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Human jaw joint hypermobility: Diagnosis and biomechanical modelling

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Summary

Patients with hypermobility disorders of the jaw joint experience joint sounds and jerky movements of the jaw. In severe cases, a subluxation or luxation can occur. Clinically, hypermobility disorders should be differentiated from disc displacements. With biomechanical modelling, we previously identified the anterior slope angle of the eminence and the orientation of the jaw closers to potentially contribute to hypermobility disorders. Using cone-beam computed tomography (CBCT), we constructed patient-specific models of the masticatory system to incorporate these aspects. It is not known whether the clinical diagnosis of hypermobility disorders is associated with the prediction of hypermobility by a patient-specific biomechanical model. Fifteen patients and eleven controls, matched for gender and age, were enrolled in the study. Clinical diagnosis was performed according to the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) and additional testing to differentiate hypermobility from disc displacements. Forward simulations with patient-specific biomechanical models were performed for maximum opening and subsequent closing of the jaw. This predicted a hypermobility disorder (luxation) or a control (normal closing). We found no association between the clinical diagnosis and predictions of hypermobility disorders. The biomechanical models overestimated the number of patients, yielding a low specificity. The role of the collagenous structures remains unclear; therefore, the articular disc and the ligaments should be modelled in greater detail. This also holds for the fanned shape of the temporalis muscle. However, for the osseous structures, we determined post hoc that the anterior slope angle of the articular eminence is steeper in patients than in controls.

KEYWORDS

biomechanical phenomena, clinical protocols, computer simulation, hypermobility, luxation, radiography, temporomandibular joint, validity

1 | INTRODUCTION

Hypermobility disorders of the human jaw joint are defined by "greater than normal range of motion in a joint, which may occur naturally in otherwise normal persons or may be a sign of joint instability".[1] It can be subdivided into different levels of severity. The recently published DC/TMD and its expanded taxonomy[2,3] differentiate between subluxations and luxations. Patients with a subluxation are able to close their mouth themselves by relaxing the masticatory muscles. Alternatively, sideway movements or self-manipulations can aid to the return of the mandibular condyles to the glenoid fossae.[4,5] Clinically, symptomatic hypermobility of the jaw joint is used as a mild subdivision of subluxations. These patients often report clicking joint sounds and jerky movements of the lower jaw during wide opening and closing of the mouth.[6-8] Patients suffering from a luxation are not capable of reducing the luxation themselves. This is only possible by someone else, for example a relative or a clinician. When a clinician performs the procedure, this may be combined with a complete sedation of the patient.[7] In the clinic, luxations are also referred to as open locks.[9] For differential diagnosis in the DC/TMD, a history suffices to discriminate between subluxations and luxations, based on the ability of the patient to self-reduce the anteriorly displaced mandibular condyles.

In the clinical setting, clicks due to hypermobility can be distinguished from those due to anterior disc displacement by their timing during opening and closing. Clicks at the end of wide opening and at the beginning of closing suggest hypermobility, while clicks at any point during opening and at the end of closing suggest an anterior disc displacement.[8,10] In addition, apart from audible clicks, palpable jerky movements of the condyle during protrusive open/close movements indicate (mild) subluxations.[11] A luxation is only diagnosed when reproduced during the actual test. As this additional test can adequately differentiate between hypermobility disorders and disc displacements,[8,12] it is often considered as gold standard.

We also approached the problem of hypermobility disorders biomechanically, by means of a model study.[13] We identified two possible morphological aspects of the masticatory system that could contribute to luxations. Firstly, regarding the angle of the anterior aspect of the articular eminence, the models predicted that steeper anterior slope angles were more likely to cause a luxation. Secondly, the models also showed that a more forwardly inclined working line of the jaw closers could contribute to luxations. The addition of masticatory muscle forces and joint reaction forces resulted in a net anterior translation of the mandibular condyle, resulting in a luxation. These two factors showed an interaction such that less steep slope angles could compensate for jaw closers with more anteriorly directed force vectors and that steeper anterior slope angles could be compensated for jaw closers with more posteriorly directed force vectors. Subsequently, we showed that different activation schemes for the jaw closers were able to reduce a luxation compared with direct closing activation.[5] Activation schemes of the jaw closers consisting of relaxation or inducing a lateral movement of the lower jaw could resolve a luxation, thus mimicking the clinical situation of subluxation.

