

Long-term changes in pharyngeal airway dimensions following activator-headgear and fixed appliance treatment

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SUMMARY The aim of this study was to evaluate changes in the pharyngeal airway in growing children and adolescents and to compare these with a group of children who received activator-headgear Class II treatment. The sample consisted of 64 children (32 males and 32 females), 32 had a combined activator-headgear appliance for at least 9 months (study group) followed by fixed appliance therapy in most patients, while the other half received only minor orthodontic treatment (control group). Lateral cephalograms before treatment (T1, mean age 10.4 years), at the end of active treatment (T2, mean age 14.5 years), and at the long-term follow-up (T3, mean age 22.1 years) were traced and digitized. To reveal the influence of somatic growth, body height measurements were also taken into consideration. A two-sample *t*-test was applied in order to determine differences between the groups.

At T1, the study group had a smaller pharynx length ($P = 0.030$) and a greater ANB angle ($P < 0.001$) than the controls. The pharyngeal area and the smallest distance between the tongue and the posterior pharyngeal wall also tended to be smaller in the study group. During treatment (T1–T2), significant growth differences between the two groups were present: the study group had a greater reduction in ANB ($P < 0.001$) and showed a greater increase in pharyngeal area ($P = 0.007$), pharyngeal length ($P < 0.001$) and the smallest distance between the tongue and the posterior pharyngeal wall ($P = 0.038$). At T2, the values for the study group were similar to those of the control group and remained stable throughout the post-treatment interval (T2–T3).

Activator-headgear therapy has the potential to increase pharyngeal airway dimensions, such as the smallest distance between the tongue and the posterior pharyngeal wall or the pharyngeal area. Importantly, this increase seems to be maintained long term, up to 22 years on average in the present study. This benefit may result in a reduced risk of developing long-term impaired respiratory function.

Introduction

Obstructive sleep apnoea (OSA), a common and frequently undiagnosed medical disorder in adults (Young *et al.*, 2002), is characterized by recurrent pharyngeal airway obstruction during sleep. Frequent episodes of hypoxia and awakenings disrupt continuous sleep and even mild OSA is associated with significant morbidity (Young *et al.*, 2002). While OSA, or sleep disordered breathing, has been reported in adolescents and children (Guilleminault *et al.*, 1976; Contencin *et al.*, 2003), it is most common in middle-aged adults (Young *et al.*, 2002). The reasons for the greater incidence of this disorder in later life are due to obesity (Martin *et al.*, 1997; Finkelstein *et al.*, 2001; Young *et al.*, 2002), family background (Mathur and Douglas, 1995; Schwab *et al.*, 2006), or natural physiological changes. It has been observed that the depth of the oropharyngeal airway decreases with age (Martin *et al.*, 1997), and that the soft palate becomes longer and thicker (Johnston and Richardson, 1999). Supine posture and physiological decrease of muscular tonus during sleep cause additional significant reductions of pharyngeal airway dimensions (Pae *et al.*, 1994; Martin *et al.*, 1997; Smith and Battagel, 2004).

When the airway and associated structures were examined, both snorers and OSA subjects exhibited reduced anteroposterior dimensions, narrower airways, reduced oropharyngeal areas, and larger tongues (Lowe *et al.*, 1986b; Battagel and L'Estrange, 1996; Battagel *et al.*, 2000). According to cephalometric studies, OSA patients seem to have shorter (Battagel *et al.*, 2000) and more retrognathic mandibles and also an anteroposterior discrepancy between the maxilla and mandible (Lowe *et al.*, 1995). Furthermore, it has been shown that there is a correlation between the minimum axial cross-sectional area of the oropharyngeal lumen and oxygen saturation and quantity, as well as the duration of apnoea episodes (Avrahami and Englender, 1995). Previous reports (Launois *et al.*, 1993; Morrison *et al.*, 1993) have emphasized the significance of the most constricted sites (retropalatal and retroglottal) of the upper airway in airflow dynamics. It is at such sites in the upper airway that critical narrowing is likely to occur, and therefore changes in the dimension of these sites are of particular importance (Ono *et al.*, 2000).

Lateral cephalometry is an established tool in the investigation of the airway in OSA subjects and it has been employed for diagnostic purposes (Battagel *et al.*, 2000;

Kuhnel *et al.*, 2005) and to monitor therapeutic response (Battagel *et al.*, 1999). Reproducibility of airway dimensions on lateral cephalograms has been studied and found to be highly accurate (Malkoc *et al.*, 2005). However, a number of limitations of lateral cephalometry have been discussed (Lowe *et al.*, 1986a; Finkelstein *et al.*, 2001), particularly inadequate description of the airway on a two-dimensional radiograph.

