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Development of the articular eminence: an argument for early orthodontic treatment.

TMJ Development

Articular eminence of TMJ is essentially flat in newborns and is progressively formed during the craniofacial growth period.^{1,2} Active movement of any synovial joint is necessary for cavitation to progress and failure of active movement, whether due to neuropathy, myopathy, constriction, or other pathology may result in arthrogyriposis in the limbs, or ankylosis in the TMJ.³ Changes in extracellular matrix, especially hyaluronan, seem important for joint cavitation and complement the contribution of muscle driven motion in creation of a functional synovial joint,⁴ while in cases of condylar agenesis, the articular eminence does not develop.⁵

The alternative to cavitation is that the joint may develop by distraction of the joint due gravity and the weight of the mandibular tissues.⁶ Distraction is postulated to contribute to the growth of the mandibular condyle as the tuberculum develops by apposition, which would imply minor and transient force incapable of cavitation. Also, the difficulty with cavitation as a major factor in development of the TM joint is the lack of expected apoptosis.⁷ Since the eminence is initially flat, the centripetal growth of the brain and cavitation conflict and create a logical difficulty considering that the superior surface of the glenoid fossa is paper thin.

The articular eminence growth pattern more closely resembles that of the face, even though it is part of the cranium.⁵ The TMJ eminence is greater than 50% its mature size, and exhibits mature morphology by the time of complete eruption of the primary dentition; prior to the complete eruption of the primary molars there is considerable growth of the eminence.^{1,5} The articular eminence height grows at a very high rate until the age of 7, in conjunction with the period of deciduous dentition development and function. The articular eminence is 70%-72% of adult dimension by the 10 years of age, after which eminence growth slows around 11 years of age, the period of mixed dentition, and may be a result of difficulty with function during the mixed dentition due to exfoliation of the succedaneous teeth.⁸ Articular eminence growth continues to approximate the somatic growth curve and by 20 years of age, the eminence is 90% to 94% of adult height, finally achieving full inclination by approximately 30 years of age,^{5,9,10} and eventually begins to decrease, especially after the fourth decade.¹¹

The initial very rapid growth of TMJ morphology is proposed as preparation to withstand the load of future masticatory function; however the slow rate of growth over time for the remaining percentage denotes susceptibility and opportunity for therapeutic intervention.^{5,12}

Occlusion as related to TMJ modeling

The roof of the glenoid fossa appears to enlarge forward by modeling while sagittal and vertical growth is achieved by deposition at the top of the tubercle, therefore the pressure



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gradient at the interface must be minor and transient as growth against a pressure gradient requires an epiphyseal plate.¹³ These changes result in a steeper slope of the eminence which take place in 3 phases, in coordination with the dentition: the eruption of the central incisors, the permanent first molars, and the permanent second molars.¹⁴ Notable is that the second molar eruption is temporally similar to canine eruption; the transition from the juvenile to adult chewing pattern appears to develop in conjunction with eruption of the permanent canines, occurring about the age of 12 years.^{15,16} This lack of transition is observed in adults with severe anterior open bite who cannot achieve normal canine function and retain the juvenile chewing pattern.¹⁵ Similarly, wide lateral excursion observed in children¹⁷ may be due to the lack of anterior and lateral guidance in young children,¹⁵

Articular eminence inclination seems related to overbite and overjet both in both protrusive and laterotrusive excursions,¹⁸ as seen above by the close relationship of the articular eminence development and occlusal development. As well, working-side interference has been observed to have an immediate, significant effect on working-side condylar movement, where the magnitude of the change is correlated with the magnitude of occlusal alteration.¹⁹ Correlation between articular fossa slopes to the palatal inclined planes of the anterior teeth (47.6 and 42.7 degrees, respectively) suggests a possible functional relationship between these 2 units, due to the protrusive movements of the mandible.²⁰

As with development of the dentition, the eminence modeling follows the demise of the dentition, especially after the fourth decade, and begins to decrease in height with dental attrition and/or tooth loss.^{11,21-23} With continued progression through to edentulism, the eminence will flatten, however, the contour can be maintained by restoration with complete dentures.²⁴

Articular Eminence Inclination and Internal Derangement

It appears that disc displacement is less likely to be found in joints with a shallow articular eminence; not only the protrusive condylar pathway angulation but also the lateral condylar pathway steepness may be important for development of disc displacement/internal derangement.²⁵⁻³⁰ Eminectomy has been advocated as effective treatment for disc displacement; however, fracture of mini-plates used for internal osseous fixation is an issue.^{28,31} The biomechanical theory of disc displacement postulates that with a steep eminence, there is tendency for the disc to rotate farther forward than normal on the condyle as the disc-condyle assembly rotates forward within the glenoid fossa during mouth opening. Meanwhile there is a stabilizing force produced by the masseter and temporalis, which with a steeper eminence, places a greater relative distalizing force relative to the disc.^{32,33} This might result in laxity of the ligaments that attach the disc to the condyle.^{34,35} The disc articulating against a steep eminence during mouth opening would gradually achieve a more anterior position relative to the condyle, predisposing the disc to anterior displacement.³²

Alternatively, there may be an effect on the lubrication of the joint as the disc is forced along the eminence. Increase in friction due to the steepness could, as the mandible rotates with opening, force the disc anteriorly a greater distance than normal exhausting the effect of weeping



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lubrication.³⁶ Individuals with a history of mandibular luxation and mandibular hypoplasia seem to display a steeper articular eminence angle.²⁸

Modeling progression:

Disc displacement is a relatively common condition with internal derangement of the TMJ characterized by increased friction of the articulating surfaces thereby causing reduced disc/condyle mobility, which has been observed to frequently progress to avascular necrosis and osteoarthritis of the condyle and eminence.^{37,38} Many cases of acquired malocclusion, facial deformity, and condylar degeneration may be the consequence of avascular necrosis.^{37,38} It is important to note that osteophyte formation may not be directly related to resorption of the condyle but rather to tension on the inferior lateral pterygoid tendon insertion due to a loss of articular support and that asymmetry in articular eminence angulation may be diagnostic of a functional shift; the retruded condyle being associated with the steeper angulation. This tension induced osteophyte formation is also often seen with functional shifts of the mandible in the absence of avascular necrosis.

