BJDOR
BRAZILIAN JOURNAL OF DENTISTRY AND ORAL RADIOLOGY

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PUBLICAÇÕES CIENTÍFICAS DE ACESSO ABERTO

Review

Classifications and Etiologies of Open Bite: A Review

Jéssica Castro Costa [1,*], Gustavo Vieira Fontenele [2], Juliany Rocha Syed [3], Julhana Alves [4], Nereida Manuela Silva Pacheco Fernandes [5]

1 Universidade Cruzeiro do Sul, Curitiba, Paraná, Brazil.
2 Universidade Nilton Lins, Manaus, Amazonas, Brazil.
3 Centro Universitário do Estado do Pará, Belém, Pará, Brazil.
4 Universidade Tuiuti do Paraná, Curitiba, Paraná, Brazil.
5 Universidade Federal da Bahia, Salvador, Bahia, Brazil.

* Correspondence: castrojeessica@gmail.com.

Abstract: The term open bite is referred to as no contact between the anterior or posterior teeth. The complexity of the open bite is attributed to a combination of skeletal, dental and habitual factors. The etiology of the open bite can be attributed to genetic, anatomical and environmental factors. However, the tendency of recurrence after conventional or surgical orthodontic treatment has been indicated. Therefore, the open bite is considered one of the most challenging dentofacial deformities to treat. The objective of this is to identify what the literature has presented on the topic, mainly in questions about early diagnosis and classification, which are essential for the good result of the intervention. The methodology used in this study was a literature review. With the realization of this study it was possible to notice that the treatment of open bite remains a great challenge for the clinician; Careful diagnosis and timely intervention with appropriate treatment modalities and selection of devices will improve treatment results and long-term stability.

Keywords: Open Bite; Orthodontics; Orthodontic treatment.

Citation: Costa JC, Fontenele GV, Syed JR, Alves J, Fernandes NMSP. Classifications and Etiologies of Open Bite: A Review. Brazilian Journal of Dentistry and Oral Radiology. 2025 Jan-Dec;4: bjd58.

doi: https://doi.org/10.52600/2965-8837.bjdor.2025.4.bjd58

Received: 18 May 2025
Accepted: 25 May 2025
Published: 28 May 2025

1. Introduction

Anterior open bite malocclusion can occur for several reasons. It may be genetic in nature, leading to a skeletal open bite, or it may be caused by functional habits that can result in a dental open bite. In younger ages, an open bite may occur due to the transitional change from primary to permanent dentition [1]. An anterior open bite occurs when the anterior teeth fail to make contact and there is no overlap between the upper and lower incisors. The anterior opening can be caused by functional habits such as thumb sucking, tongue thrusting, or prolonged pacifier use. When the thumb-sucking habit is present from the late stages of primary dentition through the early mixed dentition, it can lead to various side effects, such as flared upper teeth, retroclined lower teeth, increased open bite, and overjet. A posterior crossbite in these children, along with a decrease in intercanine and intermolar widths, is also commonly found. The more intense (and prolonged) the habit, the worse the malocclusion can be [2].

Pacifier use has also been shown to cause anterior open bites in children. Pacifier use lasting more than 18 months may cause this type of malocclusion. It is shown that as long as the sucking habit stops before the eruption of permanent teeth, the open bite can self-correct. In some cases, behavioral modification may be necessary to eliminate these oral habits [3]. A posterior open bite occurs when posterior teeth, such as molars or premolars, fail to make contact with their opposing teeth. This is more likely to occur in segments where there may be a unilateral open bite or an

open bite related to one or more specific teeth. Failure of tooth eruption due to primary failure or mechanical obstruction during the eruption phase can cause an open bite. Sometimes, lateral tongue thrust may also prevent the eruption of posterior teeth; thus, eliminating this habit can be the key to allowing eruption in these cases [3].

Dental open bite occurs in patients where the anterior teeth fail to touch; however, this is not accompanied by the skeletal tendency associated with open bite. Therefore, this type of open bite can occur in patients with a horizontal or hypodivergent growth pattern. These patients exhibit normal mandibular growth and do not present with long face syndrome. The anterior open bite in such cases may be caused by macroglossia, tongue thrusting, or thumb-sucking habits [4]. Given this context, the objective of this study is to discuss the classification and main etiologies. The methodology used was a literature review.

