Original Article

Cervical Vertebral Anomalies in Patients With Transverse Maxillary Deficiency

A. Burcu Altan, DDS, MS, PhD[1,*] and Berna Dönmez Zorkun, DDS, MS, PhD[2]

ABSTRACT

Objective: The literature indicates a correlation between vertebral morphologic anomalies and some orthodontic malocclusions. The aim of this study was to examine the occurrence of the types of cervical vertebral anomalies (CVAs) in subjects with transverse maxillary deficiency (TMD) and to compare this with the occurrence of CVA in a control group without TMD.

Materials and Method: A sample of 47 Turkish patients (17 boys, 30 girls) with TMD and another sample of 47 Turkish patients (15 boys, 32 girls) with adequate maxillary transversal dimensions was studied. A visual assessment of the cervical column was made using lateral cephalometric films. Characteristics of the cervical column were classified according to Sandham as fusions and posterior arch deficiencies of C1 (PADs). Clinically normal appearance was determined when the first 4 vertebrae could be traced as separate entities with all anatomic components present. Differences in occurrence of CVA between the groups and between genders were assessed by χ^2 independence test.

Results: In the TMD group, 10,6% of the subjects had fusion in the cervical column, and 14,9% had PAD. In the control group, 17% of the subjects had fusion of the cervical column, and 8,5% had PAD. The occurrence of PAD was significantly increased in the TMD group ($p<0.05$), while no significant difference was found between the groups for occurrence of fusion ($p>0.05$). No statistically significant gender differences were found in the occurrence of CVA in the TMD group and the control group ($p>0.05$).

Conclusion: No difference was found in the occurrence of fusion between the subjects with TMD and the subjects without TMD, and the occurrence of PAD was significantly increased in subjects with TMD. (*Turkish J Orthod*. 2014;27:136–142)

KEY WORDS: Cervical vertebral anomalies, Transverse maxillary deficiency

INTRODUCTION

Congenital anomalies of the cervical spine range in severity from benign or asymptomatic to anomalies that can potentially cause severe complications. Anomalies of the occipitocervical junction are often not detected until late childhood or adolescence, and some remain hidden well into adult life.[1] Other anomalies of the cervical spine, although recognized in early life, may not become clinically significant until adulthood.[1]

In 1982, Farman and Escobar[2] described the radiographic appearance of cervical vertebrae anomalies (CVAs) on standardized lateral cephalometric films. These anomalies include variations in the normal anatomy of the atlas, occipitalization of the atlas, progressive degrees of lipping, supernumerary vertebrae, accessory ossicles, block vertebrae, and spina bifida.[2,3] Sandham[4] classified the characteristics of the cervical column and categorized CVAs into 2 main groups: posterior arch deficiencies of C1 (PADs) and fusions.

To date, CVAs have been related to cleft lip and palate,[4–6] condylar hypoplasia,[7] skeletal deep bite,[8] skeletal open bite,[9] transverse maxillary deficiency (TMD),[10] skeletal maxillary overjet,[11] skeletal mandibular overjet,[12] craniosynostosis syndromes,[13,14] and some craniofacial syndromes, such as Klippel-Feil, Saethre-Chotzen, and Down syndromes.[15–17] On the other hand, anomalies in the cervical column morphology occur in healthy subjects with neutral occlusion and normal craniofacial morphology as well as subjects with craniofacial syndromes and severe malocclusion.[11] Sonnesen and Kjaer[11] found that fusions between the second and third cervical

[1]Assistant Professor, Kocaeli University, Faculty of Dentistry, Department of Orthodontics, Kocaeli, Turkey
[2]Assistant Professor, Trakya University, Faculty of Dentistry, Department of Orthodontics, Edirne, Turkey

*Corresponding author: A. Burcu Altan, Kocaeli University, Department of Orthodontics, Kocaeli, Turkey. Tel: 00905323182779 E-mail: burcuk12@yahoo.com

To cite this article: Altan AB, Dönmez Zorkun B. Cervical vertebral anomalies in patients with transverse maxillary deficiency. Turkish J Orthod. 2014;27:136–142 (DOI: http://dx.doi.org/10.13076/TJO-D-15-00011)
Date Submitted: February 2015, March 2015. Date Accepted: April 2015.

