Discharge Patterns of Human Tensor Palatini Motor Units during Sleep Onset

Christian L. Nicholas, PhD; Amy S. Jordan, PhD; Leila Heckel, PhD; Christopher Worsnop, MBBS, PhD; Bei Bei, DPsych; Julian P. Saboisky, PhD; Danny J. Eckert, PhD; David P. White, MD; Atul Malhotra, MD; John Trinder, PhD

1School of Psychological Science, University of Melbourne, Parkville, Australia; 2Division of Sleep Medicine, Brigham and Women’s Hospital and Harvard Medical School, Boston MA

INTRODUCTION

Obstructive sleep apnea (OSA) is a common disease with major neurocognitive and cardiovascular sequelae.1-3 Despite the high prevalence of OSA, its pathogenesis remains incompletely understood. State dependent changes in the activity of pharyngeal dilator muscles are thought to be mechanistically important in the upper airway collapse in individuals who are anatomically predisposed.4,5 The genioglossus (GG) is the most studied of these dilator muscles, as it is easily accessible and thought to be representative of the activity of other phasic muscles, i.e., those that show a burst of activity with inspiration.6 The tensor palatini (TP) is regarded as a tonic muscle; i.e., it is said to have relatively constant activity throughout the respiratory cycle.6 Multunit electromyography (EMG) of both the GG and TP muscles indicate reduced activity at sleep onset.7

We8-12 and others13-17 have applied single motor unit techniques to the recording of upper airway muscles in humans. Saboisky et al.9 have shown that GG has considerable complexity, with six distinct patterns of motor unit firing underlying the multiunit electromyographic recordings, while Chanaud and Ludlow16 have reported similar motor unit discharge patterns in laryngeal muscles. In contrast, Brown et al.15 observed little respiratory related activity in geniohyoid during quiet wakefulness. Different discharge patterns in motor units from the same muscle are thought to reflect different pre-motor inputs to the relevant motor nucleus.18 Thus, for example, the observation that the fall in activity of GG at sleep onset is primarily a result of cessation of activity among inspiratory phasic (i.e., units with firing only during inspiration) and inspiratory tonic units (i.e., those with constant activity, but increased firing rate during inspiration), suggests the reduced drive originates in brain stem nuclei that generate the respiratory pattern. The fall in activity of GG inspiratory phasic and inspiratory tonic units is a result of de-recruitment (i.e., units becoming silent) rather than major changes in rate coding (i.e., changes in firing rate). Motor units within GG that do not become silent maintain their rate of discharge during these alpha-theta transitions.11 To date, sleep transition effects have not been examined using single motor unit techniques in TP.

The tensor palatini is thought to act to stiffen the soft palate and promote velopharyngeal patency.5 Tangel et al.19 previously demonstrated that the fall in activity of TP at sleep onset is predictive of the rise of pharyngeal resistance during sleep. On the other hand, GG activity is not predictive of pharyngeal resistance during stable sleep, since its activity recovers to wakefulness levels after a transient fall at the wake-sleep transition.7 Thus, in contrast to GG, TP is thought to display a tonic pattern of activity and to show sustained rather than transient falls in activity during sleep onset and subsequent sleep.

We sought to investigate single motor unit activity in TP and thus provide insight into the various premotor inputs that control the TP during wakefulness and subsequent sleep onset. The foci of the study were the properties of TP motor units active dur-
on 16 September 2017

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METHODS

Subjects

Nine male subjects with a mean age of 21.4 (± 3.4) years and a mean body mass index of 23.4 (± 1.7) kg/m² were studied. Subjects were healthy and without sleep or respiratory complaints. The protocol conformed to the Declaration of Helsinki and had prior approval of the local institution Human Subjects Ethics Committee. Informed written consent was obtained from each subject.

General Laboratory Procedures

Data were collected on one recording night for 7 subjects and 2 nights for 2 subjects in the Sleep Laboratory, School of Psychological Science, the University of Melbourne. Subjects were requested not to consume alcohol during the day and evening before the experimental session and not to consume food or caffeine for 4 hours before coming to the laboratory. They arrived at the laboratory at approximately 21:00, and after preliminary procedures and preparing for bed, instrumentation commenced. Data collection began at approximately 23:00 and continued until approximately 03:00, depending on the continued comfort of the subject. The recording conditions were sufficiently intrusive that spontaneous arousals, and thus opportunities for subsequent sleep onset transitions, were relatively frequent, although the frequency with which sleep onset met the criteria for inclusion (see below) was low. Subjects were studied in the supine body position which was effectively maintained by the constraints of the equipment.

