Anterior Open Bite: A Review of Epidemiology, Aetiology and Management.

***Agbaje HO[a], Osiatuma VI[b], Fadeju AD[c], Kolawole KA[d], Otuyemi OD[e].**

Abstract

Anterior open bite is a vertical occlusal discrepancy with varied characterizations in the literature. The aetiology, which is multifactorial, includes oral habits, unfavourable growth patterns, and enlarged lymphatic tissue with associated mouth breathing. Its prevalence varies amongst the world's population with great preponderance towards African racial group. Several treatment options have been proposed to correct this malocclusion trait. However, proper differentiation is essential in determining the appropriate corrective measures. Dental open bites are generally more responsive to treatment with orthodontics alone, whereas skeletal open bites often require a combination of treatment techniques including orthodontics and orthognathic surgery.The functional and aesthetic challenges of this malocclusion trait cannot be overemphasized.

Keywords: **Anterior Open Bite, Epidemiology, Aetiology Management**

Authors Affiliations:

[a]Senior Resident, Orthodontics Unit, Department of Child Dental Health, Obafemi Awolowo University Teaching Hospitals, Ile-Ife, Nigeria.

[b]Senior Resident, Orthodontics Unit, Department of Child Dental Health, Obafemi Awolowo University Teaching Hospitals, Ile-Ife, Nigeria.

[c]Lecturer I, Department of Child Dental Health, Obafemi Awolowo University, Ile-Ife, Nigeria.

[d]Senior Lecturer, Department of Child Dental Health, Obafemi Awolowo University, Ile-Ife, Nigeria.

[E]Professor, Department of Child Dental Health, Obafemi Awolowo University, Ile-Ife, Nigeria.

Background

Anterior open bite was defined by Subtelny and Sakuda,[1] as an open vertical dimension between the incisal edges of the maxillary and mandibular anterior teeth. Mizrahi[2] described anterior open bite as a vertical discrepancy where upper incisor crowns fail to overlap the incisal third of the lower incisor crowns when the mandible is brought into full occlusion. Characterizations of open bites are varied in the literature. Chase[3] described open bite as a condition characterized by space discrepancies between the occlusal and incisal surfaces of the maxillary and mandibular teeth when the mandible is brought into habitual or centric occlusion. According to Ferguson[4] an anterior open bite is the absence of vertical incisor overlap between the maxillary and mandibular incisors, whereas an incomplete overbite has vertical overlap, but without tooth or tissue contact in the anterior segment. He also noted that, in the United States, both conditions have been referred to as anterior open bite. However, with a true anterior open bite, it is impossible to achieve incisor contact, whereas this can sometimes be accomplished if the mandible is protruded in patients with incomplete overbite. Otuyemi and Noar[5] described anterior openbite as a relationship that exists when there is no incisor contact and vertical overlap of lower incisors by the uppers. It may occur with an underlying class 1, class 2 or class 3 skeletal patterns.

The prevalence of anterior open bite varies with ethnicity and race in which the condition can be a major clinical challenge in management. Various studies have been reported on the causes, treatment options and stability of treatment.

This review will throw more light on the various treatment options in the management of anterior open bite.

The aim of the study therefore was to review the existing literature on the prevalence, aetiology, and treatment options available for Anterior Open Bite (AOB).

Epidemiology

There is a general variation in AOB among the world's population with great tendency towards racial predilection[5]. The prevalence of AOB ranges from 1.5% to 11% among various age and ethnic groups, and it has been shown that approximately 17% of orthodontic patients in the United States have AOB[6]. The prevalence ranges from 17% to 18% of children in the mixed dentition[7, 8]. Cozza[8] reported that the prevalence of AOB increases to 36.3% in the mixed dentition in patients with prolonged sucking habits and hyperdivergent facial characteristics.In the

