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The five types of alveolar open bites

Abstract

Anterior open bites present many challenges in clinical dentistry and require careful evaluation and treatment planning. Yet unsubstantiated theories of open bite development are erroneously used to make non biological diagnoses which provide limited treatment planning utility and misdirected orthodontic treatment. This paper identifies characteristics of alveolar bone morphology, incidence of canine intercuspation, intra-arch canine orientation, degree of dental crowding or dental spacing and degree of open bite to introduce five types of dentoalveolar open bites. These proposed categories of alveolar open bites create a straightforward and more predictable clinical model for planning and treatment.

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Introduction

The origin of open bites is divided into two categories, namely

i) Basal bone related or Skeletal.

ii) Alveolar bone related or Dentoalveolar.

Skeletal open bite reflects underlying skeletal issues which include degenerative joint disease with the hallmark presentation of its progressive nature especially when monitoring the patient over a longer period of time. The increasing severity of maxillary/mandibular discrepancies in skeletal open bites is consistent with the increasing severity of the associated degenerative joint disease. Other skeletal issues include aberrant skeletal development or congenital Class II or Class III skeletal mal relationships along with a correlation between a weakened musculature and a long face anterior bite pattern.[1–3] Other causative factors include airway obstruction, muscular dystrophy, neurological disturbances, and hereditary factors.[4,5] One very strong clinical clue that the authors believe to be very important in differentiating between a Skeletal and Alveolar open bite is the presence of posterior molar occlusion. Clinical observation of numerous cases over the past thirty years leads the authors to conclude that in the absence of first molar occlusion or minimal first molar occlusion strongly points to a Skeletal open bite. However, if first molars are in contact without facial asymmetry the clinician can or should strongly consider a dentoalveloar origin. Skeletal open bites lack molar contacts and they appear larger than Alveolar open bites and can exceed 8-10 mm.

Dentaoalveolar anterior open bites remain some of the most challenging and misunderstood case presentations in dentistry. They are defined by the lack of anterior tooth contact and are characterized by a negative overbite or lack of a proper overbite relation of maxillary and mandibular incisors with molar teeth in occlusion. Some clinical manifestations include increased mandibular facial height, lip incompetence, incisal labial inclination and profile convexity.[6,7] The prevalence of an anterior open bite varies with age and among ethnic groups and ranges from 1 to 11.5%.[8–10] The etiology of dentoalveolar open bite is complex and multifactorial and its overall etiology remains largely uncertain with numerous theories of development that include non-nutritive digital/thumb sucking, nail biting, lip biting and mouth breathing. Even with this overall uncertain etiology the dental professional continues to adhere to these and other unsubstantiated theories of development especially when diagnosing and treating dentoalveolar open bites. For example, one of the most debated theories of open bite development particularly in the classic literature and with a reported wide variation in prevalence is tongue thrust swallowing. Tongue thrust is considered a normal physiological manifestation of suckling and also occurs in transitional dentition but typically disappears with the establishment of a normal anterior overbite.[11,12] The tongue thrust diagnosis is still prevalent as a cause of anterior open bites and treatment is directed towards closure of the associated anterior open bite frequently with surgical intervention to reposition the maxilla and mandible with adjunctive treatment involving tongue re-education.[13,14] However, there is evidence to suggest that tongue thrust swallowing is an adaptive mechanism to an open bite in order to maintain an anterior seal rather than its cause.[14,15] In other words, we need to appreciate the unnatural appearance of the alveolar bone in the vertical dimension.

Hypoplasia is characterized by the malformation of or inadequate alveolar bone development because of the lingually erupted mal-aligned teeth. Hypoplasia may create a Dental Open Bite but without occlusal contact or intercuspation of maxillary and mandibular canines. Hyperplasia is characterized by the excess alveolar bone arch development due to the protruded eruption of teeth resulting in spacing. It also may create a Dental Open Bite with maxillary and mandibular canine intercuspation. The hallmark reproducible clinical finding that differentiates Hypoplasia from Hyperplasia in diagnosing Alveolar open bites is the presence or absence of maxillary and mandibular intercuspation/contact of canines. In Hypoplasia cases canines do not contact while there is contact of canines in Hyperplasia cases. Had it been the tongue that caused these open bites and not the alveolar bone growth then the canine intercuspation would have been similar in both instances. Simply stated, clinicians are erroneously looking at the tongue when they should be looking at the alveolar bone.

