

ORIGINAL ARTICLE

Correlation between centric relation–maximum intercuspation discrepancy and temporomandibular joint dysfunctionSHU SHU HE¹, XIAO DENG², PETER WAMALWA^{1,3} & SONG CHEN¹¹Departments of Orthodontics, West China College of Stomatology, Sichuan University, Chengdu, China, ²Chongqing Stomatology Hospital, Chongqing Medical University, Chongqing, China, and ³Division of Orthodontics, Department of Dentistry, Kenyatta National Hospital, Nairobi, Kenya**Abstract**

Objective. To investigate the relationship between centric relation–maximum intercuspation (CR–MI) discrepancy and temporomandibular joint dysfunction (TMD) in pre-treated orthodontic patients. **Material and methods.** The study involved an experimental group of 107 pre-treated orthodontic patients with signs and symptoms of TMD aged 18–32 years, and a control group of 70 students with no signs and symptoms of TMD aged 20–30 years. The psychological condition of subjects was evaluated using two standard questionnaires, and a clinical examination performed to assess masticatory musculature and temporomandibular joint (TMJ) function, and to establish the presence or absence of TMD. Helkimo indices, the anamnestic dysfunction index (Ai) and the clinical dysfunction index (Di), were determined. Dental casts were mounted on a semi-adjustable articulator in CR using a CR bite record taken by bilateral manipulation and verified by load testing and face bow records. Differences in condylar position between CR and MI in the three planes of space were determined using the condyle position indicator. **Results.** A positive CR–MI discrepancy, defined as a discrepancy exceeding 1 mm in the vertical or horizontal planes or 0.5 mm in the transverse plane, was found in 72.9% of the experimental and 11.4% of the control group. Comparison of the groups showed a significant difference ($\chi^2 = 22.67$, $P < 0.001$). CR–MI discrepancy was significantly correlated with Di and Ai in all subjects ($P < 0.01$). **Conclusions.** There was CR–MI discrepancy in most of the pre-treated patients with signs and symptoms of TMD. This discrepancy may be a contributory factor to the development of TMD in these patients.

Key Words: *Anamnestic dysfunction index, clinical dysfunction index, functional occlusion, occlusal disharmonies***Introduction**

Temporomandibular joint dysfunction or temporomandibular disorder (TMD) is a collective term that describes craniofacial pain problems that involve the temporomandibular joint (TMJ), masticatory muscles and associated head and neck musculoskeletal structures. This condition is characterized by fairly localized pain, limited or asymmetric mandibular movements and TMJ noises [1,2]. Other common symptoms include ear pain and stuffiness, tinnitus, dizziness, neck pain and headache. Presentation varies from mild, acute to persistent, chronic symptoms [2]. The prevalence of TMD has been reported to be between 7% and 84% in different studies [1].

The etiology of TMD is not clear, but is generally accepted to be multifactorial [1–7]. Factors that have been associated with TMD can be grossly divided into systemic, physiological and structural, and TMD may result from these factors either acting independently or interacting with each other [3]. The American Academy of Orofacial Pain groups TMD into Articular disorders (congenital or developmental, disk-derangement disorders, degenerative joint disorders, trauma, TMJ hypermobility, TMJ hypomobility, infection, neoplasia) and Masticatory muscle disorders (myofascial pain disorder, local myalgia, myositis, myospasm, myofibrotic contracture, neoplasia) [2].

The role of occlusion in the etiology of TMD has been widely debated [1,8–17]. It has been proposed

that TMD is closely associated with some types of malocclusions [8,9] and that the elimination of occlusion interferences plus achieving an occlusion system with no shift between centric relation (CR) and maximum intercuspation (MI) should be a primary goal of orthodontic treatment [10,11]. Several reviews [1,2,13–15] on this topic have, however, concluded that the current evidence suggests that occlusion is not a primary factor in the etiology of TMD. However, the evidence does not argue or conclude that occlusion or condylar position has no relevance to TMD [1,9,13–15]. The association, if any, between CR-MI discrepancy and TMD is still not very clear.

CR has been a controversial subject in dentistry for over a century, and its definition and that of other associated dental functional relationships have changed over the years, with no consensus yet having been reached [18]. The Academy of Prosthodontists defines CR as “The maxillomandibular relationship in which the condyles articulate with the thinnest avascular portion of their respective disks with the condyle in the anterior-superior position against the slopes of the articular eminence. This position is independent of teeth contact” [19]. MI is defined as “The complete intercuspation of the opposing teeth independent of condylar position” [19]. CR is a position of the condyles independent of teeth contact, while MI is a position of maxillary teeth relative to mandibular teeth and condylar position in MI is subject to tooth contact.

