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

Temporomandibular Joint Development

Articular eminence of temporomandibular joint (TMJ) is essentially flat in newborns and is 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 arthrogryposis in the limbs, or ankylosis in the TMJ.[3] Changes in extracellular matrix (especially hyaluronan) are important for joint cavitation and complement the contribution of muscle driven motion in creation of a functional synovial joint.[4] In the case 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, 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 regardless 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. By the age of 10 years, the articular eminence is 70%-72% of adult dimension, after which, eminence growth slows around 11 years of age; the period of mixed dentition. The decrease in grow may be a result of difficult function during the mixed dentition due to exfoliation of the succedaneous teeth.[8] 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] Eventually, the articular eminence begins to decrease, especially after the fourth decade[11].

The initial very rapid growth may prepare TMJ morphology to withstand the load of future masticatory function. That it gains the remaining percentage over a long period of time 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; thus the pressure gradient at the interface must be minor and transient. 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.[13] Notable is that the second molar eruption is similar, temporally, to canine eruption. This is because, 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.[14,15] 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.[14]

Wide lateral excursion observed in children[16] may be due to the lack of anterior and lateral guidance in young children,[14]

That the articular eminence inclination is related to overbite and overjet both in both protrusive and laterotrusive excursions,[17] 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.[18] 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.[19] Therefore, question is do the teeth effect the eminence, does the eminence effect the teeth or is there a mutual relationship?[12]

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. [11,20-22] With continued progression through to edentulism, the eminence will flatten, however, the contour can be maintained by restoration with complete dentures.[23]

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.[24-29] Eminectomy has been advocated as effective treatment for disc displacement, however, fracture of mini-plates, used for internal osseous fixation, is an issue.[27,30] The biomechanical theory of disc displacement prostulates 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. [31,32] This might result in laxity of the ligaments that attach the disc to the condyle.[33,34] 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.[31]

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 and the effect of weeping lubrication exhausted.[35] Individuals with a history of mandibular luxation and, interestingly, mandibular hypoplasia may tend to have a steeper articular eminence angle.[27]

Ren[36] found that the articular eminence was steeper for an asymptomatic control group than for an internal derangement group, believing that the difference was due to modeling or degeneration associated with the disorder. This may be because they did not distinguish between disc derangement with reduction and disc displacement without reduction [24] or because the mean age for the internal derangement sample was 38 years of age.[5,11,36] Decrease in observed height is probably flattening associated with avascular necrosis or osteochondritis dissecans,[37,38] which seems the body’s attempt to perform an eminectomy. Galante [39] found no correlation between ID and articular inclination, however only one cross-sectional slice was measured. It is suggested that a central cross-section is often non-descriptive and requires the additional medial and lateral cross-sectional slices.[24]

Prevalence and etiology of ID:

The glenoid fossa of the youngest infants is flat, and little articular eminence was seen which may be why there is minimal probability of disk displacement to the age of 5.[40] With age and the increase in articular eminence development, the prevalence of disc displacement also increases as observed by the 11.8% prevalence of disc displacement for children 8 to 15 years of age.[41] There is a peak incidence of symptomatic TMJ disk displacement during puberty for males and females,[42] which is about the time that the canines begin to influence the occlusion. The risk to of development of painful disc displacement in the teenaged years is 4 times greater than the risk later in life, as well as there being a female preponderance versus males (3.3:1, Isberg; 4.25:1 Katzberg).[42,43]There is another peak in incidence for women during the third and fourth decade which may be linked to hormonal factors.[42] Disc displacement is a frequent finding with pre-orthodontic adolescents [44] which may necessitate early correction of predisposing factors that are susceptible to intervention.

Disc displacement is common (33-34%) in asymptomatic volunteers and is highly correlated with patients (77-86%) with TMD[45] [43] These results are similar to those of painful TMJ patients (81.7%) when compared to patients with non-painful TMJ patients (20.9%)[46]

There is an association between internal derangement and bruxism [43], a wider range of maximum mouth opening in ID patients when positive (normal) disc and condylar translation were presented[46], and with an increased general joint laxity (39% of TMD patients with disc displacement) when compared to patients with disc displacement in the control group (9%). [47]

Modeling progression:

Disc displacement is a commonly accepted condition with internal derangement of the TMJ, characterized by increased friction of the articulating surfaces, thereby causing reduced disc/condyle mobility and frequently progressing 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 is probably not 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. This tension induced osteophyte formation is also often seen with functional shifts of the mandible n the absence of avascular necrosis.

Orthodontic manipulation of teeth and TMJ modeling:

It is important to recognize the development of the eminence in relation to 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 a attainment of final shape, yet immature size.

