ORIGINAL ARTICLE

Changes in mandibular position and upper airway dimension by wearing cervical headgear during sleep

Shigetoshi Hiyama, DDS, PhD,[a] Takashi Ono, DDS, PhD,[b] Yasuo Ishiwata, DDS, PhD,[c] and Takayuki Kuroda, DDS, PhD[d]
Tokyo, Japan

We previously reported that the wearing of cervical headgear induced forward displacement of the mandible in awake subjects. However, it was unclear whether such mandibular displacement also occurred during sleep. The purpose of this study was to examine changes in mandibular position and oropharyngeal structures that were induced by the wearing of cervical headgear during sleep. Ten healthy adults (7 male and 3 female) who gave their informed consent were included in this study. A pair of lateral cephalograms was taken with the patient in the supine position with and without cervical headgear at end-expiraton during stage 1 to 2 non–rapid-eye-movement sleep. The Wilcoxon signed-rank test was used for a statistical analysis. The amount of jaw opening was significantly decreased by the wearing of the cervical headgear ($P < .05$), although no significant anteroposterior mandibular displacement was induced. The sagittal dimension of the upper airway was significantly reduced ($P < .05$); however, no significant changes were observed in the vertical length of the upper airway. Although the hyoid bone and the third cervical vertebra moved significantly forward by the wearing of the cervical headgear ($P < .05$), the relationship among the mandibular symphysis, the hyoid bone, and the third cervical vertebra did not change. These results suggest that cervical headgear significantly reduced the sagittal dimension of the upper airway during sleep, although there was no significant anteroposterior displacement of the mandible. (Am J Orthod Dentofacial Orthop 2001;120:160-8)

Over the past few decades, many cephalometric studies have examined the effect of cervical headgear (CHG) on craniofacial growth, and the inhibition of maxillary growth has been commonly described as an important effect of treatment with this orthodontic appliance.[1-8] In addition, several cephalometric studies have examined the effect of CHG on mandibular growth. Jakobsson,[2] Wieslander,[3] and Tulloch et al[7] reported that CHG had no influence on mandibular growth; Meach[1] and Mills et al[4] showed that it inhibited forward growth of the mandible. In contrast, Sauer and Kuftinec,[5] Baumrind et al,[6,9,10] and Keeling et al[8] demonstrated that CHG facilitated mandibular growth. Thus, the previous findings conflict, and a common understanding has not yet been reached. Considering that CHG does not exert orthopedic forces directly on the mandible, its effects on mandibular growth are intriguing. Graber[11] mentioned that reflective forward mandibular thrust was associated with CHG therapy and that such “activator-like” movement could be beneficial to mandibular growth. Recently, we reported that the resting mandibular position moved anteriorly when awake subjects wore CHG and suggested that Graber’s hypothesis was valid under these conditions.[12] Moreover, the change in mandibular position seemed to be related to the reduction in the upper airway dimension caused by the pressure on the back of the neck by the neck strap.[12]

Usually, an orthodontic patient is instructed to use CHG at night during sleep. Because our previous study,[12] which examined changes in the mandibular position induced by CHG, was performed in awake subjects, it was unclear whether the mandibular displacement that was observed during wakefulness could also occur during sleep. Because the muscle tone during sleep is less than that during wakefulness,[13-15] it is likely that CHG-induced changes in both the mandibular position and upper airway dimension during sleep would be different from those in awake subjects.

From Maxillofacial Orthognathics, Graduate School, Tokyo Medical and Dental University.
[a]Postdoctoral fellow.
[b]Assistant Professor.
[c]Part-time lecturer.
[d]Professor and Chair.
Supported by a Grant-in-Aid for Scientific Research (No.10307052) from the Japanese Ministry of Education, Science, Sports and Culture.
Reprint requests to: Shigetoshi Hiyama, DDS, PhD, Maxillofacial Orthognathics, Graduate School, Tokyo Medical and Dental University, 5-45 Yushima 1-chome, Bunkyo-ku, Tokyo, 113-8549 Japan.
Submitted, May 2000; revised and accepted, October 2000.

0889-5406/2001/$35.00 + 0 **8/1/113788**
doi:10.1067/mod.2001.113788

The purpose of this study was to examine, with the use of supine lateral cephalograms, the changes in mandibular position and oropharyngeal structures that are induced by wearing the CHG during sleep.

MATERIAL AND METHODS

Subjects

Seven male and 3 female healthy adults participated in this study (mean age, 25.9 ± 1.6 years). All of the participants had normal occlusal relationships (ANB angle, 2.7 ± 2.5°) and were confirmed to have no medical history of temporomandibular disorders or sleep-disordered breathing (such as snoring or apnea). Before the study, all of the subjects gave their informed consent to participate after receiving a full explanation of the aim and design of this study.

