability to 'instruct' a

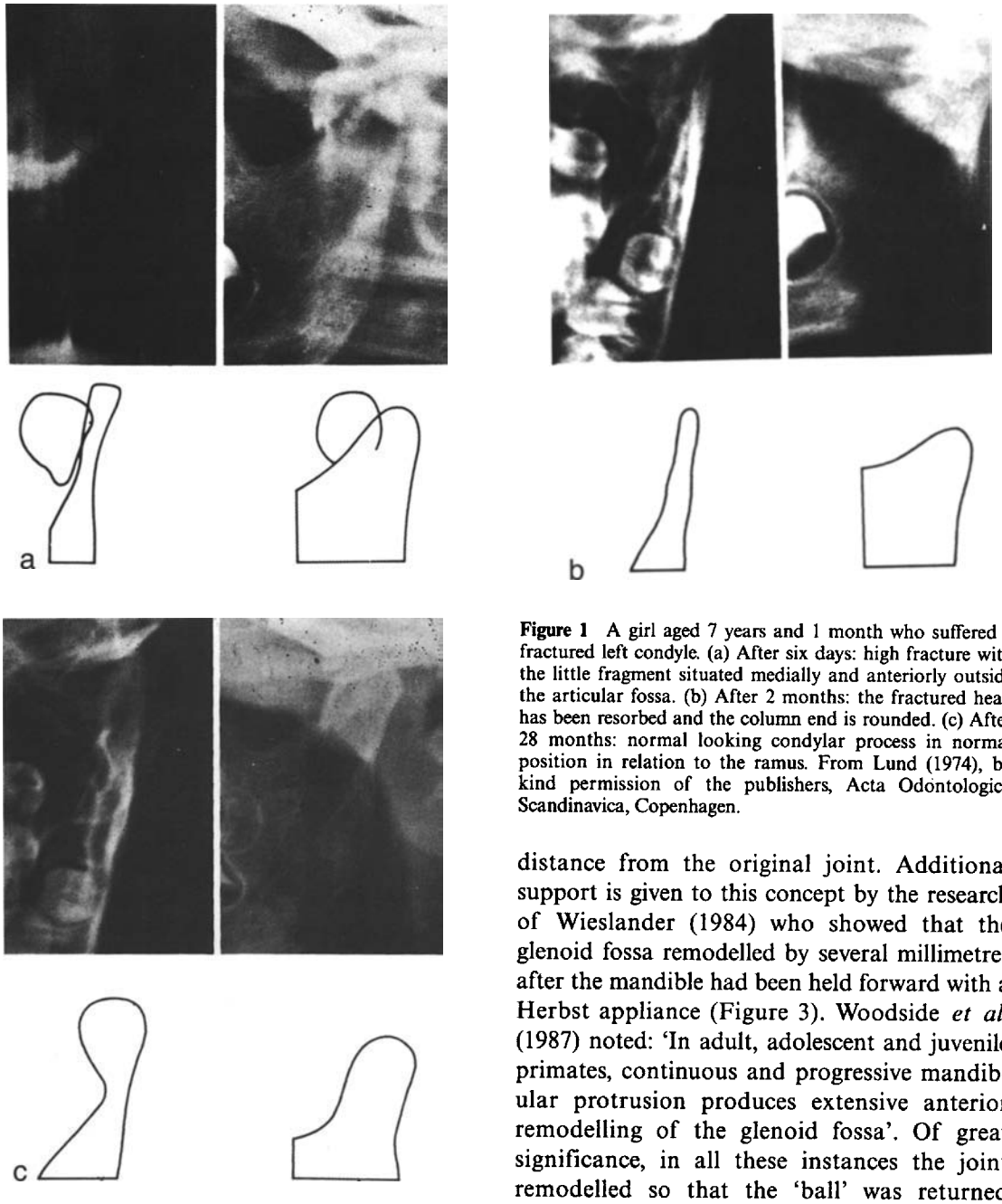

Figure 1 A girl aged 7 years and 1 month who suffered a fractured left condyle. (a) After six days: high fracture with the little fragment situated medially and anteriorly outside the articular fossa. (b) After 2 months: the fractured head has been resorbed and the column end is rounded. (c) After 28 months: normal looking condylar process in normal position in relation to the ramus. From Lund (1974), by kind permission of the publishers, Acta Odontologica Scandinavica, Copenhagen.

completely different area of bone to form a substitute joint.