From a translational viewpoint, it is not known whether the predictions from our biomechanical model correspond with the clinical diagnosis according to the DC/TMD. To this end, the generic biomechanical model should be altered to meet the anterior slope angle and the working lines of the jaw closers at an individual level. Cone-beam computed tomography (CBCT) can provide these three-dimensional data with suitable resolution for diagnostics and treatment planning.[14,15] The use of CBCT in the field of oral and maxillofacial imaging is currently widely accepted due to advantages over computed tomography like lower cost and dose.[16] It has also been shown that CBCT has high-diagnostic accuracy in the assessment of osseous TMJ structures.[17] In our aim to predict hypermobility disorders at an individual level, we use these CBCT scans that provide the morphological input to adjust the generic biomechanical model into a patient-specific biomechanical model. These predictions will be compared with the clinical diagnosis. We hypothesised that the presence of hypermobility disorders, as confirmed with clinical diagnosis, is associated with the prediction of vulnerability to open locks as performed with patient-specific musculoskeletal models of the masticatory system.

2 | MATERIAL AND METHODS

2.1 | Patients and controls

Patients were recruited through advertisement via screens in the General Dentistry Department of the Academic Centre for Dentistry Amsterdam (ACTA), by an announcement of the protocol on ACTA's patient website, and from acquaintances, ACTA students and colleagues. Two patients were referred by maxillofacial surgeons from the Emergency Department of the Academic Medical Centre (AMC), Amsterdam.

We included patients between 18 and 65 years old with a report of hypermobility disorders of the jaw joint (subluxations (including symptomatic hypermobility) or luxations). Patients were excluded for serious general health impairments, complicated dental abnormalities, osteoarthritis of the jaw joint or pregnancy. This exclusion was based on a short telephonic history, prior to enrolment in the study. We approached controls, matched for age and gender. Power analysis, based upon a pilot study,[18] showed that a sample size of ten to fifteen participants per group would be sufficient.[19] In total, fifteen patients and eleven matched controls were enrolled in the study (eight males [four patients], eighteen females [eleven patients], mean ± SD age = 33.5 ± 11.0 years).

2.2 | Ethics

The research protocol was designed according to the Helsinki Declaration and was approved by the Medical Ethical Committee of the Vrije Universiteit Medical Center (VUmc), Amsterdam, protocol number NL18726.029.07. Upon entry in the clinic, participants received additional explanation of the protocol and an information sheet about the study. After reading this and agreeing to participate, participants signed an informed consent.

2.3 | Data acquisition: clinical assessment

Upon entry in the clinic of oro-facial pain and dysfunction of ACTA, a short history was taken, followed by a clinical examination by one of two trained and calibrated clinicians (MK, FL) who were blinded to the category of recruitment (patient, control). Clinical examination was performed according to the clinical tests[8,10] and the expanded DC/TMD diagnostic rules.[3] The main diagnostic aim was to differentiate between hypermobility disorders, anterior disc displacement and controls.

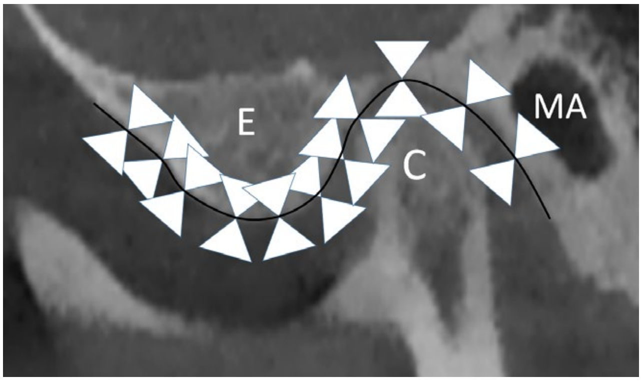

FIGURE 1 Sagittal mid condylar slice. E: articular eminence. C: mandibular condyle. MA: meatus acousticus. The hourglasses indicate the manual, graphical input points across the bony outline of the glenoid fossa and articular eminence. The solid black line represents the spline definition fitting the bony outline of these bony structures [Colour figure can be viewed at wileyonlinelibrary.com]

