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Respiratory Movement of Upper Airway Tissue in Obstructive Sleep Apnea

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Study Objectives: To measure real-time movement of the tongue and lateral upper airway tissues in obstructive sleep apnea (OSA) subjects during wakefulness using tagged magnetic resonance imaging.

Design: Comparison of the dynamic imaging of three groups of increasing severity OSA and a control group approximately matched for age and body mass index (BMI).

Setting: Not-for-profit research institute

Participants: 24 subjects (apnea hypopnea index [AHI] range 2-84 events/h, 6 with AHI < 5 events/h)

Methods: The upper airway was imaged awake in two planes using SPAtial Modulation of Magnetization (SPAMM). Tissue displacements were quantified with harmonic phase analysis.

Measurements and Results: All subjects had dynamic airway opening in the sagittal plane associated with inspiration. In the nasopharynx, the increase in airway cross-sectional area during inspiration correlated with minimal cross-sectional area of the airway ($R = 0.900$, $P < 0.001$). AHI correlated negatively with movement of the nasopharyngeal lateral walls ($R = -0.542$, $P = 0.006$). Four movement patterns were observed during inspiration: "en bloc" anterior movement of the whole posterior tongue; movement of only the oropharyngeal posterior tongue; bidirectional movement; or minimal movement. Some subjects showed different inspiratory movement patterns with different breaths. A low AHI (< 5) was associated with en bloc movement ($P = 0.002$).

Conclusions: Inspiratory movement of the tongue varied between and within subjects, likely as a result of local and neural factors. However, in severe OSA inspiratory movement was minimal.

Keywords: Obstructive sleep apnea, dynamic imaging, respiratory movement, genioglossus, lateral walls

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INTRODUCTION

Obstructive sleep apnea (OSA) has a prevalence of around 4% of the middle-aged male population.[1] It is a multifactorial disorder involving anatomical and neuromuscular alterations that predispose patients to recurrent hypoventilation and arousal during sleep.[2] One-third of the variability of the apnea hypopnea index (AHI) is ascribed to increased mechanical load, and two-thirds to blunted neuromuscular responses.[3] Passive mechanical loads are elevated in severe OSA, but some normal subjects with elevated load are able to increase neuromuscular activity and overcome airway collapse.[4] Movement of the muscles in response to neuromuscular input during sleep is poorly understood.

The upper airway muscles respond to changes in airway pressure during respiration,[5,6] but whether neural output leads to movement or muscle stiffening is unknown. The muscles of the airway, particularly the genioglossus, receive phasic and tonic input from the respiratory control center in the brainstem,[5,7] which is modulated by hypercapnia,[8-10] hypoxia,[11] and negative pressure.[12-14] Lung volume,[15,16] sex hormones,[17] and temperature[18] also influence central output to the dilator muscles. Because OSA patients have greater mechanical loads and narrower airways, description of how the muscles move during quiet breathing provides insight into how neural inputs relate to airway patency. OSA patients have shown larger genioglossus action potentials with longer duration, and during wakefulness they have an earlier onset of inspiratory phasic units.[19] At sleep onset, multiunit EMG studies document abrupt decreases in dilator muscle activity,[20-22] and tonic and phasic activity is decreased in both normal[23] and OSA subjects,[21,22] with likely suppression of the negative pressure reflex.[24] However, increased EMG activity in OSA patients may not necessarily translate to increased muscle movement. Many studies of the airway describe the behavior of the airway as a static structure, but dynamic imaging can show how the different parts of the airway interact in real time. Regional responses, particularly in the light of recent findings of compartmentalization of the nerve supply to different parts of genioglossus,[25] may play a role in regulating airway caliber, but how the muscles respond to the output from respiratory centers is unknown. Moreover, understanding the movement of different parts of the upper airway musculature may assist in understanding the heterogeneity among OSA patients and their responses to treatments that aim to manipulate the upper airway anatomy, including surgery, neural stimulation, and mandibular advancement splints.

Spatial Modulation of Magnetization (SPAMM) is a magnetic resonance imaging (MRI) technique in which a grid of saturated magnetization ("tags") is superimposed on tissues and tissue motion followed with ultra-fast imaging as the grid deforms. It was originally developed to image the myocardium,[26] but has been used to study the upper airway in rats[27] and more recently in humans.[6,28] Cheng and colleagues[6] studied upper airway movement during the respiratory cycle in young normal subjects

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and showed that the posterior tongue base moved anteriorly during early inspiration, and subsequently that an inspiratory load reduced the motion of genioglossus.[28] The aim of this study was to describe the movement and deformation of the upper airway muscles in OSA patients and matched controls during quiet breathing. The hypothesis was that movement of the upper airway muscles would vary with OSA severity during quiet breathing in wakefulness because OSA patients have a narrower upper airway[29] and increased mechanical load.[4] However, after the initial analysis, movement of tissues surrounding the airway was more heterogeneous than anticipated, so subject recruitment was expanded to describe the movement in relation to AHI.