Table 1. Distribution of the subjects according to type of malocclusion

	N	Class I ANB 0–4°	Class II ANB > 4°	Class III ANB < 0°
Transverse maxillary deficiency group	47	14	17	16
Control group	47	28	13	6

vertebrae develop in 14.3% of healthy subjects. Therefore, fusions of the upper cervical column in that range are considered normal.

TMD is a pathological condition with aesthetic[18] and functional implications, including respiratory problems.[19] The incidence is around 3–18% in patients undergoing orthodontic treatment.[18,20] Impaired nasal breathing has been attested as one of the etiologic factors causing TMD.[19] McGuinness and McDonald[21] found that the extended head posture in adolescents with posterior cross-bite decreased progressively after rapid maxillary expansion. The authors attributed this finding to the change in breathing type, from oral to nasal breathing. Furthermore, Huggare and Kylämarkula[22] observed reduced thickness in the posterior arch of the atlas in subjects with altered respiratory function. They assumed that cranial extension induced by respiratory obstruction impaired the soft tissues around the atlas (functional matrix). Similarly, Sonnesen and Kjaer[9,11] investigated the morphology of vertebrae in subjects with Class II malocclusion, increased overjet, and/or open bite, and found that vertebral anomalies were increased in association with oral respiration. Recently, Di Vece *et al.*[10] reported a statistically significant correlation between transverse maxillary constriction and cervical vertebral defects.

The aim of this study was to examine the occurrence of the types of CVA in subjects with TMD and to compare this with the occurrence of CVA in a control group without TMD.

SUBJECTS AND METHODS

A sample of 47 Turkish subjects with TMD (17 male, 30 female), aged 10.2 to 14.3 years, was obtained from the records of Cumhuriyet University, Faculty of Dentistry, Department of Orthodontics, according to the following criteria: (1) bilateral posterior crossbite and indication for rapid maxillary expansion (2) medical history and examination negative for congenital maxillofacial malformations and related syndromes, severe skeletal asymmetries, systemic muscle or joint disorders, or dentofacial deformities requiring orthognathic surgery; (3) no history of previous orthodontic treatment; (4) at least 24 permanent teeth; (5) clear pretreatment lateral cephalometric radiographs with good contrast, including those of vertebrae from C2 to C4. The control group consisted of 47 subjects (15 boys, 32 girls), aged 11.1 to 15.6 years, with adequate maxillary transversal dimensions that were obtained from same records. The inclusion criteria for the control group were the same as for the study group, with the exception of the first criteria. The distribution of subjects according to type of malocclusion is shown in Table 1.

The morphology of the cervical column was assessed from visual inspection of the first 4 cervical vertebrae as they are normally seen on standardized lateral cephalometric films. For each subject, the first 4 vertebrae and the atlantooccipital articulation were traced on matte-acetate tracing paper. Normal appearance was determined when the first 4 cervical vertebrae could be traced as separate entities with all anatomic components present (Fig. 1). Characteristics of the cervical column were classified according to the method of Sandham,[4] and the cervical vertebral anomalies were categorized into 2 main groups: (1) posterior arch deficiency at C1 (PAD) (Fig. 2), spina bifida, and dehiscence, and (2) fusion anomalies (Fig. 3), including fusion of 2 cervical bodies, block fusion when more than 2 bodies were fused, and occipitalization of C1 and the occipital bone.

Statistical Analysis

Differences in occurrence of CVA between the groups and genders were assessed by χ^2 independence test. The results were considered significant at $p \leq 0.05$. The statistical analyses were performed with SPSS software (version 16.0, SPSS, Chicago, IL, USA).