Similarly Moffett (1979) reported a case of a 70-year-old man with a long-term unresolved dislocation of his jaw. A new joint had formed, again around the head of the condyle, some distance from the original joint. Additional support is given to this concept by the research of Wieslander (1984) who showed that the glenoid fossa remodelled by several millimetres after the mandible had been held forward with a Herbst appliance (Figure 3). Woodside *et al.* (1987) noted: 'In adult, adolescent and juvenile primates, continuous and progressive mandibular protrusion produces extensive anterior remodelling of the glenoid fossa'. Of great significance, in all these instances the joint remodelled so that the 'ball' was returned towards the centre of the 'socket' (a feature which will be referred to in the Discussion). Because the joint seems so able to adapt in both adult animals and man (Woodside *et al.*, 1987), it seems quite illogical to suggest that these deformations are the cause of TMD as they clearly follow displacement of the joint. They

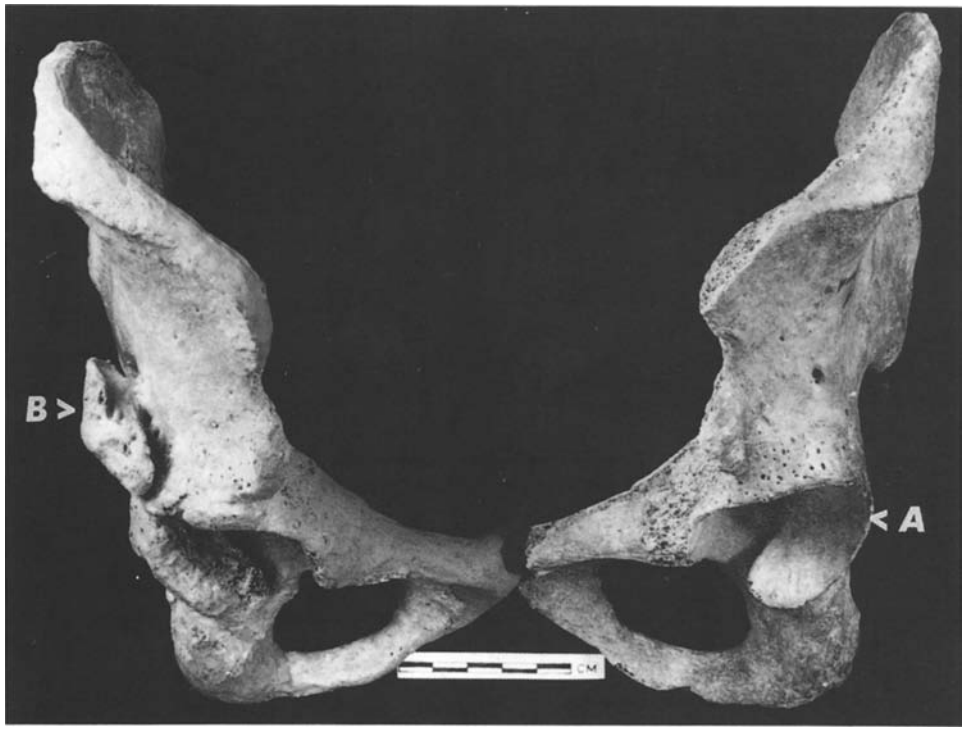

Figure 2 Pelvis of an Eskimo who had an untreated congenital dislocation of the hip (B). Note that the new socket has formed about 10 cm higher than the normal hip (A). Kindly loaned and photographed by the Smithsonian Museum, Washington, DC.

should therefore be classified as 'resultant' not 'precipitating'.

