

The effect of mode of breathing on craniofacial growth—revisited

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SUMMARY It has been maintained that because of large adenoids, nasal breathing is obstructed leading to mouth breathing and an ‘adenoid face’, characterized by an incompetent lip seal, a narrow upper dental arch, increased anterior face height, a steep mandibular plane angle, and a retrognathic mandible. This development has been explained as occurring by changes in head and tongue position and muscular balance. After adenoidectomy and change in head and tongue position, accelerated mandibular growth and closure of the mandibular plane angle have been reported. Children with obstructive sleep apnoea (OSA) have similar craniofacial characteristics as those with large adenoids and tonsils, and the first treatment of choice of OSA children is removal of adenoids and tonsils. It is probable that some children with an adenoid face would nowadays be diagnosed as having OSA. These children also have abnormal nocturnal growth hormone (GH) secretion and somatic growth impairment, which is normalized following adenotonsillectomy.

It is hypothesized that decreased mandibular growth in adenoid face children is due to abnormal secretion of GH and its mediators. After normalization of hormonal status, ramus growth is enhanced by more intensive endochondral bone formation in the condylar cartilage and/or by appositional bone growth in the lower border of the mandible. This would, in part, explain the noted acceleration in the growth of the mandible and alteration in its growth direction, following the change in the mode of breathing after adenotonsillectomy.

Adenoid face

The effect of mode of breathing on craniofacial growth has been a widely debated and controversial issue within orthodontics for decades. It has classically been maintained that because of large adenoids, nasal breathing is (partially) obstructed leading to mouth breathing and the stereotype of the adenoid face (Subtelny, 1954; Linder-Aronson, 1970), however, the complexity of this association has also been discussed (McNamara, 1981; Warren and Spalding, 1991; Trotman *et al.*, 1997; Vig, 1998). The adenoid face is characterized by an incompetent lip seal, a narrow upper dental arch, retroclined mandibular incisors, increased anterior face height, a steep mandibular plane angle, and retrognathic mandible compared with faces of healthy controls (Linder-Aronson, 1970). Comparable changes in the craniofacial structure have been described in a group of subjects with large tonsils (Behlfelt *et al.*, 1990). This development has been explained in a ‘mechanistic’ way as occurring by changes in the muscular balance. Because of mouth breathing, the tongue position in the oral cavity is low and the balance between forces from the cheeks and tongue is different compared with healthy children. This leads to a lower mandibular position and extended head posture with all the above-mentioned dental and skeletal consequences (Solow and Kreiborg, 1977; Linder-Aronson, 1979; Solow *et al.*, 1984; Figure 1).

Consequences after adenoidectomy

After adenoidectomy and facilitation of nasal breathing, accelerated mandibular growth and closure of the mandibular plane angle, but not the maxillary plane angle, have been reported, however, with a large variation in response (Linder-Aronson *et al.*, 1986; Woodside *et al.*, 1991). In a more detailed analysis, anterior face height was found to be unaffected and remained longer in the initially large adenoid subjects than in healthy controls 5 years after adenoidectomy. In the same study, growth of the mandibular ramus and condylar process of adenoidectomy patients was found to be greater than that in the control subjects (Kerr *et al.*, 1989). The changes have, as a rule, been explained by alteration in tongue position and autorotation of the mandible (Linder-Aronson, 1979; Figure 2). However, a decrease in the mandibular plane angle necessitates more growth in posterior face height/ramus height than anteriorly, since intrusion of maxillary teeth may only be possible with the use of intrusive devices or maxillary impaction with surgery (Woodside *et al.*, 1991).

Obstructive sleep apnoea

In all individuals, muscular activity is reduced and upper airway resistance increased during sleep compared with wakefulness (Worsnop *et al.*, 2000). This does not have a

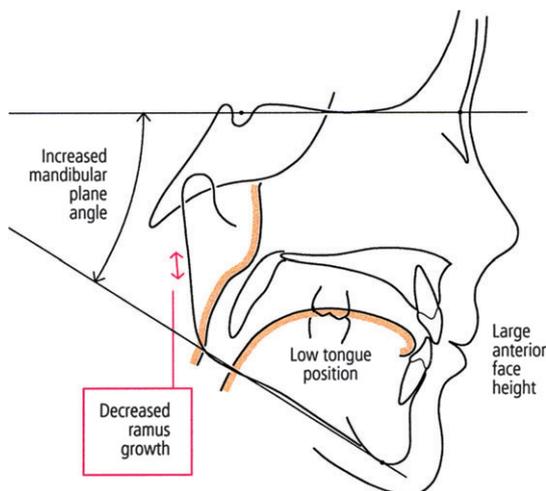


Figure 1 Tracing of a child with large adenoids. Because of mouth breathing, tongue position in the oral cavity is low and the balance between forces from the cheeks and tongue is different compared with healthy children. This leads to a lower mandibular position and extended head posture. Cephalometrically a large anterior face height and increased mandibular plane angle can be noted (adapted from Linder-Aronson, 1970, 1979). In addition, according to the present hypothesis, because of abnormal nocturnal growth hormone secretion, ramus growth is less than that in healthy subjects.