2.4 | Data acquisition: radiology

CBCT scans were performed using a NewTom 5G (QR Verona, Verona, Italy) at the Department of Oral Radiology of ACTA. The occlusal plane of each participant was set perpendicular to the floor. Scan settings were as follows: 110 kVp, 38.25 (range 22.35-55.76) mAs (3.6 seconds exposure time), and field of view of 18 × 12 cm. All CBCT data sets were stored in Digital Imaging and Communications in Medicine (DICOM) format, with isotropic voxel size of 0.3 mm.

2.5 | Data analysis: joint shape and muscle orientation

In the sagittal view of the DICOM images, bilateral glenoid fossa and eminence shape were determined in the slice midway between the condylar poles (3Diagnosys 3.1, 3Diemme, Cantu, Italy). Based on manual, graphical input of the bony outline of the glenoid fossa and eminence, a spline definition was made to fit these structures (Curve Fitting Toolbox, MatLab, R2014b, The MathWorks Inc., Natick, MA, USA) (Figure 1). The medio-lateral radius of the condyle was determined from half the distance between medial and lateral pole of the condyle. In an oblique slice, perpendicular to the medio-lateral radius, the inferior/superior radius and anterior/posterior radius of the condyle were also determined (Figure 2).

The working lines of the left and right jaw closers (masseter, temporalis and medial pterygoid muscle)[20] were derived from the DICOM images, based on assessment of their attachment sites (Masseter_origin Figure S1 and Masseter_insertion Figure S2). Origin and insertion of the jaw closers were determined according to Baron and Debussy.[21] The working lines of the deep masseter, superficial masseter, anterior temporalis, posterior temporalis and medial pterygoid were subsequently defined with respect to the participant's bite plane.

2.6 | Data analysis: adapting biomechanical model to the participant

Our biomechanical model[13,22] was adapted to fit the musculoskeletal parameters of each participant (Figure 3). The working lines of the jaw closers of the model (which were originally based on a cadaver study[23] were adjusted to meet the muscle orientation from the CBCT scan. To adequately describe the participant-specific joint morphology of the jaw joint, the spline shapes of the fossa/eminence were loaded into the model. Also, the radii of both mandibular condyles in the model were adapted to the participant's dimensions.

2.7 | Data analysis: simulations

From a closed mouth position, a forward dynamic simulation of a maximal mouth opening and closing movement was performed with MatLab (R2014b, The MathWorks Inc., Natick, MA, USA). Briefly, a forward simulation takes a predefined muscle activation pattern for the jaw openers and jaw closers. From the activation pattern, it calculates the produced muscle forces at each time step of the simulation. For each time step, the model also makes a prediction of joint