TMD is the result of previous trauma

Some clinicians and osteopaths (McCarty, 1980) consider that trauma (pre- or post-natal) may displace the cranial bones or indeed cause direct damage to the jaw or joint. Wilkes (1989), in a retrospective study of 540 patients, suggested that trauma was the single most frequent cause of subsequent TMD. However, nearly all children suffer a blow to the face at some time, but there do not seem to have been any studies to establish the global incidence of such trauma. This explanation is also at odds with the low incidence of TMD in primitive populations who are equally, if not more, exposed to damage. Nor do other joints seem so prone to trauma. Follow-up of patients who have had fractured condyles, which must involve substantial trauma to the joint, has shown that they subsequently have few objective symptoms (Dahlstrom *et al.*, 1989). Despite its popularity, the contrary evidence is so powerful that this hypothesis must be considered suspect.

Orthodontic treatment damages the joint

Whilst it has been suggested that patients who have received orthodontic treatment have a higher ratio of TMD problems than average, it should be remembered that they are also likely to have a higher ratio of dental and skeletal problems before treatment (Dibbets and van der Weele, 1992). In fact, the research suggests (Sadowsky and Polson, 1984) that orthodontic treatment causes 'no significant increase'. This opinion has been supported by a number of assessments by orthodontic departments in the USA, but as Behrents (1992) questions: 'Can an institution investigate itself?' In any event, many patients with TMD have never received orthodontic treatment.

Bruxism causes TMD

There is widespread agreement that intermittent clenching or grinding can inflict heavy loads on

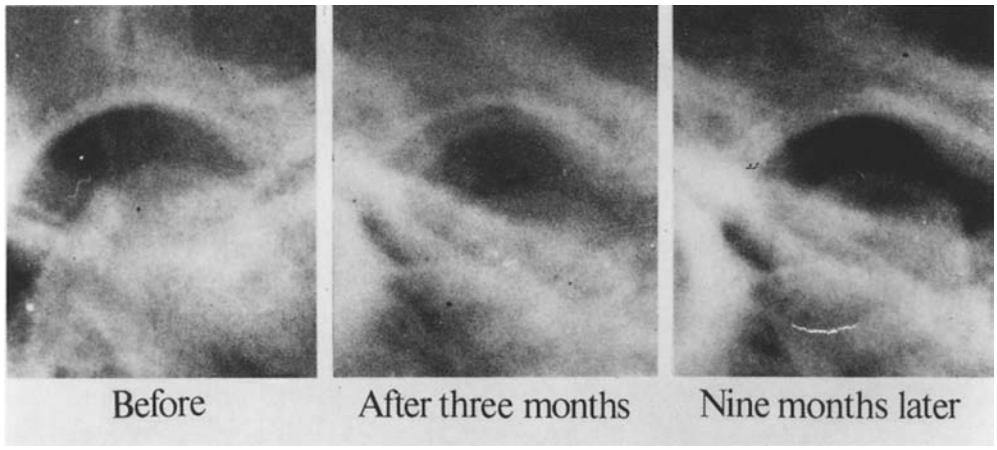

Figure 3 The articular fossa of a patient who had worn a headgear-Herbst appliance which held the mandible forward. Note the double contour of compact bone outlining the position of the joint surface before and after treatment. Kindly loaned by L. Wieslander, Basel University.

