Effect of breathing, pressure and posture on palatoglossal and genioglossal tone

Rajat MATHUR, Ian L. MORTIMORE, Mohammed A. JAN and Neil J. DOUGLAS
Respiratory Medicine Unit, Department of Medicine, Royal Infirmary, Edinburgh, U.K.
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INTRODUCTION

The upper airway in man lacks a rigid bony support and is therefore prone to inspiratory collapse. This is opposed by several dilating muscles, which thus allow continued airflow [1, 2]. The two common sites of upper airway collapse in patients with the sleep apnoea/hypopnoea syndrome are retroglossal and retropalatal [3, 4]. It has previously been shown that genioglossal activity increases with each inspiration, but the respiratory actions of the palatal muscles are poorly documented.

Palatoglossus was studied because it pulls the soft palate downwards and forwards to open the retropalatal space [5, 6]. Laxity of this muscle during sleep may lead to upward and backward movement of the palate with resultant velopharyngeal obstruction. Another upper airway muscle – genioglossus – has been shown in man [7–11] and animals [12, 13] to react to negative upper airway pressure. We have therefore tested the hypothesis that palatoglossus exhibits respiratory phasicity and is activated by negative airway pressure.

Also, because posture affects breathing during sleep and upper airway calibre [14–16] and genioglossal tone [17], we have studied the effect of posture on palatoglossal tone.

METHODS

Subjects

Data were collected from 10 awake non-snoring healthy men [24–48 (mean 36) years of age, body mass index 20–27 (mean 23.4) kg/m²] naive to the purpose of the study. Each gave informed consent to the study, which had been approved by the local ethical advisory committee. None had taken alcohol or any medication on the day of the study. All the subjects were free of any nasal, respiratory or sleep disorder.

Electrodes for recording electromyogram (EMG)

The electrodes were made of sterile silver wire (A 5766-36; Cooner Wire Company, Chatsworth, CA, U.S.A.) with an uncoated diameter of 0.125 mm and a Teflon-coated diameter of 0.175 mm. The last 2–3 mm of the wire was bared, lightly chlorided and surface coated with an aliphatic polyurethane coating (Tecoflex) to improve biocompatibility and maintain conductivity.

The wires were threaded through 23-swg needles and the tip bent over the bevel of the needle to make hooked intramuscular wire electrodes.

Key words: electromyogram, negative pressure, upper airway.

Abbreviations: A/D, analogue-to-digital; EMG, electromyogram; MANOVA, repeated measures analysis of variance; VCR, video cassette recorder.

Correspondence: Dr N. J. Douglas, Respiratory Medicine Unit, Department of Medicine (RIE), Royal Infirmary, Edinburgh EH3 9YW, U.K.
Electrode placement

We initially performed detailed cadaveric dissection of six human head and neck specimens noting the precise location of both the genioglossus and palatoglossus muscles, inserted electrodes perorally and verified by dissection that the wire electrodes were in these muscles. We then used the identical oral route of approach in our healthy subjects.

After spraying with topical lignocaine (maximum dose 10 mg), two needles each containing one electrode were inserted perorally into the palatoglossal arch of the pharyngeal fauces on the same side, parallel to the plane of the hard palate, 10 mm apart and 5 mm deep. Electrode location in each case was verified by ensuring that EMG activity increased on swallowing and on forced nasal breathing via an open mouth, manoeuvres previously shown to stress palatoglossus maximally [6]. For genioglossus, similar needles containing electrodes were inserted perorally 3 mm behind the sublingual salivary duct 5 mm on each side of the midline to a depth of 10 mm from the floor of the mouth. Electrode location in each case was verified by ensuring that EMG activity increased on swallowing and on forced protrusion of the tongue against the lower teeth.

Before starting the baseline recordings, we waited for at least 30 min after lignocaine administration to let the effect of the local anaesthesia required for electrode insertion wear off.

EMG recording

A grounding electrode was placed on the subject’s clavicle. The EMG signals were processed by a unity-gain bipolar four-channel ac preamplifier close to the electrodes (Neurolog NL 824, Digitimer, Welwyn Garden City, Herts, U.K.) and then further amplified by Isolator NL 820 to give an amplification of between 0.1 and 50000.