Experimental procedures

The subjects were instructed to stay awake all night immediately before the experiment to help ensure that they could fall asleep easily. Buccal tubes (F179-42,43; TOMY International, Tokyo, Japan) were bonded to the buccal surfaces of bilateral maxillary first molars. After the face bow (A45-0601; TOMY International) and neck strap (422-13; TOMY International) were adjusted, CHG was individually fitted for each subject. The force was adjusted to 700 g. Silver/silver chloride surface electrodes (NE-155A; Nihon Kohden, Tokyo, Japan) to record electroencephalograms (C3/A2 of the international 10-20 system) for determining the sleep stage were positioned with paste (Cardio cream Z-101BC; Nihon Kohden). The ground electrode was attached to the right ear lobe. The signals were amplified through an amplifier (Dual-channel bioelectric amplifier MEG-2100; Nihon Kohden) and monitored on an oscilloscope (Handy monitor VC-22; Nihon Kohden). Low- and high-cut filters were set at 0.5 Hz and 30 Hz, respectively. The sweep velocity of the oscilloscope was set at 25 mm/s. A thermistor (TR-712T; Nihon Kohden) to detect nasal airflow was attached to the right nostril, and the amplified signal was monitored simultaneously with the electroencephalogram on the oscilloscope.

Each subject was asked to lie on a cushioned table in the supine position with the head supported by a pillow (Fig 1). The cephalostat for the table enabled the head of the subject to be fixed without pain or discomfort by the insertion of ear rods to bilateral external auditory meatuses. The focus-film distance and object-film distance were adjusted to 170 cm and 20 cm, respectively. The exposure factors, which included the tube voltage, the tube current, and the exposure time, were set at 95 kVp and 5 mAs.

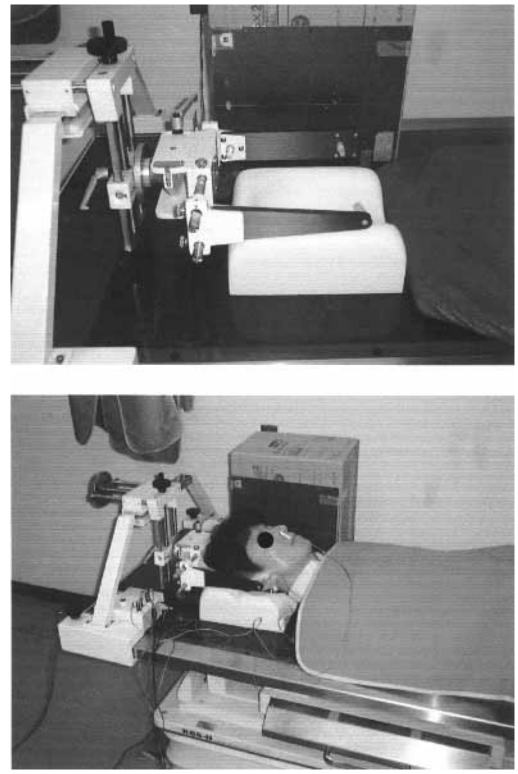

Fig 1. Equipment used in this study to take supine lateral cephalograms.

The room light was turned off, and the subject was asked to fall asleep without the CHG. A supine lateral cephalogram was taken at the end-expiration during stage 1 to 2 non–rapid-eye-movement (NREM) sleep. The room light was then turned on, and the subject was awakened to be fitted with the CHG without removal of the ear rods. Thereafter, the subject was asked to sleep again, and a supine lateral cephalogram with the CHG was taken in the same manner.

Data analysis

Lateral cephalograms of each subject were traced and superimposed on the SN plane, registered at S by a skilled orthodontist. A detailed description of the supine cephalometric protocol is given in Fig 2. The Wilcoxon signed-rank test was used to compare differences with and without the CHG for each variable. Probability values less than .05 were considered statistically significant.

Reliability

Ten supine lateral cephalograms of 5 randomly selected subjects were retraced and reanalyzed on another day by the same investigator, and the method