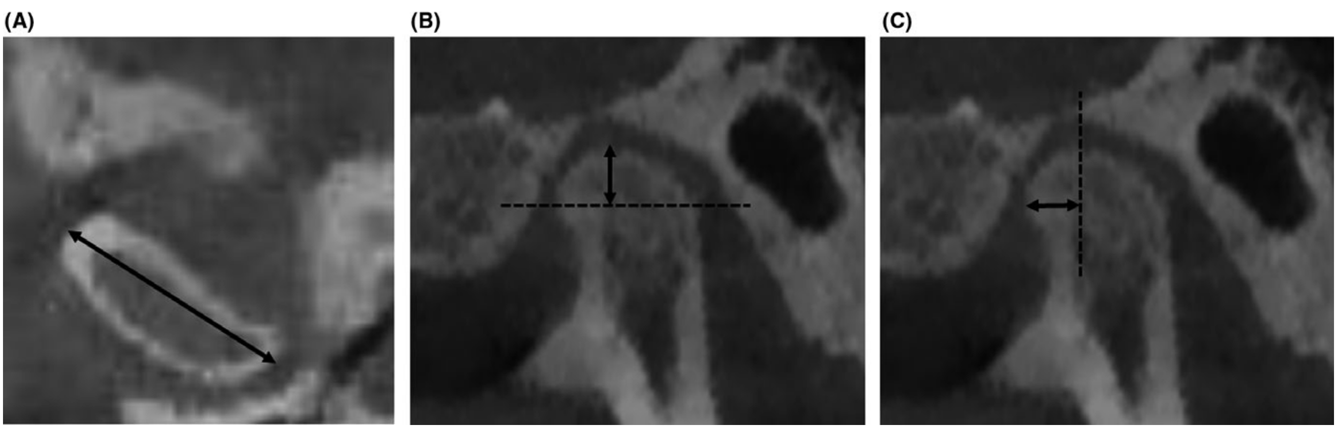

FIGURE 2 Three radii describing the mandibular condyle. A, Distance between medial and lateral pole of the mandibular condyle. The radius amounts to half of this medio-lateral distance. B, Inferior–superior radius of the mandibular condyle. Dotted line runs through the anterior most point of the condyle. C, Anterior–posterior radius of the mandibular condyle. Dotted line runs through the superior most point of the condyle

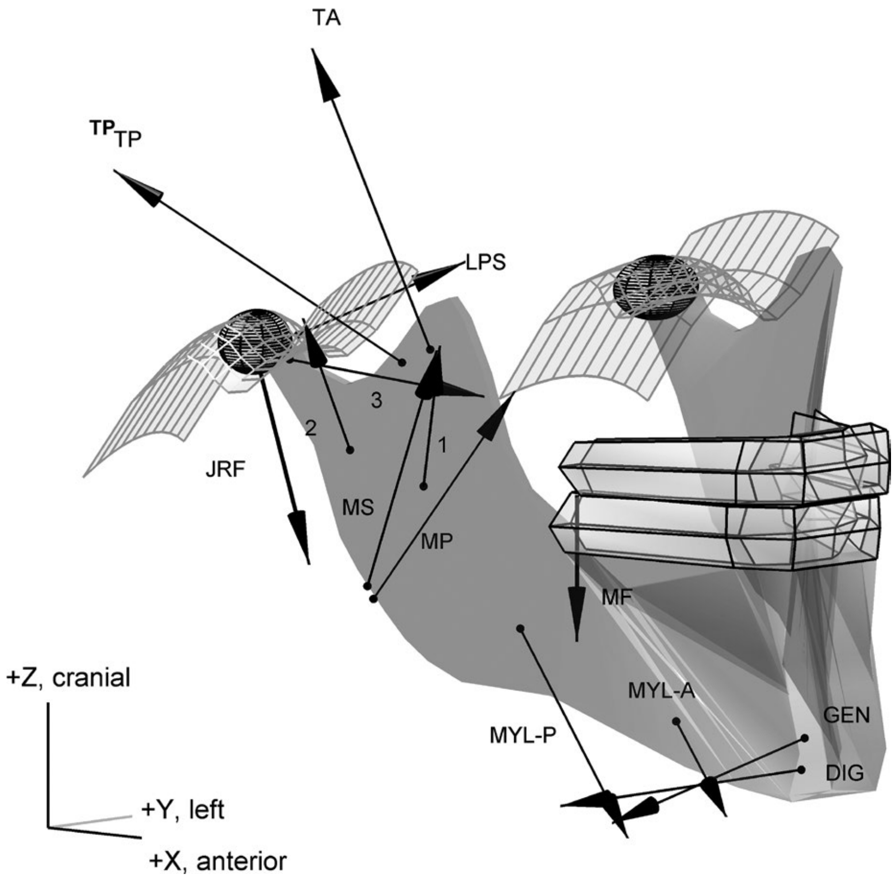

FIGURE 3 Graphical representation of the biomechanical model of the human masticatory system. Right anterior view. Arrows indicate forces (right side only). Anterior slope angle of the articular eminence (ASA). Working line of the jaw closers. More forward inclination (+15) depicted only for anterior aspect of temporalis muscle (TA). TP, posterior temporalis; MP, medial pterygoid; MA, masseter (three parts); JRF, joint reaction force; C, mandibular condyle; E, articular eminence