METHODS

This study was designed as a cross-sectional study of adult subjects. To examine the effect of OSA severity, we examined 4 groups: normal (AHI < 5), mild OSA (AHI 5-15), intermediate severe (AHI 30-50), and very severe (AHI > 50) with 6 subjects in each group. Originally 10 subjects with severe OSA and 10 age-, sex-, and body mass index (BMI)-matched normal (AHI < 10) controls were planned. This was modified include the 3 severity groups because interim analysis suggested that within the severe OSA patient group (AHI > 30), the subjects with the highest AHI (AHI > 50) behaved differently from subjects with lower AHI (AHI 30-50), even though all had severe OSA on formal classification. Normal (AHI < 5) and mild (AHI 5-15) OSA patients were studied for comparison. No subject had prior treatment for OSA. The groups were approximately matched by sex, age, and BMI. The study was approved by the Human Research Ethics Committees of the University of NSW and the Prince of Wales Hospital and conformed to the Declaration of Helsinki. Written informed consent was obtained in all subjects. All were screened for MRI safety.

Subject Selection

OSA patients were recruited through the sleep clinic at the Prince of Wales Hospital. Control subjects were recruited either through the clinic or from the community. Subjects had no other severe chronic illness or medication that would affect the muscles of the upper airway. Subjects with previous surgery to the upper airway were excluded, but minor orthodontic work was allowed. Subjects completed the Epworth Sleepiness Scale (ESS) prior to scanning.

Polysomnography

OSA subjects underwent polysomnography (PSG) using the standard technique in a clinical sleep laboratory, scored by an experienced technician and reported by a sleep physician. Electroencephalography (EEG), electrooculography, electrocardiogram, submental electromyogram (EMG) and airflow via nasal pressure transducer, oximetry, abdominal and thoracic respiratory effort were recorded. Subjects with AHI < 15 recruited through the clinic underwent clinical PSG (n = 7), and community recruits were studied at home (n = 5) using an Embletta X50 (Medcare, Iceland) with one EEG lead, airflow, oximetry, thoracic and abdominal bands. Home studies were set up, scored, and reported by a sleep physician. PSG of subjects with AHI < 15 studied in the sleep laboratory were scored by the same sleep physician for consistency. Sleep stages were scored in 30-s epochs. All studies were scored according to AASM criteria,[30] with hypopneas defined as 50% reduction in airflow accompanied by a desaturation of 3% or respiratory arousal.

MRI Technique

An available SPAMM sequence on a 3T MRI was used (Achieva, Philips Medical Systems, Best, The Netherlands). Because this sequence requires cardiac gating to initiate each scan, an artificial ECG trigger was generated to facilitate image synchronization with the respiratory cycle using LabVIEW v8.2 (National Instruments Corp., Austin, TX, USA). Once triggered, the tags were laid down (140 ms), then 8 images were taken 250 ms apart. The imaging parameters used were: flip angle 90°; repetition time 400 ms; echo time 16 ms; 256×256 matrix and a slice thickness of 10 mm, tag spacing 8.6 mm. Images were taken in the mid-sagittal plane and an axial plane through the narrowest point of the nasopharynx. A 3D anatomical scan of the pharyngeal region (T1TFE, 3D mode, 1 mm isotropic voxels, 256 × 256 matrix, 130 slices) was also conducted.

Experimental Protocol

Participants lay within the MR head-neck coil with the Frankfort plane vertical (confirmed with a fast sagittal scan). Foam padding was used to minimize head movement. Subjects were asked to stay awake and breathe through their nose with their mouth closed for the entire experiment. They were woken if their breathing became irregular, snoring was heard, or apneic pauses were observed.

Respiration was measured using an MRI compatible abdominal band 2 cm below the ribcage. The protocol for imaging the respiratory cycle with SPAMM has been described in detail previously.[6] The respiratory cycle was divided into 4 to 6 segments after observation of stable breathing. Each segment was imaged ≥ 3 times by triggering the scan at predetermined delays after peak inspiration and expiration.

Analysis

Image sequences were reviewed and sequences including swallowing or mandible movement were discarded. Movement of the tissues surrounding the airway was analyzed at the following points (see Figures 1 and 2): (1) approximately 10 mm anterior and 10 mm right lateral to the midline anterior wall of the airway; (2) 10 mm lateral to the right lateral walls; (3) a right postero-lateral point in the muscle surrounding the airway; and (4) the midpoint of the uvula. Depending on tongue size, 5 to 8 points, 8.6 mm apart were selected in a vertical line ~ 10 mm anterior to the airway wall. These points of interest were selected on the initial undeformed tagged image.