Although nasal continuous positive airway pressure remains the non-invasive treatment of choice for severe OSA in adults, various intraoral mandibular advancement devices (MAD) are successfully used for treating mild to moderate OSA and snoring (Bloch *et al.*, 2000; Marklund *et al.*, 2004). MAD are thought to prevent upper airway collapse during sleep by holding the mandible in a forward position, thereby increasing genioglossus muscle activity and pharyngeal airway space (Adachi *et al.*, 1993). It has been shown that the percentage of alterations in airway dimensions matched or exceeded the amount of mandibular advancement and that the minimum distances behind the soft palate and tongue improved while wearing these devices (Battagel *et al.*, 1999).

In growing children, similar oral appliances such as MAD (for example, activators) have been used for many decades to modify mandibular growth in skeletal Class II patients. In contrast to MAD used in adults, activators are considered to enhance skeletal growth of the mandible. Even though there are numerous studies that have examined skeletal changes with functional orthopaedic treatment in Class II children, there appears to be only one (Ozbek *et al.*, 1998) that has investigated pharyngeal airway dimension changes caused by these devices. However, that investigation had only a short observation time (1.5 years in the treatment group) and was somewhat biased since only good responders were considered for the treatment group (treated patients who did not achieve a Class I occlusion and sufficient overjet reduction were excluded) and because two types of activators were combined as one group (with and without headgear).

The aims of this long-term study were to examine physiological changes in the pharyngeal airway size in healthy children and to compare them with a group of children who received activator-headgear treatment to determine possible treatment effects.

Subjects and methods

Subjects

The 64 subjects (32 males and 32 females) participating in this study were randomly selected from the archives at the Clinic for Orthodontics and Pediatric Dentistry at the University of Zurich. Preconditions for the selection of patients in both groups were no extractions or space closure of congenitally missing teeth, no skeletal Class III

malocclusion, no extreme skeletal deep or open bite, no orthognathic surgery, no rapid maxillary expansion, remaining growth (determined by means of hand-wrist radiograph), at least three lateral cephalograms of good quality: before treatment (T1), at the end of active treatment (T2), and at the end of the long-term follow-up (T3) with the patient being at least 18 years of age.

Inclusion criterion for subjects in the study group was active treatment with a combined activator-high-pull headgear appliance as described by Teuscher (1978) of at least 9 months duration between 9 and 14 years of age. The study group consisted of 32 subjects: 16 males and 16 females. Mean ages, skeletal, and airway measurements are given in Table 1. According to the principles of the clinic, only a moderate construction bite (typically 3–4 mm) was taken, resulting in some cases in a two-step activation; care was taken to avoid increasing the vertical dimension during treatment. Therefore, a short phase with a fixed appliance was performed for 12 of the 32 patients prior to the combined activator-headgear treatment, mainly to allow an unhindered forward positioning of the lower jaw. The activator was equipped with torque-control auxiliaries for the upper incisors, and the edges of the lower incisors were embedded in acrylic. The facebow was mounted directly on the activator and the direction of the extraoral force was posterior cranial. The mean treatment time with the activator-high-pull headgear appliance was 17 ± 6.5 months (range 9–32 months), followed by fixed orthodontic treatment in 27 patients. Final treatment outcome, such as achievement of a Class I occlusion was not an inclusion or exclusion criteria.

For ethical reasons, no untreated control group was available. Therefore, individuals were chosen who had only minor treatment carried out between 9 and 16 years of age. In most cases, the treatment was undertaken for aesthetic reasons only and involved partial or full fixed orthodontic treatment. No Class II mechanics such as headgear, activator, or Class II elastics were allowed. The control group consisted of 32 subjects: 16 males and 16 females. The mean ages, skeletal, and airway measurements are given in Table 1.

Skeletal differences between the groups were inevitable due to the appliance-dictated subject selection. Patients in the activator-headgear group who received therapy primarily because of a skeletal Class II malocclusion were expected to be initially more Class II than the controls. Since a certain overlapping of the groups was desired, no skeletal parameters were defined which would result in a larger separation of the groups. Contrary to the sagittal skeletal relationship, facial divergency was very similar in both groups.