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UK the reported incidence in adults is about 4%and in children is 2-4%[9], falling from the age of nine to the early teens. This reduction is accounted for by normal occlusal development, neural maturation of the child favouring cessation of oral habits, and decrease in size of the adenoids and the establishment of a normal adultswallowing pattern. The incidence then increases again during the mid-teens presumably as a consequence of late vertical growth. It is more common in Africans and Afro-Caribbeans with a prevalence of between 5 and 10%. In a study by Kelly et al[10] the prevalence of open bite in US children was reported as 3.5% in the white population and 16.5% in the black population. Proffit et al[11] also recorded a prevalence of approximately 3.5% in patients from 8 to 17 years of age. De Muniz[12] reported 2% in Argentinean children. Al- Emran[13] found 6.6% in Saudi Arabian adolescentswhile Behbehani[14]found 3.5% in Kuwaitis. The middle-east studies are however similar to the reported Pakistani prevalence of 4%[15]. Tang[16] reported 1.5% prevalence in Hong Kong.The prevalence of AOB in the Nigerian studies[17-20] have been variously recorded as 7.0-10.2%. This is however similar to studies reported in other parts of the sub-sahara Africa[21-23]. Coetzee and Wiltshire[23]reported 10.3% prevalence in black South Africans. According to Bataringaya[24]the frequency of open bite in the Ugandan population ranged from 1-4%, which is relatively low and not consistent with other African studies. The reason for this may need further investigation.

Aetiology

The cause of an AOB is generally multifactorial and can be due to a combination of skeletal, dental and soft tissue effects.It occurs due to interaction of many causes, which include hereditary and environmental factors. Environmental factors include variations in dental eruption and alveolar growth; disproportionate neuromuscular growth or aberrant neuromuscular function related to malfunctions of the tongue or oral habits or both.Many potential aetiological factors (environmental and hereditary) have been considered, including unfavourable growthpatterns,[25,26] digit-sucking habits,[1,2]enlarged lymphatic tissue,[1] heredity[2,27] and oral functional matrices[28].

AOB can be broadly classified into two categories:

1. Dental open bite the vertical skeletal pattern is not contributory (Fig. 1).
2. Skeletal open bite the open bite is at least partly due to the vertical facial form (Fig. 2).

These can be further divided into:

1.) Dental Open Bite (Digit Sucking Habits)

Prolonged Non Nutritive Sucking (NNS), a term described by Johnson and Larson[29]to describe habits that involve digits, pacifiers, and other environmental factors, beyond the age of 3 years is the major cause of AOB in children. The interaction between an AOB and NNS habits, e.g. thumb or dummy sucking, is clear[30].

The open bite caused by NNS habits is frequently asymmetrical, being greater on the side where the digit or dummy is inserted. The thumb or finger effectively acts as a barrier to the erupting incisors, whilst allowing excessive eruption of posterior teeth. Children who digit suck for 6 hours or more each day, particularly those who sleep with a digit between the teeth all night, can develop a significant AOB. Generally, any force that impedes the eruption in the incisal region can also result in dental AOB.

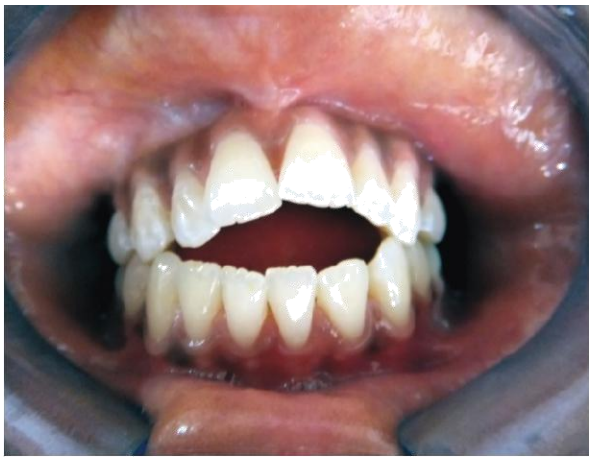

Fig. 1 Anterior open bite caused by Digit Sucking Habit in a 21-year-old female patient.

2.) Skeletal Open Bite

Open bites that develop due to excessive vertical growth are termed 'skeletal open bites'. These are usually more severe than dental open bites, often with only the terminal molars in contact. Hellman[31] , using anthropologic measurements found that subjects with open bite had shorter rami and greater total facial height. In another study by Schudy[32], clockwise rotation of the mandible was found to be a result of excessive vertical growth as it relates to horizontal growth. This kind of growth pattern occurs when vertical growth in the molar region is greater than growth at the condyle.[33] Genetic and environmental influences that encourage vertical growth in the molar region, which are not compensated by growth at the condyle or posterior ramus, will result in skeletal AOB.[34] Adverse functional activities such as mouth breathing may affect the facial architecture and enhance the development of open-bite[35].

