INTRODUCTION

Posterior crossbite is a type of malocclusion characterized by a discrepancy between the maxillary and the mandibular arch in the transverse dimension [16]. It usually results from the decreased upper arch width. The reported prevalence ranges from 7% to 23% [2,12,21,26,31]. According to the presence on either one or both sides of dental arch, posterior crossbite can be classified as unilateral or bilateral. Unilateral posterior crossbite is predominant and occurs in 80% of patients with transverse discrepancies [21]. Constriction of the maxillary arch makes contact in centric relation unstable and leads to a functional shift of the mandible upon closure. This refers to a functional unilateral crossbite, whereas in a true unilateral posterior crossbite there is no mandibular shift and crossbite is observed in both centric relation and maximum intercuspation [2]. In growing patients most cases of unilateral crossbite are functional in nature. Narrowing of the upper arch is usually symmetrical and the facial asymmetry is related to the lateral deviation of the mandible, which may promote remodeling of the temporomandibular joint and impairment of normal mandibular growth [21,33].
Classification of posterior crossbite is based on the position of the maxillary teeth. The most common form, posterior lingual crossbite, is diagnosed when the buccal cusps of the maxillary teeth are palatal to the buccal cusps of the mandibular teeth. Posterior buccal crossbite occurs when the palatal cusps of the maxillary teeth occlude buccally to the buccal cusps of the mandibular teeth. Posterior crossbite can be a result of dental or skeletal abnormalities or a combination of the two. Dental crossbite is caused by improperly inclined or malpositioned teeth. Posterior crossbite of skeletal origin is defined as a discrepancy between the width of the maxilla and the mandible. In general, the more teeth occlude in crossbite, the greater skeletal component involved. Bilateral crossbite is mostly a consequence of maxillary skeletal deficiency. If it is associated with severe skeletal class III malocclusion, it should be left untreated or corrected as a part of combined orthodontic-surgical treatment.

The etiology of posterior crossbite is complex and involves hereditary and environmental factors. The well-known causes are nonnutritive sucking habits, airway obstruction leading to mouth breathing, lowered tongue posture and short duration of breastfeeding. It has been observed that posterior crossbite develops more often in children with early extractions of primary teeth.

Early correction of posterior crossbite with a functional shift of the lower jaw is indicated. If left untreated, this type of malocclusion may cause several changes in hard and soft tissues including development of skeletal asymmetries, alteration of soft tissue growth, adaptive changes in the temporomandibular joints and attrition of primary and permanent teeth. Spontaneous correction of posterior crossbite is unlikely. The reported frequencies of self-correction range from 17% to 45%. Posterior crossbite with a functional shift of the mandible is usually corrected by a symmetrical expansion of the maxillary arch. Cases of true unilateral posterior crossbite require unilateral expansion. Overexpansion of the unaffected side should be avoided.

NON-NUTRITIVE SUCKING HABITS

Sucking behaviors in infants and young children arise mainly from the physiologic need for nutrients but also from psychological necessities. Most children up to 24 months of age engage in sucking of a digit or pacifier. It is well-established that prolonged sucking habits are associated with development of various malocclusions such as anterior open bite, class II malocclusion and posterior crossbite. The increased prevalence of posterior crossbite in children with sucking habits results from a widening of the mandibular arch and narrowing of the maxillary arch. Studies have shown that pacifier and digit habits have different impact on the dental arch and occlusion. Bishara et al. found a significantly greater incidence of posterior crossbite in children with pacifier habits than those with digit habits. On the other hand, the incidence of increased overjet was significantly greater in children with digit habits than in children with pacifier habits. Both habits resulted in similar incidence of anterior open bite and class II canine relationships. Moreover, the
authors demonstrated no significant differences in occlusal characteristics between children breast-fed for 6 to 12 months and with no habits compared to children who were not breast-fed and had habits lasting less than 12 months [4]. This may indicate that short duration of non-nutritive habits (less than 12 months) has little or no effect on occlusion. However, finger or pacifier habits lasting more than 48 months have deleterious impact on dental arch morphology [4]. Children who used pacifiers for longer than 3 years have 5-fold increased risk of posterior crossbite development [5].

**BREASTFEEDING**

The literature suggests that exclusive bottle-feeding in the first 6 months of life is a risk factor for non-nutritive sucking habits after 12 months of age. On the contrary, children who are exclusively breastfed show lower risk of non-nutritive habits development [24]. Breastfeeding has many beneficial effects on growth and development of craniofacial region. It affects the movement of masticatory muscles and promotes correct swallowing pattern and breathing [5]. There are differences in the activity of the oral and facial muscles in breastfed and bottle-fed infants. Early introduction of bottle-feeding may impede normal development of the maxillary arch and result in posterior crossbite. Children diagnosed with posterior crossbite were breastfed for shorter period of time compared to patients with no posterior crossbite [12]. Another study showed that children who were breastfed for more than 12 months had a 20-fold lower risk for posterior crossbite development compared with children who were never breastfed and a 5-fold lower risk compared with those breastfed between 6 and 12 months. [14].

