Anterior open-bite malocclusion is defined as the absence of contact between the maxillary and mandibular incisor edges consequently presenting a negative overbite (Nielsen 1991; Ngan and Fields 1997). Generally, it deteriorates the facial aspect, impairs mastication and speech, subjecting the patient to uncomfortable situations (Janson et al. 2003). The frequency of this malocclusion in the mixed dentition is high (17% [Worms et al. 1971]), and the prognosis for correction varies from good to deficient, depending on its severity and on the patient’s age. Before undertaking any treatment alternative, knowledge of the etiology of this malocclusion is important because in many instances, not only the morphological characteristics have to be corrected, but also the etiological factors have to be eliminated not only to assure treatment success, but also to provide long-lasting stability. Therefore, this chapter covers the most common etiological factors of anterior open-bite to help in managing the correction of this malocclusion in the different stages of the dentition it may present.

The etiologic factors of the malocclusions can be divided into environmental and genetic factors. However, all malocclusions are multifactorial and result from interactions of environment and genetics (Mossey 1999a, 1999b). The face and the dentition are influenced by the complex interaction of both. It can be stated that the etiology of a particular malocclusion is predominantly environmental or genetic, and this will determine how much this malocclusion can be corrected by therapeutic intervention, that is, the prognosis of orthodontic correction. The greater the influence of
environmental factors in the etiology of a malocclusion, the better the orthodontic treatment prognosis, as long as the causative factor is eliminated. When there is a strong genetic etiologic factor, most likely the best approach would consist in an orthodontic-surgical approach (Beane 1999). Because environmental open bites are more amenable to an orthodontic approach, this chapter will first cover the environmental etiologic factors and secondly the genetic factors.

**ENVIRONMENTAL FACTORS**

Anterior open bite can be considered as functional consequent to its functional etiologic factors. The most important functional factors are deleterious oral habits, (Popovich and Thompson 1973; Mahalski and Stanton 1992; Johnson and Larson 1993) and oral breathing (Proffit and Mason 1975; Lowe and Johnston 1979; Harvold et al. 1981; Linder-Aronson et al. 1986; Nagahara et al. 1996; Yashiro and Takada 1999). Some other factors may contribute in the environmental etiology such as traumatisms and pathologies (Prosterman et al. 1995).

**Deleterious habits**

In a normal occlusion, there is a balanced relationship among the oral structures, basal bones, teeth, and intra and extraoral musculature, reflecting in a correct function of the stomatognathic system (Moyers 1988). This is denominated the buccinator mechanism. Thus, the teeth are in a balanced position receiving opposing forces arising internally by the tongue and externally by the lips and cheeks (Figure 1.1) (Graber 1966).

The solution of this muscular balance for some abnormal function of the oral muscles has a negative impact on the teeth position and occlusion. Nonnutritive sucking habits, such as pacifier and thumb-sucking, atypical tongue thrust, and anterior tongue posture, all considered deleterious oral habits, can break this muscular balance.

**Pacifier and thumb-sucking**

Humans start sucking fingers, tongue, and lips during fetal life, in the maternal womb (Figure 1.2). At birth, the infant has a well-developed function of sucking to receive the nutrients essential for life. It is during suction developed in breastfeeding that the children not only get the nutrients that need to meet the physiological demands, as well as feelings of security, warmth, and acceptance necessary for their welfare and for their proper emotional development. At this stage, suction is a mean of communication of the infant with the environment (Newman 1990).

The early well-developed oral perception provides a sense of comfort, safety, and emotional satisfaction during sucking. When breastfeeding is
Figure 1.1  Balanced forces between the tongue, lips, and cheeks on the teeth and bone structures.

Figure 1.2  Prenatal thumb-sucking seen in ultrasonographic examination.

not possible, the use of bottles with orthodontic nipples that resemble the anatomy of a woman’s breasts is recommended, because they allow better contact of the tongue with the palate, as necessary for normal swallowing (Graber 1966) (Figure 1.3).