If it is possible to say that the articular eminence inclination is a function of sagittal and transverse overjet and overbite, then it is possible to influence modeling of the glenoid fossa with orthodontic treatment. 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 application. It would seem counterintuitive that that teeth should determine the shape of the glenoid fossa (within reason, not excluding muscle lengths) as teeth would be expected to be adaptive. 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,[48] This may be why the tooth determined eminence contour it is so critical. The genetic makeup of the individual may be important as well.

The genetic makeup of the individual may be important as well. Individuals with interleukin 1-ß allele-1 (related to increased EARR)[49] tend to display a decreased catabolic osseous modeling and hence decreased bone compliance. Conversely, individuals with IL-1ß allele-2 (related to periodontal disease) display increased catabolic osseous modeling.[50] This may affect the anchorage value and adaptibilty of teeth with allele-1 bone being less compliant (more anchorage) [51,52] and allele-2 bone more compliant (less anchorage). If the TMJ is, to some degree, reactive to tooth position, then there may be a genetic component to internal derangement and possibly TMD. In those with a genetic predisposition[51,52] toward decreased modeling, the teeth are less compliant to movement; the TMJ must then 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. This may explain the great variation of occlusal variables in response to TMD. It is quite possible that some individuals have an unfavorable genetic predisposition for compensation when considering contemporary refined diets. [53,54]. Clinical judgment must been used as other factors, such as parafuction, must be considered. It is possible that in a low modeling individual, a nightguard could be required to 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 physiologic[25] because catobolic 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 musculo-skeletally 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. Avacular necrosis is probably best correlated with non-growing individuals. Growing individuals with no systemic modifiers would be expected capable to modify a condylar position due to a thicker chondrocyte layer, and compliance of connective tissue (ligaments) and muscle.[50] For example, Class II growth with mandibular alveolar retrusion relative to pogonion would be expected to have a condylar position similar to normal controls and possibly a genetic type for more compliant bone. Class II growth with no mandibular alveolar retrusion relative to pogonion would be expected to have, initially, restricted condylar growth (smaller mandible) followed by a return to growth (slower) and a more posteriorly positioned condyle-fossa position. The lack of dentoalveolar retrusion may indicate an individual with a genetic type for decreased compliance/bone modeling.

If there is the possibility of modeling the articular eminence, then it could be possible to reduce the risk of anterior disc displacement.[26,31,55,56] The consideration of risk is important. TMJ adaptation to late adolescent treatment should mimic the observations of individuals with unilateral condylar fractures.[57] 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).[57] This is similar to the long term findings with progressive condylar resorption after orthognathic surgery.[58]

Transverse relationships are as important as the sagittal overjet and overbite. For example, the Class II individuals with a large overjet would display protrusive guidance on the mesial inclines of mandibular buccal cusps and the distal inclines of maxillary buccal cusps due to the relative constriction of the maxilla. Laterotrusive guidance, through contact of the mandibular buccal incline of the buccal cusps (if a premolar not the canine) and the maxillary lingual incline of the buccal cusps, would be the same if not greater vertically compared with canine guidance, due to the “wedge effect”.[59] Even though the molar or premolar buccal cusp may be 1mm shorter than a canine, the distance from the condyle influences the Class III lever system. Laterotrusive movement of this nature would create a fossa with smaller medial and possibly lateral dimesions due to the lever action of the mandible in laterotrusive movement. On left laterotrusive, the canine region would be expected to move a greater distance left, than the premolar region. In children, this is not an issue as permanent teeth (molars) occlude more tightly with each other than do those of the deciduous dentition due to cusp heights. [60]

Skeletal divergency as related to the gonial angle must be considered since a large gonial angle is related to a shallow articular eminence, while the opposite is also true, and a steep eminence is related to a steep articular eminence.[12] When a condyle is positioned more anteriorly in the glenoid fossa, the fossa will model and become more shallow[12,61] and therefore, repositioning with functional appliances may be of clinical value although effects would not be evident, on average, for 2 years.

Consequences of the Hypothesis

The recent thoughts about occlusion and the shift in functional appliance philosophy may need to be revisited prior to acceptance as axiomatic. It is recommended that the functional aspect of the teeth be viewed as a possible controlling factor in

TMJ ontogeny, which in cases, may influence a clinician toward early treatment. It is quite possible that there is a genetic basis for the occlusal variation seen with TMD which is exacerbated by our contemporary diet and the associated lack of "normal" attrition. Botox treatments may also be of some value to control facial somatotypes and guide TMJ development. The understanding of genetic factors would create research opportunities to create a test for genetic haplotype for TMD.

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