Open bite: a review of etiology and management

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Abstract

Diagnosis and treatment of open bite malocclusion challenges pediatric dentists who attempt to intercept this malocclusion at an early age. This article updates clinicians on the causes and cures of anterior open bite based on clinical data. Patients with open bite malocclusion can be diagnosed clinically and cephalometrically, however, diagnosis should be viewed in the context of the skeletal and dental structure. Accurate classification of this malocclusion requires experience and training. Simple open bite during the exchange of primary to permanent dentition usually resolves without treatment. Complex open bites that extend farther into the premolar and molar regions, and those that do not resolve by the end of the mixed dentition years may require orthodontic and/or surgical intervention. Vertical malocclusion develops as a result of the interaction of many different etiologic factors including thumb and finger sucking, lip and tongue habits, airway obstruction, and true skeletal growth abnormalities. Treatment for open bite ranges from observation or simple habit control to complex surgical procedures. Successful identification of the etiology improves the chances of treatment success. Vertical growth is the last dimension to be completed, therefore treatment may appear to be successful at one point and fail later. Some treatment may be prolonged, if begun early. Long-term clinical outcomes are needed to determine treatment effectiveness and clinicians should consider the cost-effectiveness of these early initiated and protracted plans. (Pediatr Dent 19:91–98, 1997)

Open bite was defined by Subtelney and Sakuda[1] as open vertical dimension between the incisal edges of the maxillary and mandibular anterior teeth, although loss of vertical dental contact can occur between the anterior or the buccal segment. Because different etiologic factors are involved when the open bite occurs in the anterior, as opposed to the buccal segments,[2] our discussion will be restricted to anterior open bite.

Diagnosis of open bites should be viewed first in the context of skeletal structures. Sassouni[3] classified open bites into skeletal and dental open bites. The latter have no significant skeletal abnormality. When the skeletal morphology in the vertical dimension has been classified successfully, it can be determined whether or not

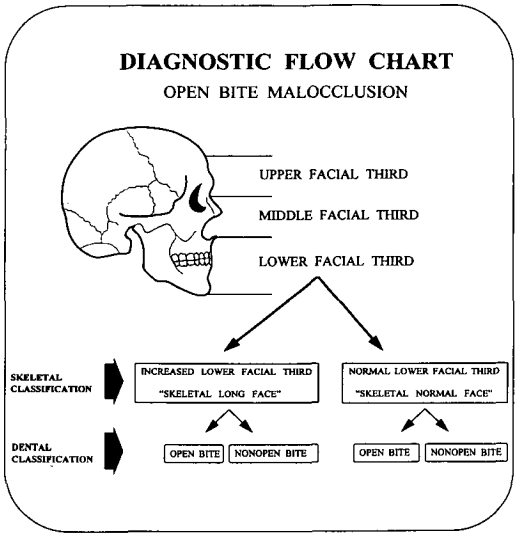


Fig 1. This diagnostic flow chart demonstrates the possibilities and relationships between skeletal and dental relationships in open bite malocclusion.

TABLE. CLINICAL AND CEPHALOMETRIC CHARACTERISTICS OF SKELETAL OPEN BITE

Clinical Characteristics	*Cephalometric Characteristics*
1. Excess anterior face height, particularly in the lower third	1. Steep palatal plane and increased percentage lower facial height
2. Lip incompetence (resting lip separation ≥ 4 mm)	2. Excess eruption of the maxillary posterior teeth
3. Anterior open bite (but not always, some incisors supraerupt)	3. Downward and backward rotation of the mandible
4. Tend to exhibit class II malocclusion and mandibular deficiency	4. Excess eruption of maxillary and mandibular incisors
5. Tend to exhibit crowding in the lower arch	
6. Tend to exhibit a narrow maxilla and posterior cross bite	

a dental open bite accompanies the skeletal relationships. Fig 1 shows that there are multiple variants of this problem.