Although 8 images were recorded, only the first 5 were analyzed because the tags "fade," and tag contrast was poor in later images. Movement (i.e., displacement) of the points was calculated using the harmonic phase (HARP) method.[6,31] HARP calculates tissue deformation by computing shifts in spectral peaks in the Fourier (spatial frequency) domain. Sinusoidal modulation of the tag grid gives sub-pixel resolution with displacement error of 0.1 pixels.[32] In the axial plane, the pre-inspiration minimal cross sectional airway (CSA) and the post-inspiration maximum CSA were measured by manually outlining the air-

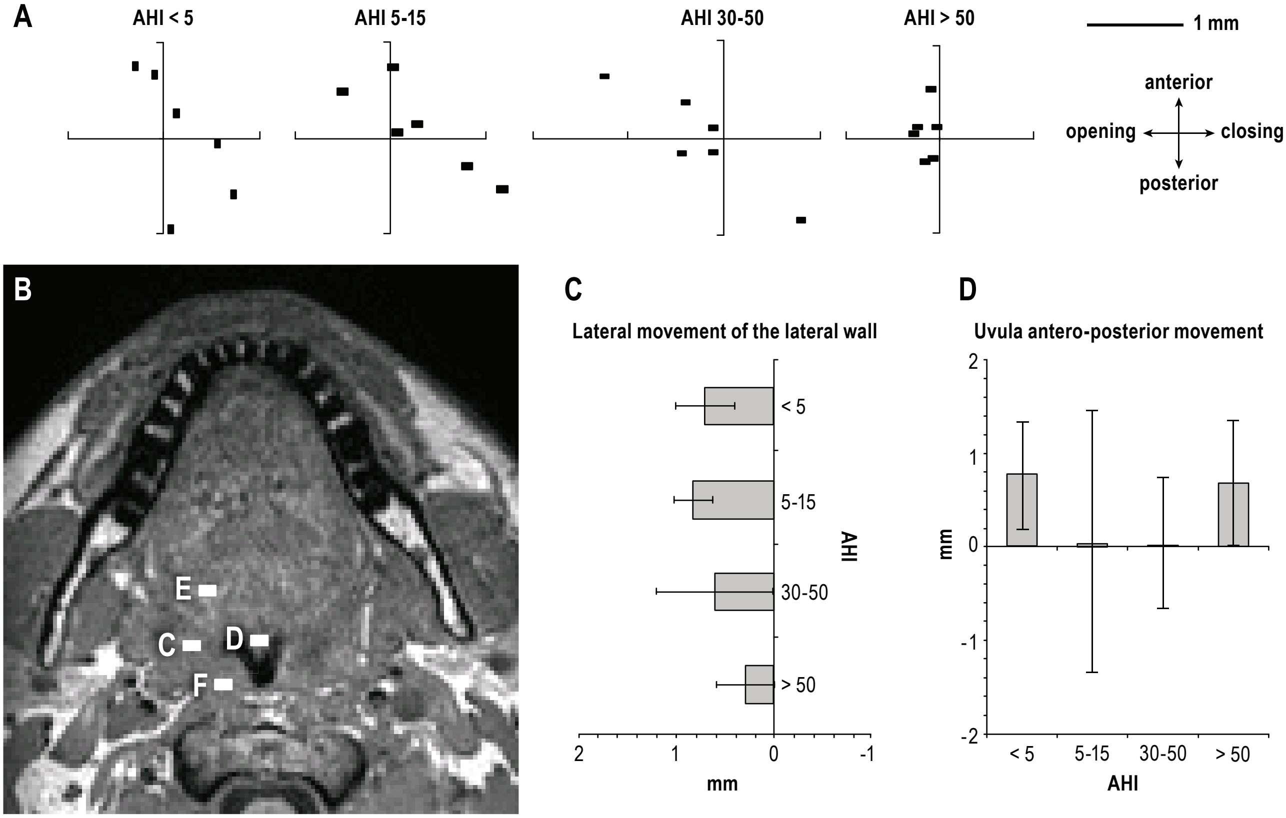

Figure 1—(A) Displacement of the lateral wall (point C) 1 second after inspiration for each AHI group. The initial position of the points is the origin. At inspiration the lateral wall point of the normal group tended to move medially, but there was a large variation in the direction of movement, although the amount of movement was less with increasing AHI. **(B)** The axial imaging plane with the points tracked: C–lateral wall point, D–uvula, E–antero-posterior point, F–postero-lateral point. **(C)** Graph of the displacement of the lateral wall (point C) compared to AHI. AHI was negatively correlated to movement of the lateral point (P = 0.006). **(D)** The antero-posterior displacement of the uvula (point D) at the beginning of inspiration. In the mild and intermediate group, the uvula moved posterior in some subjects, which resulted in large standard deviations; therefore, average is close to 0. For the graphs, movement that increased airway diameter was defined as positive.

way in the axial dynamic images using ImageJ (NIH, Bethesda, Maryland, USA). Each image in the sequence was reviewed to find the maximum or minimum airway size, depending on whether the airway was expanding or narrowing.

Sagittal inspiratory movement sequences were classified into 4 groups according to the amount of movement of the posterior tongue in the first second (Figure 3): (1) en bloc anterior movement > 1 mm; (2) bidirectional movement of the tongue where the nasopharyngeal point and the oropharyngeal point moved in opposite directions; (3) minimal (< 1 mm) movement; and (4) minimal (< 1 mm) movement of the nasopharyngeal point with > 1 mm anterior movement of the oropharyngeal point. The last group is the movement pattern in young normal subjects.[6] Classifications were independently assessed by a blinded second investigator with 97% agreement.