The disproven tongue thrust theory serves as an example of how an unsubstantiated theory of etiology is used to make arbitrary, non-biological diagnoses with subsequent misdirected orthodontic and even surgical treatment plans. It is therefore important to start with the premise that the dental or alveolar (i.e. non-skeletal) open bite is caused by inadequate (hypoplasia) or excess (hyperplasia) alveolar

bone formation. Although researchers have made significant efforts to identify etiological factors, little has been done to identify and classify specific types of non-skeletal anterior open bites. Indeed, the broad diagnostic term of open bite is vague and has limited utility in orthodontic treatment planning. The authors of this paper identify five different types of open bites and present suggested treatment protocols. We believe this new classification system will provide the clinician with valuable diagnostic information to formulate predictable treatment plans.

Methodology

Diagnostic parameters

The authors feel that the evaluation and observation of the following clinical parameters of alveolar bone hypo or hyperplasia is necessary to formulate the five different types of Alveolar open bites. Treatment for these five different types of Alveolar open bites includes following the protocols of the Fastbraces Technologies® Bracket Systems. The treatment for all Alveolar open bites is completed when the maxillary laterals overlap the corresponding mandibular dentition by 2mm.

Five types of open bites and treatment protocols

1. Type 1 Alveolar Open Bite: Hypoplasia with minor to moderate crowding of the teeth (Figure 1A). Treatment for Type 1 requires only elastics and no interproximal reduction (IPR) (Figure 1B).
2. Type 2 Alveolar Open Bite: Hypoplasia with severe crowding of the teeth resulting in blocked out canines (Figure 2A). Treatment for Type 2 requires elastics along with interproximal reduction (IPR) from molar to molar (Figure 2B).
3. Type 3 Alveolar Open Bite: Hyperplasia with minor incisor proclination defined as 2 degrees more than normal and some spacing with an anterior open bite no larger than 2mm. (Figure 3A) Treatment for Type 3 requires elastics along with interproximal reduction (IPR) from canine to canine (Figure 3B).
4. Type 4 Alveolar Open Bite: Hyperplasia with moderate incisor proclination defined as 3 to 6 degrees more than normal and some spacing with an anterior open bite between 3mm to 6mm (Figure 4A). Treatment for Type 4 requires elastics along with interproximal reduction (IPR) from molar to molar (Figure 4B).
5. Type 5 Alveolar Open Bite: Hyperplasia with severe incisor proclination defined as greater than 6 degrees more than normal and some spacing with an anterior open bite greater than 6mm but less than 8-10 mm (Figure 5A). Treatment for Type 5 requires elastics along with interproximal reduction (IPR) from molar to molar twice (Figure 5B).

In the formulation of the three types of Hyperplasia diagnoses and as illustrated in Figure 6 it is logical to infer that as the degree of proclination of anterior teeth increases so does the vertical space. As the proclination and the anterior vertical intermaxillary space of the open bite increases so does the scope and frequency of IPR in the treatment phase.

Figure 1 (A) Type 1 Alveolar Open Bite: Hypoplasia with canines not blocked out.

Figure 1 (B) Post-treatment of Type 1 after braces and elastics.

Figure 2 (A) Type 2 Alveolar open bite: Hypoplasia with canines blocked out.

Figure 2 (B) Post-treatment of Type 2 after braces, elastics and IPR molar to molar.

Figure 3 (A) Type 3 Alveolar Open Bite: Hyperplasia with minor opening.

Figure 3 (B) Post-treatment of Type 3 after braces, elastics and IPR canine to canine.

Figure 4 (A) Type 4 Alveolar Open Bite: Hyperplasia with moderate opening.

Figure 4 (B) Post-treatment of Type 4 after braces, elastics and IPR molar to molar.

Figure 5 (A) Type 5 Alveolar Open Bite: Hyperplasia with severe opening.

Figure 5 (B) Post-treatment of Type 5 after braces, elastics and IPR molar to molar twice.

