Fatiguing contractions of tongue protrudor and retractor muscles: influence of systemic hypoxia

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Although several groups of muscles are involved in the respiratory-related control of pharyngeal airway patency, the pharyngeal-dilating muscles, in particular the primary tongue protrudor muscle, the genioglossus (GG), have received the majority of physiological and clinical interest. Accordingly, abundant information on the neuromuscular control, contractile properties, and endurance performance of pharyngeal dilator muscles has been generated (for review see Ref. 31). Recent evidence suggests, however, that muscles generally considered to be pharyngeal constrictors may also be critically involved in maintaining the patency of the pharyngeal lumen (10, 13, 14, 17). For example, in the rat, cocontraction of the GG with the muscles that retract the tongue [hyoglossus (HG); styloglossus, SG] decreases pharyngeal airway collapsibility more effectively than does independent GG contraction (10).

Salamone and van Lunteren (23) have demonstrated that moderate-to-severe hypoxia adversely affects the endurance of the geniohyoid muscle, which is considered to be a pharyngeal dilator. However, the influence of hypoxia on the endurance capabilities of the tongue protrudor and retractor muscles has not been examined. The primary purpose of this investigation was to examine the endurance capabilities of the tongue protrudor and retractor muscles in normoxia and hypoxia [arterial PO₂ (PₐO₂) ~ 50 mm Hg]. Furthermore, to provide insight into the mechanisms by which hypoxia influences skeletal muscle fatigue, we also examined the influence of hypoxia on the relationship between tongue muscle electromyogram (EMG) and force during evoked contractions.

Methods

Experiments were performed on a total of 41 male SpragueDawley rats weighing 330–550 g. All procedures adhered to the guidelines established by the Institutional Animal Care and Use Committee at the University of Arizona. Rats were anesthetized with an intraperitoneal injection of urethane (1.3 g/kg); subsequent doses (0.3 g/kg) were administered if necessary. An adequate surgical plane of anesthesia was ascertained by lack of a withdrawal reflex in response to intense pressure applied to the paws and tail. At the conclusion of each experiment, animals were euthanized with an intravenous injection of pentobarbital sodium (200 mg/kg). During all surgical and experimental procedures, the rats were supine with limbs secured to the operating table. The upper jaw of the rat was secured to the surgery table by using a wire frame, which maintained the head in a stable position without interfering with the measurements of tongue force (see Measurement of tongue force). Rectal temperature was maintained at 37°C with the use of a servo-controlled heating lamp. The trachea was cannulated caudal to the larynx, and animals were ventilated with positive pressure throughout all experiments. The carotid artery and femoral vein were cannulated to enable sampling and replacement of blood. The XII nerve was exposed bilaterally, and the medial (XIImed) and lateral (XIIlat) branches were isolated. The GG and intrinsic tongue muscles are innervated via XIImed, whereas the SG and HG and intrinsic muscles are innervated via XIIlat (12).

Inspired gas concentration was controlled by mixing O₂ and N₂ with a rotometer and connecting the rotometer outflow port to the ventilator. Inspired fractional concentrations of O₂ (FIO₂) were measured with an O₂ analyzer (model OM-11, Beckman). Arterial blood samples (200–400 µl) were withdrawn at appropriate times (see Protocol) into 1-ml heparinized syringes and immediately placed on ice. Blood samples were analyzed within 30 min for PO₂, PCO₂, and pH.

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We reasoned that the loss of blood, although small, could adversely affect tongue muscle endurance performance. Therefore, the volume of blood removed was replaced with blood from a "donor" animal (see Protocol).

EMG recordings. The EMG of the GG and HG muscles was recorded by inserting two fine-wire (diameter = 0.125 mm, Formvar, California Fine Wire) electrodes into each muscle as described previously (9). The EMG signals were amplified and filtered (30–3,000 Hz) with alternating-current-coupled differential amplifiers (model 7P51K, Grass). Electrode placement was verified by passing current through the electrodes and observing the resultant tongue movements. If stimulation through the GG or HG EMG electrodes failed to evoke clear tongue protrusion or retraction, respectively, poor electrode placement was assumed and the wires were replaced. Electrode placement was also confirmed by examining the GG and HG EMG recordings during XIImed and XIIlat stimulation. If stimulation did not produce a clearly discernible compound muscle action potential (M wave) in the appropriate recording (e.g., see Fig. 1), the wires were replaced.