Method

Lateral cephalograms were taken with the Frankfurt horizontal plane parallel to the floor. For every patient, the

Table 1 Means, standard deviations, and *P* values of two-sample *t*-test comparing the activator-headgear group and the controls at the three time points.

| | <i>n</i> | T1 (pre-treatment) | | | T2 (end of treatment) | | | T3 (end of recall) | | |
|-------------------------|----------|--------------------|---------------|----------------|-----------------------|---------------|----------------|--------------------|---------------|----------------|
| | | Study group | Controls | <i>P</i> value | Study group | Controls | <i>P</i> value | Study group | Controls | <i>P</i> value |
| Age (years) | 64 | 10.2 ± 1.1 | 10.7 ± 1.6 | 0.164 | 14.6 ± 1.2 | 14.4 ± 1.4 | 0.569 | 22.2 ± 2.7 | 21.9 ± 2.3 | 0.590 |
| Body height (cm) | 56 | 140.3 ± 6.5 | 140.9 ± 9.9 | 0.800 | 164.0 ± 7.1 | 163.0 ± 8.3 | 0.630 | 172.2 ± 8.6 | 171.9 ± 8.2 | 0.882 |
| SNA (°) | 64 | 81.9 ± 3.8 | 81.5 ± 4.0 | 0.686 | 80.4 ± 3.8 | 81.8 ± 4.0 | 0.157 | 80.8 ± 3.3 | 81.9 ± 3.9 | 0.258 |
| SNB (°) | 64 | 76.3 ± 3.2 | 78.3 ± 3.4 | 0.017* | 76.8 ± 3.6 | 79.2 ± 3.6 | 0.010* | 77.8 ± 3.3 | 80.0 ± 3.8 | 0.017* |
| ANB (°) | 64 | 5.6 ± 2.0 | 3.2 ± 2.0 | 0.001* | 3.6 ± 1.7 | 2.6 ± 1.8 | 0.029* | 3.0 ± 2.1 | 1.9 ± 2.0 | 0.029* |
| SN/ML (°) | 64 | 33.7 ± 4.7 | 33.2 ± 5.0 | 0.742 | 33.0 ± 5.8 | 32.5 ± 5.3 | 0.700 | 30.7 ± 6.6 | 31.4 ± 5.9 | 0.688 |
| p (mm) | 64 | 8.3 ± 2.4 | 7.9 ± 2.4 | 0.519 | 9.0 ± 2.3 | 8.3 ± 2.7 | 0.283 | 9.6 ± 2.5 | 9.3 ± 2.5 | 0.648 |
| w (mm) | 64 | 12.6 ± 3.0 | 12.6 ± 3.3 | 0.922 | 12.9 ± 2.7 | 12.9 ± 4.1 | 0.942 | 14.1 ± 3.3 | 13.7 ± 3.5 | 0.650 |
| t (mm) | 64 | 8.5 ± 2.6 | 10.0 ± 3.7 | 0.073 | 9.8 ± 2.9 | 9.8 ± 3.6 | 0.939 | 11.1 ± 3.4 | 10.4 ± 2.9 | 0.432 |
| Length (mm) | 64 | 76.5 ± 6.6 | 81.0 ± 9.2 | 0.030* | 87.7 ± 7.6 | 86.4 ± 8.7 | 0.549 | 92.5 ± 8.6 | 91.4 ± 8.5 | 0.619 |
| Area (mm ²) | 64 | 445.5 ± 151.7 | 540.0 ± 231.6 | 0.058 | 608.7 ± 195.3 | 573.7 ± 215.4 | 0.499 | 724.2 ± 229.2 | 670.3 ± 196.7 | 0.317 |

**P* < 0.05.

three cephalograms (T1, T2, T3) were hand-traced and then scanned at 600 dpi (Duoscan HiD, AGFA, Mortsel, Belgium). The resulting 192 digitized tracings were calibrated on a known length and measured with ImageTool 3.00 for Windows (University of Texas Health Science Center, San Antonio, Texas, USA). Correction of the radiographic magnification (7.5 per cent) was also made to facilitate comparison with previous published data. The measured angles, distances, and areas are defined and illustrated in Figure 1. To study somatic growth, body height was measured at the same time as the lateral cephalograms were taken. Since one or more body height readings were missing in eight individuals, only 29 patients and 27 controls could be considered in the analysis involving body height.

Error of method

The measurements of the first cephalogram of all 64 patients were repeated by one author (MPH) after an interval of 6 weeks, and the reproducibility of the measurements (limits of agreement) was assessed using the method of Bland and Altman (1986, 1999). Furthermore, the first and second readings were compared using the concordance correlation coefficient (Lin, 1989).