When a child is bottle-fed, his physiological demand is met, but the natural need to suck is not supplied in the few minutes spent in the mother’s lap. Thus, the child can begin the compensating thumb or pacifier sucking (Graber 1966).
Figure 1.3  Breastfeeding provides the natural need to suck to the child.

Pacifier or thumb-sucking are considered as mechanisms of emotional supply of the child. Consequently, pacifier or thumb-sucking in the early child development is considered normal. Through these habits, the child releases the emotional tensions from lack of affective care resulting from conflicting relationship between child and parents, which becomes a way to draw attention from people close to them (Moyers 1988). Parental opposition to these habits can determine negative psychological consequences. When children grow and develop other means of communication with the external environment, they usually spontaneously abandon the sucking habit. Interruption of sucking habits during the deciduous dentition can provide self-correction of the anterior open bite. However, persistence of the habit until the mixed dentition represents a deviation from normality, because these habits are potent etiologic malocclusion factors, particularly for anterior open bite (Ogaard et al. 1994; Bishara et al. 2006).

Pacifier or thumb-sucking act as mechanical obstacles, preventing eruption of the anterior teeth and establishing an open bite (Moyers 1988; Proffit et al. 2007) (Figure 1.4). Anterior open-bite malocclusion due to pacifier use is characteristically restricted to the anterior teeth and circular (Figure 1.5). Anterior open bite consequent to thumb-sucking is characterized by labial inclination of spaced maxillary incisors and lingual inclination of the mandibular incisors (Figure 1.6). Anterior open bite may be associated to maxillary constriction and uni- or bilateral posterior crossbite, because, during sucking, the tongue is lowered, without contact with the maxillary posterior teeth (Moore 1996).

However, a deleterious oral habit does not always necessarily results in an open bite. First, it depends on how the habit is exercised, that is, it
Pacifier and thumb-sucking are strong etiologic factors for open-bite malocclusion. These factors are important in the etiology of this malocclusion and are known as Graber’s Trident (Graber 1958). Besides its mode of action, another important factor in the onset of an anterior open bite is the facial growth pattern. In the presence of deleterious habits, patients with vertical growth pattern have a greater tendency to manifest anterior open bite than patients with horizontal growth pattern (Schendel et al. 1976; Nielsen 1991). Therefore, the manifestation of an open bite depends on the association of environmental (Graber’s Trident) and genetic factors (facial growth pattern). This explains why there are children with habits but without an open bite (Figure 1.7).
Figure 1.5  Anterior open bite caused by the use of pacifier is characterized by being restricted to the anterior region of the dental arches and circular.

Figure 1.6  Thumb-sucking characteristically causes labial inclination of the maxillary incisors and lingual inclination of the mandibular incisors.
Prevention requires that intervention of thumb-sucking habit is started as soon as possible. Several studies (Graber 1966; Moore 1996; Vadiakas et al. 1998) have suggested the use of orthodontic pacifiers as a preventive step in thumb-sucking habit, based on findings that pacifier habit tends to be discontinued earlier than thumb-sucking habit.

**Anterior tongue posture and tongue thrust**

Atypical tongue posture and atypical tongue thrust is present in 100% of cases with anterior open bite (Fujiki et al. 2004). The tongue is considered to have a secondary role in the etiology of anterior open bite because it can maintain or aggravate the existing open bite when placed between the anterior teeth (Speidel et al. 1972; Subtelny 1973; Proffit and Mason 1975; Nielsen 1991; Proffit et al. 2007). This abnormal tongue placement may occur at rest, during speech, and swallowing. Incorrect tongue posture at rest has the greatest deleterious potential because, although the force is low, it remains during a large period of time between the teeth (Ingervall and Janson 1981) (Figure 1.8). On the other hand, during swallowing, in
which the force is much greater, the mean period of tongue thrust is between an hour (Straub 1951) to an hour and a half (Graber 1963) within 24 hours, and therefore presents a very small potential of maintaining or accentuating the open bite (Subtelny 1973; Moyers 1988; Proffit et al. 2007).