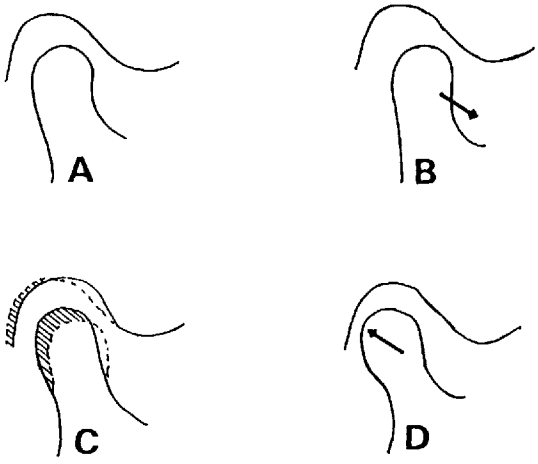

Figure 4 Artist's impression of: (A) a healthy TMJ; (B) the effect of dropping the jaw. (C) After some months in this position, the fossa and condyle adapt to re-centralize the joint. (D) The result of bringing the teeth together at that time.

the teeth and joints. Clearly, a previously damaged joint would be more likely to give trouble under these circumstances. However, many people brux and yet have no TMD problems, and so it would seem that additional factors such as irregular contacts, or previous damage, need to be present before symptoms will appear. Marks (1980) has shown that there is a close link between bruxism and allergies, and suggested that the correction of the latter might improve or even eliminate the former. However, it could be argued that the reverse is true or even that allergies tend to cause nasal obstruction, and therefore cause open-mouth postures with which both factors might be 'associated'.

The influence of diet on TMD

It seems possible that food, either by its consistency or content, could have an influence on the joint. A hard diet seems to be no disadvantage (Helkimo, 1974), but this is a relatively poorly researched area.

Stress precipitates TMD

It is recognized that emotional stress can precipitate episodes of TMD (Wadhwa *et al.*, 1993). This could be considered under the previous heading of bruxing as such patients often clench and grind their teeth. Psychological counselling has proved helpful, but cure rates are again constrained to around the 75 per cent mark. It is claimed that there are links between TMD symptoms, muscle tension, trigger points, headaches, and possibly migraine (Higson, 1985), although the last mentioned is doubted by some (Watts and Juniper, 1986). There are also characteristic placebo effects and patients in non-treatment control groups frequently report cures. In one rather bizarre instance, no less than 25 per cent of the untreated control group of symptom-free patients developed signs of TMD (Magnusson and Enborn, 1984). It seems

possible that any clinician with confidence and personality can provide minimal treatment, and still achieve a creditable 'cure' rate. Sedative medication is recommended by some (Gomersall and Stuart, 1973), but it is difficult to be sure whether any improvement is due to a reduced incidence of TMD, or a lessened awareness of the pain. Either way, long-term medication must be considered a dubious 'cure'. O'Geary (1993) expressed the opinion that 'stress by itself does not cause TMD, but will often initiate it'.

TMD results from muscle parafunction

Electromyographic studies show that TMD patients often have abnormal patterns of activity (Moss, 1975). However, this may be the result of patients attempting to avoid premature contacts, rather than the cause. Over 80 years ago, Angle (1907) suggested that the tongue and lips influence the developing occlusion, and Rix (1946) first drew attention to the tooth apart swallow. During a parafunctional swallow, the oral seal is achieved by contracting the obicularis and buccinator muscles against the tongue, and this results in obvious hypertrophy of the relevant muscles Depending on the severity of this habit, the teeth may be sucked lingually, often causing the crossbites associated with TMD (Mohlin *et al.*, 1991). Possibly for these reasons, physiotherapy (physical therapy) (Gray *et al.*, 1994) has proved helpful, but as with other cures only at the 75 per cent level.

TMD is affected by oral posture

It is difficult to measure the resting position of the jaws and lips, and almost impossible to measure that of the tongue. As a result, oral myology has tended to be a neglected area of research. There is much uncertainty about 'normal resting postures' or whether variations from normal have an influence on the joint or indeed other dental and skeletal structures. If 'rest' is synonymous with 'minimal muscular activity', then Rugh and Drago's work (1981) would suggest that the mouth should be open 8.6 mm, which seems unlikely. People who keep their mouths closed in company may not do so while alone or asleep, and often imagine their lips are together more than they are. Others may close their lips, but leave their teeth several millimetres apart.