Sleep-wake state

Two EEG channels (C3-A2 and O1-A2), left and right electromyograms, and a chin EMG were recorded to determine sleep-wake state and to identify α-to-θ transitions. Consistent with previous studies,7,11 transitions were visually identified by an experimenter blinded to the EMG recordings and were defined as having: (1) ≥ 30 sec of stable pre-transition α with ≥ 3, and up to 5, analyzable α breaths; and (2) ≥ 3 and up to 5 post-transition θ breaths. Transitions with body movements or swallows or microarousals during the θ phase were discarded. We have established in previous publications that with elimination of such events, it is rare for the recordings to be disrupted by electrode movement.8,11,12

The breath on which the EEG transition occurred was classified as an α breath if the EEG transition occurred after peak inspiratory flow. If the EEG transition preceded peak inspiratory flow, the breath was classified as the first θ breath.

Ventilation and airway mechanics

Airflow was collected by means of a full face mask (Fisher & Paykel, 431 anesthesics mask) and 2-way breathing valve (Hans Rudolph, 2600 medium). Route of breathing was not controlled or monitored. Airflow was measured by a calibrated pneumotachograph (Hans Rudolph, series 3719) placed between the mask and valve, a differential pressure transducer (DP-45, Validyne Corp) and a Morgan carrier demodulator (CD15). When attached the mask, valve, and pneumotachograph dead space was ~163 mL. To determine airway resistance, pressure was monitored in the mask with an open catheter attached to a pressure transducer (Validyne Corp) and in the airway at the level of the epiglottis using a pressure-tipped catheter (MPC-500, Millar) inserted via one nostril.

Respiratory variables were recorded as DC signals with a 15-Hz low pass filter using Grass Amplifiers (Grass Telefactor) and were digitized at 100 Hz using a 1401 interface and Spike 2 acquisition software (Cambridge Electronic Design).

Muscle activity

TP activity was recorded bilaterally using 2 monopolar, intramuscular, stainless steel, Teflon coated, fine-wire hooked electrodes (#790600, AM systems inc., WA, USA). Electrode diameter was 50.8 µm bare and 114.3 µm coated, with 0.5 mm of the tip exposed. The active electrode was referenced to a surface electrode positioned over the bony mandible. A large flexible ground strap was placed on the left shoulder (3M 9160 Universal Electrosurgical Pad, 3M Health care, MN, USA). TP electrodes were inserted via a 25-gauge hypodermic needle. The needle was bent in the middle at an angle of ~30°. To insert the electrode, the tip of the pterygoid hamulus was located via palpation at the junction of the hard and soft palate and the tissue anesthetized (lignocaine HCL). The needle was then inserted at a 45° angle along the lateral surface of the medial pterygoid plate to a depth between 10 and 15 mm into the palate. Insertions were performed by 2 experimenters (C.W. & A. J.), each of whom has considerable experience with the procedure.

Subsequently, respiratory maneuvers (sniffs, swallows, and deep breaths) were initiated to confirm the location of electrodes.19 Filters on the recording equipment were set at 0.03–3 kHz, and the signal digitized and recorded at 10 kHz.

Data Reduction

Respiratory variables

Respiratory variables were assessed on a breath-by-breath basis over each α to θ transition using a Spike-2 script (Cambridge Electronic Design). The protocol conformed to the Declaration of Helsinki and had prior approval of the local institution Human Subjects Ethics Committee. Informed written consent was obtained from each subject.
bridge Electronic Design) developed within the laboratory. The decisions of the breath detection algorithm were visually checked and edited where necessary. The respiratory variables analyzed were minute ventilation, tidal volume, cycle duration, peak inspiratory flow, epiglottal pressure at peak flow, and airway resistance at 200 mL/s. Data were averaged over α and θ breaths to give pre- and post-transition values for each transition, and these values were averaged within subjects (over both nights when 2 nights were run) and then over subjects.