Tongue thrust during swallowing is considered to be consequent to a previously established anterior open bite caused by deleterious oral habits. This is explained by the physiologic maturation of swallowing. The child without erupted deciduous teeth presents an infantile swallowing, which is normal at this stage. The characteristics of an infantile swallowing are separated jaws, active contractions of the musculature of the lips, tongue placed between the gum ridges, in contact with the lower lip, and little activity of the posterior tongue or pharyngeal musculature. When the deciduous teeth are completely erupted, around 3 years of age, there is maturation of the facial and masticatory muscles, and the child develops the mature swallowing pattern. During mature swallowing, the teeth occlude, the mandible is stabilized by the muscles supplied by the trigeminal nerve, the tip of the tongue should contact the incisive papilla, and the labial muscles are passive (Subtelny 1973; Moyers 1988; Proffit et al. 2007).

In order to swallow, negative pressure must be obtained by sealing the mouth. Therefore, the child with an open bite thrusts the tongue between the teeth and strains the lips to seal the mouth (Proffit et al. 2007) (Figure 1.9). With an open bite, maturation of the masticatory musculature occurs

![Figure 1.9](image-url) Lips strain in an open-bite child in order to seal the mouth.
differently, changing the normal pattern to an atypical swallowing pattern or tongue thrust swallow. In this type of swallowing, there is atypical tongue thrust, absence of masseter contraction, and activity of the perioral muscles. Therefore, tongue thrust during swallowing is considered to be a secondary factor in open-bite etiology: first, there is development of the open bite, and posteriorly, tongue adaptation through its placement between the anterior teeth (Proffit et al. 2007).

**Mouth breathing**

Normal breathing consists in the air flow through the nasal cavity where the nostrils promote purification, heating, and humidification of the inhaled air before reaching the lungs. Patients with nasal obstruction do not have these benefits when breathing through the mouth. In the presence of some nasal obstruction, the air flow is impaired or obstructed, and the child begins to breathe through the mouth. The etiology of nasal obstruction can be divided into upper and lower breathing obstacles. The upper obstacles are hypertrophied adenoids, allergic rhinitis, nasal turbinates hypertrophy, and nasal septum deviation. The lower obstacles are hypertrophied tonsils or frequent tonsillitis (Watson 1981; Linder-Aronson et al. 1986).

In the primary dentition, the prevalence of anterior open bite in mouth-breathing children is similar to the general population (30%), whereas in the mixed and permanent dentition, this prevalence remains almost the same in mouth-breathing individuals, but decreases in the population (between 12% and 20%) (Souki et al. 2009).

**Upper respiratory obstacles**

**Hypertrophied adenoids**

Adenoid hypertrophy is the abnormal growth of the adenoids, and in some situations, this growth is so exacerbated that can cause a complete blockage of the air passage through the upper airways. Even in situations that do not lead to a complete blockage of the air passage through the upper airways, hypertrophy of the adenoids can promote a partial blockage of the air passage through the nostrils and cause great discomfort to the patient in an attempt to breathe through the nose. Thus, the patient begins to breathe through the mouth (Gates 1996). There are two main ways to assess adenoid size: (1) indirect assessment with the nasopharyngeal mirror and (2) lateral headfilm. The use of the nasopharyngeal mirror is sometimes not possible in the young child, and in these cases, the lateral headfilm represents the best way to assess adenoid size (Figure 1.10) (Weimert 1986).
Although the abnormal growth of adenoids can cause some pediatric sleep-disordered breathing, multiple anatomic obstructions should also be considered (Guilleminault et al. 2005; Huynh et al. 2011). Children with increased sleep-disordered breathing and obstructive sleep apnea symptoms seem to share some common characteristics in the vertical plane, such as long faces, retropositioned mandibles, and lip incompetence (Pirila-Parkkinen et al. 2009; Huynh et al. 2011). Some characteristics of these patients could also be observed in the transverse plane, such as maxillary constriction, which usually occurs simultaneously with reduced transverse dimension of the upper airways, and increased nasal resistance, which consequently increases mouth breathing.

