

Etiology of anterior open bite: a review

Ätiologie des frontalen offenen Bisses: Ein Review

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Abstract

Objectives Presentation of different causes described in the literature for development of anterior apertognathia.

Methods A review about data referring to patients limited in their anterior tooth function through the positioning of their teeth has been performed. Electronic data bases, two libraries have been searched for information. Of the identified titles 357 articles and chapters were selected. 43 of these were considered.

Results Aetiology classifications are inconstant and author dependent, but mostly differentiated into genetic and environmental causes. Specifically named are: habit, tongue, airway obstruction, neuromuscular deficiency, trauma, rheumatoid disease, posture and posterior discrepancy.

Conclusions Many different factors can be relevant for the development of apertognathia and have to be considered. Further research could help differentiating which of the partially contradictory statements are true.

Keywords Open bite · Etiology · Tongue · Neuromuscular deficiency · Rheumatoid disease · Posterior discrepancy

Zusammenfassung

Ziel Die Darstellung der verschiedenen in der Literatur

beschriebenen Ursachen für das Entstehen von anterioren Apertognathien.

Methode Es erfolgte eine systematische Suche von Veröffentlichungen zu Patienten, die durch ihre Zahnstellung in der Funktion der Schneidezähne eingeschränkt sind. Elektronischer Datenbanken und zwei Bibliotheken wurden zur Datensammlung herangezogen. Von den gefundenen Titeln wurden 357 genauer bearbeitet und 43 letztendlich verwendet.

Ergebnisse Die Einteilung der Ursachen kann je nach Autor unterschiedlich sein, lässt sich aber generalisiert in genetisch und umweltbedingt einteilen. Genannt werden dabei: Habits, die Zunge im Speziellen, neuromuskuläre Defizite, Atemwegsobstruktionen, Haltung, posteriorer Platzmangel, Trauma und rheumatoide Erkrankungen.

Schlussfolgerung Viele verschiedene Faktoren können bei der Entwicklung dieser Dysgnathie relevant sein und müssen bei der Diagnose in Betracht gezogen werden. Weitere Forschung in diesem Bereich könnte zeigen, welche der zum Teil widersprüchlichen Aussagen zutreffend sind.

Schlüsselwörter Offener Biss · Ätiologie · Zunge · Neuromuskuläre Defizite · Rheumatoide Erkrankungen · Posteriorer Platzmangel

Introduction

Apertognathia is a malocclusion on which authors only agree that it is difficult to treat [5, 7, 9, 15, 18, 19, 23, 25, 27, 34, 38, 39]. Sagittal and transverse deviations have been researched, defined and successfully treated for a longer time, leaving less questions, than in the vertical plane [42]. Origins of open bites are especially important in

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growing subjects since in this group most apertognathias are self-correcting [15] and unnecessary treatment should be avoided. Yet it is difficult to find the reason for this malocclusion. Some patients might present several of the discussed causes without showing an apertognathia, while others might have the malocclusion without visible cause. It is essential to know which problems need to be eliminated before treatment can be commenced and which specialists should consequently be consulted.

Definition

There is no consensus in the literature on how exactly to define open bite, although it would seem this anomaly has distinct, easily recognizable characteristics [19]. The current literature reveals that definitions vary, sometimes by country [34]. Open bite has been specified to a certain degree by eliminating less-than-average overbites or edge-to-edge relationships, and by the claim that a certain degree of aperture and lack of contact must be present [35]. Yet the question regarding how to define a “certain degree” remains.

Apertognathia is usually considered to be located in the front, as a strictly vertical problem, and the opposite of deep bite [26]. Lateral or posterior open bite is usually specified as such since it seldom occurs [7]. A patient who presents a negative vertical incisor “overlap” clearly has an apertognathia. Opinions begin to diverge when positive overlap is present but no incisor contact can be achieved, e.g., in the United States, that would be considered an open bite, in the United Kingdom an incomplete overbite [34]. Some define this malocclusion as a clinical manifestation in which there is no contact and no vertical overlap [18, 39], others as one in which there is no contact or no vertical overlap [17].