Discussion

Placing all types of open bites in a single category implies a lack of understanding and creates limitations in treatment planning. Traditional orthodontic systems greatly influence treatment planning towards a combination of mechanotherapy and surgical orthodontics for a severe anterior open bite. Many patients wish to forgo the risks and possible complications of surgical treatment and opt for a non-surgical solution which is more difficult especially for long term stability and retention. Most often traditional orthodontic therapy in these cases will require dental extractions and high-pull headgear to aid in bite closure and intrusion of maxillary molars, respectively. Complicating matters is the adherence to Angle's arbitrary diagnostic classifications of Class I, II and III which compels the clinician to change mandibular position and functional occlusion in order to achieve a morphologic occlusion that conforms to the arbitrary ideal of Class I.

In 2014 the authors introduced a disease-based or biologically verifiable model of orthodontic diagnosis based on alveolar bone clinical morphology rather than subjective arbitrary "ideals".[16] The disease-based term Orthodontosis(TM) was introduced and defined as the non-inflammatory deficiency of the alveolar bone in the axial plane caused by the displaced root(s) of the tooth, typically palatally or lingually. The soft tissue consequence or resulting excess soft tissue and chronic inflammation leads to the diagnostic term of Orthodontitis(TM). These terms serve as the basis of the cause or illness and disease in orthodontics. In 2017 the authors created the first set of classification terms which capture our disease - based model and identify Alveolar Hypoplasia and Alveolar Hyperplasia in both the maxilla and mandible.[17] These observations led to the formulation and introduction of The Viazis Classification of Malocclusion.[18] This orthodontic classification is appropriately based on the alveolar bone morphology and was proposed as the new system for orthodontic classification as the valid replacement of Angle's invalid thesis.

The Viazis Classification system is based on the morphology of alveolar bone by identifying the pre-treatment morphologic status of alveolar bone with the ultimate orthodontic goal of creating a natural dental arch morphology with proper and natural orientation of tooth roots and with a stable occlusion irrespective of an Angle molar classification. The treatment of Alveolar Hypoplasia or

Alveolar Hyperplasia results in the establishment of the natural dental arch similar or identical to what would have resulted from the perfect/natural eruption of straight teeth. The authors believe that this fundamental treatment goal is achieved through orthodontic mechanotherapy that is best described as the facilitation of natural eruption or Orthoeruption(TM). In the case of Alveolar Hypoplasia this process induces alveolar bone remodeling and development by moving roots toward their final naturally erupted position from the beginning of treatment. In the case of Alveolar Hyperplasia, (maxillary or mandibular) treatment follows a similar sequence of attaching brackets and wires to the teeth exhibiting clinical spacing and closing of these spaces with elastic power chains. The restoration of the alveolar bone toward the establishment of a patient -specific natural dental arch also treats associated soft tissue pathology or Orthodontitis(TM) around malpositioned teeth. This is achieved with the light forces provided by following the protocols of the Fastbraces Technologies® Bracket Systems with intermaxillary elastics and relevant interproximal reduction (IPR) as needed based on the severity of the open bite.