Development of a skeletal open bite has been attributed to lack of coordination between condylar-glenoid fossa growth (horizontal factor) and vertical growth of the maxilla and the dentoalveolar processes (vertical factor). Increased expression of vertical factors rather than horizontal factors results in clockwise rotation of the mandible, leading to skeletal AOB.[32,34]

3.) Lip and Tongue Habits

Tongue thrusting has been postulated to be another cause of AOB.[36,37] Tulley[38] classified tongue thrusting as an endogenous habit or as an adaptive behaviour based largely on facial morphology and swallowing activity.The tongue is thrust forward on swallowing as an adaptive response to the presence of an AOB to prevent food/liquid/saliva escaping from the anterior part of the mouth.[39] Proffit[40] suggested that the resting position of the tongue has much greater influence on tooth position than any tongue thrust, as the duration of any thrusting activity would be too short to have a significant effect. Endogenous tongue thrust is often associated with excessive circumoral contraction on swallowing. Proffit and Mason[41] suggested that therapy for anterior tongue thrusting is not warranted with or without malocclusion before adolescence. Further, tongue therapy is most effective when combined with orthodontic treatment.

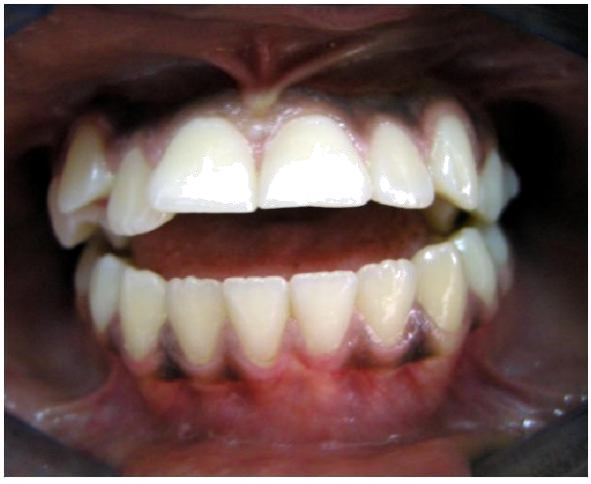

Fig 2a. Frontal view of skeletal open bite in a 15-year old female patient

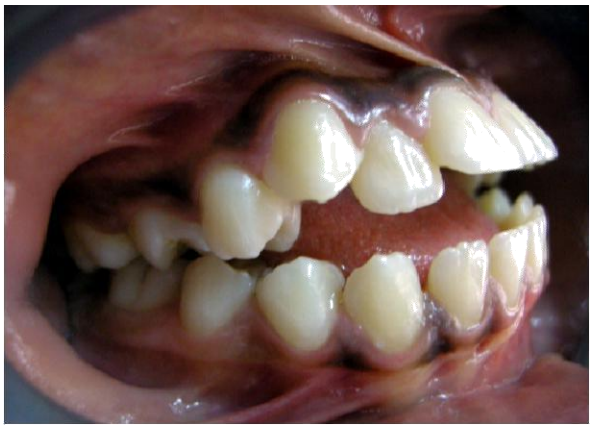

Fig. 2b Lateral View of Skeletal Open Bite in a 15-year-old female patient.

Some studies[35,42] found a correlation between orofacial musculature and facial structure suggesting a relationship between weak musculature and a long face and between tongue position and AOB pattern. Persistence of open bite is probably associated with neuromuscular imbalance or divergent growth pattern, although this is not well understood.

4.) Airway Obstruction

Some researchers[43-46]have found that mouth breathing has an effect on the facial characteristics by increasing the vertical pattern of facial growth, open bites and crossbites. Data from a longitudinal study[47] indicate that the effects of the mode of

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breathing on facial morphology are unsupported. Individuals with narrow airways and craniofacial pattern may have increased risk for obstructive sleep apnoea.[48]However, the interactions between oral breathing, maxillofacial growth and clinical symptoms associated with sleep-related breathing disorders are not clearly understood.

5.) Neurological Disturbances

Neurological disturbances that affect the oral or facial musculature may give rise to AOB. Gershater[49] reported an incidence of 32.3% in patients with learning disabilities . Carmagnani[50]reported a 64% incidence of AOB and an increased overjet in patients with the spastic form of cerebral palsy. A low level was however observed in the ataxic group. It is however not clear if neurological injuries can be the cause of changes of the craniofacial growth and development patterns resulting in malocclusion in cerebral palsy patients. Miamoto et al[51], in their study, however concluded that the main risk factors associated with the severity of malocclusion were cerebral palsy, mouth breathing, lip incompetence, and long face.