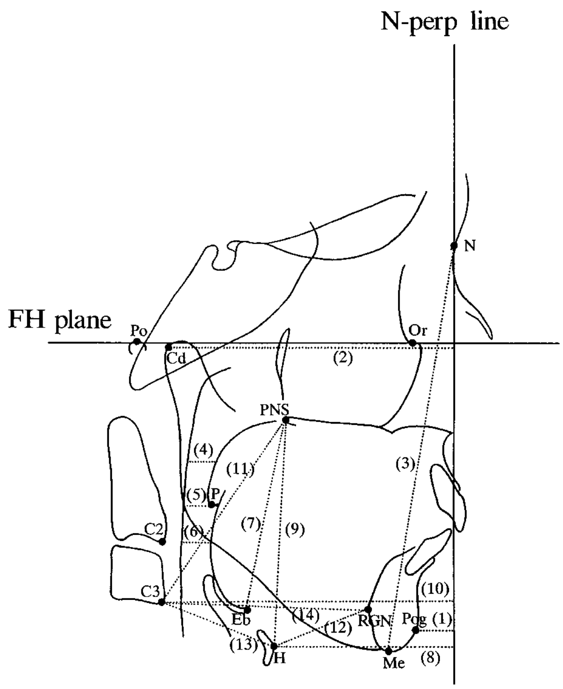

Fig 2. Diagrammatic representation of anatomic points and lines used to identify cephalometric variables. *N*, Most anterior point of frontonasal suture in median plane; *Or*, lowest point in inferior margin of orbit; *Po*, superior point of external auditory meatus; *PNS*, posterior nasal spine; *RGN*, most posterior point on mandibular symphysis; *Me*, most inferior midline point on mandibular symphysis; *H*, most superoanterior point on body of hyoid bone; *Cd*, most superoposterior point on condyle; *P*, tip of soft palate; *Eb*, most inferior point on epiglottis; *C2*, most inferoanterior point on body of second cervical vertebra; *C3*, most inferoanterior point on body of third cervical vertebra; *FH plane*, line through Or and Po; *N-perp*, line perpendicular to FH plane through N; *(1)*, N-perp to Pog (distance between N-perp and Pog; when Pog is anteriorly positioned relative to N-perp, this value is considered positive); *(2)*, N-perp to Cd (distance between N-perp and Cd); *(3)*, N-Me (distance between N and Me); *(4)*, SPPS (anteroposterior width of pharynx measured between posterior pharyngeal wall and dorsum of soft palate on a line parallel to FH plane that runs through middle of line from PNS to P); *(5)*, MPS (anteroposterior width of pharynx measured between posterior pharyngeal wall and dorsum of tongue on a line parallel to FH plane that runs through P); *(6)*, IPS (anteroposterior width of pharynx measured between posterior pharyngeal wall and dorsum of tongue on a line parallel to FH plane that runs through C2); *(7)*, PNS-Eb (distance between PNS and Eb); *(8)*, N-perp to H (distance between N-perp and H); *(9)*, PNS-H (distance between PNS and H); *(10)*, N-perp to C3 (distance between N-perp and C3); *(11)*, C3-PNS (distance between C3 and PNS); *(12)*, H-RGN (distance between H and RGN); *(13)*, C3-H (distance between C3 and H); *(14)*, C3-RGN (distance between C3 and RGN).

Table I. Changes in each cephalometric variable in male and female samples (measurement value with CHG minus that without CHG)

	*Male (mm)**	*Female (mm)**	*Significance*
N-perp to Pog	1.1 ± 2.0	1.3 ± 0.5	NS
N-perp to Cd	–0.1 ± 0.4	–0.4 ± 0.1	NS
N-Me	–0.8 ± 1.8	–1.0 ± 0.4	NS
SPPS	–0.8 ± 1.1	–1.1 ± 1.8	NS
MPS	–1.7 ± 2.0	–2.8 ± 1.2	NS
IPS	0 ± 3.1	–1.9 ± 1.5	NS
PNS-Eb	0.3 ± 2.5	0.1 ± 1.8	NS
N-perp to H	–1.8 ± 1.4	–0.1 ± 3.2	NS
PNS-H	0.6 ± 1.6	0 ± 3.0	NS
N-perp to C3	–2.4 ± 1.8	0.1 ± 2.9	NS
C3-PNS	–1.8 ± 1.1	–0.5 ± 2.5	NS
H-RGN	–0.5 ± 1.6	–0.6 ± 3.9	NS
C3-H	–0.3 ± 1.3	–0.1 ± 1.4	NS
C3-RGN	–1.3 ± 1.6	–0.1 ± 3.9	NS

Values given as mean ± SD.
*Mann-Whitney *U*-test.

error for each variable was calculated to determine the measurement reproducibility: $(\Sigma d^2/2n)^{1/2}$, where d represents the difference between the first and second measurements, and n denotes the sample size.

Furthermore, we performed a repeatability test to examine the reproducibility of the mandibular posture, upper airway dimension, and related structures during sleep. For this purpose, sets of 2 supine lateral cephalograms of 4 males and 1 female were taken without the CHG (ie, after a supine lateral cephalogram was taken without the CHG under the conditions described earlier, the subject was awakened and then asked to sleep again without the CHG). A supine lateral cephalogram was taken in the same manner. The 2 supine lateral cephalograms for each subject were traced and measured in a blinded fashion, and the differences in the 2 measurements were calculated.

RESULTS

Table I shows the changes in all of the variables in male and female subjects. Because we did not find any significant differences by gender, the male and female subjects were regarded as a single group, and the following data analysis was performed on all of the subjects without considering gender.

With regard to anteroposterior positional changes of the mandible, the N-perp to Pog distance was significantly increased by wearing the CHG, which indicated that the mandibular symphysis moved forward (by 1.2 ± 1.6 mm) by wearing the CHG during sleep. However, the N-perp to Cd distance showed no significant changes, which indicated that wearing the CHG did not induce anteroposterior condylar displacement.

With regard to vertical mandibular positional changes, although no significant changes were observed in N-Me (P = .09), wearing the CHG reduced this distance in 8 of the 10 subjects. This reflects a tendency for jaw closing (Fig 3).

