

On the Etiology and Significance of Palatal and Mandibular Tori

A recent review by Garcia, et al.¹ was most interesting and timely. I would like to comment on only one aspect, namely, the etiology of torus palatinus and torus mandibularis. Nearly all researchers in this field agree that the underlying cause of tori remains unresolved. Here, I posit a possible mechanism for the formation of maxillary and mandibular tori, including buccal exostoses. These predictions emanate from the Spatial Matrix Hypothesis.²⁻⁴

First, put simply, the Spatial Matrix Hypothesis suggests that gene-environmental interactions proceed as developmental events unfold even in a perturbed functional space (a matrix that has departed from the ideal temporo-spatial pattern encoded at the genomic level). Second, the spatial matrix hypothesis might explain the occurrence of the symptoms of TMD, which often includes clenching, bruxism, grinding and tooth wear (facets). We believe that these parafunctional habits are secondary to, or in some way compensatory for, a compromised upper airway space,^{2,5,6} which may have other presenting symptoms, such as morning headaches, snoring and obstructive sleep apnea at night, etc. Preliminary evidence of this behavioral response is beginning to emerge (Simmons and Prehn,⁷ Abstract 0668) supporting this view. Note that Drs. Prehn and Simmons⁵ suggest in Abstract P6 that they are the first to postulate that nocturnal bruxism is a compensatory mechanism of the upper airway to protect it from collapsing. In actual fact, that notion was first suggested by Singh in 2007,² following an earlier study by Singh and Olmos⁶ that was the first to associate upper airway compromise in patients presenting with temporomandibular dysfunction (TMD). The grinding and clenching activities may be an ineffectual attempt to relieve airway compromise,² in that the muscles of mastication may be acting antagonistically to the constrictors of the pharynx in this scenario. Now, let's look at a possible mechanism for the formation of maxillary and mandibular tori, including buccal exostoses given the above conditions.

During clenching, bruxism, etc., muscular forces are applied periodically to the dentition, which may present

as tooth wear (facets) over a period of time. However, tooth wear may not be obvious in all cases, depending on the qualitative and quantitative aspects of an individual's enamel, and the bone morphology that accommodates the teeth. Perhaps this latter element, the bone deformation that occurs during bruxing, has been overlooked in this scenario.

According to Proffitt,⁸ heavy forces of short duration (<50kg <1s) occur during masticatory activities, and are largely absorbed by the incompressible fluid components of the periodontium. This process results in no orthodontic tooth movement but bending of the collagen and alveolar bone can elicit a piezoelectric effect. I posit that both the maxilla and mandible undergo similar bone deformation during parafunctional activities, such as nocturnal bruxism, with the muscles of mastication acting antagonistically to the pharyngeal constrictors during airway obstruction, and the ensuing stress and stretch of the osteogenic periosteum eventually lead to bone deposition in the form of tori in a site-specific fashion. Note this present comment modifies the notion of DuBrul and Sicher⁹ that loading the jaw heavily during forceful chewing precipitates the formation of bone. However, Drs. DuBrul and Sicher did not mention signal transduction as a possible developmental mechanism in the formation of tori via the differentiation of stem cells—likely because Drs. Sicher and DuBrul were unable to exploit recent advances in molecular genetics and molecular biology. Moreover, they did refer to the putative relationship between tori and sleep disordered breathing.

Looking first at the maxilla, the midpalatal suture remains patent until at least the third decade¹⁰ and probably beyond that into later life. Repetitive, compressive stresses may lead to buckling of the maxilla about the midline. The osteogenic periosteum of the palatal vault (the midpalatal suture) would be stretched intermittently, and this tension would lead to new bone formation localized to the midline, being the epicenter of force distribution, and thereby precipitating the torus palatinus. This osteogenic model is in accord with the notion of

sutural homeostasis,⁶ and follows also from the functional matrix hypothesis.¹¹ Thus, undifferentiated stem cells would undergo mechanotransduction and differentiate locally into osteoblasts. However, if the vectors of force distribution were changed, even in the presence of bruxing, clenching, etc., this osteogenic-periosteal stretch hypothesis would predict an absence of midline palatal torus but a bulbous maxillary tuberosity, secondary to repetitive postero-inferiorly directed compressive stresses, because of altered deformations of the maxilla and site-specific osteoblastic cytodifferentiation from stem cells. Conversely, if the vectors of force acted buccally instead of palatally, buccal exostoses may be formed with the osteogenic periosteum of the buccal plate depositing new bone in focal areas of force dissemination and signal transduction by mechanoreceptors. These ideas could be further verified by fractal analysis and molecular biology techniques.