2. Literature Review

2.1 Definition of Open Bite

Open bite is a type of orthodontic malocclusion in which the upper and lower teeth do not overlap and fail to touch properly when in occlusion, leaving a space between the upper and lower rows of teeth even when the mouth is closed. Open bite was defined by Subtelny and Sakuda as an open vertical dimension between the incisal edges of the upper and lower anterior teeth, although a deficiency in vertical dental contact may occur in either the anterior or buccal segment [5].

2.1.1 Anterior Open Bite

It is defined as a malocclusion with no contact in the anterior region of the dental arches, while the posterior teeth remain in occlusion. When the malocclusion extends to the posterior segment, it is called a combined open bite [5]. Among the malocclusions most commonly encountered in clinical practice, anterior open bite is one of the most prevalent and difficult to treat. When the etiology is multifactorial, this condition leads to esthetic alterations, speech articulation difficulties, and adverse psychological outcomes. An open bite may have a dental, skeletal, or combined etiology. Dental open bite can often be treated with fixed orthodontic therapy. However, a more comprehensive approach is required for treating skeletal open bite, which may involve orthognathic surgery. Dental open bite in growing patients can be treated with myofunctional appliances, followed by a retention period with removable orthodontic devices. Nasal obstruction should be addressed during the pubertal growth phase, and tongue hyperactivity, during swallowing or even at rest, can alter the axial inclinations of the incisors, potentially leading to open bite [6].

2.1.2 Posterior Open Bite

Posterior open bite can be defined as the failure of contact between posterior teeth when the teeth are in centric occlusion. There is no occlusion between upper and lower premolars, while upper and lower molars may have slight contact [7].

2.2 Classification and Types of Open Bite

This section 2.2 discusses the classification and types of open bite, initially outlined by Worms, Meskin, and Isaacson in 1971. They categorized open bite into three main forms: simple open bite, which extends from canine to canine with a 4mm or greater gap in centric relation; compound open bite, spanning from premolar to premolar; and infantile open bite, involving the region from molar to molar. Open bites can also be classified by their nature. A false or dental open bite occurs when the teeth are proclined without any alteration of the underlying bony structures, typically not extending beyond the canines. These cases show normal facial morphology, proper skeletal relationships, and dentoalveolar issues that mimic open bite. In contrast, a true or skeletal open bite involves deformation or abnormal development of the alveolar processes and is associated with dolichofacial characteristics, maxillary hyperdivergence, and increased lower facial height and vertical dimensions.

According to the location of the open bite, they are classified as anterior, posterior, or complete. Anterior open bite may have either a dental or skeletal etiology. The dental variant results from impaired dental eruption, while the skeletal form arises from posterior facial growth. Posterior open bite is defined by the failure of one or more opposing buccal segment teeth to achieve occlusion, despite possible incisal contact. This type is rare and may be caused by tongue interposition, eruption disturbances such as ankylosis, or primary failure of eruption.

Complete open bite is further refined by Andrew Richardson's classification of anterior open bite into several categories. Transient open bite occurs during the eruption of permanent teeth and is usually self-corrected through continued alveolar development and increased anterior facial height. Open bite caused by thumb sucking arises when this habit impedes incisor eruption; elimination of the habit often leads to spontaneous resolution, particularly in younger individuals. Local pathological conditions such as cysts, dilacerations, and ankylosis can also result in anterior open bite, which can be addressed by surgically removing the underlying pathology to allow for normal dentoalveolar development.

Anterior open bite due to skeletal pathology becomes evident later in development and is associated with conditions like cleft palate, craniofacial dystosis, cleidocranial dysostosis, and achondroplasia. Non-pathological skeletal open bite is divided into three subtypes. The first presents during the primary dentition stage and typically resolves during prepubertal and pubertal growth due to compensatory dentoalveolar changes. The second appears in the prepubertal stage, resolves in puberty, but reemerges after puberty due to predominant vertical facial growth. The third subtype represents the most complex orthodontic scenario, where dominant vertical facial growth results in a worsening anterior open bite with age.

Finally, Moyers classified open bite as either simple or complex. Simple open bite is confined to the teeth and alveolar process, with the primary issue being the failure of certain teeth to erupt to the occlusal plane. Complex open bite is a result of primary vertical dysplasia and is frequently associated with Class I and II malocclusions, and less commonly with Class III.