reaction forces and ligament forces. These forces lead to the subsequent movement of the lower jaw in six degrees of freedom with respect to the skull. For in-depth modelling, details are referred to Tuijt et al.[22] At the end of the simulation, the model predicted the mandibular condyle to either remain anterior of the articular eminence (hypermobility disorder) or return to the glenoid fossa (control).

2.8 | Statistics

Various validity and predictive values for the diagnostic and the modelling approaches were calculated. Firstly, recruitment based on self-report was compared with a diagnosis based on clinical examination according to DC/TMD standards. Subsequently, model predictions of hypermobility were cross-tabulated with and tested against this diagnosis of hypermobility disorders. We used a McNemar's chi-square test, with a significance level of 0.05 (Statistics Toolbox, MatLab, R2014b, The MathWorks Inc., Natick, MA, USA). This test was corrected for the relatively small-sample size, with a mid-p calculation.[24]

3 | RESULTS

3.1 | Comparison of subject recruitment with clinical diagnosis

From the participants, who were recruited as patients, eleven of fifteen were diagnosed with hypermobility disorders according to DC/TMD standards (Table 1). Also, one of eleven controls was diagnosed with a hypermobility disorder. Therefore, the agreement was good as shown by a sensitivity of 0.73, a specificity of 0.91 and a kappa of 0.7. Overall, five participants were diagnosed differently from their recruitment. As the differential diagnosis according to the clinical DC/TMD standard was decisive, we continued with twelve patients and fourteen controls.

3.2 | Model predictions

For eighteen of 26 participants, the predictions of our participant-specific biomechanical models resulted in a final condyle position anterior of the eminence, either unilateral or bilateral, indicating a hypermobility disorder. Simulations of both conditions are reported in the supplemental material. For comparison, a simulation of a normal closing movement was added (Movie S1: normal opening and closing; Movie S2: unilateral luxation; Movie S3: bilateral luxation).

3.3 | Comparison of clinical diagnosis and model predictions

In nine of the twelve patients, diagnosed according to the DC/TMD standard, both methods agreed. This yielded a good sensitivity of 0.75. From the fourteen controls, five also led to normal simulated closing, herewith the specificity was low (0.36). Of the eighteen

TABLE 1 Cross-tab of recruitment of hypermobility disorders versus diagnosis of hypermobility disorders according to DC/TMD standards

		Recruitment: Hypermobility disorders		
Diagnosis of hypermobility disorders		+	−	Total
	+	11	1	12
	−	4	10	14
	Total	15	11	26

hypermobility predictions of the biomechanical model, nine were accordingly diagnosed clinically, leading to a positive predictive value of 0.5. The negative predictive value amounted to 0.63 (five of eight controls). Overall results coincided in fourteen of 26 participants, and thus, the accuracy was 0.54. Furthermore, the prevalence according to clinical diagnosis was 0.46 (twelve of 26 participants). The test statistic X^2 for the mid-p McNemar test amounted to 1.91, with a p-value of 0.83. Therefore, the observed agreement was considered accidental.

4 | DISCUSSION

The observed amount of agreement between clinical diagnosis of hypermobility disorders and participant-specific biomechanical prediction of hypermobility disorders was considered accidental. Therefore, our hypothesis, that there should be an association between these two methods, could not be confirmed. The hypothesis was tested using biomechanical models of the human masticatory system. It must be noted that predictions of such models are limited to the parameters or processes under consideration.[25] The models had been made patient-specific by adaptation of the geometry of the articular tubercle and muscle lines of action. Therefore, the predictions were limited to these variables.