RESULTS

In the TMD group, 10,6% of the subjects had fusion in the cervical column, and 14,9% had PAD. The fusion always occurred between C2 and C3. No statistically significant gender differences were found in the occurrence of CVA ($p>0.05$; Table 2).

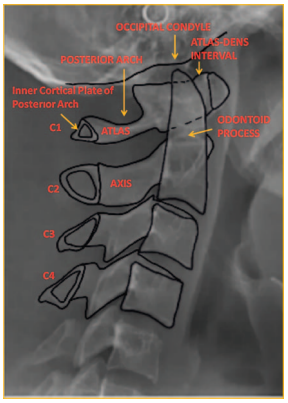

Figure 1. Radiographic view of the cervical spine with accompanying trace.

In the control group, 17% of the subjects had fusion of the cervical column, and 8,5% had PAD; PAD occurred in combination with fusion in 1 subject. The fusion always occurred between C2 and C3. No statistically significant gender differences were found in the occurrence of CVA ($p>0.05$; Table 2).

The comparison of the TMD group and the control group revealed no significant difference in the occurrence of fusion ($p>0.05$; Table 2), but the occurrence of PAD was significantly increased in TMD group ($p<0.05$; Table 2).

DISCUSSION

In previous studies, some orthodontic malocclusions, including TMD, have been associated with morphologic anomalies of cervical vertebrae.[4–12] In this study, the occurrence of types of CVA in subjects with TMD was investigated and compared with the occurrence of CVA in a control group without TMD.

Roentgenographic examination of the cervical spine may reveal a pathologic disorder in asymptomatic and symptomatic subjects.[23] Unfortunately radiographic abnormalities of the cervical spine do not always signal their existence.[3] Early detection can save patients from further neurologic injuries.[3] Hensinger[24] reported that upper cervical anomalies, such as atlanto-occipital fusion, anomalies of the odontoid process, or anomalies of the transverse ligament at a young age could lead to instability and neurologic problems secondary to minor trauma.

Ross and Lindsay[5] suggested that severe vertebral anomalies are an early developmental fault of mesenchyme. Sonnesen *et al*.[7] and Sonnesen and Kjaer[8,9,11,12] concluded that the cervical column differs phenotypically in the various skeletal malocclusion traits. According to the results of Sandham's[4] study, the occurrence of the different types of cervical vertebral anomalies in subjects with cleft does not follow a similar pattern, suggesting different etiologic mechanisms. It is not known why these malformations occur in the cervical column and why they occur with different frequencies.[11] Because vertebral bodies form around the notochord in the prenatal period, some researchers have suggested that the notochord may be responsible for the location and morphology of cervical vertebrae.[25,26] It was assumed that because the cervical vertebrae and cranial base have similar embryonic origin and the jaws are attached to the cranial base, the cranial base could be considered to be a developing link between the cervical vertebral column and the jaws.[27] Consequently, fusion anomalies were attributed as signs of deviations in the early development period or signaling among the notochord, neural tube, neural crest cells, and para-axial mesoderm.[1] Also, the mechanism involved in palatal shelf fusion during embryonic development has been attested to have an effect on the development and fusion of the posterior arch of the first cervical vertebrae.[4]

The frequency of the occurrence of the 2 types of CVA was different in the groups in this study. The most common anomaly in the TMD group was PAD, and fusion was the most common anomaly in the control group. The frequency of PAD in this study was in accordance with the frequencies reported in previous studies of orthodontic patients with deep-bite,[8] open-bite,[9] severe maxillary,[11] or mandibular overjet.[12] On the other hand, the occurrence of fusion was significantly rarer than has been reported in literature.[1,7–9,11,12]

Sonnesen *et al*.[7] reported that deviations in head posture and cranial base angle were sexually dimorphic, showing larger cervicohorizontal and cranial base angles in females than males. Additionally, a positive correlation between the aforementioned deviations and fusions of the cervical column was observed in females, whereas this correlation was not found in males. Hence, it could be hypothesized that fusion anomalies show a dimorphic pattern in their occurrence.[1] However, Sonnesen and Kjaer,[8] Faruqui *et al*.,[1] and Arntsen and Sonnesen[28] attested that there was no significant gender difference in the occurrence of CVAs. Similarly, the current study did not reveal gender tendency for occurrence of CVA.