Statistical method

Statistical analyses were performed with the Statistical Package for Social Sciences 15.01 for Windows (SPSS Inc., Chicago, Illinois, USA) and MedCalc 9.3 (MedCalc Software, Mariakerke, Belgium). For descriptive analyses, the means, standard deviations (SDs) and ranges of the measurements at the three time points were computed. Moreover, the differences in measurements between the time points were calculated, and descriptive statistics of the longitudinal changes (means, SDs, and ranges) were

produced. In order to demonstrate differences between the study and control group, a two-sample *t*-test was applied. When Levene's test indicated different variances in the

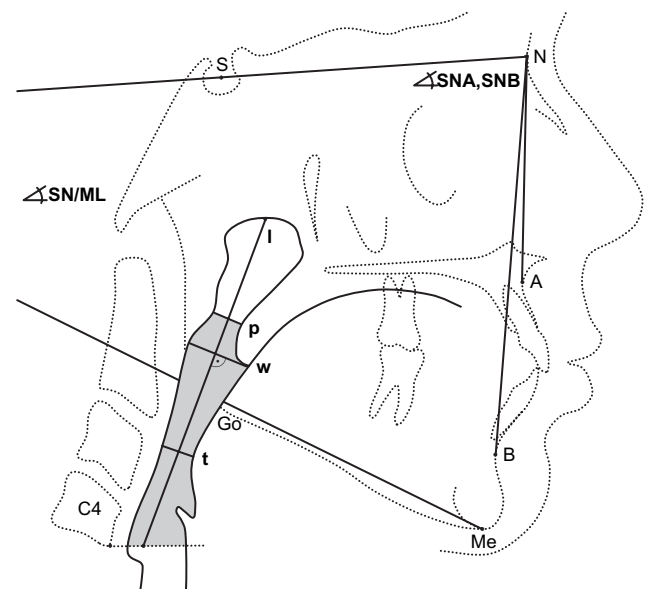


Figure 1 The cephalometric measurements used in this study to determine skeletal changes were SNA, SNB, ANB, and SN/ML (mandibular line: Menton–Gonion) angles. Pharyngeal airway was assessed with the following measurements: l—pharyngeal length, the longitudinal axis of the pharynx starting at the most cranial point of the pharynx and ending at a line parallel to Frankfort horizontal (FH) through the most anterior inferior point of the fourth vertebral corpus, p—the smallest distance between the soft palate and the posterior pharyngeal wall, w—the largest distance perpendicular to the longitudinal axis of the pharynx, between the lines p and t, t—the smallest distance between the tongue base and the posterior pharyngeal wall, area (marked grey)—the area between line p and a line parallel to FH through the most anterior inferior point of C4. When a gap was present between the tongue and the soft palate, this area was not considered. The area above line p was intentionally not included due to the possibly falsifying influence of the adenoids in growing children.

groups, the results from the Welch test were reported. Mean differences and 95 per cent confidence intervals (CIs) for the differences between groups were computed. Additionally, a paired *t*-test was used to test for significant longitudinal changes within the groups. To analyse the degree of association between two continuous variables, Pearson's correlation coefficient, with a 95 per cent CI was used. In order to analyse the relationship between one or more predictors and the observed change over time, multiple regression analyses were carried out. A *P* value of less than 0.05 was chosen to assign statistical significance for all tests. Results with a *P* value above 0.05 but smaller than 0.10 were interpreted as a tendency.

Results

Lower and upper limits of agreement according to Bland and Altman (1986, 1999) were as follows: -7.7 and 6.6 mm² for area measurement, -0.53 and 0.56 degrees for angular measurements, -0.46 and 0.37 mm for distance 't', and -0.63 and 0.61 mm for the longest distance 'l'. For all measurements, Lin's (1989) concordance correlation coefficient was above 0.99, indicating good repeatability of the measurements.

The only variables that significantly differed between the groups at T1 were SNB (2.02 degrees, *P* = 0.017), ANB (-2.41 degrees, *P* < 0.001), and the length of the pharynx 'l' (2.00 mm, *P* = 0.030), with the study group showing a smaller pharyngeal length and SNB which consequently lead to a greater ANB (Table 1). The pharyngeal area and distance 't' also tended to be smaller in the study group. At T2 and T3, differences between the groups were generally smaller, however, SNB (2.17 degrees, *P* = 0.017) and ANB (-1.14 degrees, *P* = 0.029) still remained significantly different.