Enlarged adenoids can contribute to the worsening of mouth breathing, causing an imbalance of lingual, labial, and cheek muscles (Linder-Aronson et al. 1993). This imbalance might be associated with unfavorable changes in the development of occlusion and anomalies of dental position. Although there is no clear understanding of the relationship between observed skeletal and dental changes with airway obstruction, some disruption in the normal functional environment may be responsible for these unfavorable changes. Considering the assumption that upper airway obstruction, regardless of cause, influences negatively normal facial development, the young child with airway obstruction has to be carefully handled during growth (Bresolin et al. 1983).
When the blockage of the upper airway is in the nasal cavity, treatment may be more difficult when compared with hypertrophied adenoids. Nasal obstruction can occur anywhere in the upper airways, and identification of the causative agent can be difficult to diagnose (Bresolin et al. 1983). If allergic rhinitis is the causative agent, removal of inciting allergens should be helpful. Antihistamines and decongestants frequently cause an improvement in the clinical condition of patients because they shrink the nasal mucosa (Bresolin et al. 1983).

Allergic rhinitis

Allergic rhinitis is a medical term describing irritation and acute or chronic inflammation of the nasal mucosa. The inflammation caused by allergic rhinitis results in excessive mucus production, generated by the accumulation of histamine, which causes nasal bleeding, the typical symptom of rhinitis. Allergic rhinitis occurs when an allergen, such as pollen or dust, is inhaled by an individual whose his immune system has been previously sensitized to these agents (May and Smith 2008).

Allergic rhinitis can be classified as seasonal or perennial. Seasonal allergic rhinitis occurs rarely before 6 years of age and has its signs and symptoms exacerbated in times of pollen. The perennial allergic rhinitis can occur at any time during the year and occurs more frequently in younger children. The main symptoms of allergic rhinitis are rhinorrhea (excess nasal secretion), itching, and nasal congestion and obstruction (Sur and Scandale 2010). Allergic rhinitis is one of the main causes of nasal airway obstruction in the young child (Rubin 1980).

Nasal septum deviation

The nasal septum is the part of the nose that separates the two airways and the nostrils. A deviated septum is when there is a shift from the midline or center position. In normal conditions, the nasal septum is centralized, and the air passages in the nasal cavity are symmetric. The nasal septum deviation is an abnormality in which a portion of the cartilaginous tissue deviates to one side of the nostril causing an obstruction for the airway passage on the side on which the deviation occurred. These deviations are common, and for most patients, cause no symptoms, and no treatment is required. Many people with a deviation are unaware they have it until their health is affected or discomfort is large enough. By itself, a deviated septum can go undetected for years, and thus be without any need for correction (Metson and Mardon 2013). However, the septal deviation may be severe enough to adversely affect the patient and can commonly obstruct the passage of air through the nostrils (Figure 1.11).
Open-Bite Malocclusion: Treatment and Stability

The main etiological factors of nasal septum deviation are impact trauma, such as by a blow to the face and a congenital disorder, caused by compression of the nose during childbirth (Metson and Mardon 2013). The main symptoms of a deviated septum are infections of the sinus and sleep apnea, snoring, repetitive sneezing, facial pain, nosebleeds, and breathing difficulty.