Patients can be diagnosed (or classified) clinically and/or by cephalometric analysis, as shown in the Table. Proffit characterized patients with skeletal open bite and a large total face height manifested entirely in the elongation of the lower third of the face as having long face syndrome.[4] Clinically and cephalometrically, these patients have a disproportionately long lower facial third. Lowe et al.[5] determined that although facial proportions are important, vertical facial types could be separated reliably using simple, linear extraoral measures for males and females. Fields et al.[6] demonstrated that increased interlabial gap was statistically significant between normal and long face children and adults with a mean difference from normal of 2x and 5x, respectively. Unfortunately, evidence suggests that general dentists trained to clinically detect vertically disproportionate faces are not reliable at that task.[7] In an effort to dissect this problem of vertical facial types more scientifically, Lowe et al.[8] applied quantitative assessments (Fourier and cluster analyses) to vertical and anteroposterior profiles of a great range of patients. They found distinct characterization and discrimination difficult. This same study suggested that specialists can make reliable clinical discriminations between vertical facial types after training. In summary, clinical vertical classification of patients can be accomplished, but it must be attempted with care and appropriate training.

Investigators disagree on the site of the skeletal disturbances associated with long faces. Some investigators[9] noted the maxilla was at fault, while others[6,10] indicated the lower face associated with mandibular morphology (ramus height or mandibular plane) was the location of the disturbance.

Excessive dental eruption is a confusing variable. In a study by Fields et al., no dental vertical variables were observed in adults, but long face children had significantly more vertical development, except in the maxillary anterior region.[6]

The study of facial morphology suggests that facial types, no matter how they are defined, are a complex entity. The inter-relationships of the regions make cephalometric measures highly correlated because they often look at similar morphology from slightly different perspectives.[11] Such correlations should not be viewed as confirming the identification of the source of a problem. The inter-relationships are a result of the method of analysis, not the problem of inquiry. When these correlations are taken into account, it appears that the lower face height is at fault in patients with clinically disproportionate vertical facial relationships.[6]

Once the skeletal abnormality is identified, patients can be classified as dental open bite or nonopen bite. Patients with increased lower face height may or may not have an anterior dental open bite.[10] In all patients, an open bite exists during the exchange of primary incisors to permanent incisors, which is part of normal growth and development.

In summary, both normal and long face skeletal morphology are observed in association with normal and open bite dental occlusion. In other words, the open bite dental occlusion is not indicative of a specific skeletal relationship.

Prevalence and problems related to open bites

The prevalence of skeletal long face malocclusion is unknown, but has been estimated to be 0.6% or 1,350,000 U.S. citizens.[4] The prevalence of dental open bites in U.S. children is approximately 16% in the black population and 4% in the white population,[12] with the prevalence of simple anterior open bites (involving mainly the incisors) decreasing until adolescence.[13] All children experience anterior open bites during the transition from the primary to permanent dentitions with little disruption in their oral physiology during this period, which can span 1 to 2 years.

Masticatory[14] and speech[15] problems have been attributed to open bites. The inability to incise is the chief complaint often voiced by open bite patients. Other patients indicate displeasure with their facial esthetics.[16]

Many open bites will resolve by gradually closing without treatment, and transitional open bites, which make up many of the simple open bites, are of little consequence. Complex open bites, those that extend farther distally and those that do not resolve at the end of the mixed dentition years, can be more problematic.

Relationship between temporomandibular joint dysfunction (TMD) and open bite

Several studies have related the morphologic aspects of malocclusion to mandibular dysfunction in children.[17–20] Williamson surveyed 304 pre-orthodontic patients (aged 6–16), and found that 72% of those with pain dysfunction symptoms had either open bite or deep bite.[17] In a random sample of 402 children, Egermark-Ericksson et al.[18] found a correlation between TMJ clicking and dental wear. They also found that functional malocclusion due to occlusal intereferences was more important than morphologic malocclusion in the etiology of mandibular dysfunction.[18] In a later longitudinal study on malocclusion in relation to signs and symptoms of TMD, the authors found that no single occlusal factor is of major importance in the development of TMD, but that morphological malocclusion such as crossbite and anterior open bite might be a potential risk factor.[19] In a larger longitudinal study with 7337 Japanese children, the prevalence of TMD was found to be 12.2%. In subjects with TMD, 72.9% exhibited some form of malocclusion and 5.4% had open bite. Because a large number of subjects with TMD also had malocclusion, the authors recommended early treatment to prevent severe TMD.[20]

Etiology

According to Dawson,[21] the major causes of an anterior open bite are forces that result from thumb or

finger sucking, pacifier use; lip and tongue habits; airway obstruction; inadequate nasal airway creating the need for an oral airway; allergies; septum problems and blockage from turbinates; enlarged tonsils and adenoids; and skeletal growth abnormalities. This review will demonstrate that one factor is unlikely to be the causative agent and a multifactoral etiology that most likely explains open bite problems. Our discussion can only be used as information on how to treat the condition when, and if, certain diagnostic and etiologic criteria are present.

