Commentary

Next steps in development of the diagnostic criteria for temporomandibular disorders (DC/TMD): Recommendations from the International RDC/TMD Consortium Network workshop

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Introduction

The development of the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) (1) involved expanding the taxonomy for all TMDs (2) in order to propose for future validation DC for empirically supported TMDs that were not part of the DC/TMD structure. This expanded taxonomy offers an integrated approach to clinical diagnosis and provides a framework for operationalising and testing the proposed taxonomy and diagnostic criteria in future research.

During expansion of the taxonomy, researchers identified several challenges in the diagnosis of some disorders, so the International RDC/TMD Consortium Network planned a workshop to discuss criterion improvements for five of the disorders and the biobehavioural domain. The priority areas for future advancements were identified as follows:

Arthritis

The diagnostic criteria for arthritis, a disorder that supposedly differs from degenerative joint disease, are not readily operationalised due to multiple clinical presentations. The pathophysiology of the disorder is not specific enough to clearly determine whether arthritis belongs within the joint pain diagnostic group or within the joint disease group.

Disc–condyle complex disorders

Condylar dislocation and hypo- and hypermobility of the jaw are disorders with associated changes in jaw mobility; involvement of the TMJ disc is assumed. In addition, fibrous and pressure gradient changes in the TMJ presumably have implications for disc morphology and positioning. These problems are clinically challenging because at present, we do not know whether they represent unidentified disorders that require specific interventions, or if they can be managed in the same manner that most disc displacements are currently managed.

Myofascial pain

It is not yet known whether myofascial pain can be considered a singular disorder, or whether clinically important subtypes exist. Thus, research is needed for determining whether subtypes exist, and if they do, their mechanisms and the clinical implications of defining these subtypes.

Oral movement disorders

Further progress in oral movement disorders requires better diagnostic tools and diagnostic criteria. How sleep bruxism, as a disorder, fits within the movement disorders category remains an important question.

Headache attributed to TMD

The International Classification of Headache Disorders, 2nd edition (3, ICHD-2) has been superseded by a 3rd edition, the ICHD-3 (4), which includes diagnostic criteria for Headache attributed to TMD (HA-TMD) with unknown criterion validity. The current DC/TMD diagnostic criteria for HA-TMD have excellent criterion validity. We compared both versions and recommend that the current validated DC/TMD version replace the current ICHD-3 version. Validity testing of the current and modified ICHD-3 HA-TMD versions need to establish that they have acceptable criterion validity to be credibly used in clinical or research settings. Finally, the relationship between the different primary headaches and HA-TMD, a secondary headache, needs to be explored and established. Currently, HA-TMD and tension-type headaches appear to share many clinical features.

Biobehavioural domain

The DC/TMD has identified specific instruments in widespread use for standard measurement of the core constructs deemed necessary for initial screening; however, several challenges exist in the use of those instruments. A common challenge is that various settings may prefer using other instruments that assess the same construct. To address local needs, equivalent scaling across locally selected and standard DC/TMD instruments could be created to improve generalisability of findings and would be a new goal and research opportunity. Thus, scaling methods for additional instrument development as well as using the Patient Reported Outcomes Measurement Information System (PROMIS) set of instruments are other opportunities for further development.

The purpose of this study was to present the workgroup discussions for these priority areas, and to suggest recommendations for improving the diagnostic criteria and research strategies in the next DC/TMD development phase.

Methods

The International RDC/TMD Consortium Network held its workshop on 24 June 2014 in conjunction with the annual general session of the IADR at Cape Town, South Africa. The Consortium organised six workgroups and assigned, according to their specific interests, a group leader and participants to each group. All were Consortium members in attendance at the symposium. The workshop comprised sessions for (i) general overview, (ii) group discussions and (iii) workgroup recommendations. During the general overview, the six group leaders held 30-min presentations, focusing on the shortcomings of the taxonomies and agendas for group discussions. The individual group discussions allowed face-to-face discussion of the agenda topics. Each group leader then presented the group recommendations and nominated research topics for development within the Consortium Network.

The following summarises the discussions and recommendations of each group.

Arthritis

Currently, no valid and reliable diagnostic criteria have been established for the clinical diagnosis of TMJ arthritis, defined as inflammation in articular tissues (2).

Inflammation of articular tissues may cause pain, tissue destruction and, in adolescents, mandibular growth disturbance. Signs and symptoms of arthritis lie on a continuum from no sign or symptom to any combination of pain, swelling/exudate, tissue degradation and growth disturbance; obviously, this complicates clinical diagnosis (5). Thus, use of the cardinal signs of inflammation as the sole basis for diagnosing TMJ arthritis may lack clinical utility. Cardinal inflammatory signs such as swelling, oedema and elevated temperature are seldom seen, especially in chronic TMJ arthritis. Indeed, chronic TMJ inflammation may show none of the cardinal signs, despite disease progression (6). On the other hand, TMJ arthritis may cause arthralgia but arthralgia could also be due to other factors, which trigger articular nociceptors (e.g. noxious mechanical stimuli), referred pain and general/central sensitisation.