**UPPER AIRWAY OBSTRUCTION**

The hyperplasia of adenoids and tonsils causes many health problems including Eustachian tube dysfunction, rhinosinusitis, obstructive sleep apnea, swallowing disturbances, reduced ability to smell and taste, halitosis, speech disorders and abnormal facial growth. The nasal airway obstruction forces a patient to breathe through the mouth. This in turn alters the soft tissue pressure on the jaws and teeth and affects facial growth pattern. Postural changes such as lip incompetence, lowered tongue position, proclination of the upper incisors, posterior rotation of the mandible and head tilted backwards appear as a consequence of mouth breathing [20,35,39]. Giuca et al. found higher prevalence of posterior crossbite in children with otitis media compared to healthy controls. Moreover, the authors showed that hypertrophic adenoids and tonsils were more common in otitis media group [9]. Allergic rhinitis is one of the main causes of airway obstruction and is reported to be a risk factor for increased overjet and posterior crossbite development [20]. Diouf et al. demonstrated that the grade of tonsillar hypertrophy correlates negatively with the maxillary intercanine and intermolar widths. The authors also found a significant positive correlation between the palatal vault height and degree of the tonsillar hypertrophy [7]. Snoring and obstructive sleep apnea in childhood, usually caused by adeno-tonsillar enlargement, may result in restless sleep, daytime fatigue, aggressiveness, hyperactivity and delayed
growth. Children with obstructed nocturnal breathing have narrower maxilla, increased anterior facial height, shorter lower dental arch, posterior mandibular rotation and higher prevalence of posterior crossbite [19]. Patients with genetically constricted maxilla have an increased risk of snoring and sleep disordered breathing during the period of physiological adenoid hypertrophy. The reduction in maxillary width persists throughout childhood independently of performed adenotonsillectomy. This suggests that orthodontic widening of the maxilla can be utilized in cases of chronic snoring and upper arch narrowing [18]. It is reported that maxillary expansion improves sleep characteristics by increasing sleep efficiency and total sleep time [1].

**TEMPOROMANDIBULAR JOINT AND MUSCLE FUNCTION**

Occlusal interferences, which usually appear as a result of maxillary constriction, force the mandible to shift laterally into a position of maximal occlusal contact. This causes improper stress and growth disturbances in temporomandibular joints. Untreated functional unilateral posterior crossbite may lead to mandibular and facial asymmetry [15,17]. Several studies found association between the presence of unilateral posterior crossbite and temporomandibular joint signs and symptoms such as muscle tenderness, muscle pain, headache and clicking [34,37,38]. Sonnesen et al. reported that the level of maximum bite force was significantly lower in unilateral posterior crossbite group compared to controls. The authors suggest that this is a result of the altered muscle function in children with this type of malocclusion [34]. The kinesiographic assessment of masticatory cycles in children with functional unilateral posterior crossbite revealed asymmetries and irregularities. Unilateral masticatory cycles were predominant. Orthodontic treatment with selective grinding and direct resin placement resulted in significant increase of total maximum lateral movements and improvement of the symmetry of cycles. Lateral excursions on non-crossbite side restored the normal masticatory jaw movement [25]. Ultrasound evaluation of swallowing pattern in children with unilateral posterior crossbite showed that duration of a phase IIb and the entire act of swallowing were longer in the crossbite group. In the phase IIb the tongue moves in a posterior direction towards the pharynx. The increased duration of phase IIb and the whole swallowing act might be explained by the fact that the tongue in children with crossbite has to travel a longer distance since it usually rests in the anterior part of the oral cavity. The study also demonstrated higher prevalence of infantile swallowing pattern in children with unilateral posterior crossbite [27]. Another study showed statistically significant difference in the length of the lingual frenulum between the children with and those without posterior crossbite. The crossbite group had shorter frenulum and thus lowered tongue position. This changes the equilibrium of soft tissue pressure on the upper arch and might cause maxillary constriction [23].