Thumb and finger sucking or pacifier use

In younger children, the major cause of anterior open bite (excluding open bites associated with the transition from the primary to mixed dentitions) are non-nutritive sucking habits. By adolescence, environmental causes of anterior open bite are less important than skeletal factors.

Prolonged thumb-sucking tends to create this malocclusion. A surprisingly large percentage (10–15%) of children continue to suck a thumb, finger, or other object well into the elementary school years.[12] Johnson and Larson[22] use the term non-nutritive sucking (NNS) to describe habits that involve digits, pacifiers, and other environmental influences. Two theories address the possible cause of NNS: Freud's psychoanalytical theory and the learning theory. A combined explanation suggests that all developmentally normal children possess an inherent, biologic drive for sucking. The rooting and placing reflexes are merely an expression of this drive. Furthermore, environmental factors contribute to the transfer of this sucking drive to non-nutritive sources, such as the thumb or fingers.

A typical thumb-sucker has a malocclusion characterized by an asymmetric anterior open bite due to digit position and a transverse constriction of the maxillary arch. Adair, et al. evaluated the effects of orthodontic and conventional pacifiers on the primary dentition.[23] The results showed a statistical increase in overjet in the "orthodontic" pacifier group and significantly greater incidence of open bite in the conventional pacifier group when these groups were compared. Subsequent data demonstrate no significant benefits of nonconventional pacifiers, but a tendency for open bites to close after cessation of the habit.[24]

Lip and tongue habits

Dentists and speech therapists often attribute open bite malocclusion to abnormal tongue function. Straub suggested that tongue thrusting can produce open bites but presented no data to substantiate the claim.[25] James and Townsend described different types of tongue thrusting based on the resulting deformities.[26] Tulley[27] classified tongue thrusting as an endogenous habit or as an adaptive behavior based largely on facial morphology and swallowing activity.

According to Proffit and Mason, tongue thrust is more likely to be an adaptation to the open bite, and therapy aimed at changing the swallowing pattern is not indicated.[28] Given the physiology of tooth movement, it is unlikely that tongue thrust, but rather resting tongue posture, plays a role in the etiology of open bite. Equilibrium theory suggests that light continuous forces are responsible for tooth movement and position.[29] These forces can be external (digits) or internal (tongue posture or periodontal forces). Abrupt, intermittent forces (tongue forces due to swallowing) are much less likely to be a causative factor. Proffit and Mason's recommendations,[28] then, make good clinical recommendations even today. They suggest that therapy for anterior tongue position is not warranted with or without malocclusion before adolescence. Further, tongue therapy is most effective when combined with orthodontic treatment. Speech therapy may be combined with orthodontic treatment and possibly myofunctional therapy in older children.

Airway obstruction

Patients with skeletally disproportionately long faces are often suspected of having an airway obstruction. These patients' facial appearances were characterized many years ago as adenoid facies: the cheeks are narrow, the nostrils are narrow and pinched, the lips are separated, and often there are exaggerated shadows beneath the eyes.[10, 30] This terminology prompted the erroneous notion that the familiar elongated facial pattern, with an open mouth and dull expression, was exclusively related or primarily related to an obstructive adenoid mass or some other respiratory impairment. It failed to take into account that the pathologic condition causing the obstruction could be related to disease or abnormalities of the turbinates, septum, and external nasal architecture, or an obstructing adenoid mass that may have resolved by the time an upper airway assessment is performed.

A report by Linder-Aronson in 1970[31] renewed interest in this complex relationship between respiratory pattern and facial growth and development. The author demonstrated a statistically significant relationship between obstructing adenoid tissue and certain skeletal and dental patterns. These changes included rotation of the mandible in a clockwise manner so that the mandible was in a more vertical and backward direction, causing elongation of the lower anterior face height, open bite, and retrognathia. Although statistically significant, the clinical ramifications were minimal. In another study, Hultcrantz examined the incidence of open bite in children with tonsillar obstruction and found a higher proportion of open bites than in children with unobstructed airways.[32]

Harvold showed that total nasal airway obstruction caused various developmental problems, but an open bite did develop in some animals.[33] This was mistakenly interpreted by many to indicate that mouth breathing was the cause of open bites. In reality total nasal obstruction in humans is rare and incompatible with life in the newborn.