**Overall muscle activity**

The overall level of activity recorded on each electrode was quantified using Spike-2 software by determining the average number of motor unit discharges per second that exceeded a threshold voltage (spike density)\(^9\). Electrodes that did not have identifiable activity (42% of electrodes) or had technical deficiencies, such as the electrode becoming dislodged (11% of electrodes), were not included in the analysis, leaving 47% of transition recordings with spike activity. The threshold was defined as twice the voltage amplitude of background noise. Background noise, which reflected activity of distant motor units and at times electrical interference, averaged ± 35 (SD = 12.7) µV. Spike density was determined independently for inspiration and expiration and averaged separately over α and θ breaths. Values were averaged over electrodes and transitions within subjects and then over subjects.

**Single motor unit activity**

Single motor unit activity on electrodes with decomposable spikes (38% of electrodes: 47% of electrodes had spike activity, but 9% had too much activity to be decomposed) was identified using Spike-2 analysis software (Cambridge Electronic Design). Motor units were initially identified using a spike-triggered threshold voltage and sorted by the software into templates based on their detailed morphology. Subsidiary software was then used to inspect and edit the initial software classification visually (software courtesy of Neuroscience Research Australia). Each sorted motor unit was further inspected for sudden changes in frequency (defined as a change > 40% from one inter-spike interval to the next) that likely indicated inappropriate sorting. If > 5% of these events could not be satisfactorily resolved, the unit was discarded.\(^12\) The discharge properties of the motor units were expressed as instantaneous frequency plots.

Five discharge patterns previously reported in GG by Saboisky et al.\(^7\) were identified in the current data; a sixth, *tonic other*, was not identified in this data set. The 5 discharge patterns identified were: *inspiratory phasic*, units active primarily during inspiration; *inspiratory tonic*, units active throughout the respiratory cycle with an inspiratory peak; *expiratory phasic*, units active primarily during expiration; *expiratory tonic*, units active throughout the respiratory cycle with an expiratory peak; *tonic*, units active throughout the respiratory cycle without respiratory or other modulation.

In order to determine a unit’s degree of respiratory modulation, instantaneous frequency values for the motor unit were cross-correlated with respiratory phase (obtained from a tidal volume measure derived from an integrated airflow signal) at the time of each spike, over a breath. The value was calculated for each breath and averaged over α or θ breaths. The greater the maximum cross-correlation value, the stronger the respiratory modulation. The statistic is analogous to the η\(^2\) statistic proposed by Orem and Dick,\(^20\) but modified to accommodate data with small numbers of breaths.\(^9,11\) In accord with published values,\(^8,11,12\) to be classified as inspiratory tonic or expiratory tonic, a unit had to have a cross-correlation value > 0.49 and for the maximum values to have a consistent inspiratory or expiratory phase. In allocating motor units to different discharge patterns, motor units were classified according to their α pattern. However, there was a tendency for motor units to become silent before the transition so that for 5 motor units activity was available for only 2 α breaths.

**Discharge properties of motor units**

The instantaneous frequency units were quantified to characterize the discharge pattern of the motor units (software developed by Neuroscience Research Australia). The definition of each measure has been published.\(^8,11\) Briefly the measures were: the peak, mean and tonic discharge rates of units; the timing of the onset of the phasic component; and the proportion of a breath inspiratory phasic and expiratory phasic units were active.

**Statistical Analyses**

The respiratory variables were analysed by repeated measures t-tests (α versus θ). Spike density (total number of spikes/sec) was analyzed by a 2 (inspiration versus expiration) by 2 (α versus θ) repeated measures ANOVA. The distribution of motor units with different discharge patterns during pre-sleep onset α activity was analyzed to determine if the distribution differed from chance using the \(\chi^2\) test. The α distribution was also compared to the distribution of units that ceased activity over sleep onset and the distribution of motor units during θ activity, using \(\chi^2\) analysis. Finally, statistical analysis of the discharge characteristics of motor units consisted of a series of repeated measures t-tests (α versus θ).