Roth [10], Williamson [11] and Cordray [20], among others, have argued that, while achieving functional occlusion, the mandible should seat ideally into MI during closure, without condylar deflection from CR due to occlusal interferences. Any occlusal interference that cause deflection of condyles away from CR in MI are thought to cause an imbalance between the inferior lateral pterygoid and elevator muscles, which trigger muscle hyperactivity leading to development of TMD. These authors believe that achieving CR-MI harmony after orthodontic treatment reduces the risk of development of TMD.

Studies have, however, not shown consistent results regarding the relationship between CR-MI discrepancy and TMD. Pullinger [16] and Seligman [17] showed no correlation between occlusion and TMD, and claimed that single occlusal variables can hardly be used to identify patients with TMD. They concluded that occlusal slides from retruded cuspal position (RCP) to intercuspation position can increase the odds ratio of TMD only when they exceed 5 mm. In these studies, however, diagnostic casts were not mounted on articulators, no instrumentation was used to determine CR-MI discrepancies and no precise determination of RCP was done. On the other hand, Crawford [21] compared a sample of individuals with ideal occlusion, selected from patients who had undergone full-mouth reconstruction according

to gnathologic principles, with pre-treated controls and concluded that a CR-MI discrepancy exceeding 1.0 mm in the vertical or horizontal planes and 0.5 mm in the transverse plane is likely to cause TMJ problems. Selection bias due to experimental subjects having had previous occlusal treatment may, however, have influenced these results.

The purpose of this study was, therefore, to investigate the relationship between CR-MI discrepancy and TMD by comparing an experimental group of pre-treated orthodontic patients with signs and symptoms of TMD to a control group of subjects with no such signs and symptoms. The hypotheses for this study were that there was a difference in the presence of CR-MI discrepancy between the two groups and that there was a relationship between CR-MI discrepancy and TMD.

Material and methods

The study population for this experiment comprised patients at the Department of Orthodontics, West China Hospital of Stomatology, Chengdu, China seen over a 1-year period (June 2008–June 2009) and students at Sichuan University, Chengdu, China. The study involved 177 subjects divided into an experimental and a control group.

The experimental group consisted of 107 pre-treated adult orthodontic patients. Inclusion in this group required subjects to meet the following criteria: (i) full permanent dentition with no dental prosthetic replacements and due to begin orthodontic treatment after comprehensive diagnosis and treatment planning; (ii) TMD diagnosed on the basis of the presence of signs and symptoms as described by Dawson [22] (musculature pain or tenderness, TMJ pain or tenderness, TMJ sounds, limited mouth opening, masticatory muscle fatigue, masticatory muscle spasms and mandibular deviation on mouth closing/opening); and (iii) presence of TMD confirmed by the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) [23]. This group contained 28 subjects with Angle Class I malocclusion, 61 with Angle Class II malocclusion and 18 with Angle Class III malocclusion. All new patients who consented to the experiment during the study period were interviewed and examined during selection of this group.

The control group consisted of 70 students and inclusion required subjects to meet the following criteria: (i) full permanent dentition with no dental prosthetic replacements; and (ii) no signs and symptoms of TMD as per Dawson [22] or RDC/TMD [23] criteria. During selection of this group, 900 potential subjects were initially interviewed and those with a history of TMD symptoms eliminated. Of these, 321 who reported no history of TMD

symptoms were given a questionnaire and underwent clinical examination, and 70 who met the inclusion criteria were selected to form the control group. This group contained 50 subjects with Angle Class I normal occlusion, 13 with Angle Class I malocclusion and seven with Angle Class II malocclusion. Normal occlusion was defined as having a full set of permanent teeth up to the second molars, bilateral class I molar and canine relationship, overbite and overjet of 1–3 mm and no crowding or restorations. The presence of deep bites (incisor overlap >40%), open bites (no incisor overlap) and posterior cross-bites (buccal cusps of upper posterior teeth occluding lingual to buccal cusps of lower posterior teeth) was noted in the whole sample. A summary of the demographic characteristics and distribution of malocclusions in the sample is shown in Table I. Student's *t*-test showed no significant differences in age and sex distributions between the groups.

Participation in the study was voluntary and the study was approved by the ethical board of Sichuan University. All diagnostic information obtained from the study was used in the further management of those patients who required it, at no extra cost to them.