XII nerve stimulation. The XIImed or XIIlat nerves were stimulated bilaterally by using bipolar hook electrodes (electrode diameter 0.5 mm; interelectrode distance 2 mm). The stimulating electrodes were connected in series to a stimulus isolation unit (model PSIU6, Grass) and a stimulator (model S48 Grass). To prevent efferent neural signals from reaching the tongue muscles, and to prevent antidromic impulse propagation, the XII nerves were crushed bilaterally with forceps ~1 mm proximal to the stimulating electrodes. Nerves were stimulated with "supramaximal" current, which ranged from 40 to 120 µA. Supramaximal current was established by progressively increasing the current until both M-wave and twitch force amplitude reached a plateau despite further increases in stimulus current.

Measurement of tongue force. A detailed description and critique of the tongue force measurement technique has been published previously (9). To summarize, a thread was sewn through the anterior tip of the midline frenulum of the tongue and used to connect the tongue to an isometric force transducer (model FT03, Grass). Thus retraction of the tongue loaded the transducer, whereas tongue protrusion unloaded it. This technique requires tension in the line that connects the tip of the tongue to the force transducer. The amount of tension in the line was standardized by examining the relationship between muscle twitch force and line tension for both protrudor and retractor muscles at the beginning of each experiment. XIImed or XIIlat nerves were stimulated at a range of line tensions, and the resultant protrusive and

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**Fig. 1.** Representative tracings of genioglossus (GG) and hyoglossus (HG) electromyogram (EMG) activity and tongue force during a single medial (XIImed) (A) or lateral (XIIlat) (B) XII nerve stimulation train. In both A and B, single representative M waves are also shown at an expanded time base.
retractive twitch forces were recorded. The line tension was set at the level at which the greatest twitch force was evoked and was established separately for both XIImed and XIIlat stimulation (e.g., see Fig. 2 in Ref. 9).

Protocol. The fatigue test that has been used widely adopted by many laboratories as a standard fatigue test following its original development by Burke and colleagues (2, 3). The Burke fatigue test is considered to be of general utilitarian value for comparing the fatigability of muscles and their motor units both within and across different mammalian species. The fatigue protocol used in the present study used the same stimulus parameters originally used by Burke and colleagues; however, the duration of the test was extended to 5 min (see Critique of methods for a more detailed discussion of the Burke fatigue test). The XIImed or XIIlat nerve branches were stimulated with a 0.1-ms pulse delivered at 40 Hz in trains of 330 ms (e.g., see Fig. 1). Stimulus trains were delivered once per second for 5 min. At the conclusion of the 5-min fatigue protocol, tongue muscle twitch force and M waves evoked by 1-Hz stimulation (0.1-ms pulse) were monitored at 10-min intervals for 1 h or until twitch force returned to prefatigue values. If the twitch force and M wave did not show signs of recovery within 1 h, we assumed that the XII nerves had been damaged, and the data were discarded.

The fatigue protocol was performed on two separate experimental groups, designated as normoxic and hypoxic. Fatigue tests were performed on each muscle group only once in each animal (usually 1 trial with tongue protrudors and 1 trial with retractors). The FIO2 was set at 0.21–0.25 for the normoxic control trials. We did this because, in our experience, FIO2 values of 0.21 sometimes result in mildly hypoxic PaO2, Torr values in anesthetized rats. However, the slightly elevated FIO2 values that we used resulted in PaO2 values that were slightly higher than would be expected in an unanesthetized rat at a FIO2 of 0.21 (Table 1). Nevertheless, we refer to this group of animals as “normoxic,” both for the sake of simplicity and because the resulting PaO2 values are only slightly elevated (Table 1). The FIO2 was 0.14–0.15 for the hypoxic trials.