If malocclusions are defined according to missing functions, patients must be able to incise with their incisors and chew with their posterior teeth [18, 38]. Proper articulation and pronunciation must be possible [38]. Patients with apertognathia have such problems [23]. Total functionality is only possible when the contact between teeth is complete.

Few authors claim that a case of apertognathia is not genuine if patients can incise with the mandible moved forward [34], yet that would imply that a malocclusion does not exist if it can be improved via jaw movement.

In summary, the best definition of apertognathia is a lack of contact between maxillary and mandibular teeth [40] in centric relation [9, 34, 42]. This can occur with a class I, II, or III pattern [17]. To determine just the vertical dimension, the overbite should be assessed in relation to the Frankfurt horizontal plane, the equivalent of the spatial reference plane in a patient standing upright.

Method

We conducted a thorough review of the literature (Fig. 1). Electronic databases were searched via PubMed and Web of Science by applying the following criteria:

- keywords: “open bite”, “apertognathia”, “apertognathic” and
- the years 2004 to mid-2014.

Two libraries were searched for additional information in books and magazines. The databases ScienceDirect and Google Scholar were not just helpful in retrieving articles, they also suggested further articles that we considered. Reference analysis was also part of our data acquisition. Once the basic concepts behind possible etiologies were identified, we carried out another search via less frequently mentioned causes, namely:

- keywords: “rheumatoid disease”, “neuromuscular deficiency”, “posterior discrepancy”, “airway obstruction” and
- the years 2009 to mid-2014.

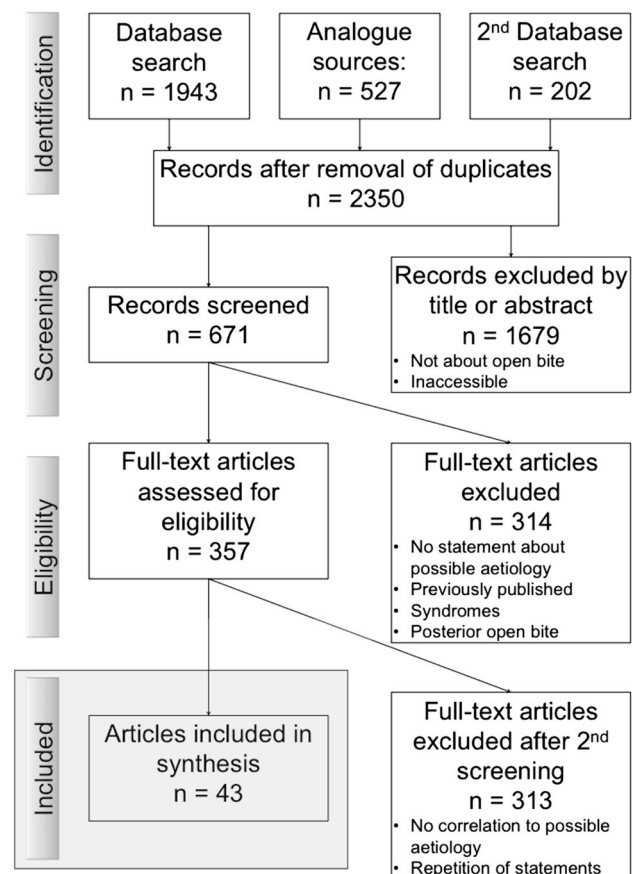


Fig. 1 PRISMA flow diagram
Abb. 1 PRISMA-Flussdiagramm

We did the same for “trauma” and “posture”, but combined those with the terms from the first search, as too many irrelevant results would appear otherwise.

Results

We identified 2350 articles and screened them by their title and abstract. Most of the 43 documents we ultimately considered were published in English.