The first step in the evaluation of each subject was assessment of the psychological status using two questionnaires: the State-Trait Anxiety Inventory (STAI) of Spielberger et al. [24] and the Depression Status Inventory (DSI) of Zung [25]. The STAI measures anxiety using 20 listed items, each with a possible score ranging from one to four, to score anxiety on a scale ranging from 20–80. Higher scores mean greater anxiety. The DSI measures depression using 20 listed items each with a possible score ranging from one to four and the sum of scores is divided by 80 to give a depression index ranging from 0.25 to 1.00. A depression index below 0.5 indicates no depression, 0.5–0.59 slight, 0.6–0.69 moderate and 0.7–1.0 severe depression.

The second step was assessment of TMJ status and function and the presence of TMD. Initial evaluation was done based on guidelines for diagnosing

TMD presented by Dawson, and further assessment of the presence of TMD was done based on the RDC/TMD [23]. Each subject completed a questionnaire to record any history of signs or symptoms of TMD, including TMJ sounds and pain, masticatory muscle pain, fatigue and spasms, mandibular movement disorders and persistent aches of the head, ear, cheek and neck. Clinical examination was done to determine maximum mouth opening, maximum left and right excursive movements, masticatory muscle tenderness on bimanual palpation, TMJ sounds during mandibular movements and mandibular path during mouth opening and closing to determine any deviations. Information gathered from history-taking and the clinical examination for the experimental group was used to determine the severity of signs and symptoms of TMD, staged according to the Helkimo indices [26]: the anamnestic dysfunction index (Ai) and the clinical dysfunction index (Di).

The third step was determination of the presence and degree of CR-MI discrepancy. Alginate impressions were taken and study casts fabricated in dental stone. The CR position was determined by the bimanual manipulation technique described by Dawson [22] and recorded using Alminax Bite registration wax (Associated Dental Products Ltd, Purton, UK). The CR determination procedure was repeated after 15 min to verify the accuracy of the record. The MI position was recorded using the same wax by asking the subject to bite into the position of maximum intercuspation. The wax-bites were stored in a sealed plastic container floating in cold water. A face-bow record of the condylar position was then taken using the Panadent articulator face bow (Panadent Corp, Grand Terrace, CA) and used to mount the maxillary cast on the Panadent semi-adjustable articulator. The mandibular cast was mounted using the CR wax-bite record. The mounted diagnostic casts were subsequently transferred to the Panadent condylar position indicator (CPI) (Figure 1). Using the MI wax-bite record, the CPI was used to measure the difference in condylar position between CR and MI

Table I. Basic demographic information and male-to-female comparison for the sample.

Variable	Group		<i>P</i>
	Experimental	Control	
<i>N</i>	107	70	
Gender; <i>n</i> (%)			0.778
Male	36 (33.6)	25 (35.7)	
Female	71 (66.4)	45 (64.3)	
Age (years); mean (SD)	24.0 (4.5)	24.4 (4.1)	0.173
Deep bite; <i>n</i> (%)	53 (49.5)	5 (7.1)	
Anterior open bite; <i>n</i> (%)	2 (1.9)	0	
Posterior open bite; <i>n</i> (%)	5 (4.7)	0	



Figure 1. The CPI used in this investigation.

in three planes of space (Figure 2). The horizontal difference was defined as x (antero-posterior displacement, A-P), the vertical difference as z (supero-inferior displacement in the sagittal plane, S-I) and the transverse difference as y (lateral displacement, L). All differences were measured and recorded to the nearest 0.1 mm using a monocular lens. A positive CR-MI discrepancy was defined as a discrepancy >1 mm in the horizontal or vertical planes and 0.5 mm in the transverse plane following the criteria established by Slavicek [27] and Crawford [21], that such discrepancies are clinically significant.

All technical procedures in the third step were done by one operator for standardization and to eliminate inter-operator error. This operator was blind to the TMD status of the subjects when he carried out these procedures. Intra-observer agreement was assessed by taking two measurements 2 weeks apart in 40 randomly selected individuals and calculated using Cohen's equation:

$$\text{kappa} = \frac{P_A - P_E}{1 - P_E}$$

where P_A is the actual intra-observer agreement rate obtained by dividing the number of consistent

observations by the total number of observations and P_E the expected intra-observer agreement rate.