Costen (1934) put forward the opinion that over-closed bites resulted in retro-positioning of the head of the condyle and were associated with TMD, together with a range of other problems.. However, it has been shown (Peterson *et al.*, 1983) that patients with over-closed bites usually posture with their teeth apart and we know that TMD patients tend to have over-closed bites and tooth-apart resting postures (Williamson *et al.*, 1990). Many TMD clinicians and some specialists in occlusion consider that a 'freeway space' of several millimetres is 'normal', and recognize 'tongue splinting' as a natural means of resting the joint. In this context, the word 'normal' can be misleading, as there is no evidence to suggest that an increased freeway space is normal, other than in the industrialized parts of the world where, perhaps not incidentally, malocclusion and TMD are endemic.

There is indeed much evidence to suggest that closed mouth postures are beneficial and it is certainly normal for neonates. Melsen *et al.* (1987) found that children who swallow with their teeth together have less malocclusion. Lundeen and Gibbs (1982) found that subjects who keep their teeth in contact have good occlusion. More recently, Linder-Aronson *et al.* (1993) have shown that patients who kept their mouths closed have increased forward growth and the naso-pharynx lengthened by the substantial amount of 10 mm more than open-mouth controls. Dibbets and van der Weele (1996) found that increased forward growth was associated with reduced signs of TMD. In contrast, Bresolin *et al.* (1984) found a clear relationship between open-mouth postures and the same group of crossbites and Class II malocclusions that Mohlin *et al.* (1991) subsequently associated with TMD problems.

The research of Proffit and Sellers (1986) with rabbits provided clear support for the concept that teeth should be in contact for a certain number of hours each day. Lee and Proffit (1995) also found that eruption in humans was highly sensitive to periods of contact and that teeth left out of occlusion for long periods can be expected to continue to erupt. Proffit and co-workers (Proffit

et al., 1993; Proffit and Fields, 1993) also demonstrated that long-faced adults and children exert lower chewing forces. It would seem that the occlusal height of any individual represents a balance between the period of time the teeth are in contact and the biting forces applied. From the above observations, the rational conclusion is that unless the teeth are in contact for between 4 and 8 hours each day, they will continue to erupt. Presumably they continue to do so until they reach the limit of alveolar support, or some other obstruction such as the tongue, if it is stored between the teeth. If over-eruption occurs, the patient would obviously be unable to close back to their previous relationship, thus perpetuating an increase in facial height. The infrequency of tooth contact might also be expected to result in poor occlusal balance. The corollory to this is that individuals who keep their mouths closed for a sufficient time each day will have good occlusion (Lundeen and Gibbs, 1982; Melsen *et al.*, 1987) and less TMD (Williamson *et al.*, 1990). If the teeth are left apart, the tongue tends to slide between them, instead of resting against the palate. Some of the consequences of such aberrations in posture have been discussed (Mew, 1981).

There has been a dramatic increase in nasal allergies in industrialized countries over the last few decades and normal 3- and 4-year-old children now leave their mouths open for about 85 per cent of the time (Glatz-Noll and Berg, 1991). In the same period, both malocclusion and TMD have become endemic in all advanced countries. The accumulated evidence quoted above leaves little doubt that both malocclusion and TMD are either caused by, or associated with tooth-apart postures. These might be said to include 'tongue splinting', 'tongue between tooth postures and swallows', 'increased freeway space', 'mouth-open postures', and according to Peterson *et al.* (1983) 'closed bites'. However, there does not seem to be an existing theory which might provide a logical link between TMD and this assortment of factors.

Discussion

The objective of this paper was to use accepted research findings to forge a theory which fitted all the available facts. Eleven theories which claim to explain the aetiology of TMD have been considered. The first five appear flawed in one or more respects and the evidence for the next three appears weak. It is only the last three that seem logical and on this basis two further theories might be suggested.