The study group had a longer treatment period (T1–T2) and showed more body height growth during that time, but neither of these factors reached statistical significance (Table 2). The annual growth rate was slightly greater in the control group. To compare the groups longitudinally, discrepancies in the growth differences of the groups were measured. During the treatment period, the study group had a significantly greater reduction of SNA (-1.81 degrees, *P* < 0.001) and ANB (-1.43 degrees, *P* < 0.001), while there was no statistically significant effect for SNB. At the same time, the study group showed statistically significant increases (compared with the controls) in the following pharyngeal parameters: area (129.50 mm², *P* = 0.007), length (5.69 mm, *P* < 0.001) and the smallest distance between the tongue and the posterior pharyngeal wall 't' (1.51 mm, *P* = 0.038). The changes in these parameters were also longitudinally significant in the study group. From T2 to T3, there were no statistically significant differences between the groups; instead, both groups underwent essentially the same changes, implying no reduction but rather a minor increase in the established changes during this period.

Using multiple linear regressions, the relationship between the observed changes in the pharyngeal area and distance 't' as the dependent variables and several predictor variables, was determined (Table 3). For the area change during T1 to T2, the only significant predictor variable was the inclusion to the study or the control group; for the distance 't' a similar tendency was found. During T2 to T3, group association (patient versus control) was no longer of relevance and the only significant predictor variable was body height growth. For the distance 't', a tendency could also be found when the change in SNB was taken into account as a predictor factor.

Table 2 Longitudinal mean changes, standard deviations, and *P* values for the activator-headgear group and the controls. Two-sample *t*-test was used for intergroup comparison and paired *t*-test for longitudinal changes within the group.

| | <i>n</i> | T1–T2 (treatment) | | | T2–T3 (post-treatment) | | | T1–T3 (overall) | | |
|---------------------------|----------|-------------------|-----------------|----------------|------------------------|-----------------|----------------|-----------------|-----------------|----------------|
| | | Study group | Controls | <i>P</i> value | Study group | Controls | <i>P</i> value | Study group | Controls | <i>P</i> value |
| Years | 64 | 4.4 ± 1.2 | 3.8 ± 1.6 | 0.064 | 7.6 ± 2.5 | 7.4 ± 2.3 | 0.795 | 12.0 ± 2.9 | 11.2 ± 2.6 | 0.235 |
| Δ body height (cm) | 56 | 23.7 ± 5.2** | 22.1 ± 7.4** | 0.362 | 8.2 ± 7.8** | 8.9 ± 7.6** | 0.749 | 31.9 ± 8.7** | 31.0 ± 9.7** | 0.712 |
| Δ SNA (°) | 64 | $-1.5 ± 1.8$ ** | 0.3 ± 1.8 | 0.001* | 0.5 ± 1.3** | 0.1 ± 0.9 | 0.166 | $-1.1 ± 1.8$ ** | 0.3 ± 1.7 | 0.002* |
| Δ SNB (°) | 64 | 0.5 ± 1.5 | 0.9 ± 1.6** | 0.334 | 1.0 ± 1.6** | 0.8 ± 1.2** | 0.518 | 1.5 ± 2.1** | 1.7 ± 1.9** | 0.764 |
| Δ ANB (°) | 64 | $-2.1 ± 1.5$ ** | $-0.6 ± 1.3$ ** | 0.001* | $-0.5 ± 1.0$ ** | $-0.7 ± 1.2$ ** | 0.580 | $-2.6 ± 1.8$ ** | $-1.3 ± 1.6$ ** | 0.005* |
| Δ SN/ML (°) | 64 | $-0.7 ± 2.3$ | $-0.8 ± 1.6$ ** | 0.789 | $-2.2 ± 2.5$ ** | $-1.1 ± 2.3$ ** | 0.059 | $-2.9 ± 3.3$ ** | $-1.9 ± 2.9$ ** | 0.186 |
| Δ p (mm) | 64 | 0.7 ± 2.8 | 0.4 ± 1.9 | 0.628 | 0.6 ± 1.6** | 1.0 ± 1.7** | 0.328 | 1.3 ± 3.1** | 1.4 ± 2.1** | 0.877 |
| Δ w (mm) | 64 | 0.3 ± 3.1 | 0.3 ± 3.0 | 0.984 | 1.2 ± 2.6** | 0.8 ± 3.0 | 0.520 | 1.5 ± 4.1** | 1.1 ± 3.0 | 0.604 |
| Δ t (mm) | 64 | 1.3 ± 3.0** | $-0.2 ± 2.7$ | 0.038* | 1.2 ± 3.1** | 0.7 ± 2.8 | 0.448 | 2.5 ± 3.5** | 0.5 ± 3.4 | 0.020* |
| Δ length (mm) | 64 | 11.1 ± 6.4** | 5.4 ± 6.8** | 0.001* | 4.8 ± 6.6** | 5.0 ± 6.8** | 0.925 | 16.0 ± 7.6** | 10.4 ± 8.1** | 0.006* |
| Δ area (mm ²) | 64 | 163.2 ± 198.9** | 33.7 ± 175.0 | 0.007* | 115.5 ± 192.6** | 96.6 ± 190.3** | 0.693 | 278.7 ± 241.0** | 130.3 ± 231.9** | 0.015* |