Data analysis

Statistical analysis was done using SPSS version 13.0 (SPSS Inc, Chicago, IL). Descriptive statistics were determined for CR-MI discrepancy, other potential TMD causative factors, signs and symptoms of TMD and Helkimo indices. The Chi-square test was used to compare differences in CR-MI discrepancy between the two groups. The level of statistical significance was set at $\alpha = 0.05$ ($P < 0.05$). Pearson's correlation was calculated to determine the correlation between CR-MI discrepancy and Helkimo indices (A_i and D_i).

Owing to the high frequency of deep bite in the sample it was controlled as a variable in correlation tests. Partial correlation analysis was performed to assess the correlation between CR-MI discrepancy and Helkimo indices (A_i and D_i) after controlling for deep bite. Two-step linear regression was used to assess the effects of CR-MI discrepancy and deep bite on TMD.

Results

Intra-observer agreement

The intra-observer agreement for the analysis of CR-MI discrepancy (third step of the analysis) was calculated as kappa = 0.782 (standard error 0.069; 95% confidence interval 0.647–0.917). A U-test of the kappa value found no significant difference, indicating consistency of the measurements.

CR-MI discrepancy

A total of 78 individuals (72.9%) in the experimental group and eight (11.4%) in the control group had a positive CR-MI discrepancy. The Chi-square test showed a statistically significant difference in incidence of CR-MI discrepancy between the groups ($\chi^2 = 22.67$, $P < 0.001$), with the experimental group having the higher incidence. Descriptive statistics of

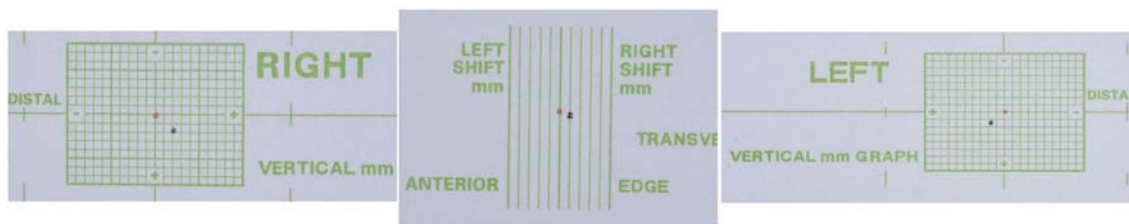


Figure 2. An illustration of the recordings made by the CPI. The red dot represents the CR position and the blue dot the MI position. The CR-MI discrepancy is measured by determining the distance between the two dots.

the CR-MI discrepancy are presented in Table II, while Table III presents the incidence of different amounts of CR-MI discrepancy in the experimental and control groups. The power of the study at the 95% confidence level was calculated as $\beta < 0.001$, $1-\beta > 0.8$, which indicated adequate power.

Psychological assessment

The mean STAI scores were 40.4 [standard deviation (SD) 5.98] and 35.2 (SD 8.64) in the experimental and control groups, respectively. The mean DSI Score was 0.37 (SD 0.06) in the experimental and 0.35 (SD 0.08) in the control group. All subjects in the control group and 97% (104 individuals) of those in the experimental group had a DSI score of <0.5 . Only 2.8% (three individuals) of those in the experimental group had a DSI score >0.5 , but they all had a DSI score <0.6 .

Other possible TMD causative factors

Arthritis, a history of external maxillofacial injury and a unilateral mastication habit were considered to be possible TMD causative factors. In the experimental group, three subjects (2.8%) had arthritis, two (1.9%) a history of maxillofacial injury, three (2.8%) unilateral mastication and two (1.9%) liked hard foods. No subject in the control group had any of these factors.

TMD signs & symptoms

Table IV presents the frequency of TMD symptoms in the experimental group. In this group, 97 subjects

(90.7%) had masticatory muscle or TMJ pain or tenderness and 93 (86.9%) had mandibular deviations on opening or closing. No subjects in the control group had signs or symptoms of TMD.

RDC/TMD assessment

In the experimental group, 57.9% (62 individuals) had group I disorders (muscle disorders), 53.3% (57 individuals) had group II disorders (disk displacements) and 26.2% (28 individuals) had group III disorders (arthralgia, osteoarthritis, osteoarthrosis). The control group contained no subjects with TMD of any type.

Helkimo's indices

Table V shows the distribution of Ai and Di scores in the experimental group. All subjects in the control group had Ai and Di scores of 0.