The sagittal dimension of the middle pharyngeal airway (MPS) was significantly reduced (by 2.0 ± 1.8 mm) by wearing the CHG (Fig 4). In contrast, the sagittal dimensions of the superior (SPPS) and inferior (IPS) pharyngeal airways showed no significant changes. The probability values for these variables were .07 and .10, respectively. Although there were no significant changes in the SPPS or IPS, these were reduced in all but 2 subjects for the SPPS and in all but 1 subject for the IPS. There were no significant changes in the vertical length of the upper airway (PNS-Eb).

The N-perpendicular to H distance was significantly decreased by wearing the CHG, which is related to forward displacement of the hyoid bone (Fig 5). The hyoid bone moved forward by 1.3 ± 2.1 mm. On the other hand, no significant superoinferior positional changes of the hyoid bone were seen. Both the N-perpendicular to C3 and C3-PNS distances were significantly decreased by wearing the CHG (by 1.7 ± 2.3 mm and 1.4 ± 1.6 mm, respectively), which reflected forward displacement of the third cervical vertebra (Fig 5).

The relationship among the mandibular symphysis, the hyoid bone, and the third cervical vertebra remained constant (Fig 6).

Fig 7 shows typical changes in oropharyngeal structures with and without the CHG in 1 subject. Counterclockwise rotation of the mandible, narrowing of the upper airway, and forward displacement of both the hyoid bone and cervical vertebra are shown.

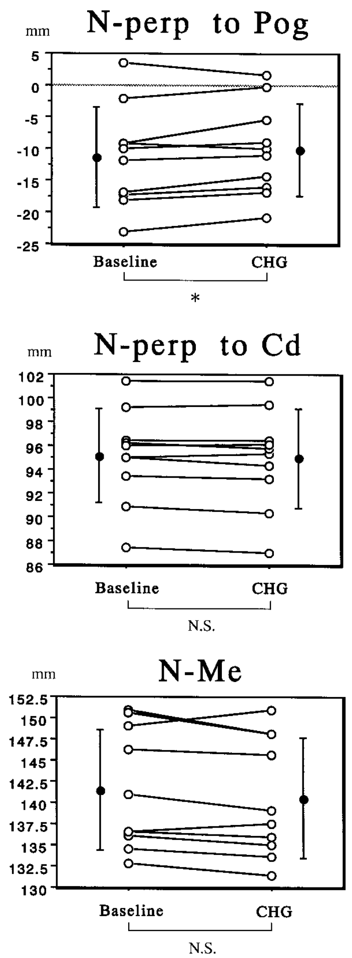

Fig 3. Changes in mandibular position. *Vertical bar* indicates mean ± SD; *baseline* indicates value measured during sleep without CHG; *CHG* indicates value measured during sleep with CHG. $^{*}P < .05$.

DISCUSSION

In our previous study,[12] we found that significant forward displacement of the mandible was induced in awake subjects who wore CHG. These results suggested that such a change in the mandibular position could dilate the upper airway space that was threatened by the pressure on the back of the neck from the neck strap. Although our findings supported Graber's proposal,[11] it was unclear whether the same change in mandibular position could be induced during sleep, when patients are usually instructed to use the appliance. If changes in the mandibular position and upper airway dimension that were induced by wearing CHG during sleep were different from those during wakefulness, a new perspective regarding treatment with CHG might be obtained.

A few studies have examined the upper airway morphologic features with supine lateral cephalograms.[16,17] Because it has been demonstrated that upper airway morphologic features and tongue posture are dependent on body position,[18,19] the effect of body position must be taken into account in a discussion of the upper airway dimension. Thus, the supine body position, one of the most popular sleep postures,[20] was adopted in this study. It has been shown that the tone of jaw-closing muscles was reduced during sleep, depending on the sleep stage.[13,15] Miyamoto et al[21] reported that the mandibular position during sleep in healthy adults was significantly affected by the sleep stage; mandibular opening progressively increased with the depth of NREM sleep. Therefore, lateral cephalograms in this study were taken in stage 1 to 2 NREM sleep. In addition, it has been reported that the position and shape of