**P* < 0.05 (significant differences between the groups).

***P* < 0.05 (significant longitudinal changes within the group).

Table 3 Multiple linear regressions on 56 observations for longitudinal changes in distance 't' and area during T1–T2 and T2–T3. Explanatory variables were group (study or control), gender, body height growth, SNB, and SN/ML change.

| | Δ distance 't' | | | | | | Δ area | | | | | |
|--------------------------------|--------------------------------|------|---------|--------------------------------|------|---------|--------------------------------|-------|---------|--------------------------------|-------|---------|
| | T1–T2 (r^2 adjusted = 0.01) | | | T2–T3 (r^2 adjusted = 0.12) | | | T1–T2 (r^2 adjusted = 0.09) | | | T2–T3 (r^2 adjusted = 0.29) | | |
| | Coefficient | SE | P value | Coefficient | SE | P value | Coefficient | SE | P value | Coefficient | SE | P value |
| Group (study = 1, control = 0) | 1.47 | 0.82 | 0.079 | 0.39 | 0.74 | 0.595 | 111.71 | 52.03 | 0.036* | −9.99 | 43.29 | 0.818 |
| Gender (male = 0, female = 1) | −0.35 | 0.82 | 0.668 | 0.97 | 0.90 | 0.283 | 2.46 | 51.55 | 0.962 | 43.99 | 52.58 | 0.407 |
| Δ body height | 0.01 | 0.07 | 0.774 | 0.17 | 0.07 | 0.016* | 5.65 | 4.11 | 0.176 | 15.19 | 3.88 | 0.001* |
| Δ SNB | −0.19 | 0.34 | 0.582 | 0.59 | 0.35 | 0.097 | −22.16 | 21.59 | 0.310 | 14.23 | 20.30 | 0.487 |
| Δ SN/ML | 0.11 | 0.26 | 0.674 | 0.24 | 0.21 | 0.247 | −1.29 | 16.60 | 0.938 | 4.17 | 12.51 | 0.741 |

* $P < 0.05$.

Considering only the changes in the control group, normal growth of the pharyngeal airway during T1 to T3 can be studied. The length (10.44 mm, $P < 0.001$), distance 'p' (1.41 mm, $P < 0.001$) and the area (130.26 mm², $P = 0.003$) demonstrated a significant increase from T1 to T3, while distances 'w' and 't' showed no significant increase and only minimal enlargement. During this period, ANB (−1.33 degrees, $P < 0.001$) and SN/ML (−1.87 degrees, $P < 0.001$) showed a significant angle reduction and were positively associated with each other ($r = 0.53$, $P = 0.002$). Again taking only the control group into account, Pearson's correlation coefficient revealed that body height growth had a significant positive association with the change in pharyngeal length ($r = 0.61$, $P < 0.001$), and area ($r = 0.41$, $P = 0.032$) and a negative association with the change in ANB ($r = -0.43$, $P = 0.027$). However, an expected correlation between the change in the pharyngeal area and that for SNB ($r = 0.20$, $P = 0.262$) or the change in SN/ML ($r = -0.22$, $P = 0.22$) was only weak.

Comparing the changes between genders longitudinally, there were no significant differences in the skeletal or airway parameters in either group during the period T1–T2. For T2 to T3, however, males showed significantly greater body height growth, pharyngeal length, and a tendency for an increase in SNB in both groups. At the long-term follow-up, the absolute values for females were similar to those of the males. Equal distribution of genders in the groups allowed pooling of data for males and females.

Discussion

It is evident that measurements on a two-dimensional cephalometric radiograph cannot reveal the transverse dimension of the airway. For this reason, three-dimensional imaging such as cone beam technology would be the preferred method. Since this technology was only introduced in recent years and has until now a relatively high radiation

dose for regular treatment monitoring, no long-term data will be available for some time. Therefore, the conventional lateral cephalogram remains a valuable and reliable diagnostic tool which has been used in numerous airway studies.