Correlation analysis

Pearson's correlation matrix of CR-MI discrepancy and Di and Ai and a partial correlation matrix of CR-MI discrepancy and Di and Ai after controlling for deep bite are shown in Table VI. Correlation between CR-MI discrepancy and Ai and Di was statistically significant in all directions on both the right and left sides, except the transverse direction for Ai. Correlation between CR-MI discrepancy and signs and symptoms of TMD was statistically significant. After controlling for deep bite, correlation between CR-MI

Table II. Descriptive analysis of CR-MI discrepancy in the experimental and control groups.

CR-MI discrepancy direction ^a	Group			
	Experimental		Control	
	Discrepancy frequency ^b ; n (%)	Amount of discrepancy: range (mm); mean (SD)	Discrepancy frequency; n (%)	Amount of discrepancy: range (mm); mean (SD)
X, left side: anterior	50 (62.5)	1.0–3.9; 1.9 (0.6)	1 (1.4)	1.3
X, left side: posterior	3 (3.8)	1.2–2.0; 1.5 (0.4)	0	–
Z, left side: inferior	50 (62.5)	1.0–3.8; 1.9 (0.6)	4 (5.7)	1.1–2.0; 1.5 (0.4)
Z, left side: superior	3 (3.8)	1.0–1.5; 1.2 (0.3)	0	–
X, right side: anterior	43 (53.8)	1.0–3.0; 1.8 (0.5)	3 (4.3)	1.0–1.6; 1.4 (0.2)
X, right side: posterior	10 (12.5)	1.0–1.9; 1.4 (0.3)	0	–
Z, right side: inferior	60 (75.0)	1.0–4.0; 1.9 (0.7)	0	–
Z, right side: superior	3 (3.8)	1.0–1.6; 1.3 (0.3)	0	–
Y: left shift	15 (18.8)	0.5–1.0; 0.7 (0.2)	0	–
Y: right shift	10 (12.5)	0.6–1.0; 0.8 (0.1)	0	–

^aDiscrepancies ≥ 1 mm in the horizontal or vertical planes and ≥ 0.5 mm in the transverse plane were considered positive CR-MI discrepancies.

^bThe discrepancy frequency refers to the number of subjects with significant CR-MI discrepancies.

Table III. Amount of CR-MI discrepancy in the experimental and control groups.

Amount of CR-MI discrepancy (mm) ^a	Number of subjects					
	Experimental group			Control group		
	A-P	S-I	T	A-P	S-I	T
0	0	3	69	0	5	56
0.1–0.4	15	1	13	32	42	14
0.5–0.9	24	32	22	34	19	0
1.0–1.4	14	17	3	3	2	0
1.5–1.9	23	19	0	1	2	0
2.0–2.4	16	14	0	0	0	0
2.5–2.9	9	9	0	0	0	0
3.0–3.4	4	9	0	0	0	0
3.5–3.9	2	2	0	0	0	0
4.0–4.5	0	1	0	0	0	0

^aOnly the side with a higher discrepancy between right and left was used to score each subject. A-P = antero-posterior CR-MI discrepancy; S-I = supero-inferior CR-MI discrepancy; T = transverse CR-MI discrepancy.

discrepancy and Di was still significant in all directions, but there was no significant correlation between CR-MI discrepancy and Ai.

Linear regression

Results of two-step linear regression to assess the effects of CR-MI discrepancy and deep bite on TMD are shown in Table VII. In the first step, CR-MI discrepancy values in all directions were introduced. CR-MI discrepancy in three directions entered into the model [$F(3, 173) = 65.06$; $P < 0.001$] accounted for 55.2% of variance in TMD

($R^2 = 0.552$). In the second step, CR-MI discrepancy and deep-bite values were introduced. CR-MI discrepancy in three directions and deep bite entered in the model [$F(4, 152) = 56.24$, $P < 0.001$] accounted for 58.6% of variance in TMD ($R^2 = 0.586$). Introducing deep bite resulted in 3.4% (change in $R^2 = 0.034$) more variance in TMD.

Discussion

It is well known that TMD is a multifactorial disease comprising a number of dysfunctions that can affect the TMJ complex and its neighboring structures in the head and neck. Known etiologic factors for TMD include parafunctional habits, psychological state and anxiety, systemic diseases and occlusion. Although

Table IV. Frequency of TMD-associated and other relevant symptoms.

TMD symptoms	Experimental group; n (%)	Control group; n (%)
Clicking	69 (64.5)	0
Masticatory muscle pain	38 (35.5)	0
Bruxism	32 (29.9)	0
TMJ pain	27 (25.2)	0
Masticatory muscle fatigue	2 (1.9)	0
Tinnitus	3 (2.8)	0
Headache	3 (2.8)	0
Neck pain	3 (2.8)	0
Arthritis	3 (2.8)	0
Sinusitis	3 (2.8)	0
Neurasthenia	2 (1.9)	0
Limited opening	2 (1.9)	0
Cheek pain	0	0

Table V. Frequency distribution of Helkimo indices scores.