After filtering between 10 Hz and 2 kHz (NL 125), the signals were rectified and integrated with a time constant of 100 ms (NL 703). The raw and integrated EMGs were displayed on oscilloscope monitor (Knott Elektronik, Germany; multichannel large-screen display SG 4100) and recorded on videotape (JVC HR-D725EK) through an analogue-to-digital video cassette recorder (VCR) (A/D) adapter (Model PCM 4/8, Medical Systems, Greenvale, NY, U.S.A.). The videotape was subsequently played back through the same VCR adapter and the output printed on a paper printer (Mark 10-1 Thermal Array Corder, Western Graphtec, Nantwich, Cheshire). A recording was also made of the output from the amplifier system with the input electrodes shorted to obtain a zero-reference baseline signal.

We did not observe any significant differences in the maximum EMG amplitudes of either muscle throughout the study [maximum voltages at the beginning and end of study: genioglossus 85 ± 5 μV (SEM) and 80 ± 4 μV, P = 0.8; palatoglossus 45 ± 5 μV and 43 ± 5 μV, P = 0.8].

Documenting the phase of respiration

Chest wall movements were measured by inductance bands placed at the level of nipples and umbilicus and displayed on the monitor as well as recorded on the videotape.

Application of negative pressure stimuli

We modified the system of Horner et al. [11] so that the subject breathed through an airtight nasal mask attached to a circuit open to atmosphere such that the inspiration occurred through a solenoid pilot-activated spring return valve (Martonair/Beech, Glasgow, U.K.; B/6SP5/122/M).

At the end of randomly selected expirations, the solenoid valve was activated to change the breathing circuit rapidly from atmosphere to a 50-l reservoir continuously evacuated to a negative pressure of -100 cmH₂O. A 4 cmH₂O spring-loaded valve (Vital Signs, Medic-Aid, Pagham, Sussex) at the subject side of the solenoid valve vented the excess negative pressure to atmosphere so that a relatively square-wave 4 cmH₂O negative-pressure pulse was applied to the subject.

EMG amplitudes were recorded 100 ms after the application of negative pressure to avoid including the slower voluntary muscle activation which might occur [11]. We determined the pressure in the mask at this time for each of the 10 subjects for all pressure applications using a differential pressure transducer (Furness, Bexhill, Sussex, U.K.) connected to the nasal mask by tubing 136 cm long. This introduced a delay of 2 ms into the pressure recording system. The pressure change was recorded on videotape as well as displayed on the monitor. The delay in the transducer to an instantaneous pressure change (bursting a balloon) was 5 ms.

In six subjects, pressure in the upper airway was measured by a catheter tip pressure transducer (Gaeltec, Dunvegan, Skye, U.K.) inserted via the nose with the tip positioned just distal to the uvula. In these subjects, inspiratory flow rate was measured by pneumotachograph and upper airways resistance calculated using the pressure difference between the mask and upper airway at an inspiratory flow rate of 151/min.

Protocol

Maximal EMG amplitudes were recorded for genioglossus by swallowing three sips of water and by forced tongue protrusion five times against the lower incisors, and for palatoglossus by breathing forcefully through the nose five times, keeping the mouth open [6], and by swallowing.

Subsequently, data were recorded for at least 30 breaths with the subject breathing through the nose,
Factors affecting upper airway electromyogram in both sitting and supine postures. The order of sitting and supine was randomized between different subjects.

In the sitting posture negative pressure was applied on each occasion through the nose at end expiration on randomly selected breaths at least 30 times and the EMGs recorded.

Then oral, nasal and pharyngeal local anaesthesia was sought by spraying the nose and oropharynx with lignocaine aerosol. Spraying was synchronized with inspiration to facilitate penetration of the aerosol. Sufficient lignocaine was administered to make the subjects unable to detect 81/min airflow through either nostril and to abolish the gag reflex to the touch of an orange stick on both the soft palate and oropharyngeal wall. This was achieved with a maximum of 200 mg lignocaine base (range 100–200 mg). Negative pressure applications were repeated at least 30 times and the study was completed within 15 min of application of this topical oropharyngeal anaesthesia.