4.1 | Diagnostics of hypermobility disorders: recruitment compared with DC/TMD standards

We found a small difference between the patient's enrolment based on history and the clinical standard. Four of fifteen participants, recruited as patients, were not diagnosed as such. It appeared to be relatively hard for patients to fundamentally understand questions about the position of the luxated jaw, or about closing problems. Misunderstandings can also occur between luxations and opening problems from a closed mouth position. We have minimised this problem by taking history by telephone, prior to enrolment. However, reported closing problems of the lower jaw could also be attributed to anterior disc displacements (ADD). Two participants, who enrolled as patients, were clinically diagnosed with ADD and had to be excluded from the patient category. The additional clinical testing that we performed therefore appeared to be necessary for accurately diagnosing a hypermobility disorder.

4.2 | Participant-specific modelling of hypermobility: morphology of the musculoskeletal system

The predictions of the model overestimated the number of participants to be susceptible to hypermobility disorders. This is indicated by the high number of false positives (eight participants), yielding a low sensitivity. We took great care to accurately describe the morphology of the patients and controls for the bony and muscular aspects of the masticatory system. Due to lack of discriminative power between patients and controls, it appears that morphology only could not differentiate between patients and controls. We tested this post hoc with an independent Student's *t* test (IBM, SPSS Statistics, Version 23) and found that only the anterior slope angle differed between patients and controls (right anterior slope angle: $t = 2{,}38$, $P = 0.026$; left anterior slope angle: $t = 1.5$, $P = 0.14$, which can be considered a strong trend due to the small number of participants). We found no difference in the direction of the working lines of the jaw closers. The variation was high for the anterior slope angles (SD 14°) as well as for the working lines of the jaw closers (masseter (SD 10°), temporalis (SD 14°), medial pterygoid (SD 15°)). However, the difference in variation between the groups was not significant for anterior slope angles, or for the working lines of the muscles (Levene's test $P > 0.05$). The large variations suggest that there might be subsets of patients with hypermobility disorders with steeper anterior slope angles who run a greater risk of experiencing luxations.

4.3 | Temporalis muscle: passive and active forces

The role of the temporalis muscle in hypermobility disorders deserves attention. The fanned shape of the temporalis allows for a multitude of working lines at the coronoid process. It appears that the passive stretch of the posterior part just above the ear is a strong contributor to a posteriorly directed force to limit anterior translation of the jaw and condyle. However, in our current model, this large variation in working lines has been simplified to two muscle slips. A more elaborate description of the temporalis[26] could provide further insight in its role in luxations of the jaw. Also, activation patterns of the various temporalis parts are not known. Earlier activation of the posterior part of the temporalis could limit further anterior translation of the condyle along the anterior slope. It has been shown that the activation pattern of the temporalis can change according to task speed. Blanksma and Van Eijden showed that the temporalis muscle shifts from the last muscle to be activated during self-selected speed of opening and closing to the first muscle during fast opening and closing, from and to the intercuspal occlusal position.[27] This nearly 200-ms earlier activation could also be beneficial during fast opening in patients suffering from subluxations or luxations. However, it is not known how well this closing speed can be controlled by patients, thus making its possible role in the management of luxations questionable.

4.4 | Model limitations and assumptions: activation pattern

To mimic an open lock, we chose an activation level for the digastric muscle of 100% to reach a maximum mouth opening and a large anterior translation of the mandibular condyle. This goal was clearly met, since open locks were predicted successfully, even ignoring possible contributions of the neck musculature to wide jaw opening.[28] For future studies, it would be a benefit to incorporate electromyography of temporalis, masseter and digastric muscles. The current ethical approval did not allow for this. Therefore, we used an older study[29] to define one of many possible activation patterns. Although newer studies regarding masticatory muscle activation during various statical and dynamical tasks are available,[27,30] incorporating their results would not have led to a better agreement between the simulation results and the clinical observations. It must be stressed that activation patterns have a very individual aspect. They can vary widely between subjects.[31] The present study suggests that personal solutions to activate masticatory muscles may help to solve hypermobility issues despite morphologic challenges.