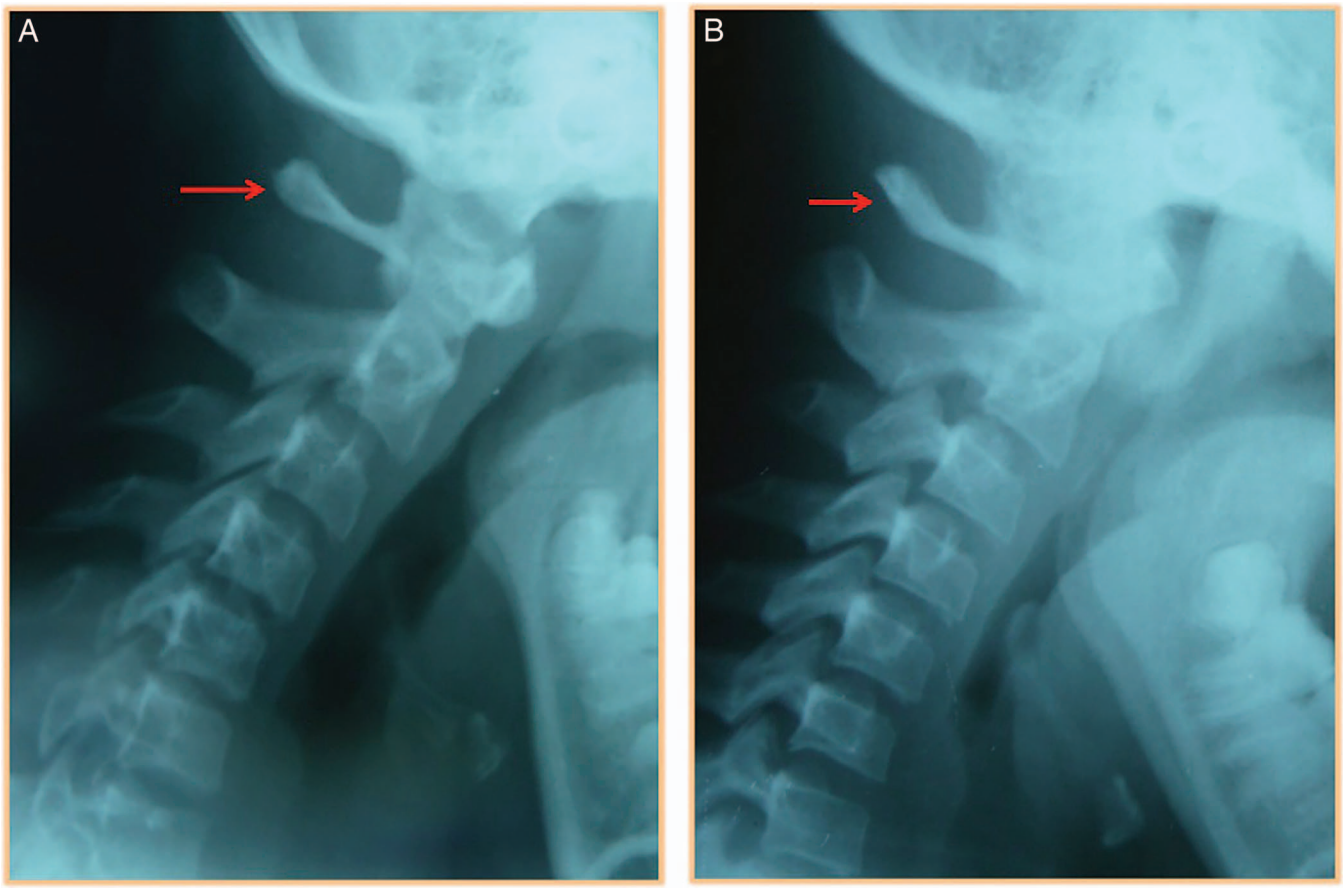

Figure 2. (A and B) Posterior arch deficiency (spina bifida) at C_1.