Two variants of the first set of analyses (discharge characteristics) were conducted. In the first method, when a unit was not active on a breath, a zero value was entered for that breath. This analysis indicated the changing contribution of each type of motor unit to the muscles output over sleep onset. In the second, breaths on which units did not fire during sleep onset were discarded from the analysis, so that the resulting values indicated the effect of sleep onset on motor units that continued to be active over sleep onset. In accord with the literature and with our previous publications, the statistical analyses reported were based on motor units, although analyses based on subjects produced equivalent mean values.

**RESULTS**

**Respiratory Measures**

Table 1 presents mean values and significance for the respiratory variables. Changes in respiratory activity were consistent with previous papers including our recent study of GG muscle activity during sleep onset.\(^11\) There was a general reduction in respiratory activity, with significant falls in tidal volume and peak inspiratory flow, and a less negative epiglottic pressure, the latter reflecting reduced respiratory drive. While minute
ventilation fell and airway resistance rose, these changes were not significant. Finally, cycle duration fell significantly.

**Overall Muscle Activity**

Overall spike density was significantly higher during inspiration than expiration (35.5 ± 10.2 spikes/s and 26.6 ± 12.4 spikes/s respectively, $F_{1,8} = 8.37, P < 0.05$) and higher during α than θ (37.4 ± 12.8 spikes/s and 24.7 ± 9.8 spikes/s respectively, $F_{1,8} = 22.00, P < 0.001$). However, the respiratory phase by sleep-wake state interaction was not significant ($F_{1,8} = 2.50, P > 0.05$).

**Distribution of Active Motor Units**

TP data were collected in the nine subjects over 121 α to θ transitions (13.4 ± 7.2/subject). As 2 electrodes were inserted in each subject, 242 sleep onset recordings were collected. Ninety-three (10.3 ± 7.7/subject), or 38%, had decomposable motor units, resulting in 128 units (14.3 ± 13.0/subject). The general properties of all the units are shown in Figure 1.

Subjects varied as to the number of motor units they contributed to the group data. In order to equalize the contribution of each subject the distribution of motor units with different discharge patterns was first expressed as percentage values for each subject, and these percentage values were then averaged over subjects. The distribution over the 5 discharge patterns during α differed significantly from chance [$\chi^2(4) = 12.55, P < 0.05$; see top panel Figure 2]. There were slightly more tonic units than other individual categories, although the difference was not large, and the total number of inspiratory modulated units (inspiratory phasic and inspiratory tonic) was greater than tonic. Further, phasic units (inspiratory phasic and expiratory phasic) made up 39% of the units. Thus, the presence of tonic units and units with a tonic component (inspiratory tonic, expiratory tonic, and tonic) were not as marked as anticipated based upon multiunit recordings of the muscle. Finally, there was a relatively high proportion of expiratory phasic units identified in TP (21%).

There were also marked differences in the units that ceased activity during sleep onset [$\chi^2(4) = 31.38, P < 0.001$]. As shown in the bottom panel of Figure 2, approximately 25% of inspiratory modulated units ceased activity during θ, while a surprising 18 of 19 expiratory phasic motor units, or 95%, also ceased activity (one such unit is illustrated in Figure 3). In contrast, tonic motor units were unaffected by sleep onset.

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**Table 1**—Mean and standard deviation values for respiratory variables during α and θ states

<table>
<thead>
<tr>
<th></th>
<th>Alpha</th>
<th>Theta</th>
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</thead>
<tbody>
<tr>
<td>Cycle Duration (s)</td>
<td>4.70 (0.77)</td>
<td>4.42 (0.52)*</td>
</tr>
<tr>
<td>Tidal volume (L)</td>
<td>0.34 (0.07)</td>
<td>0.28 (0.06)*</td>
</tr>
<tr>
<td>Minute Ventilation (L/min)</td>
<td>4.43 (1.32)</td>
<td>3.85 (1.01)</td>
</tr>
<tr>
<td>Peak Inspiratory Flow (L/min)</td>
<td>25.1 (7.30)</td>
<td>22.0 (5.63)*</td>
</tr>
<tr>
<td>Pepi Minimum (cm H2O)</td>
<td>-4.17 (1.47)</td>
<td>-3.93 (1.38)*</td>
</tr>
<tr>
<td>Resistance at 200 mL/s (cm H2O/L/s)</td>
<td>4.50 (3.00)</td>
<td>5.34 (4.06)</td>
</tr>
</tbody>
</table>

Pepi, epiglottic pressure. *P < 0.05. Breath-by-breath values were averaged over α and θ breaths within sleep onsets, over sleep onsets within subjects, and then over subjects.
consequence of some motor units being more susceptible to de-recruitment (e.g., expiratory phasic units), there was a significant difference between the distributions of motor units active during α compared to θ ($\chi^2(4) = 48.2, P < 0.001$).