Statistics

SPSS (Version 18, Chicago, IL) was used for all statistical measures. Descriptive statistics are presented as mean ± standard deviation. Alpha was 0.05. Most variables were not normally distributed, so Spearman rank correlation coefficient was used with a 2-tailed hypothesis. For comparisons between more than 2 groups, Pearson χ^2 test was performed.

RESULTS

Subject demographics, AHI, and anatomical measurements are shown in Table 1. The groups were well matched for BMI with no significant differences in BMI or age. For videos of the respiratory cycle, corresponding to the examples in Figure 2, see Videos 1-3 in the supplemental material. Because of the limited number of women in the study, the statistical analysis was also performed with the women omitted. The significant results remained unchanged. Presented here are results for the whole group.

Axial Nasopharyngeal Images

AHI and total lateral wall movement (Figure 1, point C) were significantly negatively correlated (R = -0.542, P = 0.006; Figure 1), but movement of the antero-lateral and postero-lateral points (Figure 1, points E and F) were not significantly correlated with AHI. AHI was significantly negatively correlated with maximal CSA (R = -0.501, P = 0.013) but not movement of the uvula (P > 0.05). The very severe OSA group showed little movement of the lateral walls during inspiration compared to the more heterogeneous movement of the other groups (Figure 1). Minimum CSA pre-inspiration and maximum CSA post-inspiration were strongly correlated (Spearman correlation, R = 0.900, P < 0.001, see Figure 4). Two subjects with AHI 30-50 had a

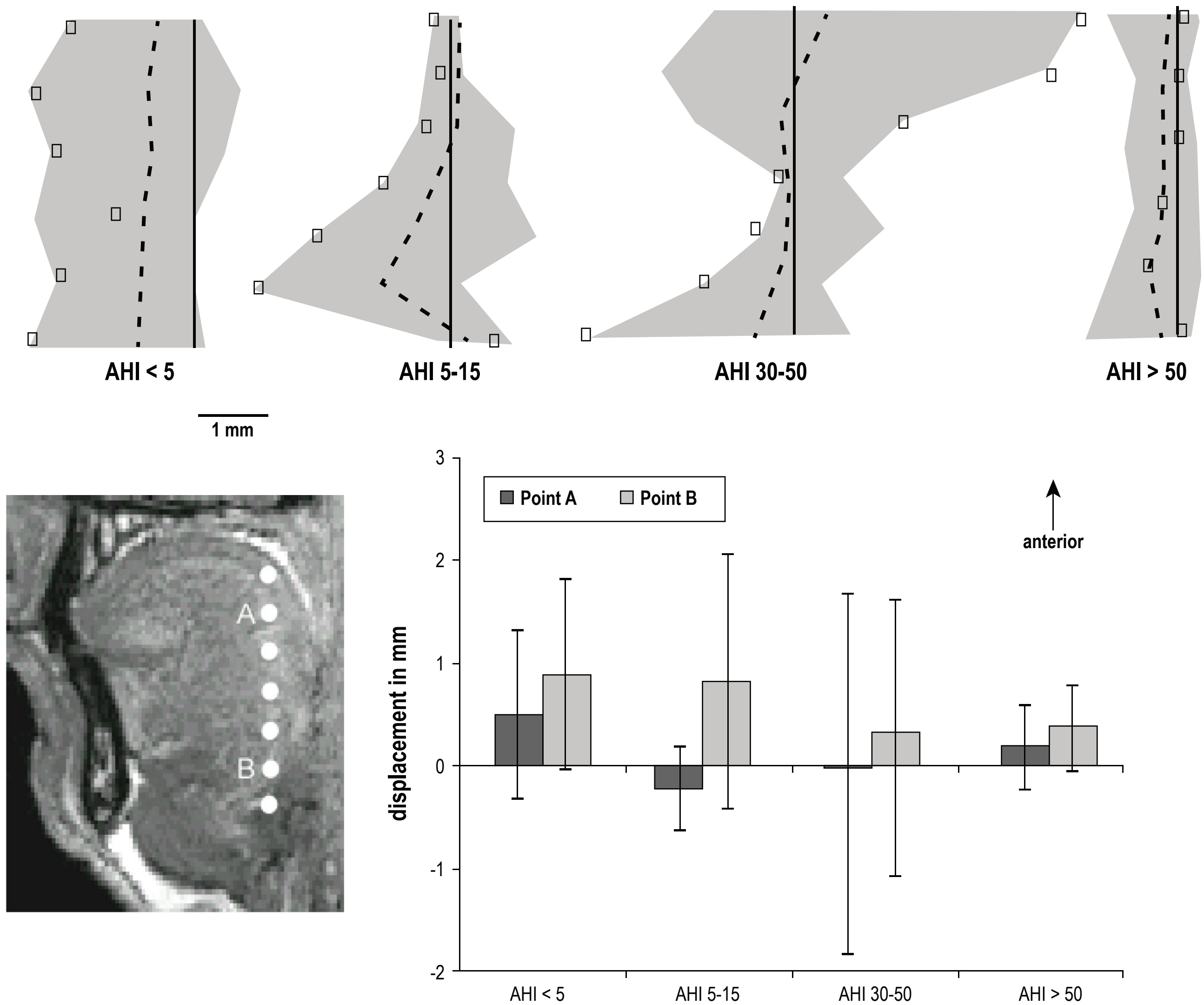

Figure 2—Top row: Patterns of movement with increasing OSA severity at the beginning of inspiration. Antero-posterior movement is exaggerated in all groups in the top row. The black vertical line is the start of movement, and the dashed line is the mean end of movement for the group after 1.5 sec. The gray shaded area is the range of movement in each group. The boxes indicate one example in each AHI group. For videos of these subjects, see Videos 1-3 in the supplemental material. In the videos, nasopharynx and oropharynx point move independently of each other. Bottom row: The anatomical image shows the points in the posterior tongue tracked to give the top line. The graph shows the mean and standard deviations of movement of points A and B shown in the anatomical image.