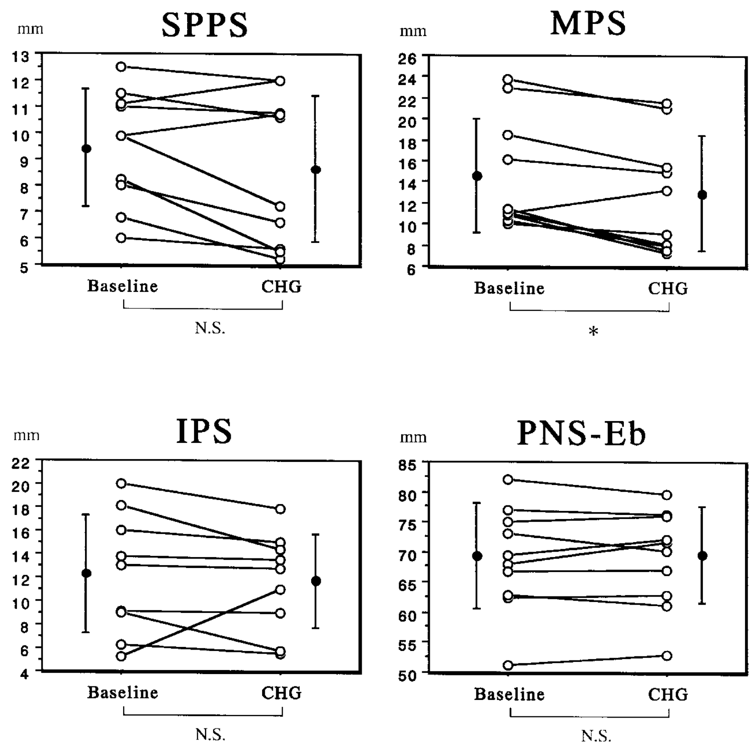

Fig 4. Changes in upper airway dimension. *Vertical bar* indicates mean ± SD; *baseline* indicates value measured during sleep without CHG; *CHG* indicates value measured during sleep with CHG. *$P < .05$.

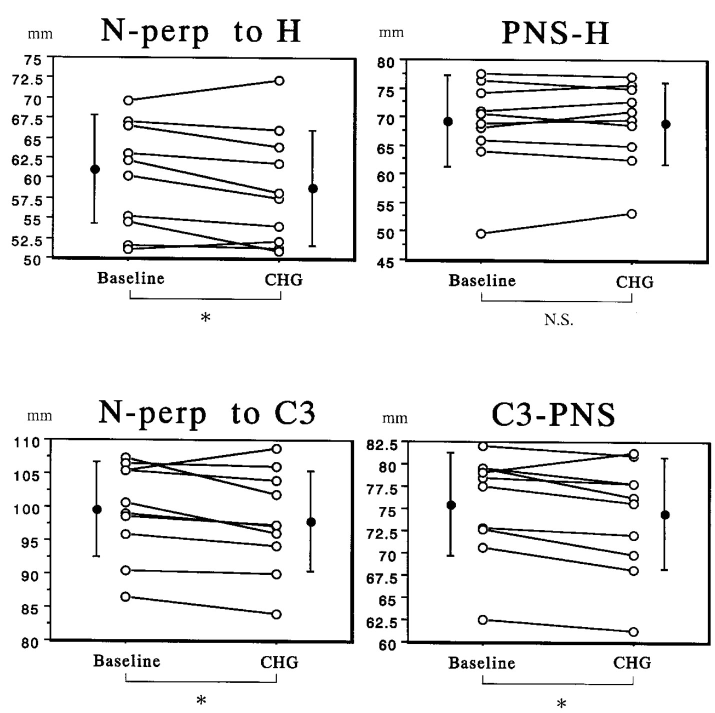

Fig 5. Changes in positions of hyoid bone and cervical vertebra. *Vertical bar* indicates mean ± SD; *baseline* indicates value measured during sleep without CHG; *CHG* indicates value measured during sleep with CHG. *$P < .05$.

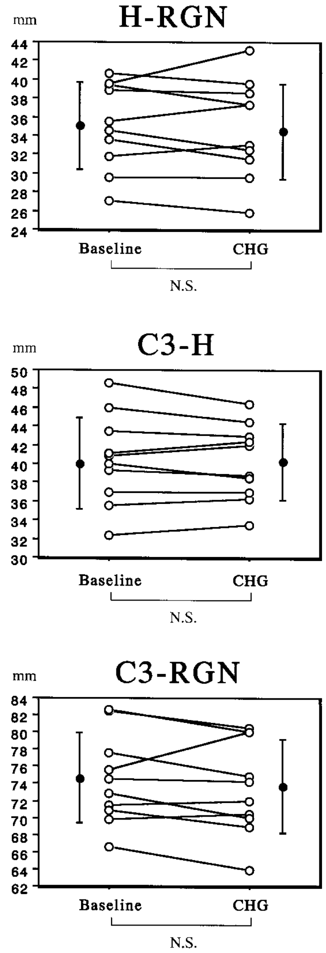

Fig 6. Changes in relationship among hyoid bone, cervical vertebra and the mandible. *Vertical bar* indicates mean ± SD; *baseline* indicates value measured during sleep without CHG; *CHG* indicates value measured during sleep with CHG. $^*P < .05$.