As mesially tipped posterior teeth can already trigger an anterior open bite [27], it is important to discover the factors that lead to the tooth and jaw positions. The etiologies have been grouped into skeletal and dental causes, supplemented by functional and habitual ones [4]. Another approach is to categorize them according to genetic, environmental [18, 23, 38], and anatomic etiologies [18]. Each of these systems has its rationale. However, clear differentiation seems to be as difficult as distinguishing dental from skeletal open bites. Both are anatomical characteristics partially determined by genetics, and influenced by functional and habitual factors—thus, the environment.

Heredity [17] plays a key role in facial patterns [27]. Evolution has led to genetically determined smaller jaws [26], more vertical facial structures, and a consequently higher risk for open bites [26]. It is not entirely clear which demographic changes lead to smaller jaws [10], but there is a genetic component [24]. Height may be the second most common facial characteristic that runs in families [26], making it an important etiological factor in patients presenting vertical excess [38]. Comparison between ethnic groups can help provide some evidence. Growth patterns often determine at least the severity [17, 27]. Although the genetics seem to correlate closely with craniofacial dimensions, dental arch variations seem to be more strongly influenced by the environment [26]. The magnitude and, in particular, duration of forces enabling even dentoalveolar effects need to be sufficient. About 6 h per day are needed to affect the equilibrium. The skeleton is not easily distorted, but it can be altered through constant functional demand [26]. Environmental pressures and forces can change physiologic stomatognathic activity [26, 30] which in turn can influence bone density [26], as well as maxillofacial growth and development [30]. Skeletal structures compensate the demands from oral functional matrices, which may lead to open bite [17]. Mandibular forms—with their functional processes—alter according to surrounding pressure and guidance [26]. Alveolar processes diminish after tooth loss if not stressed otherwise. Muscular processes adapt to muscle strength, and condylar processes compensate for mandibular forces when articulating with the skull. A relationship between anatomic form

and physiologic function is inevitable [26]. Vertical growth in the molar region must be compensated by condylar or ramus growth. If not, apertognathia can occur [6, 23]. Apertognathia can accumulate in families because they reveal hereditarily influenced reactions to the same environment [26]. Many factors interact and require consideration. Although the various etiologies of open bite are not unequivocally definable, it is only possible to influence and camouflage genetic effects, while environmental factors can be altered completely.

Discussion

Habit

Numerous habits [5, 17, 27, 41] can cause a bite to open, but until about the age of 3 years, no negative growth influence should be expected. The likelihood of acquiring an open bite rises with permanent tooth eruption [22, 26, 39]. The prevalence of apertognathia doubles when there is a sucking habit [17]. All habits involve a mechanical obstruction that impedes eruption [5, 18, 38] or allows the supra-eruption of teeth continuously lacking contact [26]. Lowering the tongue reduces maxillary oral impact [26] and may widen the mandible [1]. This imbalance between tongue [1, 39] and perioral musculature pressure [26, 39] can lead to a cusp-to-cusp relationship, keeping the mandible rotated clockwise and preventing incisor contact. Negative sucking pressure can exacerbate unwanted forces. However, muscles are assumed to have stronger influences [26].

Some say the tongue plays a role in habits [23] when it is thrust forward or in the wrong position during swallowing [41]. However, swallowing is not by definition a habit. It is a physiological, not learned act that adapts subconsciously to the surroundings [26].

Tongue

Three attributes of the tongue are controversial, namely its function, posture, and size. The tongue initiates swallowing and prevents anything from leaking out [18, 26]. Placing it anteriorly to create this seal is physiological when infants swallow [26]. In an apertognathic situation, the pattern is similar. Some postulate this as a malfunction opening the bite [5, 27, 34, 38], while others say it is always present without mentioning whether it is a cause or result [19, 25, 38]. After all, just one in ten children with a reported “thrust” swallow has an open bite. A period of transition between the two patterns seems probable, and in patients with a habit, it can be expected to last longer [26] because until the bite closes, the tongue seal is a physiological

- < 1000 swallows / day = norm
- each swallow = 1 second
- 1000 seconds pressure =
16 minutes & 40 seconds
pressure / day