large CSA, unlike the rest of the group, which is reflected in the large standard deviation in Table 1 and the outliers in Figure 4.

Sagittal Plane

Respiratory Cycle Data

Patterns of movement throughout the respiratory cycle in the sagittal plane are summarized in Figure 3. All subjects had movement of the posterior tongue, of varying magnitude, tending to open the airway just before or during inspiration. In expiration, most subjects showed posterior movement (21/23, 91%) or no movement (2/23, 9%), with most subjects (17/23, 74%) showing ≤ 1 mm movement in expiration. One subject had limited expiratory data due to a very long respiratory cycle.

Movement Related to Inspiration

There were 70 image sequences during inspiration (average 2.9 per subject, range 1-4). Figure 5 shows the percentage of different movement patterns for each AHI group (see Methods). Eleven of 24 (46%) subjects exhibited more than one pattern of movement, with 3/24 (13%) having 3 patterns. Most of the variation was whether the tongue moved forward more or less than 1 mm, but 4/24 (17%) showed en-bloc tongue movement and bidirectional movement during different breaths. Of the 15 sequences with bidirectional movement of the tongue, the nasopharyngeal region narrowed in 8 (53%), while 7 (47%) had narrowing of the oropharyngeal region. Subjects with AHI > 30 were more likely to have the minimal movement pattern, and subjects with AHI < 5 were more likely than expected by chance to demonstrate > 1 mm movement pattern ($P = 0.002$, χ^2, see Figure 5; with the women omitted, $P = 0.002$). Most subjects with AHI > 30 (7/12, 58%) had minimal movement as their predominant pattern, unlike subjects with AHI < 15 (3/12, 25%). No patient with severe OSA (intermediate and very severe groups) showed the focal oropharyngeal movement typical in young normal subjects, as previously described.[6] The amount