The first theory

Joints are highly adaptable and will always remodel to suit habitual rest positions (Figure 4A). If the mouth is held open continuously (Figure 4B), the temporomandibular joint will recontour so that the head is maintained near the centre of the fossa when in that position (Figure 4C). If following this the mouth were closed, the head of the condyle will move upwards and backwards (Figure 4D) to occupy a position frequently seen in TMD cases (Costen, 1934). Although it is commonly believed that final closure of the mandible is a purely hinge action, this is not so, as can be felt if a finger is placed on the joint.

The second theory

If the mouth is hung open, the upper incisors are likely to move lingually under the weight of the soft tissue drape (Solow, 1981). If the mouth is then closed, the anterior teeth are likely to contact slightly before the posterior, forcing the condyle back in the joint. This feature can be demonstrated in most TMD patients.

These two hypotheses suggest that the joint is placed in double jeopardy if the mouth is left open, and they also provide a specific explanation for six of the common signs of TMD: retroposed condyles, open-mouth posture, tongue between tooth posture, occlusal irregularities, lingual inclination of the teeth, and deformation of the condyle and fossa.

It would be unwise, however, to assume that any of these factors, or even a combination of them, is an actual cause of TMD. Incorrect placement of the joint alone would be unlikely to cause symptoms for which a degree of force would be required. The postural factors that have just been listed might more appropriately be considered as 'predisposing'. Of the factors originally listed, bruxism, or clenching would seem, both by the

evidence and by clinical consensus, most likely to cause the damage and should perhaps be awarded the single 'initiating' label.

These conclusions would suggest that once the initial trauma has subsided, the patient should be trained to keep their teeth in light contact for more than 4 hours out of 24. This should encourage the condyle to remodel forward to the centre of the joint.

Conclusions

1. There is little to suggest that our ancestors had any problems with their joints.
2. Mouth-open postures are common in modern environments.
3. All joints naturally adapt so the ball is near the centre of the socket in the resting position.
4. If the mandible is dropped at rest, the condyle will remodel distally to suit this position and the upper incisors will move lingually.
5. If such an individual then closes their mouth, the head of the condyle is forced distally.
6. If they also tend to clench their teeth, the joint may be damaged.

The multifactorial hypothesis for the cause of TMD appears flawed, as repeated similar failures of any particular biological structure are likely to be caused by the same 'initiating' factor, regardless of how many and how varied are the 'predisposing' factors. The evidence would suggest that TMD is 'predisposed' by open-mouth postures and tongue between tooth swallows and is 'precipitated' by intermittent clenching. There appears to be no evidence as yet against this theory, while most other theories appear flawed in one or more respects.

Address for correspondence

Mr J. R. C. Mew
Hill House
Clanricarde Road
Tunbridge Wells
Kent TN1 1PJ
UK

References

Agronin K J, Kokich V G 1987 Displacement of the glenoid fossa: A cephalometric evaluation of growth during treatment. American Journal of Orthodontics and Dentofacial Orthopedics 91: 42–48

Aldef A E 1981 Regenerated human mandibular condyle. Australian Dental Journal. 26: 216–217

Angle E H 1907 Treatment of malocclusion of the teeth. S S White Manufacturing Co., Philadelphia

Behrents R G 1992 TMJ research: Responsibility and risk. American Journal of Orthodontics and Dentofacial Orthopedics 101: 1–3

Bresolin D *et al.* 1984 Facial characteristics of children who breathe through the mouth. Pediatrics 73: 622–625

Costen J B 1934 A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporo-mandibular joint. Annals of Otology, Rhinology and Laryngology 4: 1–15

Dahlstrom, I, Kahnburg K E, Lindahl, L 1989 15 year follow-up on condylar fractures. International Journal of Oral and Maxillofacial Surgery 18: 18–23

Dawson P 1974 Evaluation, diagnosis and treatment of occlusal problems. C V Mosby Co., St Louis