**Magnitude of Respiratory Modulation**

Mean wakefulness cross-correlation values for each motor unit type are depicted in Figure 4. There was not a significant difference between inspiratory (0.69) and expiratory (0.61) tonic units compared to inspiratory (0.78) and expiratory (0.77) phasic units ($P > 0.05$). Tonic units, by definition, had the lowest cross-correlation values (0.46). Mean cross-correlation values did not change over sleep onset for any motor unit type (all $P > 0.05$). Further, the α cross-correlation values of units that became silent in θ did not differ from the α values of those units that continued (all $P > 0.05$), indicating that the strength of the phasic component within a particular discharge pattern did not predict whether the unit would cease activity during θ.

In general the discharge pattern of motor units was relatively stable, with no clear conversions between inspiratory and expiratory patterns and few between tonic and phasic patterns. While there was a tendency for respiratory modulation to be weaker during θ for some patterns, these effects were not significant (all $P > 0.05$), and falls to below 0.49 for some units were matched by rises to above 0.49 for other units. Further, the absolute change in cross-correlation values between α and θ was relatively small (0.09 for all units).

**Discharge Characteristics of Motor Units**

The discharge characteristics of motor units and the associated statistical analyses are presented in Table 2 and illustrated

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**Figure 2**—The top panel shows the distribution of motor units with different discharge patterns during α activity. The bottom panel shows the percentage of motor units with each discharge pattern during α that had become silent by the 5th θ breath.

**Figure 3**—Instantaneous frequency plot and raw EMG channels for an expiratory phasic TP motor unit that became silent immediately after a transition from α to θ EEG activity (indicated by the vertical dotted line). The insert diagram shows the over drawn spike waves for the motor unit. The breath that occurred at the transition was classified as post transition (see text). Also shown are the C3-A2 EEG and airflow (inspiration is negative, or up).
in Figures 1 and 5. Peak discharge rates were a little above, and mean rates were a little below, 20 Hz for inspiratory modulated and tonic units. Expiratory modulated units tended to have slightly lower rates. Motor unit discharge rates remained stable across sleep-wake transitions, so long as the unit continued to fire (see “continuing units” values in Table 2 and Figure 5). However, when a unit did not fire and a zero was entered for that breath (“all units” value), the mean frequency fell from \( \alpha \) to \( \theta \), indicating a reduced contribution from that unit class. Of interest was the observation that phasic units (both inspiratory phasic and expiratory phasic) tended to stop earlier in the transition than the corresponding units with a tonic component (see Figure 5).

Neither average inspiratory phasic nor inspiratory tonic discharge patterns showed significant pre-activation (see Table 2). However, 37% of individual inspiratory phasic motor units and 24% of inspiratory tonic units did show pre-activation (Figure 1). Further, the onset of firing of inspiratory phasic, but not inspiratory tonic, units was significantly later during \( \theta \) than \( \alpha \) (see Table 2), and the number of inspiratory phasic units showing pre-activation fell from 18 to 8. In addition, there was some tendency for inspiratory phasic and inspiratory tonic units that continued to be active in \( \theta \) to be more likely to show pre-activation in \( \alpha \) than units that became silent (35% vs. 21%, respectively).