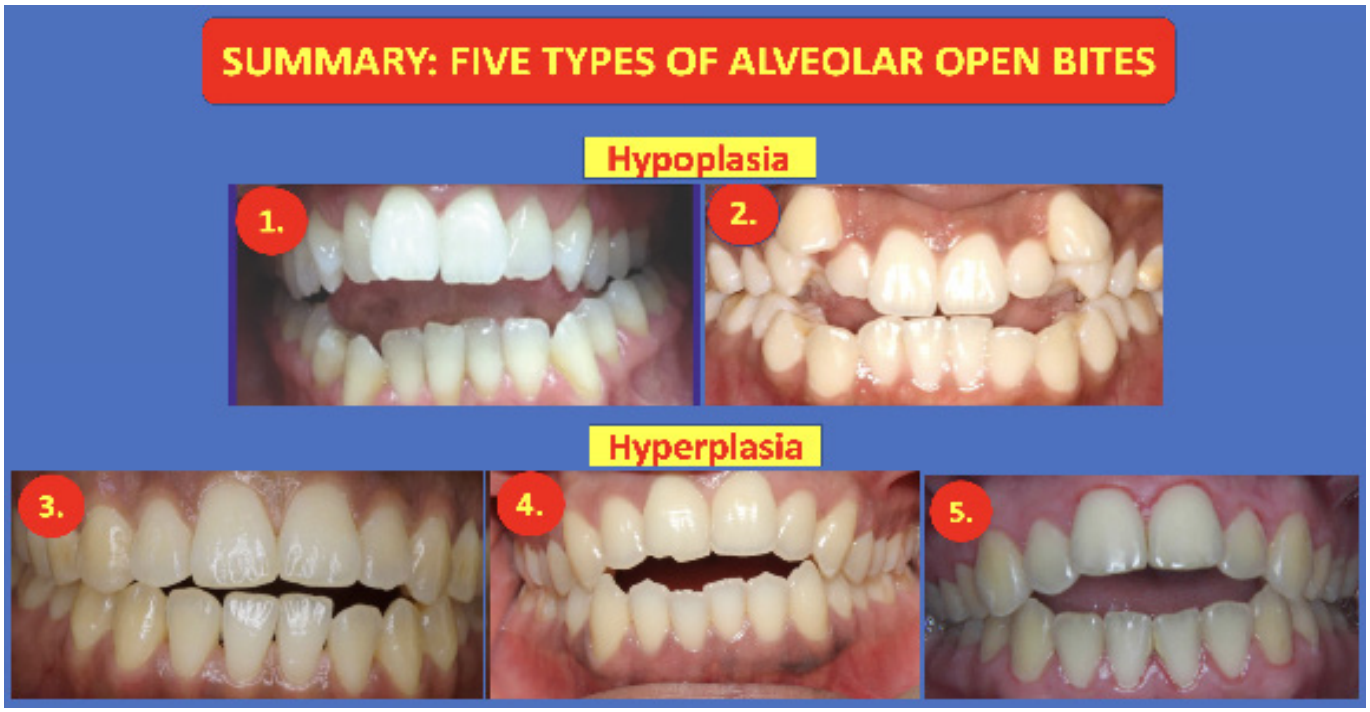

Figure 6 Summary - The Five Types of Alveolar Open Bites.

Evaluating the referenced diagnostic criteria of alveolar hypoplasia, alveolar hyperplasia, canine contact and degree of the anterior open bite space leads to what the authors believe are valuable diagnoses that corroborate clinical presentations of various open bites and lead to predictable treatment planning. As the authors consider the multifactorial nature of open bites and particularly non skeletal open bites, we theorize that the pathogenesis of our new open bite classifications is largely influenced by patient intrinsic forces – specifically, the degree of lip tightness and incompetence. The authors believe that the significance of lip incompetence in the development of Alveolar open bites is underappreciated.

The facial anatomy around the mouth is complex and includes the *orbicularis oris* or the circular muscle of the mouth which forms the underlying muscular basis of the upper and lower lips. In addition, the *orbicularis oris* is attached to at least four other facial muscles and functions to narrow the mouth, close the mouth and participates in chewing and sucking.[19] Lip incompetence or the inability to form a seal at rest is very common and even normal in children.[20,21] However, lip incompetence becomes a more significant issue in early adulthood particularly in patients that have to actively strain to form a seal. This lack of equilibrium or inharmony of lip incompetence can significantly affect tooth position with crowding and an unfavorable facial relationship. In addition, and as a result of an unfavorable lip seal, mouth breathing function can worsen the clinical presentation. The obvious lack of canine intercuspation provides proof.

While researchers and clinicians have identified and continue to evaluate the clinical manifestations and prevalence of open bites there is an ongoing adherence to unsubstantiated theories of open bite development. This information creates non biological diagnoses with misdirected treatment planning and orthodontic treatment. In addition, this information is not translated into the development of specific diagnostic parameters towards the categorization of distinct forms or classifications of open bites.

The authors propose and introduce five types of open bites which incorporate specific clinical parameters of alveolar bone morphology, evidence of canine intercuspation, intra-arch canine orientation and degree of lip incompetence towards biologically bases diagnoses. These categories create a straightforward clinical model for planning and treatment (Figure 6).

The creation of this novel classification of open bites with accompanying treatment recommendations is a logical extension of

the author's six year plus efforts to bring orthodontic diagnosis and treatment protocols in line with biologically based criteria.

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