Derrick D D (ed) The dental annual. Oxford University Press, Oxford, pp. 73–78

Dibbets J M H, van der Weele L Th 1992 Long-term effects of orthodontic treatment including extraction, on signs and symptoms attributed to CMD. European Journal of Orthodontics 14: 16–20

Dibbets J M H, van der Weele L Th 1996 Signs and symptoms of TMD and cranio-facial form. American Journal of Orthodontics and Dentofacial Orthopedics (in press)

Dibbets J M H, van der Weele L T, Boering, G 1985 Craniofacial morphology and temporomandibular joint dysfunction in children. In: Carlson D S, McNamara J A, Ribbens K A (eds), Developmental aspects of temporo-mandibular joint disorders. Monograph No. 16, Craniofacial Growth Series, Center for Human Growth and Development, University of Michigan, Ann Arbor

Dixon D C 1994 Indications and techniques for imaging the temporo-mandibular joint. In: Zarb G A, Carlson G E, Sessle B J, Mohl N D (eds) Temporo-mandibular joint and masticatory muscles disorders. 2nd edition. Munksgaard, Copenhagen, pp. 435–461

Egermark-Ericsson I, Carlson G E, Magnusson T 1987 A long term epidemiologic study of the relationships between occlusal factors and mandibular dysfunction in children and adolescents. Journal of Dental Research 66: 67–71

Garn S M 1961 Research and malocclusion. American Journal of Orthodontics 47: 661–673

Glatz-Noll E, Berg R 1991 Oral dysfunction in children with Down's syndrome: an evaluation of treatment effects by means of video-registration. European Journal of Orthodontics 13: 446–451

Gomersall J D, Stuart A 1973 Amitriptyline in migraine prophylaxis. Changes in patterns of attacks during a controlled clinical trial. Journal of Neurology, Neuro-surgery and Psychiatry 36: 684–690

Gray R J M, Quayle A A, Hall C A, Schofield M A 1994

Physiotherapy in the treatment of temporomandibular joint disorders: a comparative study of four treatment methods. British Dental Journal 176: 257–261

Helkimo M 1974 Studies on function and dysfunction of the masticatory system. Proceedings of the Finnish Dental Society 70: 37–49

Higson R H 1985 Treatment of migraine and associated conditions through dental treatment. In: Derrick D D (ed) The dental annual. Oxford University Press. Oxford

Lee C F, Proffit W R 1995 The daily rhythm of tooth eruption. American Journal of Orthodontics and Dentofacial Orthopedics 107: 38–47

Linder-Aronson S, Woodside D G, Hellsing E, Emerson W 1993 Normalization of incisor position after adenoidectomy. American Journal of Orthodontics and Dentofacial Orthopedics 103: 412–427

Lund K 1974 Mandibular growth and remodelling processes after condylar fracture. Acta Odontologica Scandinavica 32: Supplement 64

Lundeen H C, Gibbs C H 1982 Advances in occlusion. John Wright PSG, Boston

Magnusson T, Enborn L 1984 Signs and symptoms of mandibular dysfunction after introduction of experimental balancing-side interferences. Acta Odontologica Scandinavica 42: 129–135

Marks M B 1980 Bruxism in allergic children. American Journal of Orthodontics 77: 48–59

McCarty W 1980 Diagnosis and treatment of internal derangements of the articular disk, and mandibular condyle. In: Solberg W K, Clark G T (eds) TMJ problems. Quintessence Publishing Co. Inc., Chicago

Melsen B, Attina L, Suntueri M, Attina A 1987 Relationships between swallowing pattern, mode of respiration and developing malocclusion. Angle Orthodontist 57: 113–119

Mew J R C 1981 Tongue posture. British Journal of Orthodontics 8: 203–211

Moffett B 1979 Personal communication

Moffett B 1980 Temporo-mandibular joint problems. In: Solberg W K, Clark G T (eds) TMJ problems. Quintessence Pub. Co. Inc., Chicago, p.26