**DISCUSSION**

The data supported some of our hypotheses, but also produced some unanticipated observations. First, and contrary to expectations, we observed considerable complexity within TP motor unit activity, similar to that which had previously been observed in GG.\(^9\) Compared to GG there was a higher proportion of tonic units, a smaller proportion of inspiratory modulated units and a relatively high proportion of expiratory phasic units active during wakefulness. Second, reduced TP activity at sleep onset was due to de-recruitment (units became silent) of expiratory phasic units and to a lesser extent, inspiratory phasic, inspiratory tonic, and expiratory tonic units. This finding was not consistent with our hypothesis that the loss of tonic units would be the predominant mechanism producing the fall in TP activity at the \( \alpha \) to \( \theta \) transition, nor was it consistent with the pattern of de-recruitment observed in GG, as in this muscle almost all of the units that became silent were inspiratory modulated.\(^11\) Third, consistent with our *a priori* hypothesis and with a recent review,\(^18\) we did not observe a fall in the average rate of discharge (change in rate coding) in units with persistent activity during the sleep onset transition. We also observed that the motor unit discharge rate was similar to both GG\(^9\) and laryngeal muscles,\(^16\) and the proportion of inspiratory phasic and inspiratory tonic units showing pre-activation (activation before the onset of flow) was similar for TP and GG (\( \sim 30\% \)). Thus, our findings were unexpected and provide new insights into upper airway motor control in healthy humans.

The present results are consistent with and are strengthened by earlier data of Chanaud and Ludlow,\(^16\) who reported a similar range of discharge patterns in two laryngeal muscles; thyroarytenoid (which has an adductive function and an expiratory respiratory pattern on multiunit recordings), and cricothyroid (which has an abductive function and an inspiratory respiratory pattern on multiunit recordings). Thus, the upper airway muscles...
in which respiratory-related activity of single motor units has been studied have the same range and similar distribution of motor unit discharge patterns, despite being controlled by three different brainstem motor nuclei, having different overall multiunit activity patterns, and having different functions. Nevertheless, there are differences in the exact distributions of motor unit patterns, which presumably reflect each muscle’s specific functions.

In TP, motor units with both inspiratory and expiratory phasic components tended to become silent over sleep onset, although expiratory phasic units were most strongly affected. This suggests that both inspiratory and expiratory components of respiratory pattern generation are influenced by the loss of wakefulness, and that the pre-motor input to expiratory motor units in GG, which were not affected by sleep onset, may be different from that of TP. However, it should be noted that relatively few expiratory motor units were identified in GG.

The unexpected identification of a relatively high proportion of inspiratory modulated units in TP was noteworthy, given the widespread view that TP is a tonic muscle. However, as spike density identified a significant inspiratory modulation in the muscle, it is possible that we have overestimated the proportion of inspiratory modulated units. Nevertheless, the current data leave little doubt that respiratory modulated motor units are present in TP. Indeed, there is a range of evidence supporting this view. Animal studies clearly show inspiratory phasic activity in TP, and anatomical studies in animals have shown that the trigeminal motor nucleus receives input from the

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**Table 2**—Mean and standard deviation values for motor unit discharge characteristics during α and θ

<table>
<thead>
<tr>
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<th>Alpha</th>
<th>Theta</th>
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<tbody>
<tr>
<td></td>
<td>α Cont</td>
<td>α All</td>
</tr>
<tr>
<td>Inspiratory phasic (N = 49)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak f (Hz)</td>
<td>21.3 (4.7)</td>
<td>21.5 (4.4)</td>
</tr>
<tr>
<td>Mean f (Hz)</td>
<td>17.5 (3.7)</td>
<td>17.9 (3.6)</td>
</tr>
<tr>
<td>% Duration</td>
<td>36.5 (9.6)</td>
<td>38.0 (11.2)</td>
</tr>
<tr>
<td>Onset Time (s)</td>
<td>0.11 (0.3)</td>
<td>0.23 (0.3)</td>
</tr>
<tr>
<td>Inspiratory tonic (N = 29)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak f (Hz)</td>
<td>22.0 (4.3)</td>
<td>21.0 (5.6)</td>
</tr>
<tr>
<td>Mean f (Hz)</td>
<td>19.1 (4.1)</td>
<td>18.0 (5.1)</td>
</tr>
<tr>
<td>Tonic f (Hz)</td>
<td>13.6 (5.3)</td>
<td>11.8 (7.8)</td>
</tr>
<tr>
<td>Onset Time (s)</td>
<td>0.35 (0.5)</td>
<td>0.39 (0.5)</td>
</tr>
<tr>
<td>Expiratory phasic (N = 19)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak f (Hz)</td>
<td>15.3 (2.6)</td>
<td>13.1 (4.6)</td>
</tr>
<tr>
<td>Mean f (Hz)</td>
<td>12.3 (2.1)</td>
<td>10.5 (3.3)</td>
</tr>
<tr>
<td>% Duration</td>
<td>33.6 (18.4)</td>
<td>33.6 (17.5)</td>
</tr>
<tr>
<td>Expiratory tonic (N = 5)</td>
<td></td>
<td></td>
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<tr>
<td>Peak f (Hz)</td>
<td>19.3 (2.0)</td>
<td>17.4 (1.8)</td>
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<tr>
<td>Mean f (Hz)</td>
<td>16.2 (2.2)</td>
<td>14.8 (1.8)</td>
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<tr>
<td>Tonic f (Hz)</td>
<td>15.3 (2.1)</td>
<td>13.3 (3.2)</td>
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<tr>
<td>Tonic (N = 26)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean f (Hz)</td>
<td>18.0 (5.3)</td>
<td>17.4 (4.9)</td>
</tr>
</tbody>
</table>