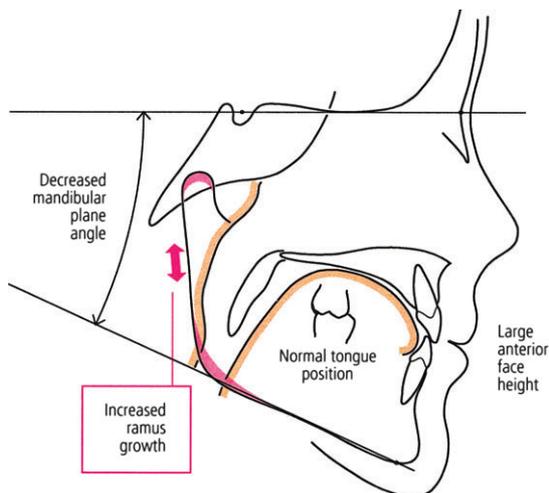


Figure 2 Tracing of a child after adenotonsillectomy. Because of normalization of breathing and tongue position, the mandibular plane angle has been found to decrease and mandibular growth accelerated with no changes in anterior face height (adapted from Linder-Aronson *et al.*, 1986; Woodside *et al.*, 1991). In addition, according to the present hypothesis, because of normalization of secretion of growth hormone and its mediators, accelerated mandibular growth and change in its growth direction can be explained particularly by increased ramus growth. In other words, more intensive growth in the condylar cartilage and/or in the lower border of the mandible at the muscular attachment area.

notable effect on breathing in anatomically and functionally ‘healthy’ individuals. On the other hand, reduction of muscular tonus in children with large adenoids and tonsils, or with other underlying abnormal upper airway anatomy,

may lead to airway obstruction and eventually to obstructive sleep apnoea (OSA). Interestingly, these children have been found to have similar craniofacial characteristics as adenoid face children (Guilleminault *et al.*, 1996; Shintani *et al.*, 1997; Agren *et al.*, 1998; Zucconi *et al.*, 1999; Kawashima *et al.*, 2000, 2002; Zettergren-Wijk *et al.*, 2006). The first treatment of choice of OSA children is removal of adenoids and tonsils (Nieminen, 2002; Guilleminault *et al.*, 2004). It can thus be postulated that some children with a clinical diagnosis of an adenoid face could nowadays be diagnosed as having OSA. Of particular interest is the recent cephalometric study on 5-year-old children with polysomnographically verified OSA (Zettergren-Wijk *et al.*, 2006). This study showed that OSA children have a different facial morphology compared with age-matched controls. The mandibular plane angle was found to be posteriorly inclined, anterior face height to be greater, and posterior face height smaller, in the OSA than in the control children. At the 5-year follow-up after adenotonsillectomy, no major craniofacial differences were noted. In a closer look at the growth changes it becomes evident that anterior face height remained greater in the OSA children than in the control children (difference on average 2.5 mm), but it increased on average by a comparable amount in both groups of children. Yet, the mandibular plane angle was decreased in the OSA children. This may be explained by the described greater posterior face height growth (ramus growth) in the OSA than in the control children (OSA children 5 mm, control children 3 mm).

OSA children with large adenoids and tonsils have also been found to have somatic growth impairment due to abnormal nocturnal growth hormone (GH) secretion (Goldstein *et al.*, 1987; Bar *et al.*, 1999; Nieminen *et al.*, 2002). Following adenotonsillectomy, a significant increase in the serum levels of GH mediators, i.e. insulin-like growth factor I (IGF I) and its binding protein, has been reported. Consequently, normalization and even catch-up of somatic growth have been observed (Bar *et al.*, 1999; Nieminen *et al.*, 2002). Could the craniofacial characteristics, particularly the height of the mandibular ramus, in adenoid face children and changes after removal of adenoids and tonsils, be partly explained by changes found in the hormonal status?

Growth of the mandibular ramus

Endochondral bone formation in the condylar cartilage and bone apposition in the lower border of the mandible (gonial region) contribute to the growth in height of the mandibular ramus. Studies on mandibular condylar cartilage have shown that the cartilage not only is a passive growth site, but also is endowed with some tissue-separating potential (Coprav *et al.*, 1986; Rönning and Peltomäki, 1991). It has also been maintained to be active in displacing the condylar process downwards (Kantomaa, 1984). In addition, the mandibular condylar cartilage seems to be a target and

production site of hormonal agents as evidenced by IGF I receptor and IGF I messenger RNA expression in the cartilage (Visnapuu *et al.*, 2001, 2002). Patients with GH deficiency have been shown to have a small posterior face height compared with age and gender-matched healthy controls (Pirinen *et al.*, 1994; Karsila-Tenovuo *et al.*, 2001). Furthermore, administration of GH to patients with GH deficiency, such as those with Turner syndrome or in bone marrow transplant patients, has clearly shown that mandibular growth, and particularly mandibular ramus growth, is accelerated compared with control children (Dahlöf *et al.*, 1991; Rongen-Westerlaken *et al.*, 1993; Simmons, 1999; Forsberg *et al.*, 2002). The increase in mandibular ramus height by GH can be explained by two possibilities. Firstly, increased endochondral bone formation in the condylar cartilage and secondly, increased bone apposition in the lower border of the mandible through the anabolic effects of GH on the masseter and medial pterygoid muscles (Vogl *et al.*, 1993).

Conclusion

Taking into account the recent evidence from children with OSA, it can be postulated that the craniofacial structure before, and its change after adenotonsillectomy, in patients with large adenoids and tonsils (classically, regarded as mouth breathing patients) are not only caused by a mechanistic alteration in the muscular balance and head and tongue position due to the change in the mode of breathing but also caused by a more complex sequence of epigenetic events. Because of abnormal nocturnal secretion of GH and its mediators in children with obstructed breathing, mandibular ramus growth is less than that in healthy subjects (Figure 1). After normalization of hormonal status, ramus growth is enhanced by more intensive endochondral bone formation in the condylar cartilage and/or by appositional bone growth in the lower border of the mandible at the muscular attachment area (Figure 2). This growth enhancement would, in part, explain the noted acceleration in the growth of the mandible and change in its growth direction after alteration in the mode of breathing following adenotonsillectomy. Finally, it is noteworthy that in many cases the growth acceleration is not sufficient to solve the already formed malocclusion and skeletal discrepancy, and therefore, orthodontic treatment is also indicated.

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