Arterial blood was sampled immediately before and after the fatigue tests. A volume of blood equivalent to that withdrawn was subsequently infused via the venous catheter. Donor blood was obtained from littermates 1–5 days before the experiment and refrigerated in heparinized syringes. Blood-gas values are presented in Table 1. Both EMG and force data were not always collected from a given animal. For the normoxic group, tongue protrusion and retraction force data were obtained from 11 and 13 rats, respectively, and protrudor and retractor EMG recordings were made in 10 rats. In the hypoxic group, protrudor and retractor muscle force data were collected from 13 rats and EMG data from 10 rats.

Data analysis. Tongue force, EMG activity, and output of the stimulator were recorded on videocassette recorder tapes after pulse-code modulation (model 4000, Vetter); tongue force was also recorded directly onto a polygraph chart recorder (model 79, Grass). Subsequently, computer software programs were used to analyze the EMG waveforms and tongue force (Windaq, Dataq Instruments, Akron, OH, and SPIKE II, Cambridge Electronics, Cambridge, UK). Tongue force was quantified by calculating the force-time integral of each 330-ms train in the 5-min fatigue test. Baseline shifts in the force record often occurred during XIImed stimulation (e.g., see Fig. 2). Therefore, computer software was used (SPIKE II, Cambridge Electronics) to calculate a new baseline for each individual stimulus train. The EMG activity was quantified by measuring the peak-to-peak amplitude, peak-to-peak duration, and total area of the M wave, as described by Enoka et al. (7). For the normoxic and hypoxic fatigue tests, the mean tongue force and EMG responses were calculated for the first and last 10 s of the first minute and for the last 10 s of each minute thereafter. Force as well as M-wave area and peak-to-peak amplitude were expressed as a percentage of the value observed during the first 10-s epoch of the fatigue test.

Statistical differences in tongue force and EMG parameters within and across normoxic and hypoxic fatigue tests were determined with a two-way ANOVA and the Student-Newman-Keuls post hoc procedure. The relationship between tongue force and M-wave area was examined by using linear-regression analyses. Statistical significance was set at P < 0.05. All data are presented as means ± SE.

RESULTS

Representative recordings of tongue protrusion force and the GG M wave during XIImed stimulation in both normoxia and hypoxia are shown in Fig. 2. During normoxia (Fig. 2, left) GG M-wave amplitude and tongue protrusion force were relatively constant throughout the protocol. In contrast, when the experiment was performed during systemic hypoxia (Fig. 2, right), GG M-wave amplitude declined, M-wave duration increased, and tongue protrusion force declined progressively. Examples of changes in the HG M-wave and tongue retraction force evoked by XIIlat stimulation during both a normoxic and hypoxic fatigue test in separate animals are depicted in Fig. 3. Tongue force was relatively constant and HG M-wave amplitude declined slightly over the duration of the test in normoxia (Fig. 3, left). XIIlat stimulation during systemic hypoxia, however, resulted in a substantial drop in tongue retraction force (Fig. 3, right) as the test progressed. In addition, a decline in HG M-wave amplitude and an increase in M-wave duration accompanied the decrease in tongue retraction force during hypoxia.

The average changes in tongue protrusion and retraction force during XIImed and XIIlat stimulation, respectively, in both normoxia and hypoxia are presented in Table 1. Blood-gas values during normoxic and hypoxic fatigue trials

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<th>Normoxia</th>
<th>Hypoxia</th>
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<td></td>
<td>Pre</td>
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<tr>
<td>XIImed stimulation</td>
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<td></td>
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<tr>
<td>PaO2, Torr</td>
<td>119±7</td>
<td>112±7</td>
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<td>PaCO2, Torr</td>
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<tr>
<td>pH</td>
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<tr>
<td>XIIlat stimulation</td>
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<tr>
<td>PaO2, Torr</td>
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<td>PaCO2, Torr</td>
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<tr>
<td>pH</td>
<td>7.34±0.04</td>
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Values are means ± SE. Pre, before fatigue trial; Post, after fatigue trial; XIImed, medial XII nerve branch; XIIlat, lateral XII nerve branch; PaO2, arterial PO2; PaCO2, arterial PCO2.