2.3 Etiology

Anterior open bite, like any other malocclusion, results from certain causes, whether of hereditary origin, acting pre- or postnatally on orofacial tissues [16].

2.3.1 Hereditary Factors

Open bite anomaly is most associated with hereditary patterns of facial growth. Horizontal skeletal dysplasias are generally considered to have a genetic basis, and vertical dysplasias may also follow inherited trends. In efforts to understand the determinants of craniofacial growth, three major theories have been proposed. The first theory posits that bone, like other tissues, is the primary determinant of its own growth. The second theory suggests that skeletal growth is mainly directed by cartilage, with bone adapting in a secondary and passive manner. The third and more integrative theory argues that the primary determinant is the surrounding soft tissue matrix in which skeletal structures are embedded, and that both bone and cartilage respond secondarily to changes in this matrix.

2.3.2 Non-Hereditary Factors

Studies have emphasized the contribution of abnormal tongue function, deleterious oral habits, abnormal swallowing patterns, and speech problems to the open bite phenomenon. Tongue dysfunction may either cause or result from abnormal swallowing behavior [18]. The location of the open bite deformity depends on the predominant forces and the ability of teeth and supporting structures to resist change. For example, abnormal swallowing with strong forward tongue thrust can

lead to anterior open bite. The severity of the condition is also influenced by persistent thumb sucking, finger/lip sucking, mouth breathing habits, and weak lip musculature [18].

2.3.3 Sucking Habits

Several factors influence the extent of dental and soft tissue damage caused by sucking habits, including the duration, frequency, intensity, and position of the habit. Thumb or index finger sucking is commonly observed in children up to the age of four or five and is generally considered a normal behavior at this stage, rarely leading to permanent malocclusion. However, if the habit continues into the mixed or permanent dentition phase, it may contribute to the development of an anterior open bite. The impact varies depending on whether the child actively sucks the finger or merely allows it to rest passively in the mouth. This variation in intensity and persistence is directly linked to malocclusions of differing severities. Persistent thumb sucking can apply an upward and forward force on the anterior maxillary complex, potentially altering normal facial growth patterns.

2.3.4 Abnormal Tongue Function

The cause-and-effect relationship between abnormal tongue function and anterior open bite remains unclear. However, four main factors are considered relevant: activity, posture, age and growth, and adaptability. Regarding activity, some studies have found that simply placing the tongue between the teeth is not enough to cause an open bite. Others argue that tongue posture at rest may be more influential than its activity during swallowing. In terms of posture, any disruption in the balance between the skull, mandible, and tongue can potentially contribute to an open bite. For example, nasal airway obstructions—such as enlarged nasal cartilage or chronic upper respiratory infections—may lead to forward head posture and anterior tongue positioning due to a lowered mandible.

Age and growth also play a role. During childhood, the tongue occupies a significant portion of the oral cavity to accommodate dietary and developmental needs. This enlargement may persist as macroglossia, often related to endocrine disorders, promoting tongue thrust and resulting in anterior open bite. Adaptability refers to the body's compensatory mechanisms. Excessive backward rotation of the mandible may lead to open bite and lip incompetence, prompting overactivity of the mentalis and tongue muscles to achieve lip seal during swallowing.

Additionally, iatrogenic factors can influence the development of open bite. Orthodontic procedures such as molar extrusion and distalization can increase the mandibular plane angle and result in a hyperdivergent facial appearance. Similarly, maxillary expansion to correct posterior crossbite may create a wedge effect, clinically manifesting as increased lower facial height.

2.3.5 The Role of Facial Growth in Anterior Open Bite

Growth rotation of the mandible occurs primarily due to two mechanisms. The first involves differential vertical growth among the condylar growth, the combined suture growth, and the alveolar growth of the maxilla and mandible. When the vertical growth of the nasal, zygomatic, and frontal sutures, along with extrusion of the upper and lower molars, exceeds the vertical component of condylar growth, the mandible tends to rotate in a clockwise direction. Studies have shown that this clockwise rotation is a result of greater vertical growth in the molar region compared to the condylar region, with extreme cases leading to anterior open bite.