Despite the above contentions, the osteogenic-periosteal stretch hypothesis needs to be able to explain the formation of mandibular tori. Indeed, mandibular tori may present even in the absence of a torus palatinus, and these mandibular tori are most often found bilaterally in the premolar region on the medial aspect of the mandible. This observation can also be explained on the basis of the osteogenic-periosteal stretch hypothesis. First, the morphology of the mandible permits its body (corpus) to bend more easily in the mental foramen region, which has a reduced bone volume locally, due to the neurovascular bundle emanating from the mental foramen. The body of the mandible does not have any additional, robust, supporting structure anterior to the mylohyoid line. The same cannot be said of the ramus, which is enclosed by the muscles of mastication and oriented differently. However, it is understood that parafunctional activity, such as nocturnal bruxism, can lead to hypertrophy both of the masseter and its bony attachment, the angle of the mandible, precipitating an antegonial notch—a finding that is often associated with patients who present with symptoms of TMD. These consequences may also be explained by the functional matrix hypothesis.¹¹ However, the functional matrix hypothesis cannot easily explain the localization of tori in the mandibular bicuspid region. According to the osteogenic-periosteal stretch hypothesis, the chin is pre-

vented from undergoing excessive deformation due to the mental process. Humans lack the simian shelf but have instead developed an external chin to strengthen the weakest part of mandible. Thus, the morphology of the mandible localizes torus formation to the premolar region as the mandibular body buckles medially due to a combination of muscular compression and tooth orientation directed by the maxilla. With the teeth in occlusion, the buccal overjet and curve of Spee will ensure that the mandibular dentition is bent inwards (medially). The corollary of this conjecture is that if teeth are missing or extracted there would be less force available in that region and should produce smaller deformations and, consequently, smaller or absent tori on the tooth deficient side. Indeed, the presence of a unilateral mandibular torus would be proof positive of the veracity of this developmental mechanism, and preliminary data has been collected by the author in this regard (**Figure 1**).



Figure 1

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Therefore, generally speaking, smaller deformations should produce smaller palatal tori with concomitantly smaller mandibular tori. Furthermore, in a case of Angle's Class III malocclusion, the direction of vectors would be changed; thus mandibular tori may not be prominent at all in some cases.

Patients with buccal exostoses likely develop these features secondary to nocturnal bruxism associated with disordered sleep breathing. In other words they have been grinding/clenching the teeth and will probably show signs of tooth wear. Ideally, these patients should have sleep studies done to rule out obstructive sleep apnea. Despite the above contentions, evidence for the osteogenic-periosteal stretch hypothesis of torus formation is currently lacking; however, it can be tested by using various analytical techniques, such as finite-element analysis, 3-D modeling,¹² fractal analysis and molecular biology. In addition, tori, like many clinical features, exhibit complexity. This complexity is both structural (in terms of the clinical presentation of the abnormalities) but also statistical—in the sense that it is virtually impossible to predict the quantity and localization of tori prior to their development. It is important to understand the notion of complexity here, which is an extension of the chaos theory, i.e., the deterministic nature of these systems does not make them easily predictable. Nevertheless, this osteogenic-periosteal stretch hypothesis takes genetic/familial predisposition into account, as children inherit jaw form from parents, and the osteogenic-periosteal stretch hypothesis predicts that tori should be preventable if the perturbed spatial matrices of the orofacial system are diagnosed early and cor-

rected appropriately. Clinically, children should be screened for wear facets in the deciduous dentition and, if observed, referral to a sleep physician should be considered to rule out covert or overt obstructive sleep apnea. Similarly, in adults, it is likely that palatal and mandibular tori are manifestations of undiagnosed sleep disordered breathing, and may represent a valuable diagnostic sign in the triad of TMD, sleep disordered breathing and malocclusions.

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