Some differences can be highlighted when comparing skeletal and dental features in children with chronic nasal-breathing obstruction secondary to nasal septum deviation and nose breathing. Regarding linear dimensions in the vertical plane, children with chronic nasal-breathing obstruction due to nasal septum deviations usually present increase of upper anterior facial height (N-palatal plane) and total anterior facial height (N-Me) in comparison with nose-breathing subjects. Considering the angular relationships of the horizontal planes of the face, such as sella-nasion, palatal plane, and occlusal planes to the mandibular plane, these angles are usually greater in mouth breathing due to nasal septum deviations in comparison with nose-breathing subjects. Additionally, the gonial angle (Ar-Go-Me), palatal height, and overjet are significantly higher in mouth-breathing subjects. Regarding the anteroposterior position of the jaws, a retrognatic maxilla and mandible is frequently observed in mouth-breathing patients due to nasal septum deviations in comparison with nose-breathing subjects. Some differences can also be observed in the occlusal aspect. Mouth-breathing children due to nasal septum deviations usually present Class II malocclusion, whereas most of nose-breathing subjects present normal occlusion (D’Ascanio et al. 2010)

However, the association between nasal resistance and open bite is of only 8.2% when evaluating children from 7 to 15 years of age (Lopatiene and Babarskas 2002).

Figure 1.11 Nasal septum deviation obstructing the airway passage through the nostrils seen in a computed tomography examination.
Lower respiratory obstacles

Hypertrophied palatine tonsils

The tonsils are two structures forming part of the lymphatic system that are located at the entrance of the upper airway and which has as main function to help the body’s defense against respiratory infections or preventing the entry of organisms through the mucosal respiratory system. Because it represents one of the first sources of defense against microorganisms, it is a region potentially subject to infections and is one of the major immunocompetent tissues of the oropharynx. It is a part of Waldeyer’s ring, which is also formed by the nasopharyngeal tonsil or adenoid (NT), the paired tubal tonsils (TT), the paired palatine tonsils (PT), and the lingual tonsil (LT) (Merati and Rieder 2003).

Tonsillitis is an infection of the tonsils that causes inflammation and represents the most common acute manifestation of tonsillar pathology (Figure 1.12). The main symptom of tonsillitis is sore throat, and the pain is usually worse when swallowing. Other symptoms can include fever, general ill feeling, headaches, and vomiting. In a clinical view, the tonsils may present with normal size or enlarged and often with the presence of erythematous areas. The presence of exudates and secretions is common but does not occur in all clinical situations. In fact, not all of these signs and symptoms are present in all patients diagnosed with tonsillitis (Merati and Rieder 2003).

Tonsillar hypertrophy is the enlargement of the tonsils, but without the presence of inflammation. The obstructive tonsillar hypertrophy is the
main indication for tonsillectomy, which is the surgical removal of the tonsils. The main signs and symptoms that may be observed in patients with tonsillar hypertrophy are sleep disturbances in a wide spectrum of severity, loud snoring, irregular breathing, coughing, night choking, interrupted sleep apnea, dysphagia, and excessive daytime sleepiness (Merati and Rieder 2003).

Radiographs are not particularly useful in evaluating tonsil size. Direct visualization is far more useful when compared with lateral cephalograms that gives a rough impression of tonsil size (Figure 1.13) (Weimert 1986). A large variation in tonsil size is frequently common.

Nowadays, there are specific indications for surgical removal of tonsils, and the most common indication is recurrent infection (Figure 1.14) (Weimert 1986).