Orthodontic manipulation of teeth and TMJ modeling:

It is important to recognize there is a relationship between development of the eminence and the teeth. The eminence height is half of the adult height at the age of 2, which is correlated to reduced (if not ½) anterior guidance and cuspal steepness of the primary dentition compared to the permanent dentition. As the permanent maxillary incisors erupt, there is hint of change progressively until 7 or 8, while the maxillary lateral incisors are beginning to erupt and the progressive increase in central incisor root length stabilize anterior guidance. Not until the maxillary canine erupts is there attainment of final shape, albeit immature size.

Probable is that the articular eminence inclination is a function of sagittal and transverse overjet and overbite, conceptually allowing therapeutic modeling of the glenoid fossa. The difficulty is that the modeling of fossa form has begun in the mixed dentition and largely complete by the time the maxillary canine is erupting (and the permanent 2nd molar); this is approximately the recommended time for functional appliance therapy application. It would seem counterintuitive that the teeth should determine the shape of the glenoid fossa (within reason, not excluding muscle lengths) rather than adapt; however, that the dentoalveolar region is most symmetrical as compensatory changes occur to minimize the effects of underlying skeletal asymmetry in order to allow for a functional occlusion,³⁹ This may be why tooth determined eminence contour is more logical.

The genetic influence of an individual may be important as well. Individuals with interleukin 1-β allele-1 (related to increased EARR)⁴⁰ tend to display a decreased catabolic osseous modeling and hence decreased bone compliance. Conversely, individuals with IL-1β allele-2 (related to periodontal disease) display increased catabolic osseous modeling.⁴¹ This may affect the anchorage value and adaptability of teeth with allele-1 bone being less compliant (more anchorage)^{42,43} and allele-2 bone more compliant (less anchorage). There may also be



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correlations with alveolar densities in which tooth adaptability is decrease in individuals with greater bone density.⁴⁴ If the TMJ is partially reactive to tooth position then there may be a genetic component to internal derangement component of TMD. In those with a genetic predisposition^{42,43} toward decreased alveolar modeling, the teeth are less compliant to movement and the TMJ must accommodate through eminence and condylar modeling, or the teeth must accommodate with attrition. In those with the genetic predisposition toward increased bone modeling, the tooth movement may play a greater role in accommodation, which may explain the great variation of occlusal variables in response to TMD. It is possible that some individuals have an unfavorable genetic predisposition for compensation when considering contemporary refined diets.^{45,46} Clinical judgment must be used as other factors, such as parafunction, should be considered. It is possible that in a low modeling individual, a nightguard could be required to minimize forces to the TMJ to prevent avascular necrosis, in conjunction with occlusal adjustment. It is postulated that osteonecrotic modeling of the condyle can be pathologic, for contemporary refined diets, rather than physiologic²⁶ because catabolic condylar modeling may be erratic and create a skeletal openbite in the absence of attrition. It would seem preferable for the occlusion to adjust or be adjusted contiguous with the musculoskeletal stable position. Again, this does not in any way address parafunction or divergence from a musculoskeletal position, nor would it be expected a permanent solution as age dependant flux of the orofacial capsular matrix would still not be compensated with appropriate tooth movement. Avascular necrosis is probably best correlated with non-growing individuals. Growing individuals with no systemic modifiers would be expected capable of modifying the condylar position due to a thicker chondrocyte layer, and more compliant connective tissue (ligaments) and muscle component;⁴¹ although rate of chondrogenic adaptation seems to display individual variation as hypo-, normal and hyper-responsive.⁴⁷ **(Figure 2)**

If there is the possibility of modeling the articular eminence, then therapeutic management could reduce the risk of anterior disc displacement.^{27,32,48,49} The consideration of risk is important. TMJ adaptation to late adolescent treatment should mimic the observations of individuals with unilateral condylar fractures;⁵⁰ on average, 2 years were required for condylar adaptation needed to normalize chewing cycles. (inclusion criteria 16 to 70 years of age, sample size had decreased by 2 years).⁵⁰ This is similar to the long term findings with progressive condylar resorption after orthognathic surgery.⁵¹ When a condyle is positioned ventrally in the glenoid fossa with a functional appliance, the fossa will model and become more shallow^{12,52} and therefore, repositioning with functional appliances may be of clinical value although effects may not be fully evident, on average, for 2 years.

Conclusion

It is recommended that the functional aspect of the teeth be viewed as a possible controlling factor in TMJ ontogeny, which may influence a clinician toward early functional treatment. A genetic basis for the occlusal variation seen with TMD and is exacerbated by our contemporary diet and the associated lack of “normal” attrition, seems supported in literature. The



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understanding of genetic factors could create opportunities for developing genetic testing relevant to the study of internal derangement or structural adaptation components of TMD.

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