4.5 | Model limitations and assumptions: posterior capsule

After assembling the participant's data sets, all preliminary test simulations ended in a condyle anterior of the articular eminence. The mandibular condyle slipped off the anterior slope at its most anterior aspect. In the previous version of our model, we already incorporated a lateral ligament, as this is the strongest part of the capsule. However, at maximum opening, this ligament did not become stretched during the simulations and did not limit anterior translation. The anterior translation of the condyle could be stopped by the posterior part of the capsule. In the previous version of the model, this was neglected. As it becomes taut at maximum opening, we added the posterior part of the capsule to limit the anterior translation to one centimetre anterior of the apex of the articular eminence. The mandibular and cranial attachment sites were chosen based on the anatomical descriptions of the condylar neck just below the condyle and inferior of the external hearing canal.

4.6 | Model limitations: articular disc/compression forces

The current version of our model contains a precise description of the bony contour of the fossa/eminence complex. The joint reaction forces are estimated by a penalty-type contact criterion, based on the amount of penetration of the mandibular condyle into this complex. This represents the deformation of the disc and superior articular cartilage layer during loading. The assumption of a homogenous layer of cartilage has the least influence of model predictions at maximum mouth opening. The thinnest part of the disc, the intermediate zone, is then compressed at the anterior position of the condyle. However, the disc can also have a displaced position. In the clinic, it is very important to assess whether the disc is anteriorly displaced and, if this is the case, whether the posterior band of the disc reduces at maximum mouth opening. As stated, the current version of the model does not contain a description of the shape of the disc, and therefore, the influence of disc displacements could not be investigated. In future studies, the addition of a finite element model of the disc would result in a hybrid rigid body-finite element model.[32] This could provide further insight in the role of the disc in anterior disc displacements and hypermobility disorders.

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REFERENCES

1. Anderson DM, ed. *Dorland's Illustrated Medical Dictionary*, 32nd edn. Philadelphia: Elsevier Saunders; 2012.
2. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Groupdagger. *J Oral Facial Pain Headache*. 2014;28(1):6-27.
3. Peck CC, Goulet JP, Lobbezoo F, et al. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. *J Oral Rehabil*. 2014;41(1):2-23.
4. de Leeuw R. Internal derangements of the temporomandibular joint. *Oral Maxillofac Surg Clin North Am*. 2008;20(2):159-168.
5. Tuijt M, Koolstra JH, Lobbezoo F, Naeije M. How muscle relaxation and laterotrusion resolve open locks of the temporomandibular joint. Forward dynamic 3D-modelling of the human masticatory system. *J Biomech*. 2016;49(2):276-283.
6. Kalaykova S, Naeije M, Huddleston Slater JJ, Lobbezoo F. Is condylar position a predictor for functional signs of TMJ hypermobility? *J Oral Rehabil*. 2006;33(5):349-355.
7. August M, Troulis MJ, Kaban LB. Hypomobility and hypermobility disorders of the temporomandibular joint. In: Miloro M, Ghali GE, Larsen PE, eds. *Waite PD Peterson's principles in oral and maxillofacial surgery*, 2nd edn. Hamilton, ON: BC Decker Inc.; 2004:1033-1048.
8. Huddleston Slater JJ, Lobbezoo F, Chen YJ, Naeije M. A comparative study between clinical and instrumental methods for the recognition of internal derangements with a clicking sound on condylar movement. *J Orofac Pain*. 2004;18(2):138-147.
9. Shorey CW, Campbell JH. Dislocation of the temporomandibular joint. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2000;89(6):662-668.