Impaired nasal breathing has been found to be one of the etiologic factors causing TMD.[19] Huggare and Kylämarkula[22] observed reduced thickness of the posterior arch of the atlas in subjects with altered respiratory function. Similarly, Sonnesen and Kjaer[9,11] found that vertebral anomalies are increased in association with oral respiration. Considering the literature, Di Vece *et al*.[10] hypothesized that the morphology of the cervical vertebrae could be affected by TMD. To explain this phenomenon, the authors[10] asserted an adaptation process associated with oral breathing, depending on the soft-tissue stretching hypothesis of Solow and Kreiborg.[29] According to this hypothesis, respiratory obstruction starts neuromuscular feedback that can alter craniocervical posture, leading to stretching of soft tissues that can increase the pressure they exert on skeletal tissues, modifying the direction of bone growth. Similarly, some authors believe that external agents such as pressure,[30] body posture,[31] and facial components[32] can modify the height of vertebral bodies. On the other hand, the clinical effects of breathing pattern on TMD are not predictable.[10] Although there is significant evidence that poor nasal breathing will lead to mouth-nasal breathing, its impact on dentofacial growth is still unclear.[33] Souki *et al*.[34] observed that individual facial genotypes had different sensitivity on developing malocclusion; following the exposure to mouth breathing, a wide variety of interarch relationships could be found. It has been also shown that adenoidal/tonsillar hyperplasia or the presence of rhinitis have no association with the prevalence of Class II malocclusion, anterior open bite, and posterior crossbite in that sample of mouth breathers.[34]

In the study of Di Vece *et al*.,[10] the relationship between CVA and TMD was investigated, and a significant correlation was reported. For visual analysis of the cervical vertebrae, morphologic anomalies known as vertebral defects were classified as round or concave by assessing the contact points of the edges of the vertebrae.[10] This method was different from the one used in the current study and in several studies[1,7–9,11,12] that evaluated the association between CVA and orthodontic anomalies. In the most used method, the classification of