An important goal with diagnostic criteria for arthritis should be the possibility of early

identification of patients with ongoing TMJ arthritis with high risk of chronicity and damage because there is evidence that early arthritis treatment allows less damage, suffering and treatment (7, 8). The American College of Rheumatology recently updated their classification criteria for rheumatoid arthritis with a primary focus on establishing clinical findings important for early diagnosis of cases with high risk of chronicity and damage whilst not excluding more established cases (7). This approach seems reasonable to implement in the future development of the extended DC/TMD taxonomy. By then, clinical symptoms and signs other than the cardinal signs should be considered for inclusion in the diagnostic criteria to enable early and more specific diagnosis. Examples of such signs could be pain from the TMJ on jaw movement, pain from the TMJ on loading and recent progressive occlusal changes.

In systemic arthritides as well as monoarthritic conditions, TMJ pain on jaw movements has been found to be strongly related to an inflammatory intra-articular milieu (9–12). TMJ pain on jaw movement thus seems to be useful clinical symptom or sign when attempting to diagnose TMJ arthritis. In future revisions of this taxonomy, TMJ pain on jaw movement should be considered as an additional clinical feature added into the taxonomy. The criterion of local pain for a diagnosis of arthralgia, part of the common TMDs, is met either from palpation of the TMJ or from jaw movement; by extension, that criterion for TMJ arthralgia should undergo review in terms of it being more specific to another disorder.

It is difficult to distinguish 'arthralgia' from 'arthritis' as they overlap to a great extent but not totally. 'Arthralgia' is perhaps more of a clinical finding of joint pain, whereas 'arthritis' may comprise pain but it does not always comprise the clinical finding 'arthralgia'. At the same time, 'arthralgia' may be due to other factors than arthritis, for example overstretching of the joint and sensitisation, peripheral or central. The discussion in Cape Town leads to the suggestion to consider that the diagnosis 'arthralgia' should be subdivided into 'arthralgia due to arthritis', 'arthralgia due to noxious mechanical stimuli', 'arthralgia due to referred pain', 'arthralgia due to general/central sensitisation', 'idiopathic arthralgia', etc. This would be relevant as these diagnoses, as with arthritis, would be more based on the underlying

pathophysiology and as the treatment options could differ to some extent.

To complicate matters further, in rheumatology, the definition of 'definite synovitis' in a particular joint is a swollen or tender joint. This is fine for most joints but probably not for the TMJ as swelling is very rare and the pressure pain threshold for mechanical pressure over the TMJ is mainly modulated by systemic factors rather than local intra-articular inflammatory mediators (12). This means that the rheumatological definition of synovitis is probably not appropriate for the TMJ.

One major unsolved issue is lack of an established reference standard for arthritis. TMJ synovial fluid sampling to determine inflammatory mediator content may be a step forward (9). Studies have reported indications of a strong relationship between TMJ inflammatory activity and elevated biomarker levels of tumour necrosis factor, interleukin-1 beta, interleukin-1 receptor antagonist, serotonin or glutamate, or reduced levels of tumour necrosis factor receptor II or interleukin-1 receptor II in TMJ synovial fluid (9–13). Indeed, preliminary data from analysis of the TNF or IL-1 content in TMJ synovial fluid indicate that TMJ resting pain, TMJ pain on palpation or TMJ movement pain has high sensitivity for inflammatory activity in the TMJ; no TMJ resting pain, TMJ pain on palpation or TMJ movement pain has a high specificity for inflammatory activity; and TMJ movement pain is strongly related to the degree of inflammatory activity in the TMJ.

Workgroup recommendations. The workgroup recommended exploring use of TMJ synovial fluid levels of specific biomarkers as a reference standard, conducting studies to clarify whether clinical findings beyond what the DC/TMD examination already contains should be included, and considering clinical findings that may improve identification of patients with early-stage TMJ arthritis.

The workgroup also suggested a systematic review of standards for the diagnosis of arthritis comprising all joints, together with rheumatological and orthopaedic expertise. Another suggestion was to form a group to propose new diagnostic criteria for TMJ arthritis (of local or systemic genesis), including a simple diagnostic flow chart, after gathering scientific data. A final suggestion was to consider subgroups of

arthritis (with and without pain, with and without tissue destruction) in future discussions and development of diagnostic criteria.

Disc–condyle complex disorders

Disc–condyle complex disorders typically result in changes in jaw mobility, including hypo- and hypermobility. A classification scheme for these joint disorders is outlined in the recently published DC/TMD (1, 2, 14) and includes three categories with nine separate disorders: disc disorders (disc displacements), hypomobility disorders other than disc disorders (joint adhesions, ankyloses) and hypermobility disorders (dislocations) (Table 1). From a clinical perspective, disc–condyle complex disorders may result in acute pain and more enduring functional impairment, ranging from joint sounds, jaw deviation and limited movement (particularly opening and contralateral movements) to an inability to close the mouth.

Community sampling suggests some joint disorders are relatively common; for example, disc displacements are estimated to occur in 18–35% of the non-

clinical population (15). However, epidemiological data are not available for all disorders, including those variables that may be important in aetiology and progression, such as age of onset, sex, pain, range of mandibular movement, frequency of symptoms, and stage and degree of morphological and pathological changes in the disc/condyle complex (14–18).

A recent study demonstrated no association between an individual's intra-articular status and reported pain, function and disability (19). The study focused on disc displacements with and without reduction, and whilst this was a cross-sectional study, it suggests these disorders have minimal impact. Investigation of the impact of the other hypo- and hypermobility disorders is worthy of consideration. Importantly, epidemiological data, including the impact on the individual and society, will help determine the priorities of research into these disorders, including development of valid diagnostic criteria.