6.) Muscular Dystrophy

Vertical dentofacial aberrations have been observed in patients with reduced muscle function, as can be found in myotonic dystrophy.[52] Similarly, a high prevalence of AOB has been noted in Duchenne muscular dystrophy (DMD) patients, which appear to be strongly related to the involvement of the orofacial muscles in the disease.[53,54] Progressive muscular dystrophy, or Duchenne muscular dystrophy (DMD), is a recessive neuromuscular disease affecting the short arm of the X chromosome in the p21-2 position[55]and has a prevalence of 1:3500 male newborn.The decrease in tonic muscle activity that occurs in muscular dystrophy allows the mandible to rotate downwards away from the rest of the facial skeleton.[56] This results in increased anterior facial height, a posterior growth rotation of the mandible, excessive eruption of the posterior teeth, narrowing of the maxillary arch and AOB that worsens with growth.

7.) Iatrogenic Open Bite

Poor mechanics during fixed-appliance treatment may cause extrusion of the molar teeth or intrusion of the incisors during overbite correction, which open the bite. Failing to prevent overeruption of second molars when bite planes or functional appliances are used may also give rise to an AOB.[57]

8.) Pathological Open Bite

Localized AOB may be associated with cleft lip and palate, acromegaly or trauma to the facial skeleton, such as mal-union of condylar and bilateral fractures of the body of the mandible.[58,59]

Clinical Features/ Diagnosis

The importance of making a clinical distinction between the types of AOB traits becomes evident when formulating a treatment plan. This clinical decision can benefit from the use of cephalometric analysis.It must be decided if the AOB is a true skeletal dysplasia or a habitual problem involving only the dentoalveolar structures as this will aid in possible prevention or early treatment, and will ensure that the mechanics utilized will not worsen the condition.[60]

AOB may occur as simple or complex .[61]Simple AOBs are characterized by vertical separation of anterior teeth, extending up to premolars, whereas in complex AOB, the vertical separation extends right up to the molars. Based on severity, vertical separation of 0-2 mm is moderate, 3-4mm is severe and more than 4mm is considered extreme[2].

Normal facial proportions with or without a history of parafunctional habits, proclined incisors, undererupted anterior teeth, normal or slightly excessive molar height, and thumb or finger sucking habit characterize a dental open bite. The open bite is generally found in the anterior region within the area of the cuspids and incisors. This type can be treated efficiently by only orthodontic means.
A skeletal open bite is usually characterized by vertical maxillary excess, excessive eruption of posterior teeth, downward rotation of the

mandible, and normal or excessive eruption of anterior teeth. The Frankfort Mandibular Plane Angle (FMPA) is usually increased.

Generally, a dental open bite has a better prognosis than a skeletal open bite.

The characteristics of individuals with a skeletal open bite include one or more of the following: excessive gonial, mandibular and occlusal plane angles, small mandibular body and ramus, increased lower anterior facial height, decreased upper anterior facial height, retrusive mandible, increased anterior and decreased posterior facial height, class II tendency, divergent cephalometric planes, steep anterior cranial base,[62]and inadequate lip seal.[25]

Cephalometric Findings

There is no consensus on cephalometric criteria for determining the presence of open bites. Dental AOB do not show any distinguishing features. Skeletal open bites show increased lower anterior facial height as compared to posterior facial height, steep mandibular plane, obtuse gonial angle, and increased maxillary posterior dentoalveolar height. There is increase in the angle between the Sella-Nasion (S-N) plane and the occlusal plane. In addition, cephalometric studies of individuals with obstructive sleep apnoea[63]and mouth breathing,[64] have found a characteristic cephalometric pattern, which includes: long face and increased lower anterior facial height suggesting a hyper divergent pattern of skeletal open bite.[65] Cangialosi[60] in the "classical study" suggested that patients with AOB had increased posterior-to-anterior facial height and upper-to-lower facial height ratios. In addition, the S-N -to-mandibular plane, the gonial and the maxillary-mandibular plane angles are all increased in the open bite group. The studyemphasized the great variation that can occur in the dental and skeletal morphology in patients with open bites. Dung and Smith[66]also defined some measurements for diagnosis of an open bite tendency, which included SN-to-mandibular plane angle of 40° or less, a posterior-to-anterior facial height ratio of 0.58 or less, and an upper-to-lower facial height ratio of 0.7 or less. The cephalometric analysis (Kim's Analysis) of open bite proposed by Kim[67]includes two factors: the overbite depth indicator used for an appraisal of the vertical component, and the anteroposterior dysplasia indicator for the horizontal component. These factors are used to determine the open bite tendency and Class 2 or 3 skeletal pattern tendencies, respectively.