Fig. 2 Swallowing pressure per day
Abb. 2 Beim Schlucken pro Tag generierter Druck

necessity [39]. A mature pattern is never achieved in 10–15 % of patients [26], again a percentage higher than that of apertognathic adults. Swallowing as a result only [18, 26] has also been proven in studies demonstrating only low force levels [12], even lower levels than in a normal pattern [26]. The total amount of force is, at less than 20 min per day, not nearly enough to influence tooth positions (Fig. 2) [26]. Not until the resting posture [12, 17, 26, 27, 34] has been altered can enough impact be created to trigger malocclusion. A faulty resting posture may be associated with a visceral swallowing pattern, but posture is likely to be more important [12, 26, 34]. This “relative” macroglossia can change tooth positions, just as seldom-occurring genuine macroglossia can [5, 18], which is not easily influenced. Indicators are flared anterior teeth [39] and a tongue with lateral indentations, extending onto occlusal surfaces [39].

Airway obstruction

Nasal airway obstruction such as adenoid hyperplasia [17, 19], allergic swelling, or other anatomic blockages [14] can lead to oral respiration [27] with a more backward head [22, 26] and low tongue position. Just as in patients with habits, this can result in a more hyperdivergent growth pattern [22] with apertognathia [1, 19, 26, 31, 38]. People with a dolichofacial-type face are more likely to be mouth breathers. However, a minority of oral respirators develops this malocclusion [26]. When assessing the risk of developing apertognathia, one needs to distinguish habitual lip separation at rest from one for breathing [26]. These patients are characterized as having adenoid faces with darkly circled eyes [23], and narrow cheeks and nostrils [23]. Their postural adaptation to the breathing pattern can lead to altered muscular strength [23].

Neuromuscular deficiency

There is debate about the relation between muscles and open bite [5, 11, 17, 25, 26, 30, 31]. The facial muscles influence bone formation at their insertion points according to activity [26], and they are important in the functional, rotational adaptation of the lower to upper occlusal plane.

When there is too little activity, the planes may diverge and anterior open bite can develop [30]. Weaker chewing muscles supposedly lead to supra-eruption of the posterior buccal segments [39]. When this occurs nowadays, it is attributed to the consumption of processed foods [26, 38]. However, people display a vertical growth pattern before the musculature’s activity becomes discernable [26]. On the other hand, some researchers propose that apertognathia leads to low muscle tone, instead of being the result thereof [25, 26]. It may be caused by the reduced palatal arches these patients often have [25]. Those with apertognathia are likely to be missing the input of anterior guidance. Without such input, they assume a motoric pattern involving narrower and shorter chewing cycles and lower muscular activation [25]. This seems likely, since the muscular activity of open and deep bite patients resemble each other after orthodontic surgical treatment [11].

Trauma

Trauma is usually easy to differentiate. Dentoalveolar or skeletofacial trauma [39] can affect occlusion [26, 40]. Traumatic forces against the primary dentition can trigger ankylosis in the secondary dentition, just as a direct force can, resulting in a partially missing overbite.

A blow against the mandible can lead to arrested condylar growth, and an ankylosed or fractured condyle, resulting in an apertognathia [39, 40]. A Le Fort I fracture can also lead to deflective occlusal contacts [40] in conjunction with anterior bite opening.

Rheumatoid disease

Rheumatoid disease in the temporomandibular joint [33] induces condylar degeneration. Although less common [5], it can result in acquired apertognathia [28, 39, 40]. When joint and surrounding structures degrade, shape changes occur [28]. A loss of condylar height leads to a retrognathic open bite [28]. Inflammatory and noninflammatory causes have been reported separately, or secondary to each another.