Much of the controversy appears to result from the lack of objective criteria used to assess facial morphol-

ogy and respiratory behaviors. Recent developments in evaluation of both facial morphology and respirometric variables make it possible to explore this relationship further.

Considerable progress also has been made in quantifying the mode of respiration. Previously, investigators used undisclosed, subjective, or unreliable methods to evaluate and label respiration as either nasal, oral, or a combination of these two modes.[34–36] Lateral cephalometric radiographs have been used to quantitatively evaluate airway size and patency.[37] Although positive correlations have been found between airflow and airway measurements from cephalometric radiographs,[38] the validity of evaluating a three-dimensional structure with a two-dimensional radiographic projection is questionable.[39]

Several investigators used measures of nasal resistance to determine airway dynamics.[31, 40, 41] Although nasal resistance measurements are valid and reliable when used appropriately, this method does have certain limitations.[42] Nasal resistance cannot be correlated with respiratory mode, the proportional nasal and oral components of breathing.[43, 44]

A system to measure respiratory behavior objectively should provide continuous monitoring of successive respiratory cycles, measure both inspiratory and expiratory airflow, provide simultaneous measurements of oral and nasal airflow, interfere minimally with normal respiratory behavior, and have a high degree of reliability and reproducibility.[45] Such methods have been developed.[45–48]

Warren[42] demonstrated a method to assess nasal airway impairment using a technique to measure a minimum nasal cross-sectional area. This method involved modifying the theoretical hydraulic principle and assumed that the smallest cross-sectional area of a structure can be determined if the differential pressure across the structure is measured simultaneously with rate of airflow through it. This technique enables clinicians to estimate the size of the nasal airway's minimum cross-sectional area during breathing and gives some indication of the potential for nasal impaired or normal respiratory function. Warren et al.[48] also described an alternative approach for measuring oral and nasal respiration and tested its reliability.

Fields et al.[49] demonstrated that the normal and long face groups had similar tidal volumes and minimum nasal cross-sectional areas, but the long face subjects had significantly less nasal component of respiration. These results illustrate that groups without significant differences in airway impairment can demonstrate significantly different breathing modes that may be behaviorally based instead of airway dependent. Postural changes may be responsible for the morphologic changes of the face and may have been established early as an adaptation for previous airway deficiencies. The adaptive posture may have resulted in altered muscle forces that can impact dental and skeletal structures. Solow et al.[50] advanced this theory that was noted by Warren and Spalding.[51]

Because of conflicting results, these studies suggest that one should have a clinically reliable evaluation of the airway before intervention, so that any treatment is directed at a valid etiologic agent.

Skeletal growth abnormalities

In 1931, Hellman[52] suggested that open bite is due primarily to skeletal deficiencies. In a study of 43 treated and untreated open bite cases, he found the percentage of successful treatments was equal to the percentage of self-correcting cases in the untreated group. Using anthropologic measurements, he found that subjects with open bite had shorter rami and greater total facial height. In another study by Schudy,[53] clockwise rotation of the mandible (as viewed from the patient's right) 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. 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 anterior open bite.[54] Similarly, forces that impede the eruption in the incisal region also result in anterior open bite.

In summary, vertical malocclusion develops as a result of the interaction of many etiologic factors. In young children, digit habits and pacifiers are the most common etiologic agents. In the mixed dentition years, other than the normal transitional open bite, some open bites are probably attributable to lingering habits, while others are clearly skeletal in nature. In the adolescent and the adult, it is difficult to assign singular causation. The influence of the tongue, lip, and airway on the development of malocclusion remains to be substantiated. Variations in growth intensity, the function of the soft tissues and the jaw musculature, and the individual dentoalveolar development influence the evolution of open bite problems.

Cures (treatment considerations)

To state that there are cures for open bite malocclusion is misleading. To indicate that some approaches are more rational than others is fair. Unfortunately, the long-term clinical outcomes are not well documented. The discussion presents some data and some clinical impression. The treatment for open bite problems ranges from observation or simple habit control procedures to complex surgical procedures. This is complicated by the fact that vertical growth is the last dimension to be completed.[55] This means that sometimes a simple treatment will prevail, while at other times, may appear to be successful at one point only to fail later. It also implies that some treatments may be extremely long, if begun early. The cost-effectiveness of these protracted plans must be questioned.