**TREATMENT**

The orthopedic expansion of the maxilla is achieved by the separation of the mid-palatal suture. The sutural opening can be usually performed up to age of 16 in females.
and up to age of 18 in males; however, this is highly individual and depends on the cessation of the skeletal growth [3,13]. Persson and Thailander studied the degree of obliteration in the midpalatal suture on histological sections of autopsy material. The earliest suture closure was observed in a 15-year old female, while the oldest person without ossification was 27 years old. This study confirms that the total obliteration of the midpalatal suture is very variable [29]. As was mentioned above, early correction of posterior crossbite with a mandibular shift is required because it prevents from the development of skeletal asymmetries in the adulthood. The treatment in the deciduous dentition should be limited to the correction of the premature occlusal contacts in primary teeth, most often the canines. The success rate of this method is only 30% to 50%. However, other techniques can be employed; it is recommended to postpone the treatment to the phase of early mixed dentition when the first permanent molars erupt. If the posterior crossbite is of dental origin, tipping and/or translation are used to move individual teeth to correct position. If the posterior crossbite is caused by skeletal transverse discrepancies maxillary base expansion is the treatment of choice [21]. Correction of dental crossbites can be accomplished with various appliances such as removable expansion plate, transpalatal arch, quad helix, W arch and 2 x 4 appliance [3,21]. The acrylic-based removable appliance usually has a single expansion screw located at the midline between the premolars or primary molars. The retention is provided by Adams or ball clasps. Turning the screw once or twice a week applies pressure to the palatal surfaces of the teeth and the alveolar processes and produces movement. Removable appliances usually expand the maxillary arch by tipping the teeth and the alveolar processes buccally, although the separation of midpalatal suture is possible in primary and early mixed dentition. Skeletal expansion can be achieved with jackscrew appliances which are fixed to the teeth by bands or bondable acrylic pads. Haas and Hyrax appliances incorporate an expansion screw mostly attached to orthodontic bands on the first permanent molars and first premolars. Haas appliance includes an acrylic button molded to the palate, in which the jackscrew is embedded and to which the bands are attached. In the Hyrax appliance the heavy round wires are soldered to the screw and connected with the bands [3,41]. Maxillary base expansion can be produced by rapid maxillary expansion (RME) or slow maxillary expansion (SME). RME modality is usually defined as two turns of the expansion screw daily (0.5 mm) [10]. The initial force tips the maxillary molars buccally until the speed of screw activation exceeds the speed of root movement. After that, the midpalatal suture separates and skeletal expansion begins [21]. Reported side effects of RME include external root resorption, higher levels of pain and discomfort especially during initial activation compared with SME, microtrauma of the midpalatal suture and relapse [10,22]. Slow maxillary expansion is defined as one turn of the jackscrew (0.25 mm) every other day. SME is thought to provide the maximum rate at which the sutures can adapt, with minimum tearing and hemorrhaging compared with RME [10]. RME treatment produces both orthodontic (tipping and translation of the teeth) and orthopedic (sutural expansion) effects [13]. SME is as effective as RME in determining skeletal expansion [22]. Generally, skeletal changes are approximately 30% to 50% of the amount of dental changes. It is advised to overcorrect the posterior crossbite so that the buccal incline of the maxillary molars’ palatal cusps contacts with
the lingual inclines of the buccal cusps of the mandibular molars. This allows for physiologic rebound that will occur in the upper arch and for compensatory buccal uprighting of the lower teeth as a result of the changes in the soft tissue pressure [3]. The appliance should be left intraorally in a passive state for retention of 4 to 6 months [3,10]. A significant side effect of orthopedic maxillary expansion is downward displacement of the maxilla and bite opening caused by the tipping of the posterior teeth. Some authors suggest that use of bonded jackscrew appliance prevents bite opening. More rigid appliance is thought to reduce dental tipping and additionally by biting on the acrylic pads, muscles stop the maxilla from being displaced downward [41]. A comparison of the effectiveness of quad helix, expansion plate, and composite onlay in correction of the unilateral posterior crossbite in mixed dentition showed that treatment with quad helix is the most effective. Expansion plate was unsuccessful in one third of the cases because of lack of patient’s cooperation [30].

REFERENCES


ABSTRACT

Posterior crossbite is caused by a transverse discrepancy between the maxillary and mandibular arches. The etiology is multifactorial with both genetic and environmental factors. Nonnutritive sucking habits, upper airway obstruction leading to mouth breathing and short duration of breastfeeding are well-established causes of maxillary constriction. Temporomandibular signs and symptoms such as muscle tenderness, headache and clicking are more common in patients with unilateral crossbite and a lower jaw deviation. The early treatment of unilateral posterior crossbite with a functional shift of the mandible is required due to possible development of facial skeletal asymmetries.

STRESZCZENIE

Zgryz krzyżowy w odcinku bocznym spowodowany jest niezgodnością w wymiarze poprzecznym między łukami zębowymi szczęki i żuchwy. Etiologa jest wieloczynnikowa, o podłożu zarówno genetycznym, jak i środowiskowym. Dobrze znawanymi przyczynami zwężenia szczęki są nawyk ssania, niedrożność górnych dróg oddechowych skutkująca ustnym torem oddychania oraz krótki okres karmienia naturalnego. Dolegliwości ze strony stawu skronio-żuchwowego takie jak tkliwość mięśni, bóle głowy oraz objawy akustyczne są spotykane częściej w pacjentów z jednostronnym bocznym zgryzom krzyżowym ze zbaczaniem żuchwy. Wskazane jest wczesne leczenie jednostronnego bocznego zgryzu krzyżowego ze zbaczaniem żuchwy ze względu na prawdopodobieństwo rozwoju szkieletowej asymetrii twarzy.