The progression of disorders is important to better understand the distinction between the disorders in the current classification scheme. Whilst a limited subset of disc displacements with reduction progress to a non-reducing state (20, 21), the progression of other disorders such as dislocations is unknown. Neither is it known whether the disorders represent unique entities that require specific interventions, or if they can be grouped and then managed in the same manner. For example, it is not known whether there is a cause–effect relationship between adhesions or adherence and disc displacement without reduction (18). Initially, conservative management is recommended for these disorders (15, 22), but if this fails, there is insufficient evidence to support or refute other strategies (23). In summary, the factors that may predict worsening symptoms, disorder progression and management success are not well understood.

Whilst the aetiologies and pathophysiologies of these disorders are not clearly understood, it is assumed that the anatomical (structural) and biomechanical (functional) environments contribute to these disorders. Cross-sectional studies suggest that the anatomical variables contributing to the aetiology include a steep anterior wall of the condylar fossa, a high articular eminence, incongruence between condyle/disc and fossa, ligament laxity, muscle angulation and lateral pterygoid attachment to the disc (15). Putative biomechanical aetiological contributors

Table 1. Joint Disorders taxonomy and validity

Joint disorders	Validity of diagnostic criteria
A. Disc disorders	
1. Disc displacement with reduction	Sensitivity 0·34; specificity 0·92
	Reference standard: Imaging
2. Disc displacement with reduction with intermittent locking	Sensitivity 0·38; specificity 0·98
	Reference standard: Imaging
3. Disc displacement without reduction with limited opening	Sensitivity 0·80; specificity 0·97
	Reference standard: Imaging
4. Disc displacement without reduction without limited opening	Sensitivity 0·54; specificity 0·79
	Reference standard: Imaging
B. Hypomobility disorders other than disc disorders	
1. Adhesions/Adherence	
2. Ankylosis	
a. Fibrous	
b. Osseous	
C. Hypermobility disorders	
1. Dislocations	
a. Subluxation	Sensitivity 0·98; specificity 1·00 (based on history only)
b. Luxation	

include high compressive, tensile and/or shear forces resulting from, for example oral parafunction, trauma or impaired joint lubrication (18). Further, the interaction of two or more of these structural and functional variables may more likely result in a joint disorder. For example, biomechanical modelling suggests that a combination of elevator muscle angulation and eminence morphology results in hypermobility disorders (24).

Diagnostic criteria for all nine disc–condyle complex disorders have been proposed; however, the criteria for four of these have not been assessed for criterion validity. Of the other five disorders, the diagnostic criteria of three do not have acceptable sensitivity and specificity for a definitive diagnosis (i.e. sensitivity greater than 70% and specificity greater than 95%) (25). Diagnostic schemes that produce relatively high false negatives (i.e. low sensitivity) are not necessarily a priority research area if the disorder has little clinical consequence, such as disc displacements with reduction. For a definitive diagnosis of disc–condyle complex disorders, additional testing of diagnostic imaging has been recommended (1, 2). Whilst TMJ magnetic resonance imaging is the reference standard for disc displacement diagnoses, image interpretation without calibration has been shown to be unreliable (26, 27).

Workgroup recommendations. To better understand disc–condyle complex disorders, consideration needs to be given to the prevalence and incidence of these disorders in the community, and their progression and impact. Reference standards and criterion validity for all of the disorders need to be established, and those already established (e.g. imaging for disc displacements) should be considered further in the light of the cost and impact of the disorder. The expanded taxonomy has nine disc–condyle complex disorders, and research should focus on whether these are distinct entities. For example, are temporomandibular joint adhesions, disc displacement without reduction and fibrous ankylosis related? Consideration also needs to be given to management strategies that are effective in reducing impact or preventing the disorder's progression.

The variables influencing the development and progression of disc–condyle complex disorders are unknown, and a multicentre longitudinal trial should be considered a priority. Current and past research can help inform standardised variables of interest.

For example, demographic (e.g. age, sex), clinical/functional (e.g. range of mandibular movement, duration of locking, parafunctional habits, pain characteristics, degree of inflammation, comorbid conditions and treatment type, frequency and duration) and structural (e.g. jaw muscle angulation, joint morphology and severity of disc displacement) variables have been suggested as possible influences that should be considered. It is important to select such variables carefully, and those with clinical utility will lend themselves to better acceptance. Such a trial will help establish diagnostic criteria for those disorders without criterion validity and provide data on predictive variables for positive and negative progress.

Myofascial pain

The expanded taxonomy for TMD lists four mutually exclusive muscle pain disorders, and empirical data show that myalgia can be differentiated into three clinical subtypes (Table 2) (1, 2). The distinction into local myalgia, myofascial pain and myofascial pain with referral rests on a thorough palpation of the masseter and temporalis muscle with a pressure of about 1 kg for 5 s. The task of the workgroup was to reflect on the importance of distinguishing the myalgia subtypes, and more specifically myofascial pain, and thereafter discuss the research avenues and clinical implications of defining these subtypes. It is important to underscore that 'myofascial pain' per the DC/TMD is not the same clinical entity described in the original RDC/TMD (25). In fact, Group I Muscle Disorders of the RDC/TMD are broadly covered under the umbrella diagnosis of 'myalgia' and this includes myofascial pain with limitation of opening which is no longer a DC/TMD muscle disorder diagnosis following a recommendation from the International Consensus Workshop that cited lack of data supporting its clinical utility as a specific diagnosis (1, 28)