Treatment techniques can be categorized as habit, appliance, or surgical. Simple techniques are those in

which the etiologic factor is removed and the bite closes by the normal eruptive process, or closure is enhanced using orthodontic appliances. More difficult procedures are those in which intrusion (either active or relative intrusion achieved by inhibiting eruption of the posterior teeth) is attempted with orthodontic appliances. In some cases, orthognathic surgery is the last and only resort. Often treatment approaches are combined when the etiology is unclear.

Habit therapy

In young children engaged in NNS, treatment consists of controlling the habit, which alone may be sufficient to allow the teeth to erupt to a normal position. Johnson and Larson[22] suggest that therapy should begin when the benefit to the patient outweighs the risks (dental, emotional, and psychologic) of habit discontinuation. Treatment may involve habit awareness, time out, contract of reward or punishment, positive reinforcement, and sensory attenuation procedures (procedures designed to interrupt the sensory feedback from NNS such as orthodontic appliances, chemical aversion, and hand wraps). A habit device can be incorporated into the maxillary expansion appliance to correct both the transverse (maxillary constriction) and vertical problems (Fig 2). Because patient compliance and cooperation are essential in eliminating NNS habits, a child must want to terminate the habit before intervention begins.

Early or interceptive treatment of anterior open bite with cribs or retraining exercises aimed at tongue control remains a controversial issue. Worms et al.,[13] in a study examining 1408 Navajo children ranging from 7 to 21 years for occlusal discrepancies, found spontaneous correction of 80% of the anterior or simple open bites. Appliances such as tongue cribs have been used to treat anterior open bites by redirecting an anteriorly positioned tongue. Erverdi et al.[56] studied the effect of crib therapy to treat anterior open bite. The most significant findings were the eruption of the mandibular and maxillary incisors and intrusion of the mandibular first molars, which decreased lower face height. These findings were considered to result from the posterior tongue posture. Myofunctional therapy periodically resurfaces as a treatment method. At this time, no scientific evidence supports myofunctional therapy as effective in correcting open bites.[25]

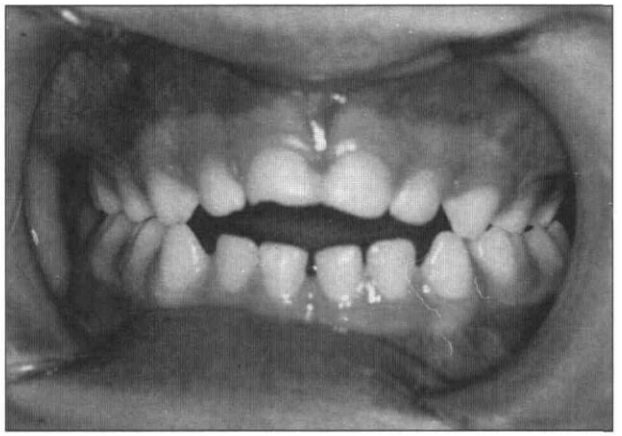
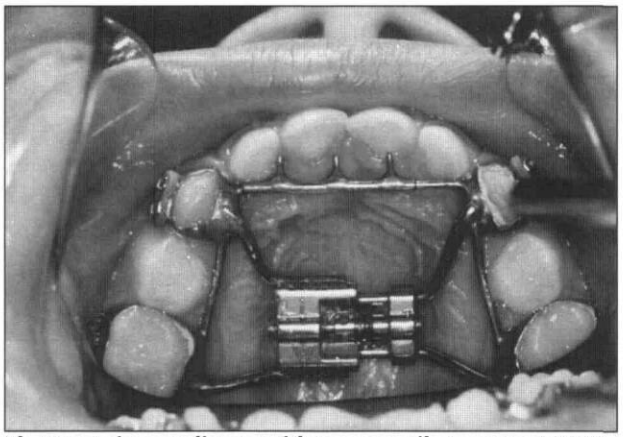


Fig 2a. A 5-year-old boy presented with anterior open bite and constricted maxilla due to NNS habit.