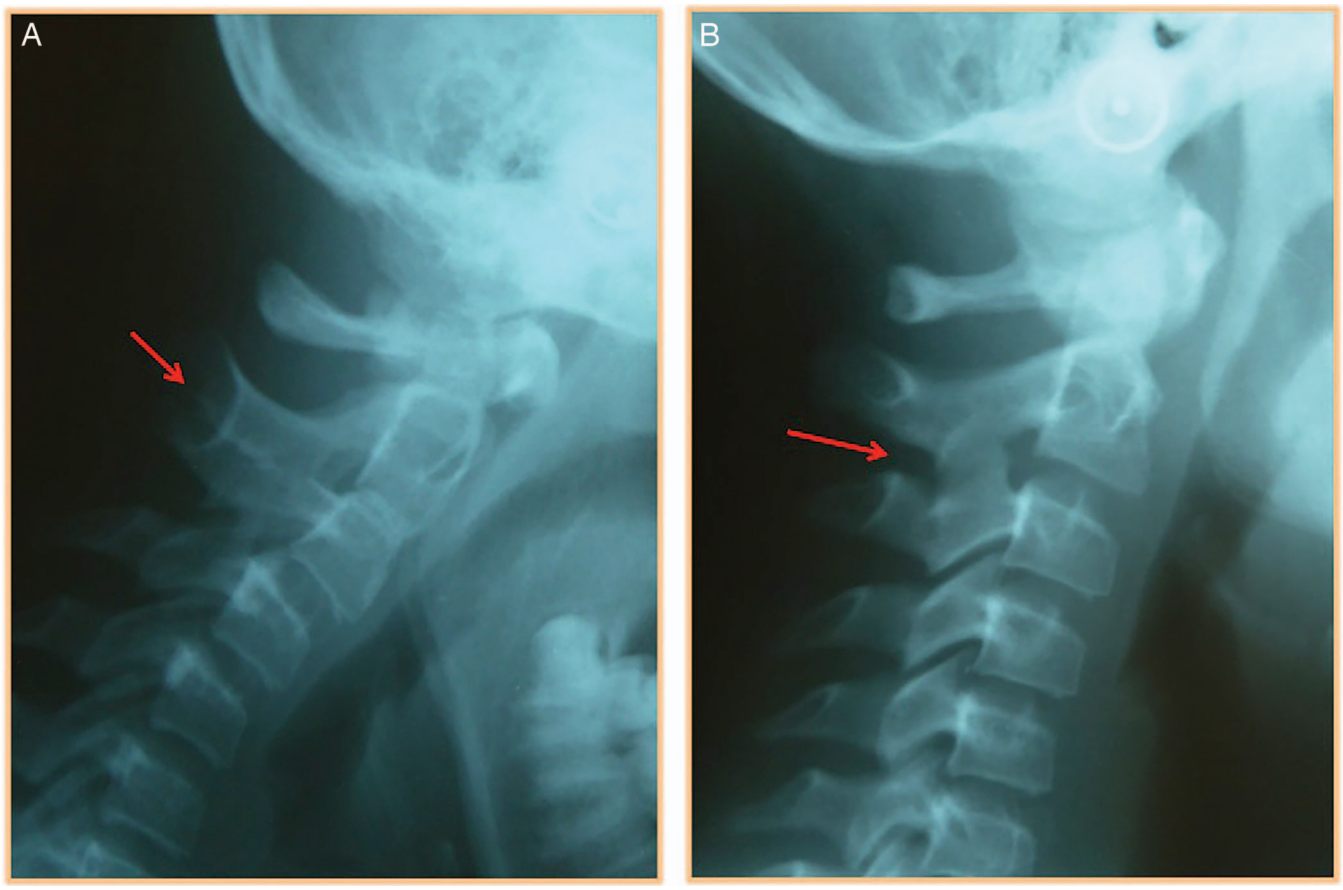

Figure 3. A, B. Fusion of the posterior arches of C2 and C3.

Sandham,[4] CVA divided into 2 groups; PAD and fusions. According to this classification, we observed a significant difference between groups in the occurrence of PAD, though there was no difference for the occurrence of fusions. The similarity between our findings about PADs and the findings of Di Vece *et al*.[10] might depend on the similarity of the etiologic mechanisms that was incorporated into the development of these anomalies. Likewise, the reason for the incompatibility of the findings about fusions might be also the difference in etiologic mechanisms, as Sandham[4] reported.

In the literature, several dental and skeletal malocclusions, as well as some congenital anomalies, have been related to anomalies of the cervical vertebral column.[4–12] On the other hand, it has been reported that variations of cervical column morphology also occur in healthy subjects with neutral occlusion and normal craniofacial morphology.[11] In our study, the control group was obtained from an orthodontic population to avoid any additional radiographic exposure to subjects as lateral cephalometric radiograph is a routine procedure required for orthodontic treatment planning. The subjects in this group did not have any major skeletal discrepancies or deviating craniofacial morphology but had an orthodontic malocclusion to some degree. To discard subjects with severe skeletal malocclusion,

Table 2. Comparison of the groups for the occurrence of posterior arch deficiencies of C1 (PAD) and fusion (χ^2 test)

		PAD			Fusion		
	N	**Girls**	**Boys**	***p***	**Girls**	**Boys**	***p***
Transverse maxillary deficiency group	47	5	2	0.65	3	2	0.85
Control group	47	2	2	0.41	6	2	0.64
p				0,036*			0,370

* $p < 0.05$.

no subject who needed orthognathic surgery was included into the groups. The distribution of the subjects according to type of skeletal malocclusion was balanced in the study group. We preferred subjects with skeletal Class I primarily for the control group. On the other hand, if the control group had consisted of subjects with Class I only; we could not evaluate the effect of TMD on CVA because the occurrence might depend on other types of skeletal malocclusions as well, as has been reported in the literature.[4–12] It would have been better if the study group had included more subjects with skeletal Class I malocclusion. However, most of the patients with TMD had either skeletal Class II or Class III malocclusion.