The longitudinal changes in the control group in this study revealed that not all pharyngeal dimensions increased during adolescence. While the length of the pharynx 'l' steadily increased, the smallest distance between the tongue base and the posterior pharyngeal wall 't' did not significantly increase. The present findings for distance 't' are in agreement with an earlier study (McNamara, 1984), which found no appreciable changes with age and showed the average value of this measurement to be between 10 and 12 mm (9–11 mm without magnification). The present results for pharyngeal length are also consistent with other findings later in life, between 20 and 50 years of age, which show continued vertical growth in pharyngeal length and a reduction in pharyngeal depth (Johnston and Richardson, 1999). Growth differences in pharyngeal dimensions between males and females were generally small, and at T3 both genders had reached similar absolute values, which is in line with a previous study (Abu Allhaja and Al-Khateeb, 2005).

At T1, the study group, which was, on average, skeletally more Class II than the controls, showed a tendency for a smaller pharyngeal area and distance between the tongue base and the posterior pharyngeal wall 't'. These findings agree with other studies that showed a weak but significant correlation between the skeletal configuration (ANB) and inferior pharyngeal airway space (Ceylan and Oktay, 1995; Abu Allhaja and Al-Khateeb, 2005). Clinical relevance was established by a study of 121 patients demonstrating an increasing severity of OSA with increasing overjet (Bates and McDonald, 2005), measured on lateral cephalograms.

During T1 to T2, the study group showed significantly greater increases compared with the controls in the distance

't', pharyngeal length, and pharyngeal area. During T2 to T3, these established changes remained and the groups showed similar growth. Taking into account the norm for the distance 't', which is 10–12 mm (McNamara, 1984), it becomes clear that the activator-headgear group was initially below the average and could reach normal values during treatment in an airway measurement that normally does not demonstrate any significant growth. This is illustrated in Figures 2 and 3, where the study group, which started with lower values, surpassed the controls during treatment and showed no decrease in the changes during the follow-up period. The body height measurements helped to verify that both groups had similar growth rates and that there was no bias in the selection of patients and controls.

Even though determining skeletal changes was not the primary goal of this study, it is evident that the combined activator-high-pull headgear appliance had a significant impact on the maxilla. While SNA remained constant in the controls, there was a reduction in the study group. However, this did not reduce the smallest distance between the soft palate and the posterior pharyngeal wall 'p'. In another study (Kirjavainen and Kirjavainen, 2007), retropalatal airway space was even increased during cervical headgear treatment in Class II children, whereas the oro- and hypopharynx remained unchanged. SNB demonstrated similar increases in both groups but the potential effect of the activator-headgear appliance on lower jaw growth could not be verified from the present data. Nevertheless, since the study group had a more retrognathic mandible at T1, perhaps resulting in diminished future lower jaw growth if left untreated (Lux *et al.*, 2005; Franchi *et al.*, 2007), the similar growth rate alone could be a positive effect of the appliance. To support this hypothesis, an untreated Class II group would be needed.

The positive impact of activator-headgear therapy on the airway dimensions cannot be explained just by the established skeletal changes. The present data suggest that the underlying mechanism is more complex, also having an effect on the soft tissues. Different posture of the tongue caused by increased genioglossal muscle tonus or other soft tissues changes may play an important role and are probably induced by forward positioning of the mandible during activator-headgear treatment. This effect seems to be similar to the way MAD work in adults. A study on the effect of MAD in subjects with OSA showed that the minimal distance behind the tongue improved by 0.8 mm while wearing these devices (Battagel *et al.*, 1999). In the present study, the minimal distance behind the tongue 't' improved by 2.5 mm on average in the activator-headgear group during the observation period. Even though this seems to be a small increase, it is nevertheless clinically significant, considering that a 0.8 mm increase with a MAD has a positive effect on OSA.

Another possible explanation for the improvement in the activator-headgear group could be a 'catch-up growth' concept, where children with small oropharyngeal dimensions would have a greater intrinsic stimulus to increase their capacity for respiratory function (Ozbek *et al.*, 1998). Nonetheless, it is noteworthy that individual differences in normal growth and in the response to treatment are considerable. Even a small diameter change can result in a substantial volumetric change and can have a great effect on airflow because the resistance decreases to the fourth power as the radius increases.