Table 1. Blood-gas values during normoxic and hypoxic fatigue trials
Fig. 4. For both protrudor and retractor muscles, application of hypoxia significantly attenuated tongue muscle endurance relative to the normoxic condition (P < 0.05). Average GG and HG M-wave parameters during the normoxic and hypoxic fatigue trials are given in Table 2. GG and HG M-wave amplitude and area (both expressed as a percentage of the initial value) were significantly lower during hypoxia compared with normoxia (P < 0.05). In addition, GG and HG M-wave peak-to-peak and total duration (ms) were longer during hypoxia vs. normoxia (P < 0.05).

Figure 5 depicts protrudor and retractor muscle M-wave area and force data from individual animals during both normoxic and hypoxic fatigue trials. Each data point from the fatigue trial is included in this figure (i.e., M-wave and force data from the 0-, 1-, 2-, 3-, 4-, and 5-min points from all animals). For both protrudor and retractor muscles, M-wave and force are not correlated during normoxia. In contrast, a significant correlation between M-wave amplitude and force is present during hypoxia.

DISCUSSION

Summary. Our primary finding is that fatigue of tongue protrudor and retractor muscles in vivo, measured with a standard fatigue protocol, is significantly greater during systemic hypoxia compared with during normoxia. The influence of hypoxia on force and neural excitation of tongue muscles during a fatigue protocol was demonstrated by comparing performance on fatigue tests in hypoxia and normoxia in separate animals. EMG recordings from both tongue protrudor and retractor muscles demonstrate that hypoxia reduced M-wave peak-to-peak amplitude and area, whereas it increased M-wave duration.

Critique of methods. A detailed critique of the technique used to quantify tongue muscle force has been
published previously (9). One of our primary concerns is the validity of comparing fatigability in protrudor and retractor muscles. In our experimental preparation, the tongue retractor muscles contract against the force transducer, and, as a result, the contractions are isometric. Conversely, protrusion of the tongue unloads the force transducer, permitting the protrudor muscles to shorten during contraction. Thus, although the stimulus pattern was identical for protrudor and retractor muscles, the fatigue task was different. Because the mechanisms of muscle fatigue are highly task dependent (8), accurate comparisons between tongue protrudor and retractor muscle endurance performance cannot be made from the present data. Nevertheless, the fatigue task was the same within each muscle group across normoxic and hypoxic trials. Therefore, quantitative examination of the influence of hypoxia on the endurance performance of tongue protrudor and retractor muscles can be made with confidence.

A second methodological concern is in regard to the number of fatigue trials performed in each experimental animal. We chose to examine both tongue protrudor and retractor muscle fatigue in the same animal. Stimulating XIImed or XIIlat activates the tongue protrudor or retractor muscles in isolation of the other muscle group, and therefore fatigue in one muscle group should have a negligible influence on the other. Furthermore, the normoxic and hypoxic trials were performed in separate animals, and therefore a given

Table 2. GG and HG M-wave parameters during normoxic and hypoxic conditions

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<tr>
<th></th>
<th>GG</th>
<th>HG</th>
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<tr>
<td></td>
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<tr>
<td></td>
<td>0</td>
<td>1</td>
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<tr>
<td>P-P amplitude, %initial</td>
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<tr>
<td>Normoxia</td>
<td>100</td>
<td>89 ± 4</td>
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<tr>
<td>Hypoxia</td>
<td>100</td>
<td>77 ± 5</td>
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<tr>
<td>Area, %initial</td>
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</tr>
<tr>
<td>Normoxia</td>
<td>100</td>
<td>96 ± 5</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>100</td>
<td>100 ± 6</td>
</tr>
<tr>
<td>P-P duration, ms</td>
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<td></td>
</tr>
<tr>
<td>Normoxia</td>
<td>0.86 ± 0.07</td>
<td>0.90 ± 0.06</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>0.93 ± 0.03</td>
<td>1.43 ± 0.15</td>
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<tr>
<td>Total duration, ms</td>
<td></td>
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<tr>
<td>Normoxia</td>
<td>6.24 ± 0.64</td>
<td>6.68 ± 0.70</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>7.24 ± 0.52</td>
<td>8.96 ± 0.53</td>
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Values are means ± SE. GG, genioglossus; HG, hyoglossus; P-P, peak to peak. Although the majority of individual data points are not different between hypoxia and normoxia, the overall normoxic response was significantly different from the hypoxic response for all M-wave parameters, P < 0.05. *Different from time 0, P < 0.05; †hypoxic response different from corresponding normoxic data point, P < 0.05.
muscle group was not tested twice in the same animal. In this manner the confounding effects of performing more than one fatigue test on the same muscle group, in the same animal, were eliminated.