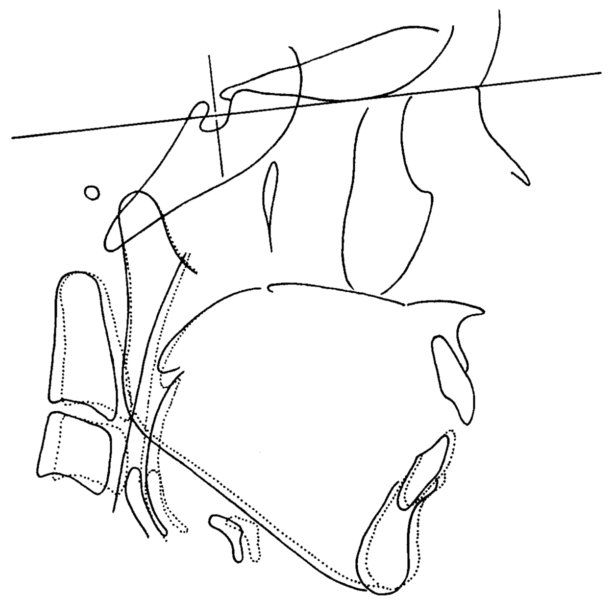

Fig 7. Representative CHG-induced changes in oropharyngeal structures in 1 subject. *Solid line* indicates during sleep without CHG; *dotted line* indicates during sleep with CHG. Note counterclockwise rotation of mandible, forward movement of hyoid bone and cervical vertebra, and reduction of sagittal dimension of upper airway.

the oropharyngeal soft and hard tissues change in association with the respiratory phase,[22-24] which suggests that the respiratory phase must be carefully determined to evaluate the oropharyngeal morphologic features. All cephalometric radiographs in this study were taken at the end of expiration.

Method errors for each variable are given in Table II. These values ranged from 0.21 mm to 0.52 mm, which are comparable with the results in previous studies.[12,24,25] No systematic errors were found, and the reliability of the analysis in the present study seemed to be high.

The results of the repeatability test are shown in Table III. The differences among the cephalometric variables in the 2 supine lateral cephalograms were relatively small. Therefore, the mandibular posture, upper airway dimension, and related structures were shown to be highly reproducible when supine lateral cephalograms were taken under defined conditions with regard to the respiratory phase and sleep stage.

Mandibular position

Although significant forward displacement of Pog was demonstrated by the wearing of the CHG, there were no significant changes in the mandibular position when evaluated with regard to Cd. These findings suggested that no anteroposterior mandibular displacement occurred by wearing the CHG, and only counterclockwise rotation of the mandible was induced. This was also confirmed by the change in N-Me, which tended to decrease by wearing the CHG.

The underlying mechanism of counterclockwise rotation of the mandible is unclear; however, a possible explanation is that it has been reported that jaw closing leads to an increase in the upper airway dimension.[26] The upper airway dimension tended to decrease by the wearing of the CHG in our study (Fig 4). Thus, the decrease in the amount of jaw opening may be a protective response to upper airway narrowing that is induced by the CHG. It is unknown whether such a protective reflex is active during sleep. However, Hollowell and

Table II. Method error*

Cephalometric variables	*Method error (mm)*
N-perp to Pog	0.35
N-perp to Cd	0.22
N-Me	0.21
SPPS	0.47
MPS	0.21
IPS	0.25
PNS-Eb	0.49
N-perp to H	0.25
PNS-H	0.52
N-perp to C3	0.21
C3-PNS	0.40
H-RGN	0.25
C3-H	0.45
C3-RGN	0.49

*$(\Sigma d^2/2n)^{1/2}$, where d represents the difference between the first and second measurements, and n denotes the sample size.

Table III. Repeatability test

Cephalometric variables	*Difference (mm)*
N-perp to Pog	0.5 ± 0.3
N-perp to Cd	0.2 ± 0.2
N-Me	0.4 ± 0.2
SPPS	0.2 ± 0.1
MPS	0.5 ± 0.2
IPS	0.5 ± 0.3
PNS-Eb	0.9 ± 0.1
N-perp to H	0.3 ± 0.2
PNS-H	0.7 ± 0.1
N-perp to C3	0.2 ± 0.3
C3-PNS	0.2 ± 0.2
H-RGN	0.4 ± 0.1
C3-H	0.3 ± 0.4
C3-RGN	0.6 ± 0.3

Values given as mean ± SD.

Suratt[27] showed that the mandibles of patients with obstructive sleep apnea (OSA) were opened more than the mandibles of normal subjects during sleep, and such jaw opening during sleep was proposed as a factor that could lead to upper airway obstruction in patients with OSA. Therefore, the CHG-induced change in mandibular position observed in the present study may be related to the mechanism that protects the upper airway.[28] On the other hand, the pressure on the back of the neck from the neck strap of the CHG would push the soft tissue surrounding the neck anteriorly, which should augment the soft tissue volume in the submental region and result in passive superoanterior rotation of the mandible, regardless of the neuromuscular mechanism.[28]