Management

There is general agreement among orthodontists that patients with AOBs are challenging to treat, and relapse is common after treatment with orthodontics alone or combined with orthognathic surgery.

Depending on the age of the patient and severity of the open bite, four treatment modalities are usually employed:

(1) Advice about cessation of early problems or parafunctional habits
(2) Interceptive treatment
(3) Camouflage treatment using orthodontics only, and
(4) A combined orthodontic and surgical approach.

Numerous orthodontic techniques have been proposed to obtain bite closure, extrusion of the incisors, intrusion of the posterior teeth, and mesialization of the posterior teeth, as well as uprighting the incisors after dental extractions.[68-70]Due to the variety of theories on the cause, a wide variety of treatments have been advocated for correcting AOB[65,71-74]by either eliminating the cause or correcting dentofacial changes, with the objective of improving mastication, respiratory function and swallowing. However some studies[62,75]have reported high relapse rates.

The orthodontic literature describes various methods for treatment and retention aimed at decreasing the risk of open bite relapse, including functional and fixed appliances, tongue spurs or cribs, [76,77]elastics, [78,79]wires, [67]molar intrusion,[78] orthognathic surgery, [80] extractions, partial glossectomy,[81] and

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orofacial myofunctional therapy (OMT). [82,83]In order to obtain autorotation of the mandible by intruding the posterior teeth orthopaedic appliances, such as high-pull headgears, [84]bionators, [85]Fränkel functional regulators, [65]and Teuscher activators[86]have been used. Open bite correction using bite-blocks with repelling magnets on the upper and lower posterior teeth was also reported.[87] However, besides precise impressions of the upper and lower jaw, this technique demands absolutely correct placements of the magnets. In addition, spring- loaded bite-blocks in the lower jaw[88]were suggested for open bite correction as they exert an intrusive force on the posterior teeth due to the spring mechanism. One disadvantage of this technique may be the breakage of the springs as reported by Kuster and Ingervall.[72]Other treatment options include the use of multi-loop edgewise archwires,[59]invisalign system,[89] mini-implants,[90] and orthognathic surgery.[80]

Stability and Relapse

When considering the patient's investment of time, discomfort, and money, the issue of stability becomes even more important.There is general agreement among orthodontists that patients with AOBs are challenging to treat, and relapse is common after treatment with orthodontics alone or combined with orthognathic surgery.[62] One reason is that vertical growth and eruption of posterior teeth may continue until the late teenage years or early twenties, with vertical growth of the maxilla being the last stage of maturation. [91]Relapse of open bite can occur because of tongue size or posture,[37]digit-sucking habits,[92] respiratory problems, [93]condylar resorption, [94]and unfavorable growth patterns.[32]However, no known characteristics are clear predictors of relapse. [95] Thus, clinicians should pay more attention during retention phase and long-term studies on post-treatment changes and stability should be encouraged.[96]

Conclusion

There is a general variation in anterior open bite among the world's population with great tendency towards racial predilection. Successful treatment of anterior open bite greatly relies on both diagnosis, including cephalometric analysis, and management. Although there are many different treatment modalities available, stability after treatment is still a critical issue as evidence on long-term stability of various treatment options is lacking. Thus, clinicians should pay more attention during retention phase and long-term studies on post-treatment changes and stability should be encouraged.

Contributors

Agbaje H.O. was responsible for the acquisition of materials for the review and the write-up. Osiatuma VI was also responsible for the acquisition and drafting of the article. Fadeju DA was responsible for collection of materials and drafting of the article. Kolawole KA was responsible for critical revision and final approval of the article. Otuyemi OD was responsible for the concept, drafting, critical revision and final approval of the article.

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