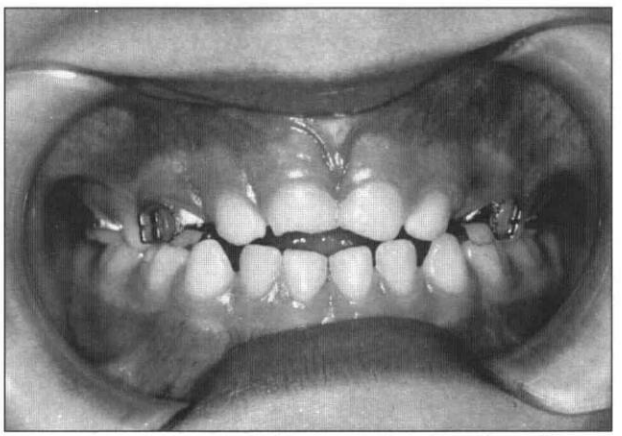


2b. Expansion appliance with tongue crib to correct NNS habit. A Hyrax™ rapid maxillary expansion appliance was chosen rather than a simpler W-arch or quadhelix due to the ability of this rigid appliance to prevent a compulsive thumb sucker from imbedding the appliance in palatal tissues.

Appliance therapy

Appliance therapy usually has one of several goals: to impede dental eruption and thereby control vertical development, to reduce or redirect vertical skeletal growth with intraoral or extraoral forces, or to extrude anterior teeth. Bite blocks often are used as a component of orthodontic appliances to intrude or control eruption of the posterior teeth. Bite blocks made of wire or plastic fit between the maxillary and mandibular teeth at a slightly increased vertical dimension. The stretched muscles theoretically place an intrusive force on the posterior teeth, which in turn helps control eruption. With limited eruption, skeletal growth is directed more anteriorly and less vertically.

Dellinger[57] describes the use of the Active Vertical



2c. Correction of NNS habit, normalization of maxillary arch width and improvement in anterior open bite after 3 months of appliance therapy.

Corrector™ (AVC), which is a removable or fixed appliance that intrudes the posterior teeth in both the maxilla and mandible by reciprocal forces. This appliance reportedly corrects open bites by actually reducing anterior facial height. Haydar and Enacar[58] used a Frankel™ appliance (FR4) to correct open bites, and showed that it did decrease the open bite significantly, but produced mainly a dentoalveolar rather than skeletal result. Aragao's function regulator[59] was shown to normalize open bite.

Magnets also have been incorporated into bite blocks to exert an intrusive force on the molars with a result of decreasing the open bite.[60] Kuster and Ingervall compared the use of spring-loaded bite blocks with bite blocks with repelling magnets. Their results showed an average improvement in open bite of 1.3 mm in the spring-loaded group and 3.0 mm in the magnet group. There was a tendency toward relapse, but they felt this might be counteracted by a long phase of active retention.[61] Iscan compared spring-loaded bite blocks with passive bite blocks and found no significant difference between the two.[62] Continuous force appears from clinical reports to be able to intrude posterior teeth. This control is required until vertical growth is completed. Maintaining correction is the most difficult task.

In correcting skeletal open bite problems, intraoral appliances, such as activators, bionators, Frankel™ regulators (most with the inclusion of posterior bite blocks), have been used to control vertical maxillary growth of the mixed dentition. Weinbach and Smith[63] showed that a bionator can be used to treat open bite problems, especially if accompanied by a class II molar relationship.

Another appliance approach uses extraoral devices to impede the vertical skeletal and dental growth pattern, such as a high-pull headgear. The biggest problem with the headgear is that it is almost impossible to obtain a pure vertical force. Wieslander suggests that for the headgear to obtain a skeletal effect, it must be worn 12–14 hr/day with a force of 10–16 oz (400–450 g) per side.[64] Schudy advocated a high-pull headgear along with a mandibular splint covering the second molars and anterior vertical elastics to treat open bites.[65] Pearson suggests controlling the vertical force by using intrusive forces on the mandibular posterior by light mandibular headgears, which he states can be helpful in reducing lower molar height increases and gaining control of the occlusal plane angle.[66, 67]

When patients have increased vertical development and a class II malocclusion, the potential exists to use headgear in combination with a functional appliance incorporating posterior bite blocks.[68, 69] Ngan demonstrated that open bite complicated by a class II vertical growth pattern can be treated during the mixed dentition with favorable results by using a combination of an activator and high-pull headgear.[70] Dermaut[71] studied the effect of headgear activator of Van Beek and found that the use of combined activator and headgear controlled the increase in lower anterior face height. This combined approach of functional appliance and headgear provides some skeletal and dental control.