Analyses

All EMGs were expressed as a percentage of maximal for both muscles. Peak inspiratory and tonic end-expiratory EMG values were measured breath by breath in both postures and results averaged for 30 breaths. The EMGs immediately before negative pressure application were compared with those 100 ms after pressure application [11], and averaged over 30 breaths. Results were analysed using repeated-measures analysis of variance (MANOVA) with the SPSS/PC software package.

RESULTS

Satisfactory raw and integrated EMG signals were obtained from genioglossus in all 10 subjects. For palatoglossus, satisfactory signals were obtained in eight subjects (Fig. 1) and the data from these eight analysed.

Palatoglossal EMGs were significantly higher on inspiration than on expiration (P = 0.016). However, there was no significant effect of change of posture on palatoglossal activity (P = 0.13; Fig. 2).

Genioglossus also exhibited strong respiratory phasicity with significantly higher inspiratory than expiratory EMGs (P = 0.001). Genioglossal EMGs were significantly higher supine than sitting (P = 0.01; Fig. 3).

Upper airway inspiratory resistance between the nares and retrouval space was higher supine than sitting (5.4 ± 0.6 and 3.7 ± 0.7 cmH₂O s⁻¹; P < 0.01) and higher on inspiration than on expiration (P < 0.025) in both postures.

After negative pressure application, the peak pressure in the nasal mask was −4.1 ± 0.1 cmH₂O with a 0–90% rise time of 10 ± 1 ms. One hundred milliseconds after the negative pressure application, retrouval pressure was −3.3 ± 0.01 cmH₂O in comparison with a maximum spontaneous inspira-
tory pressure during quiet breathing of \(-2.3 \pm 0.01\) cmH\(_2\)O.

Both palatoglossus \((P<0.001)\) and genioglossus \((P=0.02)\) exhibited increased EMG activity with negative pressure application (Fig. 4). This increase in EMG in response to negative pressure remains statistically significant after surface anaesthesia for both palatoglossus \((P<0.05)\) and genioglossus \((P=0.005)\). Surface anaesthesia did not alter the response of genioglossus to negative pressure \((P=0.9)\), but did reduce the palatoglossal EMG response \((P<0.05;\) Fig. 4). Maximal EMG activity remained unchanged after surface anaesthesia.

**DISCUSSION**

This study demonstrates that both genioglossus \([1, 18]\) and palatoglossus show respiratory activity, that palatoglossus as well as genioglossus \([7, 8, 11]\) is activated by negative upper airway pressure and that genioglossal \([17]\) but not palatoglossal activity increases in the supine position.

We believe that our study of genioglossus differs importantly from previous ones \([7, 10, 11]\) as the applied pressure was in the physiological range and the response was measured rapidly thereafter, excluding any voluntary effects.

The response of the palatoglossus muscle to negative airway pressure has not previously been assessed. However, our results are compatible with a recent preliminary finding that palatoglossal activity increased in response to an inspiratory resistive loading of \(25\) cmH\(_2\)O s\(^{-1}\) in eight normal men \([19]\).

Our data indicate the presence of a reflex pathway of palatoglossal activation by negative pressure, similar to that previously demonstrated and herein confirmed for genioglossus \([20]\). We attempted to identify the location of the receptors mediating this upper airway dilator muscle reflex using topical oropharyngeal anaesthesia. Our failure to abolish this reflex after surface anaesthesia could indicate that such receptors are not located in the surface mucosa but may be, for example, in muscle spindles.