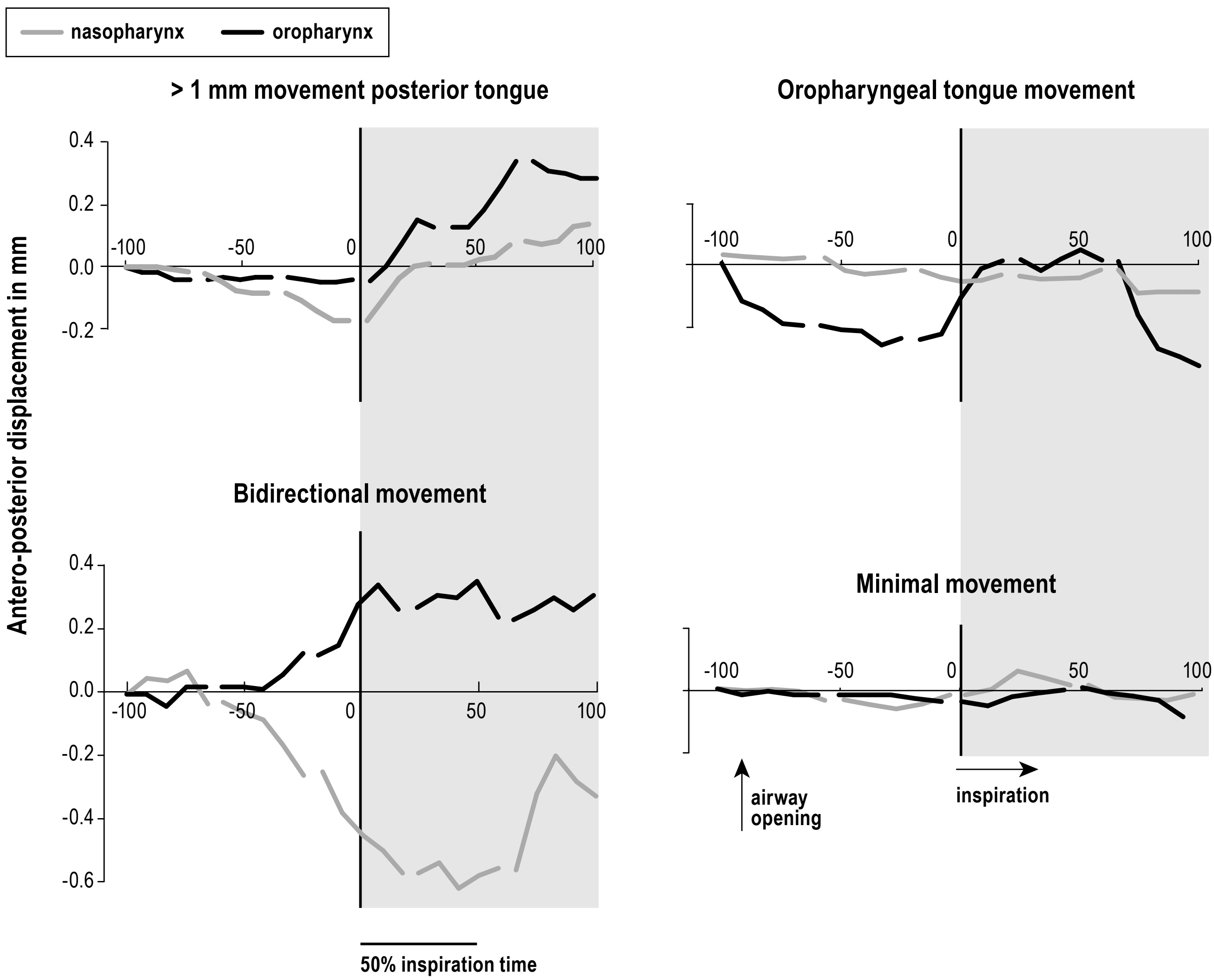

Figure 3—Typical examples of movement over the respiratory cycle for the four patterns of movement. The gray shaded region is inspiration. Inspiration starts at time 0 and the percentage of inspiratory time is shown on the x axis.

Table 1—Demographic, anatomical, and polysomnography data for each group (mean ± SD)

	Normal	Mild	Intermediate severe	Very severe	P value
AHI	< 5	5-15	30-50	> 50	
number	6	6	6	6	
Mean AHI (events/h)	3 ± 1	10 ± 2	34.2 ± 3.9	70.3 ± 14.5	
Sex (M:F)	6:0	4:2	5:1	5:1	
Age (years)	44 ± 13	55.5 ± 17.1	53.3 ± 17.8	44.7 ± 12.8	0.496
BMI (kg/m²)	30 ± 3	34.1 ± 4.9	30.6 ± 3.9	31.8 ± 4.5	0.786
Minimum nasopharynx (mm)	3.7 ± 1.2	4.3 ± 1.6	3.8 ± 1.7	3.8 ± 2.6	0.933
Minimum oropharynx (mm)	10.7 ± 2.6	9.5 ± 0.5	12.7 ± 4.2	10.2 ± 4.3	0.397
CSA (mm²)	113 ± 21	78 ± 34	120 ± 90	74 ± 19	0.314
MPA (degrees)	26 ± 8	25 ± 8	27 ± 4	30 ± 9	0.726
Craniocervical angle (degrees)	96 ± 5	93 ± 3	96 ± 8	101 ± 5	0.148
Craniocaudal airway length (mm)	79.5 ± 9.4	77.8 ± 8.6	82 ± 13.3	84.0 ± 5.0	0.685
Number retrognathic (n/6)	1	1	1	2	0.860
ESS	5 ± 2	5.3 ± 2.5	9.4 ± 3.2	9.2 ± 3.5	0.064

The minimum nasopharyngeal and oropharyngeal diameters were measured in the sagittal plane. AHI, apnea hypopnea index; BMI, body mass index; CSA, cross sectional area; MPA, mandibular plane angle; ESS, Epworth Sleepiness score.

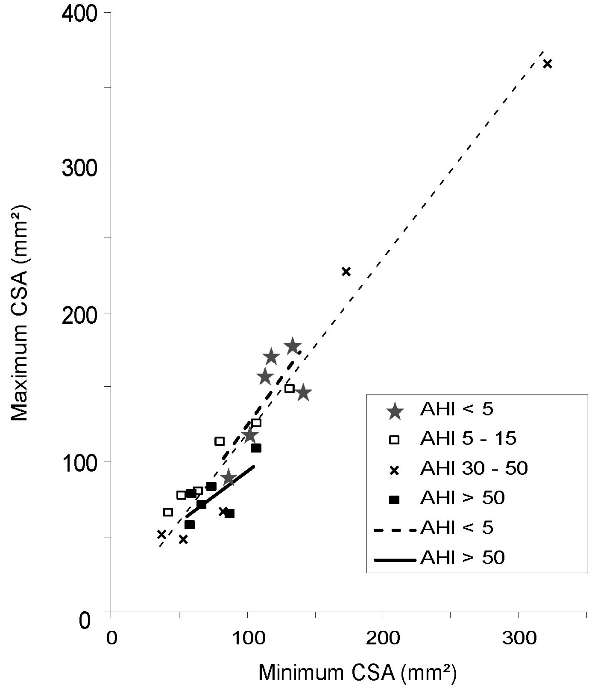

Figure 4—Maximum CSA versus minimum CSA with dashed line of best fit for the entire dataset (CSA max = 1.16 × CSA min + 6.08, R = 0.900). The lines of best fit for the normal and very severe OSA subgroups are also shown, although not statistically significant.