Criterion validity for myalgia is established and excellent, as it is for the clinical subtype 'myofascial pain with referral'. However, using the diagnostic criteria for local myalgia and myofascial pain without having assessed their validity for these subtypes is a significant obstacle in research settings. The argument that sensitivity and specificity are likely within acceptable ranges, considering the reliability of the palpation test and the high values reported for the diagnostic criteria for myalgia and myofascial pain with referral,

Table 2. Diagnostic criteria for the three subtypes of Myalgia

Myalgia subtypes	Local myalgia	Myofascial pain	Myofascial pain with referral
History criteria	Positive for both of the following: 1 Pain in the jaw, temple, in the ear or in front of ear 2 Pain modified with jaw movements, function or parafunction		
Examination criteria	Positive for all of the following: 1 Confirmation of pain location(s) in the temporalis or the masseter muscle(s); AND 2 Report of familiar pain with palpation of temporalis or masseter muscle(s); AND		
	Report of pain localised to the site of palpation	Report of pain spreading beyond the site of palpation but within the boundary of the muscle	Report of pain at a site beyond the boundary of the muscle being palpated
Sensitivity/Specificity	Unknown	Unknown	0·86/0·98

cannot justify bypassing a validation study (29). The new taxonomic structure also raises the question of the dual level of diagnosis with myalgia corresponding to a broader and non-specific diagnosis as opposed to any of the three clinical subtypes. Guidance as to what is most suitable in clinical and research settings would certainly be helpful. In other words, knowing whether a diagnosis of myalgia allows selection of the most appropriate treatment, and if this level of diagnosis is specific enough to answer important research questions, would be very valuable. At this point in time, no differentiated treatment algorithms exist for myalgia subtypes, and future research should try to identify potential differences and similarities in treatment modalities.

Whilst the classification scheme for the myalgia subtypes suggests an apparent hierarchy, little is known about how these three conditions relate to each other. One can hardly disregard the possibility that local myalgia, myofascial pain and myofascial pain with referral are presentations of a single disorder at different points in time, and thus represent a continuum from mild and remittent local pain to more regional and continuous severe pain. Notwithstanding the importance of an Axis II diagnosis, and despite the questionable value of tender muscle spot quantification as a predictor of treatment response, more thorough muscle palpation allowing differentiation between the three subtypes of myalgia may be worthwhile if, indeed, it is helpful for prognostic consideration and early disease modifying treatment (30).

The distinguishing features of each myalgia subtype also raise the possibility of dealing with at least two if not three separate disorders. Different pathophysiologic processes may well explain why pain evoked by palpation remains localised or is referred elsewhere. A supporting argument for a dichotomy between local myalgia and myofascial pain with referral is the existence in the latter condition of painful foci in muscles defined as trigger points. Active muscle trigger points generating spontaneous pain are seemingly associated with local changes in the biochemical milieu, which points to a pathologic entity that is presumably distinguishable from non-specific muscle hyperalgesia (31). Confirmation studies of such changes in trigger points are urgently needed to show that it is more than simply epiphenomenon associated with deeper and long-lasting muscle pain.

There is an open debate about the reference standard for muscle trigger point identification that is central to the diagnosis of 'myofascial pain syndrome' in other body areas. Interestingly, the trigger point phenomenon was recently documented in the temporalis and masseter muscles of patients and controls using four basic criteria: (i) palpable taut band in a muscle, (ii) point tenderness upon pressing the taut band, (iii) local twitch response elicited by the snapping palpation of the taut band and (iv) referred pain in response to sustained compression of the taut band (32). To improve the reliability and diagnostic validity of trigger point identification, replacing the 'local twitch response' and 'pain referral' criteria with

'nodule in a taut band' and 'painful limitation induced by movements' has been suggested (33). Importantly, only active trigger points cause pain known to reproduce the patient's pain complaint; trigger points that do not are labelled 'latent' and can be observed in both symptomatic and asymptomatic subjects (31). Thus, it seems that thorough palpation of the muscles is likely to uncover the presence of trigger points as defined above when applying sufficient pressure for at least 5 s. If pain referral is no longer a required criterion for the identification of trigger points, 'myofascial pain' and 'myofascial pain with referral' are then potentially the same disorder and similar to the 'myofascial pain syndrome' known to occur in other parts of the body, if taut bands are found in both types of myofascial pain as defined per the DC/TMD.

Workgroup recommendations. The task ahead for answering any of the above questions is complex but imperative to solving issues regarding the taxonomic framework for myalgia and its different subtypes. Above all, criterion validity for the diagnosis of local myalgia and myofascial pain must first be established with acceptable sensitivity and specificity. Thereafter, the distinctive myalgia subtypes should be assessed through well-designed research protocols based on the multidimensional, integrated approach recently proposed in the ACTTION-American Pain Society Pain Taxonomy (AAPT) (34). Hence for each myalgia subtype, distinguishing features must be delineated according to five major dimensions: (i) core diagnostic criteria; (ii) common features; (iii) common medical comorbidities; (iv) neurobiological, psychosocial and functional consequences; and (v) putative neurobiological and psychosocial mechanisms, risk factors and protective factors (34).

This approach may be the key for clearly substantiating whether both types of myofascial pain per the DC/TMD are a single disorder representing a distinctive clinical entity of local myalgia due to significant differences in pathophysiological and psychosocial mechanisms, response to treatment and prognosis.