Mohlin B, Pilley J R, Shaw W C 1991 A survey of craniomandibular disorders in 1000 12-year-olds. European Journal of Orthodontics 13: 111–123

Moss J P 1975 Function-fact or fiction. American Journal of Orthodontics 67: 625–645

O'Geary S 1993 The functional TMJ. Dental Practice 31: 1–3

Peterson T J, Hugh J D, McIver J E 1983 Mandibular rest position in subjects with high and low mandibular plane angles. American Journal of Orthodontics 83: 318–320

Proffit W R, Fields H W 1993 Occlusal forces on long and normal face children. Journal of Dental Research 62: 571 (Abstract)

Proffit W R, Sellers K T 1986 The effect of intermittent forces on the rabbit incisor. Journal of Dental Research 65: 118–122

Proffit W R, Fields H W, Nixon W I 1993 Occlusal forces in normal and long-faced adults. Journal of Dental Research 62: 566

Riolo M L, Brandt D, TenHave T R 1987 Associations between occlusal characteristics and symptoms of TMJ dysfunction in children and young adults. American Journal of Orthodontics and Dentofacial Orthopedics 92: 467–477

Rix R E 1946 Deglutition and the teeth. Dental Record 66: 103–108

Rugh J D, Drago C J 1981 Vertical dimension: a study of clinical rest position and jaw muscle activity. Journal of Prosthetic Dentistry 45: 670–675

Sadowsky C, Polson A M 1984 Temporomandibular disorders and functional occlusion after orthodontic treatment. American Journal of Orthodontics 86: 386–390

Schellhas K P, Pollei S R, Wilkes C H 1993 Pediatric internal derangements of the temporomandibular joint: Effect on facial development. American Journal of Orthodontics and Dentofacial Orthopedics 104: 51–59

Schiffman E, Fricton J R 1988 Epidemiology of TMJ and craniofacial pain. In: Fricton J R, Kroening R J, Hathaway K M (eds) TMJ and craniofacial pain; Diagnosis and management. IEA Publishers, St Louis, pp. 1–10

Sicher H 1949 Oral anatomy. C V Mosby Co., St Louis

Solberg W K 1986 Temporomandibular disorders. British Dental Journal 160: 157–161; 195–200; 231–236; 273–277; 317–322; 351–356; 379–385; 421–428

Solow B 1981 The dento-alveolar compensating mechanism. British Journal of Orthodontics 7: 145–161

Stringert H G, Worms F W 1986 Variations in skeletal and dental patterns in patients with structural and functional alterations of the temporomandibular joint. American Journal of Orthodontics 89: 285–297

Wadhwa L, Uteja A, Tewari A 1993 A study of clinical signs and symptoms of temporomandibular dysfunction in subjects with normal occlusion, untreated, and treated malocclusions. American Journal of Orthodontics and Dentofacial Orthopedics 103: 54–61

Watts P G, Juniper R P 1986 Migraine and the TMJ. The final answer? British Dental Journal 161: 170–173

Wieslander L 1984 Intensive treatment of severe Class II malocclusion with a headgear-Herbst appliance in the early mixed dentition. American Journal of Orthodontics 86: 1–13

Wilkes C H 1989 Internal derangement of the temporomandibular joint: pathologic variations. Archives of Otolaryngology—Head and Neck Surgery 115: 469–477

Williamson E H, Hall J T, Zwemer J D 1990 Swallowing patterns in human subjects with and without temporomandibular dysfunction. American Journal of Orthodontics and Dentofacial Orthopedics 98: 507–511

Witzig J, Spahl T 1991 The clinical management of basic maxilofacial orthopedic appliances, Vol. 3. T S G Publishing Co., Massachusetts

Woodside D G, Metaxas A, Altuna G 1987 Influence of functional appliance therapy on glenoid fossa remodeling. American Journal of Orthodontics and Dentofacial Orthopedics 92: 181–198