In the present study, no significant mandibular displacement was induced by wearing the CHG during sleep. This was different from the result in our previous study,[12] in which CHG induced forward displacement of the mandible in awake subjects. This discrepancy can be explained by the reduction in muscle activity during sleep.[13-15] When we consider the effect of CHG on mandibular growth, the finding that the change in the mandibular position in response to wearing CHG was different between wakefulness and sleep may represent a new conceptual basis regarding treatment using CHG. Because wearing CHG during sleep does not induce significant forward displacement of the mandible, its stimulating effect on mandibular growth could not be anticipated in contrast with so-called functional appliances, which force the mandible to move forward. On the contrary, the forward displacement of the mandible that is induced by wearing CHG while awake would presumably facilitate mandibular growth. There is a common interpretation about the effect of CHG on maxillary growth. Nevertheless, there are discrepancies concerning the effect of CHG on mandibular growth. This is perhaps due to variations in the effects of CHG, depending on the time of the day when the patients were instructed to use the CHG. We should be aware of the fact that CHG exerts orthopedic forces constantly on the maxilla; however, the changes in mandibular position induced by the wearing of CHG differ between the awake and sleep states. This should have unexpected and differential effects on mandibular growth. We should remember that dentoskeletal growth and development are influenced by various physiologic factors. For example, Lee and Proffit[29] examined circadian rhythms of tooth eruption and found that greater tooth eruption occurred during the evening observation period and that the rate of tooth eruption was greater in children who were supine and relaxed than in children who were upright and active. This daily rhythm was suggested to be related to fluctuations of hormonal levels that affected metabolic activities. Recently, such diurnal variation in tooth movement in response to orthodontic force was also demonstrated in rats.[30] These findings make it reasonable to consider that such metabolic variations might also affect mandibular growth. Therefore, considering the actual effect of CHG on mandibular growth, various factors should be considered besides the period when CHG is used. Future studies, including clinical cephalometric studies, are needed to clarify these issues.

Upper airway dimension

In this study, a significant reduction in the middle upper airway dimension was demonstrated by the wearing of CHG during sleep. In addition, both the superior and inferior upper airway dimensions also tended to decrease by wearing CHG. The reduction in the sagittal dimension of the upper airway by wearing the CHG during sleep may modify the respiratory function during sleep. Recently, OSA has been well recognized as a sleep-related respiratory disorder that is characterized by recurrent upper airway narrowing or obstruction during sleep. Pirila-Parkkinen et al[31] reported that CHG therapy might contribute to the occurrence of OSA in children, especially those with a retrognathic mandible. Because CHG is frequently used to treat patients with maxillary protrusion accompanied by retrognathia, careful attention must be paid when using CHG during sleep in these patients. Evaluation of the upper airway with the use of lateral cephalograms and a questionnaire regarding clinical signs and symptoms related to sleep-disordered breathing should be helpful for detecting such contraindications in orthodontic treatment.

On the other hand, the vertical length of the upper airway (PNS-Eb) showed no significant changes by the wearing of CHG. The force vector of CHG is predominantly in the anteroposterior direction, and therefore orthogonal to the upper airway, which may have little influence on the vertical length of the upper airway.

Changes in the position of the hyoid bone and cervical vertebra, and their relationships with the mandible

When the CHG was in place, the third cervical vertebra moved forward, and the hyoid bone simultaneously moved in the same direction. As a result, the distance between the third cervical vertebra and hyoid bone (C3-H) remained constant. C3-H, which is closely related to the sagittal dimension of the upper airway, was shown to be stable during wakefulness independent of external disturbances including mandibular set-back surgical procedures for the treatment of mandibular prognathism.[32,33] This indicates the existence of an important neuromuscular mechanism to maintain the homeostasis of respiration.[28] The finding that the distance between the hyoid bone and cervical vertebra remained constant strongly implies that such a mechanism for protecting the upper airway also functions during sleep.

On the other hand, there was no constant change in the vertical position of the hyoid bone (PNS-H) by the wearing of CHG during sleep. This finding is consistent with that of our previous study[12] in which no significant change was observed in this measurement in awake subjects. This was also consistent with the fact that the vertical length of the upper airway did not show a constant CHG-induced change.

No significant mandibular displacement was recorded by the wearing of the CHG, and only counterclockwise rotation of the mandible was observed. As a result, Pog and RGN moved forward geometrically. The hyoid bone, cervical vertebra, and RGN moved constantly forward by the wearing of the CHG, and these changes resulted in no changes in the distances among these structures.

We thank all of the subjects in this study and Mr T. Chiba and Mr T. Morizono for their technical assistance in radiography.