Oro-facial movement disorders

The expanded taxonomy of the DC/TMD (2) includes two types of oro-facial movement disorders: oro-facial dyskinesia and oromandibular dystonia. Although the literature in this area is limited, the operational definitions and diagnostic criteria for both conditions are apparently unequivocal (e.g. 35–37). Nevertheless, several questions remain unanswered, concerning not only the definitions themselves but also related issues such as aetiology, consequences, differential diagnosis and treatment of oro-facial movement disorders. Specifically, similarities and differences between sleep and awake bruxism on the one hand and other oro-facial movement disorders on the other hand need further clarification. These topics are addressed below by focusing on five questions, followed by recommendations for future research.

The first question was whether the definitions of the oro-facial movement disorders in the expanded DC/TMD have sufficient face validity. Although both definitions apparently have sufficient face validity, an overall definition of oro-facial movement disorders that fully covers the entire collection of possible conditions is currently lacking. Thus, the following definition is proposed: 'Orofacial movement disorders are characterised by hyperkinesia/hypertonia or hypokinesia/hypotonia; possibly involving the face, lips, tongue, and/or jaw; and being focal, segmental, or part of a generalised movement disorder'. This definition includes all conditions that are characterised by either too much or too little muscle activity, tone or both; all possible anatomical structures that can be affected; and the possibility that the movement disorder is either localised in the oro-facial area alone or part of a larger, that is segmental, or even generalised problem.

The second question focused on similarities between the suggested aetiological factors for sleep and awake bruxism and those for other oro-facial movement disorders. For sleep and awake bruxism (38), a multifactorial aetiology has been proposed where peripheral, psychosocial, intrinsic (biological) and extrinsic factors may be involved (for reviews, see 39–41). The literature (35, 42, 43) and clinical experience suggest that a classification of possible aetiological factors similar to the one for bruxism can be constructed:

1 *Peripheral factors*: edentulism, malfunctioning prostheses, dento-alveolar trauma and over-closure in the vertical dimension of occlusion.
2 *Psychosocial factors*: psychiatric conditions and psychosocial factors that have been associated with bruxism.

3 *Intrinsic (biological) factors*: genetics and dopamine dysfunction (basal ganglia).

4 *Extrinsic factors*: extrinsic factors that have been associated with bruxism and medication (various types).

Clearly, when this listing is compared to the one for sleep and awake bruxism, it must be concluded that there is a considerable overlap between bruxism and other oro-facial movement disorders.

The third question was whether there are any similarities between the suggested consequences of sleep and awake bruxism and those of other oro-facial movement disorders. Based on the above-cited literature and clinical experience, there seems to be an enormous overlap between the suggested consequences of sleep and awake bruxism and those of other oro-facial movement disorders. For example, both conditions may yield tooth wear; fracture or failure of teeth, restorations, prostheses and implants; accelerated bone loss in edentulous patients; oro-facial pain; temporomandibular joint (TMJ) degeneration; soft tissue damage; and muscle hypertrophy. Additionally, oro-facial movement disorders may cause TMJ luxation, speech or swallowing impairments, social embarrassment and chewing difficulties; this last consequence may lead to inadequate food intake, weight loss and even cognitive impairment, especially in the elderly population (44).

The fourth question was how a dentist or dental specialist can recognise oro-facial movement disorders in the clinic. The first step is to develop a diagnostic grading system for oro-facial movement disorders analogous to the one that was recently developed for sleep and awake bruxism (38):

1 A possible diagnosis of oro-facial movement disorder is based on the outcomes of questionnaires, an oral history, or both, yielding descriptions of the behaviour.

2 A probable oro-facial movement disorder diagnosis can be established by the dentist or dental specialist on the basis of questionnaires, an oral history and a clinical examination focusing on observations like tooth wear; fracture or failure of teeth, restorations, prostheses and implants; accelerated bone loss in edentulous patients; oro-facial pain; TMJ luxation; TMJ degeneration; soft tissue damage; speech or swallowing impairments; muscle hypertrophy;

direct observation of the behaviour; and qualitative or quantitative sensory testing (in the case of sensory nerve conduction deficits).

3 A neurologist is needed to establish a definite diagnosis of oro-facial movement disorder, using additional diagnostic techniques such as surface or intramuscular electromyography (in the case of motor nerve conduction deficits) and functional imaging (e.g. MRI, PET).

The fifth and final question was: Is there a role for the dentist or dental specialist in the treatment of other oro-facial movement disorders than sleep and awake bruxism? The answer is yes, but this depends on the aetiological factors affecting the individual patient; when possible, treatment should be aetiology-based.

Workgroup recommendations. As a next step, the existing literature on oro-facial movement disorders should be systematically assessed to ascertain that the proposed definition covers all conditions mentioned in previous publications, and to discover any possible comorbidities of oro-facial movement disorders.

The literature should be systematically assessed to find evidence for the proposed classification of possible aetiological factors. Additionally, large-scale cross-sectional assessments of general population samples would be valuable to establish risk indicators, as would longitudinal follow-up studies to assess risk factors and experimental animal studies to determine possible aetiological factors. Following this, suggested aetiological factors should be compared to what is currently known for bruxism.

A systematic literature assessment of the possible consequences of oro-facial movement disorders in relation to those of sleep and awake bruxism is needed to provide the necessary evidence for this overlap. In addition, cross-sectional assessments of patient population samples should be made to further establish possible or probable consequences of oro-facial movement disorders whilst longitudinal follow-up studies may serve to unequivocally establish the consequences of these conditions. The outcomes should then be compared to the current evidence for sleep and awake bruxism.