Another appliance aimed at controlling the vertical growth that may cause an open bite is the chin cup. Pearson reported that the use of a vertical-pull chin cup could result in a decrease in mandibular plane angle and an increase in posterior facial height compared with the growth of untreated individuals with a resultant decrease in open bite tendencies.[72] However the chin cup generally has poor compliance rates.

Straight wire appliances and leveling the arches may spontaneously correct mild open bites.[73] This has some efficacy if the upper arch has a curve of Spee and the lower does not. Injudicious leveling of the lower arch usually opens the bite and is contraindicated. Some open bites can be treated by stepping the arch wires to close the bite combined with use of vertical elastics. Viazis published a case report using rectangular NiTi wires and elastics to close an anterior open bite.[74] Care must be taken not to erupt the teeth extensively when the patient has increased facial height. Excessive and unesthetic dentoalveolar height can result from this approach if smiling reveals extensive gingival display.

Arat and Iseri compared fixed appliance treatment with functional treatment to correct open bite. During fixed appliance therapy, marked increases in the maxillary and mandibular posterior dentoalveolar height were observed, and the mandible rotated backwards. On the other hand, with functional appliances, forward and upward rotation of the mandible was noted with the center of rotation at the premolars.[75] These data vividly emphasize an important point. If functional appliances are used for phase I therapy and are followed by phase II fixed appliances, all the gains from phase I can be lost in phase II. Incorporating removable bite blocks with fixed appliance therapy has shown some clinical success if continued into retention and the nongrowing years.

In summary, any of the mixed dentition approaches must take several factors into account. First, facial growth can make these efforts unsuccessful in the long run. Fixed appliance therapy with its extrusive biomechanics, must not reverse gains previously made. Combinations of techniques may be essential even during the finishing and retentive phases. For that reason, it may be best to tackle only mild or moderate problems or those in patients who are near the end of growth, and not severe open bite problems. Second, any treatment aimed at controlling eruption in one arch must guard against compensatory eruption in the opposing arch.

Surgical management

One method of surgical correction is to extract second and/or third molars if they are the only source of centric contacts.[21] Glossectomies have been used to correct open bite problems associated with abnormal tongue habits. Their effectiveness in closing anterior or posterior open bite problems has not been substantiated.[4]

Surgical procedures to improve the patency of the airway must be undertaken with caution. Documenting the amount and location of the obstruction is a prerequisite. In many cases, a more conservative medical approach may serve the same purpose when the obstruction is related to allergies. This is especially important because it is recognized that a reduction in tonsilar and adenoid tissue occurs near adolescence, and other children appear to "outgrow" certain allergies.

Severe skeletal open bites in patients who are not growing are often treated by combined orthodontic-surgical approach. Superior repositioning of the maxilla, via total or segmental maxillary osteotomies, is indicated in skeletal open bite patients with excess vertical maxillary growth. Maxillary impaction allows forward and upward rotation of the mandible, therefore decreasing the lower face height and eliminating anterior open bite.[4] This upward and forward autorotation often makes mandibular reduction or reduction genioplasty necessary as well. Superior repositioning of the maxilla is one of the most stable orthognathic surgical procedures. In a study of 61 patients who had a LeFort I downfracture with the maxilla moved superiorly at least 2 mm, only three patients (5%) had significant relapse; 95% were vertically stable.[76] These excess problems are best approached when growth is nearly completed so that residual growth does not obviate the correction. Such procedures can be completed earlier in females than males.

Summary

The problem of open bite is multifactorial. Diagnosis should be viewed in the context of the skeletal structure and the dental structure. Anterior open bite accompanied by a normal lower face height can be treated successfully using appliance therapy if the etiology can be identified as a habit or obvious environmental influence. The influence of tongue, lip and airway on the development of this malocclusion remains to be substantiated. Reliable and valid otolaryngology consultation should be obtained if nasal airway obstruction is suspected.

Open bite problems of skeletal nature require orthopedic intervention. Severe skeletal open bite in nongrowing patients usually requires treatment with orthodontic-surgical procedures. The treatment of open bite remains a challenge to the clinician, and careful diagnosis and timely intervention will improve the success of treating this malocclusion.

A portion of the section on "Airway Obstruction" was excerpted from Fields et al.: "Relationship between vertical dentofacial morphology and respiration in adolescents". Am J Orthod Dentofac Orthop 99:147:154, 1991.

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