REFERENCES

1. Meach CL. A cephalometric comparison of bony profile changes in Class II Division 1 patients treated with extraoral force and functional jaw orthopedics. Am J Orthod 1966;52:353-70.
2. Jakobsson SO. Cephalometric evaluation of treatment effect on Class II Division 1 malocclusions. Am J Orthod 1967;53:446-57.
3. Wieslander L. The effect of force on craniofacial development. Am J Orthod 1974;65:531-8.
4. Mills CM, Holman RG, Graber TM. Heavy intermittent cervical traction in Class II treatment: a longitudinal cephalometric assessment. Am J Orthod 1978;74:361-79.
5. Sauer GJ, Kuftinec MM. Cervera headgear. J Clin Orthod 1981;15:351-6.
6. Baumrind S, Korn EL, Isaacson RJ, West EE, Molthen R. Quantitative analysis of the orthodontic and orthopedic effects of maxillary traction. Am J Orthod 1983;84:384-98.
7. Tulloch JFC, Proffit WR, Phillips C. Influences on the outcome of early treatment for Class II malocclusion. Am J Orthod Dentofacial Orthop 1997;111:533-42.
8. Keeling SD, Wheeler TT, King GJ, et al. Anteroposterior skeletal and dental changes after early Class II treatment with bionators and headgear. Am J Orthod Dentofacial Orthop 1998;113:40-50.
9. Baumrind S, Korn EL, Molthen R, West EE. Changes in facial dimensions associated with the use of forces to retract the maxilla. Am J Orthod 1981;80:17-30.
10. Baumrind S, Korn EL. Patterns of change in mandibular and facial shape associated with the use of forces to retract the maxilla. Am J Orthod 1981;80:31-47.
11. Graber TM. Orthodontics: principles and practice. 3rd ed. Philadelphia: Saunders;1972. p. 579-81.
12. Hiyama S, Ono T, Ishiwata Y, Kuroda T. Changes in mandibular position by wearing the cervical headgear [abstract in English]. Orthod Waves 1999;58:344-52.
13. Chase MH, McGinty DJ, Sterman MB. Cyclic variation in the amplitude of a brain stem reflex during sleep and wakefulness. Experientia 1968;24:47-8.
14. Parmeggiani PL. Behavioral phenomenology of sleep (somatic and vegetative). Experientia 1980;36:6-10.
15. Tangel DJ, Mezzanotte WS, Sandberg EJ, White DP. Influences of NREM sleep on the activity of tonic vs. inspiratory phasic muscles in normal men. J Appl Physiol 1992;73:1058-66.
16. Pracharktam N, Hans MG, Strohl KP, Redline S. Upright and supine cephalometric evaluation of obstructive sleep apnea syndrome and snoring subjects. Angle Orthod 1994;64:63-74.
17. Lowe AA, Ono T, Ferguson KA, Pae EK, Ryan CF, Fleetham JA. Cephalometric comparisons of craniofacial and upper airway structure by skeletal subtype and gender in patients with obstructive sleep apnea. Am J Orthod Dentofacial Orthop 1996;110:653-64.
18. Brown IB, McClean PA, Boucher R, Zamel N, Hoffstein V. Changes in pharyngeal cross-sectional area with posture and application of continuous positive airway pressure in patients with obstructive sleep apnea. Am Rev Respir Dis 1987;136:628-32.
19. Miyamoto K, Özbek MM, Lowe AA, Fleetham JA. Effect of

body position on tongue posture in awake patients with obstructive sleep apnea. Thorax 1997;52:255-9.
20. Cartwright RD, Lloyd S, Lilie J, Kravitz H. Sleep position training as treatment for sleep apnea syndrome: a preliminary study. Sleep 1985;8:87-94.
21. Miyamoto K, Özbek MM, Lowe AA, et al. Mandibular posture during sleep in healthy adults. Arch Oral Biol 1998;43:269-75.
22. Britton PD. Effect of respiration on nasopharyngeal radiographs when assessing adenoidal enlargement. J Laryngol Otol 1989; 103:71-3.
23. Schwab RJ, Gefter WB, Pack AI, Hoffman EA. Dynamic imaging of the upper airway during respiration in normal subject. J Appl Physiol 1993;74:1504-14.
24. Nakagawa F, Ono T, Ishiwata Y, Kuroda T. The morphological changes in the oropharynx in association with the respiratory phase [abstract in English]. J Jpn Orthod Soc 1996;55:337-44.
25. Solow B, Tallgren A. Head posture and craniofacial morphology. Am J Phys Anthropol 1976;44:417-36.
26. Kuna ST, Remmers JE. Neural and anatomic factors related to upper airway occlusion during sleep. Med Clin North Am 1985;69:1221-42.
27. Hollowell DE, Suratt PM. Mandible position and activation of submental and masseter muscles during sleep. J Appl Physiol 1991;71:2267-73.
28. Bosma JF. Evaluation of oral function of the orthodontic patient. Am J Orthod 1969;55:578-84.
29. Lee CF, Proffit WR. The daily rhythm of tooth eruption. Am J Orthod Dentofacial Orthop 1995;107:38-47.
30. Igarashi K, Miyoshi K, Shinoda H, Saeki S, Mitani H. Diurnal variation in tooth movement in response to orthodontic force in rats. Am J Orthod Dentofacial Orthop 1998;114:8-14.
31. Pirila-Parkkinen K, Pirttiniemi P, Nieminen P, et al. Cervical headgear therapy as a factor in obstructive sleep apnea syndrome. Pediatr Dent 1999;21:39-45.
32. Takagi Y, Gamble JW, Proffit WR, Christiansen RL. Postural change of the hyoid bone following osteotomy of the mandible. Oral Surg Oral Med Oral Pathol 1967;23:688-92.
33. Nakagawa F, Ono T, Ishiwata Y, Kuroda T. Morphologic changes in the upper airway structure following surgical correction of mandibular prognathism. Int J Adult Orthod Orthognath Surg 1998;13:299-306.