Previous studies also found that different genes act in different regions,[35,36] and this might be the focus of future studies on the pathogenesis. Genetic studies with a prenatal insight into normal and pathologic associations in development in the cranial base, the jaws, and the cervical region are necessary to explain the etiology of skeletal deviations in craniofacial morphology.

CONCLUSION

- The occurrence of fusion was 17% and the occurrence of PADs was 8,5% in the examined orthodontic population.
- No difference was found in the occurrence of fusion between the subjects with TMD and the subjects without TMD; while the occurrence of PAD was significantly increased in subjects with TMD.
- No gender tendency was observed in the occurrence of cervical vertebral anomalies in both groups.

REFERENCES

1. Faruqui S, Fida M, Shaikh A. Cervical vertebral anomalies in skeletal malocclusions: a cross-sectional study on orthodontic patients at the Aga Khan University Hospital, Pakistan. *Indian J Dent Res*. 2014;25:480–484.
2. Farman AG, Escobar V. Radiographic appearance of the cervical vertebrae in normal and abnormal development. *Br J Oral Surg*. 1982;20:264–274.
3. Vastardis H, Evans CA. Evaluation of cervical spine abnormalities on cephalometric radiographs. *Am J Orthod Dentofacial Orthop*. 1996;109:581–588.
4. Sandham A. Cervical vertebral anomalies in cleft lip and palate. *Cleft Palate J*. 1986;23:206–214.
5. Ross RB, Lindsay WK. The cervical vertebrae as a factor in etiology of cleft palate. *Cleft Palate J*. 1965;36:273–281.
6. Rajion ZA, Townsend GC, Netherway DJ, Anderson PJ, Yusof A, *et al*. A three-dimensional computed tomographic analysis of the cervical spine in unoperated infants with cleft lip and palate. *Cleft Palate Craniofac J*. 2006;43:513–518.
7. Sonnesen L, Pedersen CE, Kjaer I. Cervical column morphology related to head posture, cranial base angle, and condylar malformation. *Eur J Orthod*. 2007;29:398–403.
8. Sonnesen L, Kjaer I. Cervical vertebral body fusions in patients with skeletal deep bite. *Eur J Orthod*. 2007;29:464–470.
9. Sonnesen L, Kjaer I. Cervical column morphology in patients with skeletal open bite. *Orthod Craniofac Res*. 2008;11:17–23.
10. Di Vece L, Faleri G, Picciotti M, Guido L, Giorgetti R. Does a transverse maxillary deficit affect the cervical vertebrae? A pilot study. *Am J Orthod Dentofacial Orthop*. 2010;137:515–519.
11. Sonnesen L, Kjaer I. Anomalies of the cervical vertebrae in patients with skeletal Class II malocclusion and horizontal maxillary overjet. *Am J Orthod Dentofacial Orthop*. 2008;133:188.e15–e20.
12. Sonnesen L, Kjaer I. Cervical column morphology in patients with skeletal Class III malocclusion and mandibular overjet. *Am J Orthod Dentofacial Orthop*. 2007;132:427.e7–e12.
13. Hemmer KM, McAlister WH, Marsh JL. Cervical spine anomalies in the craniosynostosis syndromes. *Cleft Palate J*. 1987;24:328–333.
14. Thompson DN, Slaney SF, Hall CM, Shaw D, Jones BM, Hayward RD. Congenital cervical spinal fusion: a study in Apert syndrome. *Pediatr Neurosurg*. 1996;25:20–27.
15. Anderson PJ, Hall CM, Evans RD, Hayward RD, Harkness WJ, Jones BM. The cervical spine in Saethre-Chotzen syndrome. *Cleft Palate Craniofac J*. 1997;34:79–82.
16. Frost M, Huffer WE, Sze CI, Badesch D, Cajade-Law AG, Kleinschmidt-DeMasters BK. Cervical spine abnormalities in Down syndrome. *Clin Neuropathol*. 1999;18:250–259.
17. Shen FH, Samartzis D, Herman J, Lubicky JP. Radiographic assessment of segmental motion at the atlantoaxial junction in the Klippel-Feil patient. *Spine (Phila Pa 1976)*. 2006;31:171–177.
18. Betts NJ, Vanarsdall RL, Barber HD, Higgins-Barber K, Fonseca RJ. Diagnosis and treatment of transverse maxillary deficiency. *Int J Adult Orthodon Orthognath Surg*. 1995;10:75–96.
19. Kilic N, Oktay H. Effects of rapid maxillary expansion on nasal breathing and some naso-respiratory and breathing problems in growing children: a literature review. *Int J Pediatr Otorhinolaryngol*. 2008;72:595–601.
20. Hartgerink DV, Vig PS, Abbott DW. The effect of rapid maxillary expansion on nasal airway resistance. *Am J Orthod Dentofac Orthop*. 1987;92:381–389.
21. McGuinness NJ, McDonald JP. Changes in natural head position observed immediately and one year after rapid maxillary expansion. *Eur J Orthod*. 2006;28:126–134.
22. Huggare J, Kylämarkula S. Morphology of the first cervical