Helkimo index level	Experimental group		Control group	
	n (%)	Mean score	n (%)	Mean score
DI				
0	0	0	70 (100)	
1	17 (15.9)	2.3	0	0
2	62 (57.9)	6.4	0	0
3	28 (26.2)	17.5	0	0
AI				
0	15 (14.0)	–	70 (100)	–
1	55 (51.4)	–	0	–
2	37 (35.6)	–	0	–

Table VI. Pearson's correlation matrix of CR-MI discrepancy and DI, AI. Values in parentheses represent partial correlation coefficients after controlling for deep bite.

	1. Left side condylar A-P def	2. Left side condylar S-I def	3. Right side condylar A-P def	4. Right side condylar S-I def	5. Transverse deflection	6. DI	7. AI
6	0.64** (0.55**)	0.62** (0.58**)	0.55** (0.49**)	0.68** (0.63**)	0.39** (0.43**)	–	
7	0.32** (0.02)	0.21* (0.02)	0.30** (0.11)	0.33** (0.09)	0.15 (0.20)	0.47**	–

* $P < 0.05$; ** $P < 0.01$; A-P def = anterior-posterior deflection.; S-I def = supero-inferior deflection.

previous large-sample epidemiological studies have shown that the type of malocclusion has no or only a weak correlation with TMD [28–30], some types of malocclusions, such as posterior cross bite, open bite and deep bite, have been reported to be important in the occurrence of TMD [8,9]. The role of CR-MI discrepancy in the etiology of TMD remains controversial however and whether this discrepancy is one of the causes of TMD is unclear.

Studies have reported few and inconsistent associations between malocclusion, functional occlusion factors and TMD [12,13,15–17,28,31]. It is important to note that no articulation of diagnostic study models or precise determination of CR position was done in most of these studies, since none of them focused on the relationship between CR-MI discrepancy and TMD.

In the present study the experimental group had a statistically significantly higher incidence of CR-MI discrepancy than the control group. During correlation analysis, deep bite was controlled as a variable since it has been linked with TMD development [8] and its incidence differed between the two groups. Other factors that have been associated with TMD were not included because their incidence was very low in both groups. We found a positive correlation between CR-MI discrepancy and TMD before and after controlling for the deep-bite effect. Two-step linear regression to assess the effects of CR-MI discrepancy and deep bite on TMD found the

effect of deep bite to be much less than that of CR-MI discrepancy. A high correlation between signs and symptoms of TMD and CPI values has also been reported by Crawford [21], which concurs with our findings. It may be argued that CR-MI discrepancy is an important factor in the development of TMD.

The most popular method of evaluating TMD in epidemiological studies involves using Helkimo's indices Ai and Di. In this study we used Ai and Di to quantitatively evaluate signs and symptoms of TMD, and found strong positive correlations between Ai and Di and the degree of CR-MI discrepancy. This implies that CR-MI discrepancy may be a causative factor of TMD, and also that the greater the CR-MI discrepancy, the more serious the clinical signs and symptoms of TMD.

Mechanisms by which CR-MI discrepancies could lead to development of TMD have been proposed by Dawson [22] and Roth [10]. They purport that when CR interferences are present during jaw closure the inferior lateral pterygoid muscle, which stays passive when CR-MI is in harmony, contracts non-physiologically to pull the condyle out of CR to achieve MI. The elevator muscles are, therefore, thought to be hyper-activated and the balance between the elevator and depressor muscles is broken, leading to masticatory muscle spasms and pain. It is claimed that if the occlusal interferences are not removed, chronic hyperactivity of the muscles will lead to articular disk derangement and forward displacement, which causes TMJ clicking, and further progression will result in intracapsular disorders, osteoarthritis and even condylar resorption. Occlusion is considered an integral part of the whole masticatory system and discrepancies between occlusion and other parts of the masticatory system, such as muscles, will lead to the development of TMD. It is important, however, to note that these proposed mechanisms are speculative and have not been proved by scientific studies.

Some studies have shown that psychosocial factors play an important role in the occurrence and development of TMD [4–7]. In this study we assessed the psychological states of all subjects and only three had significant psychosocial issues. Hence, psychosocial factors could not have been the major causative factor of TMD in the study participants. Furthermore, in

Table VII. Linear regression of CR-MI discrepancy and deep bite on TMD.