To be able to breathe, the mouth-breathing child remains most of the time with the mandible in a lowered position to keep the mouth open. The tongue follows the mandible and consequently will not establish contact with the palate in the rest position, as usual, during nose breathing (Proffit et al. 2007). The absence of lateral contact of the tongue with the palate results in predominance of lingual forces of the buccinator muscle, and this consequently will result in smaller transverse development of the palate, which can cause posterior crossbite (Figure 1.15). Concurrently, there will be greater posterior vertical development of the palate. Besides, the tongue can rest over the anterior teeth, restricting their vertical development and contributing to anterior open bite. Notice that mouth breathing is a common etiologic factor for posterior crossbite and anterior open bite. That is the reason why many times, these malocclusions are associated. The associa-
Hypertrophied tonsils are also considered as a contributing factor for anterior open bite because it causes mouth breathing and anterior tongue posture. Increase in tonsil size impairs the air flow through the lower respiratory airway (Moyers 1988; Proffit et al. 2007). Therefore, to free the lower airway and breathe normally, the child has to open his/her mouth to be able to project the tongue anteriorly, and since the mouth is open, there will be also mouth breathing. The anteriorly projected tongue can be placed between the teeth, preventing the normal vertical development of the incisors and maintaining and/or aggravating the open bite (Subtelny 1973; Harvold et al. 1981).
Tongue thrust due to tonsillar hypertrophy can also occur in the premolars and molars region, causing a posterior open bite. This condition usually occurs when there is early loss of posterior deciduous teeth during the stage of occlusal development. Thus, the tongue loses its lateral shield and ends up placed in spaces between the posterior teeth during swallowing, limiting eruption of permanent teeth.

**Traumatisms**

Dental traumatisms can cause ankylosis—which possibly comes from some kind of injury—which causes changes in the periodontal ligament, forming a bone bridge joining the cementum and the lamina dura. Ankylosis of any primary tooth may cause, beyond its retention, a delay or even an ectopic eruption of the permanent successor and may cause open-bite malocclusion (Mandava and Kumar 2009) (Figure 1.16).

**GENETIC FACTORS**

**Growth pattern**

In orthodontics, patients can be classified into three groups according to their growth pattern: horizontal, normal, or vertical growth pattern.
Etiology of open-bite malocclusion

Patients with horizontal growth pattern usually present low mandibular plane and gonial angles, decreased lower anterior face height, deep overbite, increased free-way space, decreased molar and incisor dentoalveolar heights, and greater biting forces than patients with vertical growth pattern (Isaacson et al. 1971; Peterson et al. 1983; Trouten et al. 1983; Dung and Smith 1988; Nanda 1988, 1990; Janson et al. 1994). Patients with normal growth pattern have balanced characteristics between these extreme growth patterns. Consequent to these characteristics, patients with vertical growth pattern are also more susceptible to the environmental influences predisposing to open bite, and consequently present this malocclusion most frequently (Schudy 1964; Dung and Smith 1988; Nanda 1988, 1990). This does not mean that every subject with a vertical growth pattern will present an open bite because there may be a compensatory eruption mechanism (Dung and Smith 1988; Betzenberger et al. 1999). However, subjects with an open bite usually will present more vertical characteristics than subjects with a balanced or horizontal growth patterns (Trouten et al. 1983; Nanda 1988, 1990).

Generally, when the growth pattern causes severe skeletal deformities either in the vertical and/or in the anteroposterior planes, the best treatment consists in an orthodontic-surgical approach (Beane 1999).

Pathologies

Craniofacial anomalies

Some congenital deformities and syndromes can cause malocclusion, including anterior open bite. Cleidocranial dysostosis is a congenital deformity usually associated with heredity, which may be associated with the presence of anterior open bite (Daskalogiannakis et al. 2006).
Treacher Collins syndrome involves hypoplastic mandible, glossoptosis, small size of the pharynx and nasopharynx, and occasionally choanal atresia can cause severe breathing problems, and consequently, open-bite malocclusion (Shete et al. 2011) (Figure 1.18).

Excessive activity of the tongue, in the act of swallowing or even at rest, can change the incisors axial inclinations and cause an open bite. In patients with some degree of neurologic impairment, this is observed quite frequently (Pedrazzi 1997) The compensatory coordination of the tongue movement, the movement of the soft palate and the pharyngeal constrictor muscle activity, still occur during swallowing in these patients (Fujiki et al. 2000).

Juvenile rheumatoid arthritis is a disease with oral involvement with anterior open bite, limited mouth opening, and in most of the cases, compromises the temporomandibular joint (Savioli et al. 2004; Carvalho et al. 2012).

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