OSA children with large adenoids and tonsils have been found to have somatic growth impairment due to abnormal nocturnal growth hormone (GH) secretion. After adenotonsillectomy, which is the first treatment of choice in

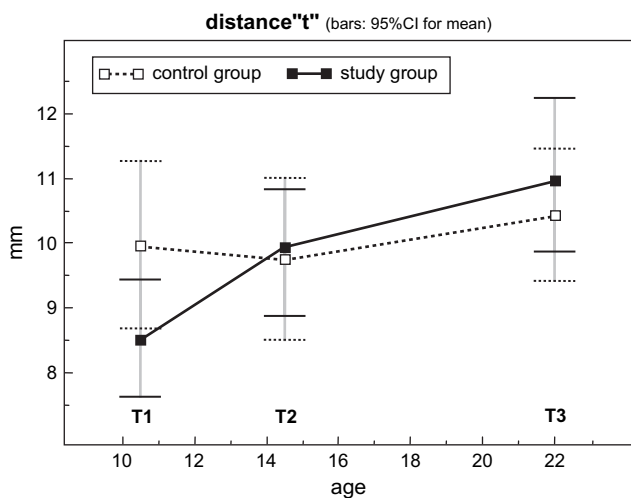


Figure 2 Longitudinal development of the smallest distance between the tongue base and the posterior pharyngeal wall 't' at the different time points. The growth rate was significantly different between the groups during the treatment phase (T1–T2), but not during the follow-up period (T2–T3).

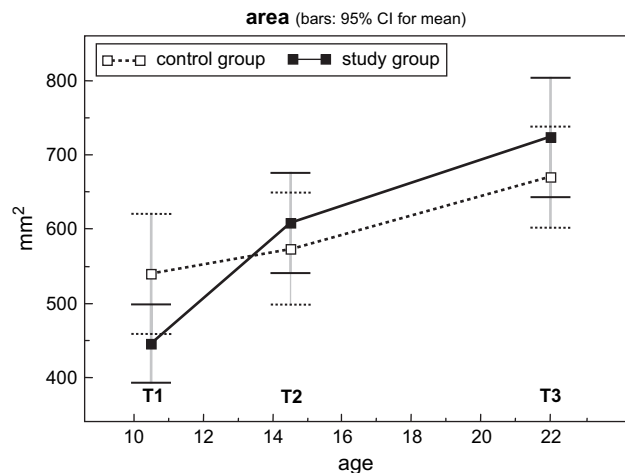


Figure 3 Longitudinal development of the pharyngeal area defined in Figure 1 at the different time points. The growth rate was significantly different between the groups during the treatment phase (T1–T2), but not during the follow-up period (T2–T3).

OSA children, a significant increase in serum levels of GH mediators and a normalization of somatic growth can be observed (Bar *et al.*, 1999; Nieminen *et al.*, 2002). Most likely, the explanation for this improvement is the demonstrated link between insufficient sleep patterns or stages and nocturnal release of GH (Spath-Schwalbe *et al.*, 1995; Steiger, 2002). The same occurrence seems also plausible in OSA children who have severe mandibular retrognathism as their primary cause of airway obstruction. In these children, activator-headgear therapy could also stimulate GH secretion by normalization of nocturnal breathing and thereby have a positive effect on mandibular growth. It has been demonstrated that GH increases endochondral bone formation in the condylar cartilage and enhances ramus height by augmented bone apposition in the lower border of the mandible (through the anabolic effects of GH on masticatory muscles; Vogl *et al.*, 1993). Of course, such a positive sequence achieved by activator-headgear treatment is currently hypothetical and must be confirmed.

Snoring and OSA are described as two aspects of the same basic disorder, namely sleep-related narrowing of the upper airways which differ only in severity (Lugaresi and Plazzi, 1997). The patency of the upper airway depends on the balance between the negative intrapharyngeal pressure developed during inspiration and its counteraction by dilating muscles (Friberg, 1999). It is clear that upper airway collapse most often results from a combination of anatomical factors that predispose the airway to collapse during inspiration, plus neuromuscular compensation that is insufficient during sleep to maintain airway patency (Young *et al.*, 2002). Therefore, it is possible that small pharyngeal dimensions established early in life may predispose to OSA and snoring later when subsequent soft tissue changes (Martin *et al.*, 1997) caused by age, obesity, or genetic background further reduce the available oropharyngeal airway. Consequently, it can only be regarded as beneficial if functional orthopaedic treatment in children (Ozbek *et al.*, 1998) or surgical mandibular advancement (Achilleos *et al.*, 2000) results in a permanent increase in pharyngeal airway dimensions.

Conclusions

The present findings indicate that activator-headgear therapy (followed by fixed appliance treatment) has the potential to increase pharyngeal airway dimensions, such as the smallest distance between the tongue base and the posterior pharyngeal wall or the pharyngeal area. Importantly, this achieved increase seems to be maintained in the long term, up to 22 years on average according to the findings of the present research. However, as in all orthodontic studies involving growing children, individual differences in normal growth and in the response to treatment were substantial. The evident increase in the airway dimension

obtained by functional orthopaedic treatment may reduce the risk of developing OSA in later life. This treatment can therefore be considered beneficial not only from the dental point of view but also from a general medical perspective.

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