Diagnostic criteria could be derived from systematic assessments of the literature and also from consensus discussions with experts. Based on such efforts, a

comprehensive set of diagnostic criteria could be composed, for possible, probable and definite diagnoses of oro-facial movement disorders. Reliability, validity and diagnostic cut-off criteria for the created system should then be established, followed by item reduction based on the above steps, a Delphi procedure, or both.

A systematic assessment of the literature, randomised clinical trials, and subsequent development of evidence-based guidelines will be pivotal in the development of evidence-based treatment strategies for oro-facial movement disorders.

Importantly, from the above questions, it can be gathered that medical (i.e. neurological) input in this highly multidisciplinary topic is badly needed, in both the research and the clinical settings.

Headache attributed to temporomandibular disorders

The current DC/TMD Headache attributed to TMD (HA-TMD) has excellent criterion validity (sensitivity 89% and specificity of 87%) compared to a TMD headache reference standard, and these criteria have achieved broad acceptance in the TMD community (1, 2, 45). With the same reference standard, the criterion validity of the ICHD-2 (3) has a sensitivity of 84% and a specificity of 33% (45). Compared to the ICHD-2, HA-TMD in the DC/TMD has significantly higher specificity ($P < 0.001$) (45). The low specificity of the ICHD-2 was due, in part, to the need for a positive imaging finding of intra-articular temporomandibular joint (TMJ) disorders, reduced or irregular opening and TMJ noise – all of which can be present in headache patients without other TMD signs or symptoms, including pain. The DC/TMD for HA-TMD do not require imaging findings to demonstrate the presence of a TMJ disorder (1). The criteria require instead that the patient have a pain-related TMD diagnosis. This approach seems more logical because it directly links TMD-related headache to pain-related TMD (e.g. TMJ arthralgia or masticatory myalgia) (1, 2) rather than imaging findings, which can be present in asymptomatic individuals (45–48).

Recently, the ICHD-3 (beta version) proposed a new version for HA-TMD, with unknown criterion validity, that contains criteria from both the ICHD-2 and the DC/TMD for HA-TMD (4). Criterion B in the ICHD-3, like in the ICHD-2, still requires 'Clinical and/or imaging evidence of a pathological process affecting the temporomandibular joint (TMJ), muscles of mastication and/or associated structures'. As previously noted relative to the ICHD-2, imaging findings decrease the accuracy of the diagnostic criteria and could allow an inaccurate diagnosis of HA-TMD (i.e. false positive) (45). Thus, criterion B could improve if it were limited to clinical findings per the DC/TMD version, where the patient must have a diagnosis of pain-related TMD (e.g. TMJ arthralgia or masticatory muscle myalgia) in order to be given a diagnosis of HA-TMD.

Criterion C-2 in the ICHD-3 requires three new history items: C-1, headache developed in temporal relation to the onset of the TMD; and C-2, headache worsened significantly in parallel with progression of the TMD, or headache significantly improved or resolved in parallel with improvement in or resolution of the TMD. These criteria are potentially problematic in several situations. When TMD and headache onset is recent, criterion C-2 is of little use as the short duration of the condition may be insufficient for one of these relationships to develop or be recognised by the patient. Additionally, if the headache has existed for some time, and improved, resolved or worsened in parallel with the TMD as it improved, resolved or worsened, patients may find it difficult to remember such associations, especially if they had been unaware that they also had TMD or that a possible relationship might exist. Furthermore, if patients did know that they have TMD and headache, they may still be unable to recall this relationship from the past or may erroneously remember this relationship (i.e. recall bias).

Table 3 shows a comparison of the DC/TMD and ICHD-3 HA-TMD criteria using McNemar's test for correlated data as we tested these two criteria in the same 48 subjects (32 with TMD and 16 normal subjects). Three blinded TMD experts rendered diagnoses; interexaminer reliability (GEE Kappa procedures) was excellent ($\kappa \geq 0.79$). Additionally, the subjects completed the three history items proposed in the ICHD-3 for criterion C-1 and criterion C-2. The ICHD-3 criteria (with the three history items) and the DC/TMD criteria (without the three history items) were compared for ipsilateral diagnosis of right- and left-side headache. The data were analysed for each side as criterion C-4 in the ICHD-3 states: 'headache, when unilateral, is ipsilateral to the side of the tem-

Table 3. Comparison of DC/TMD and ICHD-3 criteria for *Headache attributed to TMD*

ICHD-3 TMD headache criteria (Right side)	Reference standard: DC/TMD headache criteria (Right side)		
	Yes	No	Total
Yes	15	0	15
No	5	28	33
Total	20	28	48

ICHD-3 TMD headache criteria (Left side)	Reference standard: DC/TMD headache criteria (Left side)		
	Yes	No	Total
Yes	15	0	15
No	6	27	33
Total	21	27	48

ICHD-3: International Classification of Headache Diseases. DC/TMD: Diagnostic Criteria for Temporomandibular Disorders.

poromandibular disorder'. As per Table 3, relative to the DC/TMD for HA-TMD, the ICHD-3 headache criteria detected 15 of 20 (75%) of right-side HA-TMD cases and 15 of 21 (71%) of left-side HA-TMD cases. These diagnostic detection rates were statistically different: $P = 0.025$ and $P = 0.014$, respectively. Thus, the ICHD-3 criteria have a sensitivity between 71% and 75% and a specificity of 100% when the DC/TMD criteria are considered the reference standard. These results suggest that the ICHD-3 criteria significantly underdiagnosed HA-TMD when compared to the DC/TMD validated criteria.