The second mechanism concerns the growth direction of the mandibular condyle, which may be vertical, sagittal (posterior and superior), or a combination of both. The clinical implication of this is significant: extreme mandibular rotation has the potential to either resolve or exacerbate existing occlusal issues. Anterior open bite associated with backward rotational growth of the mandible is likely to worsen over time. Although craniofacial growth is typically seen as beneficial for correcting

occlusal discrepancies, it can be detrimental if it occurs in an unfavorable direction or magnitude. In particular, skeletal open bite patterns tend to deteriorate as growth continues.

Research has identified that individuals with backward rotation patterns often present with a glenoid fossa positioned more superiorly, which contributes to a shortened ramus. Furthermore, recent findings suggest that the tongue grows at a rate different from that of the surrounding dentoalveolar and muscular structures. While the tongue is relatively large in early childhood, its proportional influence decreases during puberty and adulthood. This evolving balance between the tongue and surrounding tissues may account for the spontaneous resolution of some cases of tongue thrust–related open bite and the improvement of endogenous tongue thrust with age.

3. Discussion and Conclusion

Open bite malocclusion is considered one of the most challenging problems to treat orthodontically. The causes of open bite are multifactorial and may arise from genetic and/or environmental factors. Generally, open bite can be classified into two categories: skeletal and dental [6, 7, 8, 17, 19]. Dental open bite can be treated with orthodontic therapy, but true skeletal open bite may require surgical intervention along with orthodontic treatment [14]. Open bite may present as an esthetic, functional, and psychological concern for patients [2, 3, 5, 9, 11, 16, 18].

Functional problems include speech defects, chewing difficulties, and swallowing issues, which may impair child development. In the mixed dentition phase, the prevalence of open bite can be observed in up to 17% of cases [15]. Recurrent adenoid infections can lead to improper tongue positioning, persistent infantile swallowing, and deleterious oral habits, which may be observed as partial eruption of the incisors [1]. Harmful oral habits such as thumb or lip sucking, pacifier use, mouth breathing, and tongue thrusting result in dentoalveolar anterior open bite [4, 13, 21, 22, 23].

This condition can often be corrected with orthodontic treatment alone, especially if diagnosed at an early age and if the associated habits can be eliminated. Mouth breathing is frequently associated with dry rhinitis and speech difficulties, particularly with fricative consonants. Vertical dentofacial dysplasia tends to relapse. This is observed in both deep bite and open bite malocclusions. Vertical dysplasia manifested as anterior open bite is multifactorial in nature [7, 14, 17, 18, 23, 24]. Among environmental factors, harmful oral habits such as thumb, finger, or lip sucking, mouth breathing, and tongue thrust, often accompanied by macroglossia, are most relevant [4, 7].

Neuromuscular deficiencies contribute to the skeletal component of open bite. Leptoprosopic patients with muscular dystrophy present with supra-eruption of the posterior buccal segment, leading to anterior open bite [3, 13, 15, 19]. In cases of trauma, it may be skeletal-facial or dentoalveolar in nature. Pronounced anterior open bite is commonly observed following condylar cranial trauma, which may result in interrupted growth or condylar ankylosis, presenting as altered vertical mandibular development. Dentoalveolar trauma, particularly involving the incisors, can result in anterior open bite. Ankylosis of damaged teeth is often observed before the patient completes facial growth [17].

Regarding systemic diseases, degenerative conditions such as idiopathic condylar resorption and juvenile rheumatoid arthritis are often associated with condylar resorption [12, 13]. Studies also highlight the role of genetics. Inherent growth potentials are regulated by the individual's genetic constitution. For instance, control of sagittal, transverse, and vertical dimensions is often inherited within families, as seen in the Habsburg jaw. Growth and rotational patterns occurring during late maturation stages are also attributed to the patient’s genetic pool. Facial types such as hyper- and leptoprosopic allow for vertical molar eruption, contributing to an excessive vertical skeletal growth pattern [2, 6, 9, 14, 15].

The relationship between open bite and skeletal morphology has been extensively studied by Cangialosi. His studies show that open bite patients have greater anterior facial height than posterior facial height; the proportion of lower facial height is greater than upper facial height in open bite patients; and open bite patients present with a steep mandibular plane angle and a large gonial angle [1, 19].

Funding: None.

Research Ethics Committee Approval: None.

Acknowledgments: None.

Conflicts of Interest: None.

Supplementary Materials: None.

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