Topical oropharyngeal surface anaesthesia administered in this study with \(100-200\) mg lignocaine spray completely abolished the palatal and oropharyngeal gag reflexes and the subject's ability to sense an airflow of a \(8\) l/min through either nostril. Thus, we believe that complete or near complete nasal, nasopharyngeal and oropharyngeal surface anaesthesia was achieved. We did not assess the extent of laryngeal or lower airway anaesthesia produced. However, Horner et al. \([20]\) reduced the genioglossal response to negative pressure by a combination of upper airway topical anaesthesia with \(0.2\) mg cocaine to the nose and \(40\) mg lignocaine spray to the larynx plus blocking the internal laryngeal branches of the superior laryngeal nerve by injection of \(1.2\) mg lignocaine. Also, White \([21]\) in a provisional report, indicated a reduction in the genioglossal EMG response to negative pressure in four of five subjects after \(10\) % lignocaine spray to the oropharynx and \(0.25\) % bupivacaine gargle. The disparity between our results and these other studies \([20, 21]\) could relate to differences in the density or distribution of the local anaesthesia or to the different muscles being sampled. Horner et al. \([20]\) used surface electrodes and a sample from different groups of muscle fibres to those in the current study, and White \([21]\) presumably used his standard approach to genioglossus, which is anterior to the site of insertion in the current study. Genioglossus is a heterogeneous muscle, and this could also explain the disparity. However, it is impossible to exclude the possibility that the surface anaesthesia used in the current study was insufficiently dense, especially in the larynx. It is possible that the greater effect of lignocaine on the palatoglossal limb and genioglossal response to negative pressure could be due to differing sites of afferent receptors. For example, the receptors for the palatoglossal reflex might be high in the upper airway at a level effectively anaesthetized in the study, whereas the receptors for the genioglossal reflex might be at the laryngeal level, which was perhaps not adequately anaesthetized.

There are numerous possible mechanisms for the observed increased upper airway muscle activation in response to negative pressure or inspiratory resistive loading. Chemoreceptors do not seem to mediate this reflex \([22]\). Reduction in lung volume with negative pressure with pulmonary stretch receptor-mediated reduced vagal inhibitory activity on upper airway motor neurons remains a possible...
explanation of these results. However, previous studies [10, 20] have found that little if any of the response to negative airway pressure is due to lower respiratory receptor activation. Moreover, reanalysis of our data to compare the EMG before stimulus with that 100 ms after stimulus on occasions when there was no change in lung volume detected by inductance plethysmograph does not change our results [before stimulus and 100 ms after stimulus]. EMGs at the same 'lung volume' (percentage of maximum) genioglossus, 6% ± 2% (SEM) and 13% ± 3%, P = 0.01; palatoglossus, 8% ± 2% and 23% ± 6%, max, P < 0.001).

We assessed the responses to negative pressure by comparing the EMGs 100 ms after pressure application with those immediately before pressure application as this has been the standard approach [11, 21]. We also analysed these data post hoc by comparing the EMG 100 ms after pressure application with that 100 ms after the initiation of the preceding spontaneous breath. This analysis confirmed that the activity of both palatoglossus (P < 0.05) and genioglossus (P < 0.02) was significantly increased after application of negative pressure. Comparison of the end expiratory genioglossal EMGs before and after lignocaine application (Fig. 4) shows them to be the same, as are EMGs obtained 100 ms after pressure application. Thus, the conclusion that the genioglossal response to negative pressure is not influenced by lignocaine cannot reflect any change in EMG due to changes in the respiratory cycle.

In this study, we found that upper airway resistance increased on lying down. Radiological investigations have shown that the retroglossal airway widens while the retropalatal airway narrows on lying down in both normal subjects and sleep apnoea/hypopnoea syndrome patients [16]. Both peak inspiratory and tonic expiratory genioglossal EMGs increase in the supine posture [17, 18]. Our finding that palatoglossal activity remains unchanged with the assumption of supine posture while genioglossal activity increases may explain the radiological observations [16] that the soft palate moves back on lying down. Interpretation of the EMG findings crucially hinges on the insertion of the electrodes in the appropriate muscles. We believe that this was achieved, basing this contention on our cadaveric dissections, obtaining the predicted increases in EMG activity with manoeuvres designed to stress these particular muscles and the reproducible EMG response to these manoeuvres throughout the study period. The relationship between EMG activity and the force of muscle contraction varies as a function of muscle length [23, 24], velocity of muscle shortening [25], the twitch tension characteristics of the active motor units [24] and the elastic and viscous properties of the attachments between velar muscles [26]. We have assumed in this study that EMG activity correlates with muscle force, an assumption we know of no way to test for in palatal muscles in humans other than by indirect reference to airways resistance or critical pressure measurements.

REFERENCES