Lastly, the suitability of the Burke fatigue test for the rat should be discussed. The original Burke fatigue test (2, 3) was applied to motor units in a widely tested cat hindlimb muscle, the medial gastrocnemius. The Burke fatigue test on rat muscles having fast- and slow-twitch motor units that may have faster contraction times and, therefore, require higher stimulus frequencies to attain their peak force (4, 15, 16). The answer to this question pertains primarily to experimental strategy rather than physiological mechanisms. Totosy de Zepetnek et al. (29), for example, have used the Burke test on rat tibialis anterior muscle because it allowed them to compare their results both within and across a variety of different mammalian species. Similar reasoning led us, as well as others (see Refs. 6, 7, 19), to choose the Burke fatigue test.

Pharyngeal muscle endurance in hyperoxia and normoxia. Solomone and van Luteren (23) and Van Lunteren and colleagues (30–32) have examined the contractile and endurance properties of several pharyngeal muscles. Comparison of the endurance capabilities of the feline geniohyoid and diaphragm muscles with the Burke test in situ showed that the geniohyoid had much better endurance than the diaphragm [2-min fatigue index (FI) = 0.67 for the geniohyoid, and 0.15 for the diaphragm] (32). Further experiments from the same group of investigators examined the in vitro endurance capabilities of the GG, sternohyoid, sternothyroid, and diaphragm muscles with the Burke fatigue test (31). Consistent with prior data (32), the endurance capacity of the pharyngeal muscles was better than that of the diaphragm. Interestingly, of the three pharyngeal muscles studied, the GG had the greatest fatigue resistance. Gilliam and Goldberg (12) examined the in vivo fatigability of rat tongue muscles by stimulating XIImed or XIIlat using a modified Burke protocol. These investigators concluded that the tongue protruder and retractor muscles have good endurance properties relative to prior reports in rat limb muscle (2-min FI of 0.67 for the tongue retractor muscles; 0.76 for the tongue protruder muscles).

The data from the present study are consistent with previously published data (see above) indicating that the pharyngeal muscles have good endurance capabilities. Indeed, our data indicate that the tongue protruder and retractor muscles have a greater fatigue resistance than was reported previously by Gilliam and Goldberg (12). The 5-min FI reported here (Fig. 4) for both tongue protruder and retractor muscles exceeds previously reported values obtained after 2 min of repetitive stimulation (12). However, although the aforementioned experiments were performed in normoxia, rats were not mechanically ventilated and blood gases were not reported. Our experience is that rats under these conditions are mildly hypoxic (unpublished observations), and this may account for the observed differences in FI. In addition, XII nerve branches were stimulated at a higher rate in the Gilliam and Goldberg experiments (60 Hz) than in the present study (40 Hz), which likely contributed to the lower FI observed in their study. Nevertheless, a general conclusion that may be drawn from the present data, as well as previously published data, is that the pharyngeal musculature, in particular the tongue protruder and retractor muscles, has good endurance capabilities when studied by using in vitro and in vivo animal preparations.