During joint function, load causes the articular disc to become temporarily deformed as a kind of adjustment. Biomechanical properties like stiffness dictate the nature and amount of load that can be endured [16]. Constant remodelling keeps joint form, function, and occlusal relationships in equilibrium [2, 36]. Tissues can undergo irreversible injury when forces exceed adaptive capacities [36] either because forces are excessive or the tissue’s capacity to adapt is diminished through continuous strain, age, inflammation or otherwise, leading to stiff or fragile discs [36]. As causes of inflammatory degeneration, various types of arthritis [33, 39] are usually mentioned. Many

studies have also demonstrated that compression leads to gradual osseous condylar resorption [2]. Occlusal discrepancies can cause temporomandibular disorders [8, 23], although many people with malocclusion do not have a joint problem [8]. However, in a joint with reduced adaptive capacity, occlusal relationships can be decisive by preventing excessive load of the structures. Though opinions differ on correlations between occlusal interferences and temporomandibular joint disorders, most have detected an association with apertognathic patients [23, 40].

Posture

Postural changes in the tongue, mandible, or head can cause open bites [23, 26, 27]. As mentioned earlier, secondary long-term changes in soft tissue equilibrium affect this. A more backward head posture with a maxilla rotating counterclockwise aggravates jaw separation, allowing molars to supra-erupt [26]. Evolutionary changes in humans to an upright body and head posture have resulted in skulls with small anteroposterior dimensions [30, 31]. The smaller jaws [26] give modern humans retrognathic profiles, accompanied by vertical growth tendency. The downward growth of the maxillary occlusal plane in particular leads to the bite opening [30, 31] if the mandible stops adapting [30].

Posterior discrepancy

Posterior discrepancy is associated with apertognathia [3, 15, 29–31]. Evolutionarily smaller jaws are partly responsible for crowding [10, 26, 31], the most common malocclusion [13, 26, 32]. It also correlates with divergent jaw growth [13]. Research has focused more specifically on the main patient complaint of anterior crowding [20, 24] even though it occurs throughout the dentition [20]. As crowding worsens throughout dental development [13, 32], it is indicated that the teeth erupting last are crucial [32]. In the restricted space allotted to the molars, room for all three molars is often missing, leading to posterior crowding [31]. There is always potential to disturb the periodontal equilibrium of eruptive forces, as is apparent when teeth proceed to erupt after the loss of their antagonist [26]. Molars are the last teeth to erupt—one of many changes during growth [30]. Postpuberty growth spurts occur, and this growth proceeds because third molar development has not usually finished by the end of puberty. It is called the fourth physiological bite raising. The first is due to the eruption of the deciduous molars, the second to the first permanent molars, the third to the second permanent molars, and the last to the third molars [21].

Due to the lack of adequate space, third molars become impacted [31, 32] and physiological bite raising becomes

pathological. The third molars seek mesial development, putting pressure on already sufficiently erupted teeth, resulting in mesial tipping [32] and supra-eruption [3, 15, 32]. Occlusal changes occur [15] that steepen [29] or flatten the planes [29, 37]. Since mandibular position is determined by molars [38], a vertical increase can turn molars into hypomochlions, leading to functional mandibular dislocation [15, 30]. The condyle can be loaded, thereby regulating growth; rotational mandibular modulation can also take place [30]. If adaptive capacities are exhausted, divergent occlusal planes lead to cases of apertognathia [15, 27, 29, 30]. A 1 mm molar supra-eruption can trigger an anterior bite opening of 2–3 mm [5, 26, 39], predisposing anyone to this malocclusion [26, 31].

Conclusion

When patients present missing incisor function, many various influences need to be considered. When making the differential diagnosis, it is important to know about any possible trauma. Etiologies like habits and posterior discrepancy should be eliminated to enable proper treatment. Some diagnoses necessitate cooperation with colleagues from other specialties such as otorhinolaryngology, speech therapy, or allergology. Patient records are likely to contain more than one potential etiology to which this malocclusion could be attributed, a factor that may explain some of the variation in this malocclusion.

Further research is necessary to clarify contradictory study findings. Fortunately for our patients, extreme forms of apertognathia are seldom, making it difficult to recruit enough study subjects for research to demonstrate statistical validity.

Compliance with ethical guidelines

Conflict of interest Christina Rijpstra and Jörg Alexander Lisson state that there are no conflicts of interest. The accompanying manuscript does not include studies on humans or animals.

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