		β	t	P	R^2
Step 1	Z	0.68	11.64	<0.001	0.463
	X	0.48	7.33	<0.001	0.537
	Y	0.14	2.48	0.014	0.552
Step 2	Z	0.42	6.49	<0.001	0.463
	X	0.25	3.75	<0.001	0.537
	Deep bite	0.21	3.67	<0.001	0.563
	Y	0.17	3.06	0.005	0.586

Z = supero-inferior CR-MI discrepancy; X = antero-posterior CR-MI discrepancy; Y = transverse CR-MI discrepancy.

our study, very few subjects had arthritis, a history of maxillofacial injury, a unilateral mastication habit or a liking for hard foods and these factors should not have had a significant impact on the findings.

Some clinicians [32–35] have found stabilization splints to be effective in alleviating TMD symptoms in patients. Although several mechanisms of action have been proposed to explain their efficacy, including relaxing the muscles and seating the condyles in CR [22,36–38], which would suggest eliminating CR-MI discrepancies beneficial in alleviating symptoms of TMD, many other types of splint have been shown to be effective in the management of TMD, which discredits this argument.

From our findings we suggest that, in clinical practice, it is prudent for orthodontists to check whether definite CR-MI discrepancies exist in late-teenage and adult patients, and consider achieving CR-MI harmony to be one of the goals of orthodontic treatment [20]. By doing this the health of the masticatory system and long-term stability may be safeguarded.

Conclusions

We found a significant correlation between CR-MI discrepancy and signs and symptoms of TMD. The degree of CR-MI discrepancy had a strong positive correlation with the severity of signs and symptoms of TMD, and may be a reliable indicator of the presence and severity of TMD. Further studies with larger sample sizes and more random sampling methods are required before more concrete conclusions can be made regarding this relationship.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

- [1] Luther F. TMD and occlusion part I. Damned if we do? Occlusion: the interface of dentistry and orthodontics. *Br Dent J* 2007;202:38–9.
- [2] Scrivani SJ, Keith DA, Kaban LB. Temporomandibular disorders. *N Engl J Med* 2008;359:2693–705.
- [3] Mohlin BO, Derweduwen K, Pilley R, Kingdon A, Shaw WC, Kenealy P. Malocclusion and temporomandibular disorder: a comparison of adolescents with moderate to severe dysfunction with those without signs and symptoms of temporomandibular disorder and their further development to 30 years of age. *Angle Orthod* 2004;74:319–27.
- [4] McNeill C. Management of temporomandibular disorders: concepts and controversies. *J Prosthet Dent* 1997;77:510–22.
- [5] Manfredini D, Landi N, Bandettini Di Poggio A, Dell’Osso L, Bosco M. A critical review on the importance of psychological factors in temporomandibular disorders. *Minerva Stomatol* 2003;52:321–30.
- [6] Pankhurst CL. Controversies in the aetiology of temporomandibular disorders. Part 1. Temporomandibular disorders: all in the mind? *Prim Dent Care* 1997;4:25–30.
- [7] Parker MW. A dynamic model of etiology in temporomandibular disorders. *J Am Dent Assoc* 1990;120:283–90.
- [8] Tanne K, Tanaka E, Sakuda M. Association between malocclusion and temporomandibular disorders in orthodontic patients before treatment. *J Orofac Pain* 1993;7:156–62.
- [9] Andrade Ada S, Gameiro GH, Derossi M, Gaviao MB. Posterior crossbite and functional changes. A systematic review. *Angle Orthod* 2009;79:380–6.
- [10] Roth RH. Functional occlusion for the orthodontist. *J Clin Orthod* 1981;15:32–51.
- [11] Williamson EH. Occlusion: Understanding or misunderstanding. *Angle Orthod* 1976;46:86–93.
- [12] Gesch D, Bernhardt O, Mack F, John U, Kocher T, Alte D. Association of malocclusion and functional occlusion with subjective symptoms of TMD in adults: results of the Study of Health in Pomerania (SHIP). *Angle Orthod* 2005;75:183–90.
- [13] Mohlin B, Axelsson S, Paulin G, Pietila T, Bondemark L, Brattstrom V, et al. TMD in relation to malocclusion and orthodontic treatment. *Angle Orthod* 2007;77:542–8.
- [14] Rinchuse DJ, Kandasamy S. Evidence-based versus experience-based views on occlusion and TMD. *Am J Orthod Dentofacial Orthop* 2005;127:249–54.
- [15] Gesch D, Bernhardt O, Kirbschus A. Association of malocclusion and functional occlusion with temporomandibular disorders (TMD) in adults: a systematic review of population-based studies. *Quintessence Int* 2004;35:211–21.
- [16] Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. *J Prosthet Dent* 2000;83:66–75.
- [17] Seligman DA, Pullinger AG. The role of functional occlusal relationships in temporomandibular disorders: a review. *J Craniomandib Disord* 1991;5:265–79.
- [18] Rinchuse DJ, Kandasamy S. Centric relation: A historical and contemporary orthodontic perspective. *J Am Dent Assoc* 2006;137:494–501.
- [19] The glossary of prosthodontic terms. *J Prosthet Dent* 2005;94:10–92.
- [20] Cordray FE. Centric relation treatment and articulator mountings in orthodontics. *Angle Orthod* 1996;66:153–8.
- [21] Crawford SD. Condylar axis position, as determined by the occlusion and measured by the CPI instrument, and signs and symptoms of temporomandibular dysfunction. *Angle Orthod* 1999;69:103–15; discussion 15–16.
- [22] Dawson PE. Functional occlusion: from TMJ to smile design. St. Louis, MO: Mosby; 2006.
- [23] Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord* 1992;6:301–55.
- [24] Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, Jacobs GA. Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press; 1983.
- [25] Zung WK. Depression status inventory and self-rating depression scale. In: Guy W, editor. ECDEU Assessment Manual for Psychopharmacology—Revised. Rockville, MD: U. S. Department of Health Education and Welfare Public Health Service Press; 1976. p. 172–8.
- [26] Helkimo M. Studies on function and dysfunction of the masticatory system. II. Index for anamnestic and clinical dysfunction and occlusal state. *Sven Tandlak Tidsskr* 1974;67:101–21.
- [27] Slavicek R. Clinical and instrumental functional analysis and treatment planning. Part 4. Instrumental analysis of