vertebra in children with enlarged adenoids. *Eur J Orthod*. 1985;7:93–96.

23. McRae DL. The significance of abnormalities of the cervical spine. *Am J Roentgenol Radium Ther Nucl Med*. 1960;84:3–25.
24. Hensinger RN. Congenital anomalies of the cervical spine. *Clin Orthop*. 1991;264:16–38.
25. Kjaer I. Neuro-osteology. *Crit Rev Oral Biol Med*. 1998;9:224–244.
26. Sadler TW. Embryology of neural tube development. *Am J Med Genet C Semin Med Genet*. 2005;135C:2–8.
27. Sonnesen L. Associations between the cervical vertebral column and the craniofacial morphology. *Int J Dent*. 2010;2010:295728.
28. Arntsen T, Sonnesen L. Cervical vertebral column morphology related to craniofacial morphology and head posture in preorthodontic children with Class II malocclusion and horizontal maxillary overjet. *Am J Orthod Dentofacial Orthop*. 2011;140:e1–e7.
29. Solow B, Kreiborg S. Soft-tissue stretching: a possible control factor in craniofacial morphogenesis. *Scand J Dent Res*. 1977;85:505–567.
30. Gooding CA, Neuhauser EB. Growth and development of the vertebral body in the presence and absence of normal stress. *Am J Roentgenol Radium Ther Nucl Med*. 1965;93:388–394.
31. Bridges PS. Vertebral arthritis and physical activities in the prehistoric southeastern United States. Am J Phys Anthropol. 1994;93:83–93.
32. Bench RW. Growth of the cervical vertebrae as related to tongue, face and denture behavior. *Am J Orthod*. 1963;49:183–214.
33. Warren DW. Effect of airway obstruction upon facial growth. *Otolaryngol Clin North Am*. 1990;23:699–712.
34. Souki BQ, Pimenta GB, Souki MQ, Franco LP, Becker HM, Pinto JA. Prevalence of malocclusion among mouth breathing children: do expectations meet reality? *Int J Pediatr Otorhinolaryngol*. 2009;73:767–773.
35. Acampora D, D'Esposito M, Faiella A, Pannese M, Migliaccio E, *et al*. The human HOX gene family. *Nucleic Acids Res*. 1989;17:10385–10402.
36. Krumlauf R. Hox genes in vertebrate development. *Cell*. 1994;78:191–201.