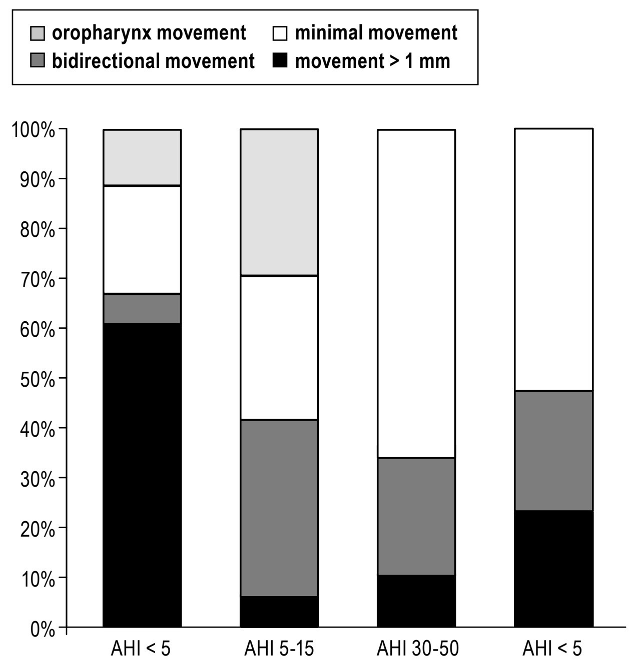

Figure 5—Distribution of movement patterns of the upper airway observed for groups with different levels of AHI.

of movement varied between subjects, but with less variation in subjects with AHI > 50 compared to the other groups (Figure 2). Maximal movement showed no significant correlation with AHI in the sagittal plane.

DISCUSSION

This is the first study to quantify respiratory-related movement of the soft tissues of the upper airway in OSA. This was measured during wakefulness. The main findings are: (1) a high AHI (> 50) was associated with minimal movement of the posterior tongue and the lateral walls of the nasopharynx; (2) in all subjects, the CSA of the narrowest part of the nasopharynx increased during inspiration in proportion to the minimal CSA; (3) intermediate severity OSA patients had heterogeneous and variable movement patterns. It was striking that subjects with the highest AHI typically had little movement of the tissues surrounding their airway. The amount of movement of the lateral and nasopharyngeal points correlated negatively with AHI. Although four patterns of sagittal tongue movement were observed during inspiration, no subject with severe OSA showed the oropharyngeal movement pattern seen previously in young, normal BMI subjects,[6] and the normal subjects in this study tended to have en bloc anterior motion of the whole tongue. While our results revealed heterogeneity of tissue movement, the very severe OSA group was more homogeneous, which suggests a common final outcome in very severe OSA. The minimal movement pattern in these subjects may reflect a failure to dilate the narrow upper airway.

Why changes to muscle, neural output, and mechanics associated with increasing AHI lead to decreased movement of the tissues during wakefulness is unclear. Extra-luminal tissue pressure[33] and tongue and soft tissue volume[34] are increased in OSA patients compared to matched controls, and these changes increase the load required to dilate the airway in OSA. Breathing against increased inspiratory load in normal subjects (with large airways) decreased oropharyngeal tongue movement,[28] which may underlie what was observed here. However, we speculate that minimal movement reflects an inadequate neuromechanical response to increased load in OSA subjects, because the normal matched controls in this study exhibited coordinated airway dilation during inspiration. Our results suggest less effective dilation of the airway with increasing AHI. Some studies of muscle function have shown little difference in fatigability, muscle strength,[35] twitch tension,[36] or force-frequency relationships[37] in OSA patients compared to snorers or non-snorers; but other studies have shown increased maximal tongue force production[36,38] with decreased time to task failure.[39] It is unclear why these studies show conflicting results, but variation in voluntary activation and supraspinal fatigue are likely to be involved.[40] Only up to 11% of maximal protrusion strength of the genioglossus is used during quiet breathing in OSA subjects,[41] which suggests that the muscle in OSA patients should be able to dilate the airway under increased load, although OSA patients may have subclinical swallowing dysfunction.[42] In normal subjects with increased mechanical load, the dynamic response of the upper airway tissues compensate to overcome the load, but OSA patients are not able to overcome their passive critical closing pressure.[4] In this study, the coordinated opening of the airway during inspiration in control subjects may depict how normal subjects overcome increased load.

Whether there is muscle pathology present in OSA patients has been debated,[38,43,44] but it seems more likely given current evidence that the muscle is normal but a motor neuropathy is present.[45] Histopathological studies[46-48] have shown direct evidence of denervation with overexpression of neural cell adhesion and evidence of axonal regrowth in the palatial muscles of OSA patients.[43] Changes in single motor unit EMG[19,49] in OSA patients are also consistent with a mild motor neuropathy involving chronic denervation and reinnervation of muscle fibers. Genioglossus multiunit EMG is increased in OSA both in wakefulness and in sleep compared to controls,[13,22] which, in part, guided our original hypothesis that there would be more movement in the more severe group to compensate for a narrow airway. Single motor unit EMG studies found the active motor units had larger motor unit action potential areas.[19,49] They fired slightly earlier, and a subset (inspiratory phasic units) had modestly increased firing rates in OSA.[19,49] The increased multiunit EMG in OSA may largely reflect peripheral changes in motor unit size. If overall drive is increased in OSA patients[13,22] but the muscle does not move, this would be functional evidence for motor neuropathy. Neuropathy plus increased mechanical load may lead to the minimal dilatory movement seen here. However, many additional factors (including chemosensitivity, level of arousal, and extra-luminal pressure) will also influence motion of the upper airway. There was little change in the background or peak firing frequency of the expiratory phasic and tonic units in single motor units studies of OSA patients compared to controls,[19] which may account for the observation that expiratory data was similar in all four groups in this study.