- mandibular casts using the mandibular position indicator. *J Clin Orthod* 1988;22:566–75.
- [28] Gesch D, Bernhardt O, Kocher T, John U, Hensel E, Alte D. Association of malocclusion and functional occlusion with signs of temporomandibular disorders in adults: results of the population-based study of health in Pomerania. *Angle Orthod* 2004;74:512–20.
- [29] Dworkin SF, Huggins KH, LeResche L, Von Korff M, Howard J, Truelove E, et al. Epidemiology of signs and symptoms in temporomandibular disorders: clinical signs in cases and controls. *J Am Dent Assoc* 1990;120:273–81.
- [30] Szentpetery A, Fazekas A, Mari A. An epidemiologic study of mandibular dysfunction dependence on different variables. *Community Dent Oral Epidemiol* 1987;15:164–8.
- [31] Gazit E, Lieberman MA. The intercuspatal surface contact area registration: an additional tool for evaluation of normal occlusion. *Angle Orthod* 1973;43:96–106.
- [32] Kurita H, Ikeda K, Kurashina K. Evaluation of the effect of a stabilization splint on occlusal force in patients with masticatory muscle disorders. *J Oral Rehabil* 2000;27:79–82.
- [33] Tsuga K, Akagawa Y, Sakaguchi R, Tsuru H. A short-term evaluation of the effectiveness of stabilization-type occlusal splint therapy for specific symptoms of temporomandibular joint dysfunction syndrome. *J Prosthet Dent* 1989;61:610–3.
- [34] Davies SJ, Gray RJ. The pattern of splint usage in the management of two common temporomandibular disorders. Part III: Long-term follow-up in an assessment of splint therapy in the management of disc displacement with reduction and pain dysfunction syndrome. *Br Dent J* 1997;183:279–83.
- [35] Carraro JJ, Caffesse RG. Effect of occlusal splints on TMJ symptomatology. *J Prosthet Dent* 1978;40:563–6.
- [36] Dylina TJ. A common-sense approach to splint therapy. *J Prosthet Dent* 2001;86:539–45.
- [37] Gray RJ, Davies SJ. Occlusal splints and temporomandibular disorders: why, when, how? *Dent Update* 2001;28:194–9.
- [38] Al-Ani Z, Gray RJ, Davies SJ, Sloan P, Glennly AM. Stabilization splint therapy for the treatment of temporomandibular myofascial pain: a systematic review. *J Dent Educ* 2005;69:1242–50.