Criterion C-3 of the ICHD-3 is a mix of clinical provocation tests that have not been operationalised and therefore have unknown reliability and validity. In addition, unlike the DC/TMD headache criteria, the ICHD-3 does not require that the provocation tests replicate the subject's headache. Finally, the ICHD-3 criteria do not incorporate the DC/TMD criterion requiring that jaw movement, jaw function or jaw parafunction change the headache – and these criteria are considered the hallmark of pain-related TMD (1).

Workgroup recommendations. Two sets of diagnostic criteria for the secondary headache, HA-TMD, is not in the best interests of clinicians and researchers because having two versions will cause confusion and make research results using different versions incomparable. We propose the following principles to unify the ICHD-3 diagnostic algorithms and reduce the potential for confusion:

1 Retention of the proposed ICHD-3 template defining diagnostic criteria for secondary headaches where *no* validated criteria exist.
2 Use of validated diagnostic criteria when they do exist.

These principles would set a precedent for how validated diagnostic criteria can become part of the ICHD-3, especially when they deviate from the current ICHD-3 template (4). Thus, we are recommending that the ICHD-3 use the current validated DC/TMD for HA-TMD.

Recommendations for future research are:
1 Determine the criterion validity for a modified version of the ICHD-3 HA-TMD (see Table 4) and for the current ICHD-3 version using a credible TMD headache reference standard and then test validated versions in heterogeneous population(s) and different clinical settings. The modified version attempts to incorporate features of HA-TMD from both the ICHD-3 version and the validated DC/TMD version.
2 Determine criterion validity for the validated DC/TMD HA-TMD version in heterogeneous populations and different clinical settings.
3 Establish the relationship of HA-TMD with different primary headache types as all ICHD-3 diagnostic criteria for secondary headaches have the criterion state that any primary headache type can be present with HA-TMD.
4 Determine whether TMD is a trigger of migraine.

Table 4. The 3rd edition of the International Classification of Headache Disorders (ICHD-3) for *Headache attributed to Temporomandibular Disorders*: Criteria, issues and changes between current version (V1) and proposed version (V2) for future criterion validity assessment

HA-TMD criteria	Issues
A.	
Any headache fulfilling criterion C	Determine whether this applies to all primary headache types or is limited to specific primary headaches types (e.g. tension-type headache)
B.	
V1:Clinical and/or imaging evidence of a pathological process affecting the TMJ, muscles of mastication and/or or associated structures	Use V1 or V2.
V2:Clinical evidence of pain affecting the TMJ, muscles of mastication and/or associated structures (e.g. TMJ arthralgia or masticatory myalgia)	
C.	
Evidence of causation demonstrated by *at least two* of the following:	Are 2 criteria needed?
1. Headache has developed in temporal relation to the onset of the TMD	Is this criterion needed?
2. Either or both of the following:	
a. Headache has significantly worsened in parallel with progression of the TMD	Are these criteria needed?
b. Headache has significantly improved or resolved in parallel with improvement in or resolution of the TMD	
3. V1: Headache is produced or exacerbated by active jaw movements, passive movements through the range of motion of the jaw and/or provocative manoeuvres applied to temporomandibular structures such as pressure on the TMJ and surrounding muscles of mastication	Use V1 or V2 or a different version? V1 would need to be operationalised
V2: History of headache changed by jaw movement, function or parafunction, AND familiar headache is produced or exacerbated by provocation tests of palpation of the temporalis or masseter muscle(s), TMJ(s), or range of motion of the jaw	
4. Headache, when unilateral, is ipsilateral to the side of the TMD pain	Determine the clinical utility of having this criterion
D.	
Not better accounted for by another ICHD-3 diagnosis	

Sensitivity and specificity have not been established; TMJ = temporomandibular joint.

5 Assess how components of pain are either similar or different in order to probe the underlying mechanisms. For example: Are patients with local pain in the temporal area different from those with pain referred to the temporal area? Are there subgroups of patients who respond differently to local anaesthetic blocks to the temporalis muscle(s)?

Biobehavioural domain

To assess core Axis II constructs, the DC/TMD identified a set of instruments focusing on TMD, pain disorders in general, psychological status and physical symptom reporting (1). These instrument recommendations build on the RDC/TMD (25), the psychometric review from the RDC/TMD Validation Project (49) and the revisions proposed in the 2009 International Consensus Workshop (28). Despite a more inclusive set of instruments which should map better to clinical needs, new challenges emerge for Axis II; these challenges and the possible responses will shape the implementation of the DC/TMD protocol and how it continues to evolve.

One challenge is that some settings may prefer legacy instruments (i.e. Axis II from the RDC/TMD) or alternative instruments (e.g. the Beck Depression Inventory) for assessing corresponding DC/TMD constructs. Local needs should always be considered in instrument selection, but one consequence of choosing an outside instrument is lack of comparability with other DC/TMD research settings which would be a significant limitation in the overall implementation of the DC/TMD. Clearly, different measures of the same construct can and should be compared (50), and item response statistical models can create equivalent scaling (51, 52).