For “θ continuing units,” only breaths on which the unit fired were included in the mean value; while for “θ all units,” all breaths were included with a zero entered for breaths on which the unit was silent (αP < 0.05; βP < 0.01; γP < 0.001; significantly different from α; where a variable did not apply to the unit type it has been omitted from the Table).

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**Figure 5**—Mean discharge frequency for motor units with different discharge patterns during α and θ breaths. Closed symbols represent the average of motor units that continued to fire and reflect the rate of discharge of continuously active units. Open symbols represent the average at each breath position with a zero included when a unit did not fire and reflect the contribution of each motor unit type to the fall in overall muscle activity. Thus, the difference between the two representations of the data is due to de-recruitment, an effect that was also apparent in peak values (not shown).
pre-Bötzinger complex. While most studies identify purely tonic activity in multiunit TP recordings, the presence of an inspiratory phasic component has been reported in a number of studies. The current data are also consistent with the presence of inspiratory modulated motor units in other muscles that do not have an overall inspiratory phasic pattern, such as, for example, the thyroarytenoid.  

There are several reasons why the activity of respiratory modulated motor units may not be manifest in TP multiunit activity under resting conditions. First, comparison of the current TP data with equivalent data for GG suggests that the proportion of units with phasic activity is likely to be less than in GG, resulting in a lower probability of multiunit recordings incorporating such phasic units. Second, there is likely to be a tendency for the activity of inspiratory and expiratory motor units to combine, thereby producing a multiunit EMG envelope from the TP that appears tonic. Third, the size (extent of axonal branching) of phasic motor units may be smaller in TP. Thus, while the exact proportion of respiratory modulated units remains uncertain, it is evident that TP contains such units, indicating a capacity for respiratory modulated activity. However, that capacity is not usually manifest in multiunit recordings under low drive conditions.

The identification of a sizable number of expiratory phasic motor units during wakefulness and their almost complete cessation of firing over sleep onset was unexpected. However, it is likely that the impact on muscle activity would have been similar to that of the loss of inspiratory modulated units, as although a smaller proportion of inspiratory units became silent, they had higher discharge rates, and there were more units active during wakefulness. Thus, both inspiratory and expiratory activity was reduced, a pattern consistent with the likely action of TP to stiffen the palatal region, rather than dilate the airway during inspiration. It should also be noted that in this study we only followed units for up to five breaths following the α to θ transition. However, it is known that multiunit TP activity continues to fall well into sleep; whether this is achieved through further losses to inspiratory modulated units or to tonic units remains unknown.

Previous studies have established that reductions in GG activity are due to de-recruitment (units stopping at sleep onset), while increases in activity are due to recruitment (arousal from sleep and hypercapnia) and not to significant changes in rate coding (changes in the discharge rate). The same pattern occurred in TP, suggesting a similar pattern of motor control. As has been pointed out by Bailey, this pattern may be due to a number of potential properties of upper airway muscles. One possibility is that respiration requires relatively undifferentiated activity, rate coding being more marked in muscles involved in fine motor coordination. Another is that upper airway muscle motor units may be operating at the upper limit of their dynamic range, preventing an increase in discharge rate in response to increased respiratory drive. Finally, as only a small proportion of upper airway muscle motor units are likely to be active during relaxed wakefulness and sleep, there would be scope for recruitment. However, two observations complicate these interpretations. First, the properties of upper airway motor units change markedly during nonrespiratory maneuvers, with large increases in discharge rate above that observed during relaxed respiration. Second, increases and decreases in respiratory drive are both associated with recruitment and de-recruitment, suggesting that upper airway motor units are not at the end of their dynamic range during relaxed wakefulness. Together these observations suggest that the pattern of motor control of upper airway muscles may be specific to particular behaviors.