10. Huddleston Slater JJ, Lobbezoo F, Onland-Moret NC, Naeije M. Anterior disc displacement with reduction and symptomatic hypermobility in the human temporomandibular joint: prevalence rates and risk factors in children and teenagers. *J Orofac Pain*. 2007;21(1):55-62.
11. Widmalm SE, Dong Y, Li BX, Lin M, Fan LJ, Deng SM. Unbalanced lateral mandibular deviation associated with TMJ sound as a sign in TMJ disc dysfunction diagnosis. *J Oral Rehabil*. 2016;43:911-920.
12. Marpaung CM, Kalaykova SI, Lobbezoo F, Naeije M. Validity of functional diagnostic examination for temporomandibular joint disc displacement with reduction. *J Oral Rehabil*. 2014;41(4):243-249.
13. Tuijt M, Koolstra JH, Lobbezoo F, Naeije M. Biomechanical modelling of open locks of the human temporomandibular joint. *Clin Biomech (Bristol, Avon)*. 2012;27(8):749-753.
14. Parsa A, Ibrahim N, Hassan B, Motroni A, van der Stelt P, Wismeijer D. Influence of cone beam CT scanning parameters on grey value measurements at an implant site. *Dentomaxillofac Radiol*. 2013;42(3):79884780.
15. Oliveira ML, Tosoni GM, Lindsey DH, Mendoza K, Tetradis S, Mallya SM. Influence of anatomical location on CT numbers in cone beam computed tomography. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2013;115(4):558-564.
16. Parsa A, Ibrahim N, Hassan B, van der Stelt P, Wismeijer D. Bone quality evaluation at dental implant site using multislice CT, micro-CT, and cone beam CT. *Clin Oral Implants Res*. 2015;26(1):e1-e7.
17. Honey OB, Scarfe WC, Hilgers MJ, et al. Accuracy of cone-beam computed tomography imaging of the temporomandibular joint: comparisons with panoramic radiology and linear tomography. *Am J Orthod Dentofacial Orthop*. 2007;132(4):429-438.
18. Smeets EC. Open Lock of the Human Temporomandibular Joint. Master Thesis 2015, ACTA.
19. Hoozemans MJ, Burdorf A, van der Beek AJ, Frings-Dresen MH, Mathiassen SE. Group-based measurement strategies in exposure assessment explored by bootstrapping. *Scand J Work Environ Health*. 2001;27(2):125-132.
20. Schumacher GH. *Funktionelle Morphologie der Kaumuskulatur*. Jena, Germany: VEB Gustav Fischer Verlag; 1961:1-53.
21. Baron P, Debussy T. A biomechanical functional analysis of the masticatory muscles in man. *Arch Oral Biol*. 1979;24(7):547-553.
22. Tuijt M, Koolstra JH, Lobbezoo F, Naeije M. Differences in loading of the temporomandibular joint during opening and closing of the jaw. *J Biomech*. 2010;43(6):1048-1054.
23. van Eijden TMGJ, Korfage JAM, Brugman P. Architecture of the human jaw-closing and jaw-opening muscles. *Anat Rec*. 1997;248:464-474.
24. Fagerland MW, Lydersen S, Laake P. The McNemar test for binary matched-pairs data: mid-p and asymptotic are better than exact conditional. *BMC Med Res Methodol*. 2013;13:91.
25. Koolstra JH. Number crunching with the human masticatory system. *J Dent Res*. 2003;82:672-676.
26. Desailly E, Sardain P, Khouri N, Yepremian D, Lacouture P. The convex wrapping algorithm: a method for identifying muscle paths using the underlying bone mesh. *J Biomech*. 2010;43(13):2601-2607.
27. Blanksma NG, Van Eijden TMGJ. Electromyographic heterogeneity in the human temporalis and masseter muscles during static biting, open/close excursions, and chewing. *J Dent Res*. 1995;74(6):1318-1327.
28. Koolstra JH, van Eijden TMGJ. Functional significance of the coupling between head and jaw movements. *J Biomech*. 2004;37:1387-1392.
29. Møller E. The chewing apparatus. An electromyographic study of the action of the muscles of mastication and its correlation to facial morphology. *Acta Physiol Scand* 1966;260:1-229.
30. Widmalm SE, Lillie JH, Ash MM. Anatomical and electromyographic studies of the digastric muscle. *J Oral Rehabil*. 1988;15:3-21.
31. Koolstra JH, van Eijden TMGJ. A method to predict muscle control in the kinematically and mechanically indeterminate human masticatory system. *J Biomech*. 2001;34:1179-1188.
32. Koolstra JH, Van Eijden TMGJ. Combined finite-element and rigid-body analysis of human jaw joint dynamics. *J Biomech*. 2005;38(12):2431-2439.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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