A second challenge relates to subject burden. An increasing number of psychosocial constructs are of interest in both clinical and research settings. To assess these, increased subject burden may lead to decreased reliability due to subject fatigue. A prominent solution is computerised adaptive testing (CAT), which is based on real-time integration of information from item response statistical models; items presented to the individual are optimised in order to maximise measurement precision and minimise the number of items (53–56). Extensive testing (57) of this approach indicates that it is ready for routine implementation.

A third challenge is fundamental to the goals of Axis II assessment: instrument-level measurement solely for purposes of classification versus dimensional measurement of the targeted characteristic. Overall, the DC/TMD instruments for depression, anxiety and physical symptom status reduce subject burden, reduce provider burden, increase validity and simplify interpretation compared to the corresponding instruments in the RDC/TMD. Yet, finding the right balance between classification versus dimensional measurement with respect to utility of the Axis II assessment tools is an ongoing process and clearly will never have a single approach that works equally well for every setting. Alternative instruments should be selected based on careful consideration of these stated principles.

A fourth challenge focuses on screening versus comprehensive assessment. A minimal assessment framework needs to be standardised. For example, the 4-item Patient Health Questionnaire (PHQ-4), the Graded Chronic Pain Scale and the Pain Drawing can comprise an acceptable minimal set for biobehavioural screening; this would allow the DC/TMD to promote a fundamental, simplified face for Axis II use. Whether the assessment of parafunctional behaviours (via the Oral Behaviors Checklist [OBC]), a construct with widespread interest and broad relevance to dentistry, should be included as part of that minimal set of instruments for biobehavioural screening is a complex question, in part because validity and utility have yet to be demonstrated. The goals of screening need clarification, in that an increasing number of biobehavioural constructs have supporting evidence for their relevance to TMD pain and its treatment, but the value of more information is realised only when it can be utilised in the consulting room. In addition, repeated assessments across time is an intrinsic char-

acteristic of biobehavioural management, and the optimal timeframes for obtaining more information, whether by interview, screening or comprehensive assessment instrument, are in reality specific to a given patient and related to treatment priorities. Training and guidance are needed for optimal joint use of interview and instruments within the sequencing of evaluation, immediate treatment and long-term management, and for the clinician's next steps in the light of information gained by biobehavioural assessment.

A fifth challenge for the DC/TMD Axis II emerges in response to a new taxonomy project for chronic pain disorders, the ACTTION-American Pain Society Pain Taxonomy (AAPT) (34). Some of the five dimensions addressed in this new taxonomy project are subsumed within Axis II of the DC/TMD. As the evolution of the RDC/TMD to the DC/TMD is one template for the AAPT project, further evolution of the DC/TMD in reflecting this 5-axis approach will probably be timely as well as synergistic.

Workgroup recommendations. The complexity of these issues suggests that the Consortium needs to consider taking on additional roles in advocacy and education (e.g. training in Axis II implementation). Political changes would be essential for some of the clinical procedures deemed important for implementation even in settings that may also have high medical literacy and financial resources. A long-term workgroup was recommended for conducting relatively simple (possibly survey) studies to address some of the issues emerging from this discussion. One study could be a survey of existing practices: What is actually done in biobehavioural assessment and treatment? During the era of the RDC/TMD, the authors assumed that Axis II would be used as intended, and for the DC/TMD, we have made the same assumption. However, that assumption was not necessarily true for the users of the RDC/TMD, and it is not likely to be true for users of the DC/TMD. This gap in usage versus intention exists for many reasons. For example, one group has replaced the Graded Chronic Pain Scale with a structured instrument that assesses multiple aspects about a patient's pain experience. They approach the standardised assessment of all constructs embedded in the DC/TMD Axis II via single-item screening questions, followed by a longer questionnaire if the single-item screener is endorsed. This approach achieves a

different balance between patient burden and derived information. Whilst the gap between usage and intention of the DC/TMD Axis II occurs from a desire to maximise efficiency in clinical assessment, this situation also raises psychometric issues.

Clearly, the DC/TMD Axis II is a work in progress. Usage and interpretation of Axis II ranges across settings represented by members of this workgroup as follows: (i) none: not using the Axis II instruments, and hence no interpretation; (ii) basic: very specific selection of instruments for screening, and straightforward interpretation (e.g. refer to psychologist); and (iii) complex: administering core Axis II instruments as well as additional instruments, and interpreting in a multidimensional manner. To illustrate the challenges that go into matching biobehavioural assessment with intended usage for a given population, one well-informed clinical setting uses the following instruments: Graded Chronic Pain Scale, pain drawing, three pain catastrophising questions, the Survey of Pain Attitudes scale and the PHQ-4.

To move this complex discussion and these challenging short- and long-term goals forward, a trial administration of selected screening instruments was proposed to determine the minimum effective screeners empirically. A parallel project would be to assess how biobehavioural information is used in clinical decision-making.

Conclusion

As stated in the bylaws, the RDC/TMD Consortium Network is an international organisation that facilitates collaborative research at multinational university or healthcare delivery sites that strongly support research conducted at the highest standards of scientific excellence. The objectives of the consortium include promoting international and collaborative researches aiming to a more complete understanding of TMD and biopsychosocial complexity. The consortium conducts at least one scientific meeting per year in conjunction with meetings of the IADR to disseminate research related to TMD and discuss criticisms. According to the critical analysis presented in this paper, continuous discussion is vital, and research challenges in the described domains must be addressed. The present paper could serve as an agenda in upcoming years for research carried out under the umbrella of the consortium.

Acknowledgments

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