There was some evidence in the data to suggest that inspiratory tonic and expiratory tonic units that de-recruited, did so later in the transition than inspiratory phasic and expiratory phasic units that became silent (Figure 5). Further, units that showed pre-activation were less likely to de-recruit over sleep onset. This suggests that units with tonic and pre-activation components in wakefulness maintain excitability further into sleep.

Despite the strengths and novelty of our study, we acknowledge the following limitations. First, based on our recording techniques, we sampled only from specific areas within the TP of our subjects. Thus, we must work under the assumption that the portions of muscle sampled were representative of the entire muscle’s activity. Indeed, based on our experience in recording GG muscle activity, we would predict that the spatial distribution of motor units would be relatively homogeneous throughout the muscle. In addition, we would view our technique of using implantable, hooked-wire electrodes as a strength since it allowed us to study recruitment strategies. Second, our sample size consisted of 9 subjects over 11 nights, yielding 128 motor units. One could argue that a larger sample size would be desirable, and indeed further work is planned in this area. However, our sample size is more than comparable to most intensive physiological studies, particularly given the labor-intensive nature of motor unit electrophysiology. Third, while our analyses were limited to sleep onset, we argue that this is the most critical phase in understanding the effect of sleep on upper airway patency. Fourth, our detailed comparison of the current data with our earlier work on GG, while critical in understanding the comparative roles of the two muscles in maintaining airway patency, was limited by the subject samples being different individuals. Fifth, the use of a mask and pneumotachograph to measure ventilation would have increased dead space and would likely have increased end tidal CO₂, potentially modifying muscle activity. However, all of our previous studies of single motor units in upper airway muscles have been conducted under equivalent conditions. Finally, our sample included only young healthy male subjects. While this had the advantage of comparability with our study of GG, which consisted primarily of young men, it does limit our conclusions to the population studied. We would advocate further research into gender and aging effects on upper airway control. In addition, studying the pathophysiology of OSA patients would be of interest.

These findings provide important new insight into TP motor control during wakefulness and sleep onset. In particular, rather than consisting of predominantly tonic motor units that reduce their activity at sleep onset, a high proportion of TP motor units showed either inspiratory or expiratory respiratory modulation, with a reduction in both types of units at sleep onset. We speculate that this arrangement provides flexibility in the muscle’s activity while preserving its respiratory role of stiffening the palatal region of the airway throughout the respiratory cycle. Given recent insights into neurochemical control of the trigeminal motor system, pharmacological targets for OSA are likely to emerge once the critical units to target have been defined.
ACKNOWLEDGMENTS

The study was conducted in the School of Psychological Science, The University of Melbourne. Research was supported by NHMRC project grant #566781 and NIH R01HL085188-01A2.

DISCLOSURE STATEMENT

This was not an industry supported study. Dr. Jordan has consulted for Apnex Medical. Dr. Malhotra has received research and consultant income from the NIH, AHA, Apnex Medical, Philips Respironics, SGS, SHC, Pfizer Pharmaceuticals, Merck Pharmaceuticals, Apnicure, Medtime Pharmacy, Sepracor Inc. and Cephalon. Dr. White is the chief medical officer of Phillips Respironics. Dr. Eckert has received research support from Sepracor Inc. and serves as a consultant for Apnex Medical. Dr. Worsnop serves on the national health and Medical Research Council of Australia. He received research support from the University of Melbourne, Austin Hospital, GlaxoSmithKline, AstraZeneca, Pfizer Pharmaceuticals, Boehringer Ingelheim, Medimark, Alfred Hospital and Novartis Healthcare. The other authors have indicated no financial conflicts of interest.

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