Bidirectional motion of the tongue in quiet breathing, where one part of the genioglossus dilates the airway but another region narrows (see supplemental Video 1, subject 3), has not been previously reported. Bidirectional motion was almost exclusively seen in OSA patients and may reflect regional differences in mechanical load, but could also be consistent with possible non-uniform neuropathy leading to impaired coordination. The genioglossus has two neuromuscular compartments, the horizontal (inferior, probably associated with oropharyngeal dilation) and oblique (superior).[25] Previous work[6] showing oropharyngeal movement alone and this study suggest that the two compartments can move independently and/or in concert during quiet breathing. Within subject breath-to-breath variation in the pattern of genioglossus movement suggests that ongoing adaptation to conditions in the upper airway during wakefulness is achieved by varying neural drive to the two compartments, although this needs to be verified with electromyographic techniques. Regional tongue movement could be an adaptive response to local negative pressure and local conditions leading to regional airway dilation but we speculate this is maladaptive when it results in airway narrowing. Decreased coordination or differential activation between the compartments could explain the bidirectional motion, resulting in paradoxical airway narrowing. The tongue acts as a muscular hydrostat,[6,50,51] which means that contraction in one area of the tongue leads to corresponding volumetric elongation elsewhere. This may displace tissue into the airway if the displaced region is unable to respond.

In addition to being sufficiently loud that it would be difficult to image subjects while asleep, the SPAMM technique has limitations for imaging the respiratory cycle. The main one is that dividing the respiratory cycle was necessary due to tag fading after ~1s, so respiratory cycles are pieced together from separate breath segments. There could be breath-to-breath variation that might mask more complex motion patterns when averaged over three breaths as done here. We have timed all breath segments using the abdominal band, having previously shown that inspiratory timing taken from the abdominal band was accurate to within 100 ms,[6] however airway opening may occur when a critical narrowing or pressure is reached rather than at a particular time point. For this reason we analyzed the pre-inspiratory as well as post-inspiratory sequences to measure the minimum and maximum CSA associated with inspiration. Displacement of the tissues over the respiratory cycle did not return to zero in some cases possibly due to accumulated small errors and breath-to-breath variation. It could also be that where there is large tongue movement, it may not return to precisely the same position after each breath. The number of patterns of movement was unexpected and not part of the original hypothesis as the study of young normal subjects had not demonstrated this.[6] It is unlikely that the limitations in the SPAMM technique and analysis methods used could result in errors which appeared as multiple movement patterns because all the sequences were visually assessed for movement and the quantified measurements were consistent with what was visually observed. Furthermore, the analysis method has been shown to have sub-pixel accuracy (see Methods). Heterogeneity of airway movement and the limited sample size in this study decreased the power of the study to find correlations with anatomical and other variables, but age or BMI are unlikely to account for the differences seen in this study because the groups were deliberately matched for these variables. A separate study is underway to assess the effect of these variables on upper airway movement in normal subjects. It has previously been debated that AHI may not be a good marker for disease severity.[52] Differing patterns of movement are not explained by differing levels of drowsiness in participants because the two groups of severe OSA had a similar ESS.

This study reveals altered movement of the upper airway during quiet breathing in OSA in wakefulness. Although the tongue responded dynamically from breath to breath in a regional manner in some subjects, different patterns of movement occurred with increasing OSA severity. More severe OSA patients were less likely to have airway dilation during inspiration and did not have the oropharyngeal movement pattern commonly seen in normal subjects. The markedly reduced movement in very severe OSA may be the end stage of the disorder at a neuromechanical level. In these subjects the tongue did not move in response to the increased background load. In spite of heterogeneous responses in intermediate severity groups, the lack of movement and airway dilation of the most severe OSA group was consistent and may be evidence for motor neuropathy in OSA patients. Some subjects with intermediate severity OSA had regional airway narrowing which may be maladaptive. Overall, these results suggest that regional control of the airway is abnormal even during wakefulness in OSA.

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DISCLOSURE STATEMENT

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SUPPLEMENTAL MATERIAL

Video 1 shows genioglossus motion during inspiration for four subjects in different AHI categories. **Subject 1:** AHI < 5. Beginning of inspiration. **Subject 2:** AHI 5-15. Beginning of inspiration. **Subject 3:** AHI 30-50. Beginning of inspiration. **Subject 4:** AHI > 50. Beginning of inspiration.

Video 2 shows genioglossus motion during the entire respiratory cycle, for a subject with AHI < 5.

Video 3 shows genioglossus motion during the entire respiratory cycle, for a subject with AHI > 50.