Voluntary tongue protrusion tasks have been used to examine the endurance capabilities of the GG muscle in human subjects (22, 24). For example, Scardella et al. (24) examined GG endurance by having subjects protrude the tongue against a force transducer. Protrusions were maintained until a predetermined percentage of maximum force could no longer be sustained. Thoracic inspiratory muscle endurance was also examined to enable comparisons with tongue muscle endurance. In addition, inspiratory loading and hypercapnia were used to augment respiratory-related drive to the tongue and inspiratory muscles and to determine how this influenced the endurance performance of these...
In these experiments, tongue force declined more readily than did inspiratory pressure after inspiratory loading and hypercapnia, leading Scardella and colleagues to conclude that the human GG fatigues more readily than the thoracic inspiratory muscles. However, the interpretation of these studies may be confounded by the technique used to measure GG force. Protrusion of the tongue against an immovable force transducer requires stiffening of the body of the tongue and therefore contraction of the intrinsic tongue muscles (30). Thus the experiments of Scardella et al. probably examined intrinsic tongue muscle endurance as well as GG endurance. More studies are required to accurately describe the endurance capabilities of human tongue protrudor and retractor muscles.

Pharyngeal muscle endurance: influence of hypoxia. Hypoxia has previously been shown to adversely affect pharyngeal muscle endurance performance. Using an in situ canine preparation, Salomone and van Lunteren (23) examined the influence of hypoxia on geniohyoid muscle fatigue. The F1 of the geniohyoid muscle after 2 min of repetitive stimulation was attenuated by severe (PaO2 < 40 Torr) but not mild (PaO2 = 45–65 Torr) hypoxia. The results of the present study demonstrate for the first time that the endurance performance of both the tongue protrudor and retractor muscles is impaired by hypoxia (Figs. 4 and 5). The degree of hypoxia used in the present experiments (i.e., PaO2 ~50 Torr) did not, however, influence geniohyoid muscle endurance in the experiments of Salomone and van Lunteren (23). Therefore, our data indicate that, in the rat, the tongue protrudor and retractor muscles are more susceptible to hypoxia-induced fatigue than has been reported previously for other upper airway muscles.

How does hypoxia accelerate muscle fatigue? Metabolic changes within the tongue muscles probably contributed to increased tongue muscle fatigue during hypoxia. One possibility is that the O2 demand of the tongue muscles exceeded O2 supply during hypoxia. Certainly, if the amount of O2 present in a skeletal muscle fiber does not meet the demands of oxidative phosphorylation, muscle force production will be compromised. Alterations in intramuscular concentrations of ATP, ADP, Pi, and H+ can influence muscle fatigue (5), and changes in these parameters may be more prominent during hypoxia if O2 was limiting. Moreover, declines in muscle pH increase the rate of skeletal muscle fatigue (1), and intracellular muscle pH is lower during hypoxic than during mild hyperoxic exercise (21).

Our M-wave data (Fig. 5, Table 2) suggest that mechanisms acting proximal to the muscle contractile apparatus also contribute to the potentiation of fatigue by hypoxia. For example, protrudor and retractor muscle M-wave area was significantly less in the hypoxic compared with the normoxic trials (Table 2). Moreover, declines in M-wave area and force were significantly correlated during hypoxia (Fig. 5), suggesting that a portion of the force decrement observed in hypoxia can be attributed to the decline in EMG. Fuglevand (11) points out that decrements in EMG amplitude during fatiguing contractions (similar to that seen during hypoxia in the present experiments) can result from diminished sarcolemmal excitability, possibly due to fatigue-related changes in transmembrane electrolyte gradients. Recent data collected by Overgaard and colleagues (18) lend support to this hypothesis. These authors have performed a series of experiments by using an in vitro rat soleus muscle preparation in which motor nerves are stimulated and both contraction force and M-wave area are quantified. Experimentally altering Na+-K+ gradients by bathing muscles in 85 mM Na+ and 9 mM K+ buffer solution produced parallel declines in tetanic force and M-wave area (~50% decrease relative to control). Subsequently, stimulation of Na+-K+ transport with a β2-adrenergceptor agonist resulted in the recovery of both M-wave area and force. These experiments demonstrate that alterations in transmembrane Na+-K+ gradients can produce changes in muscle force and M-wave area that are qualitatively similar to those observed in the present in vivo studies during hypoxia. Our working hypothesis is that contraction induced alterations in transmembrane electrolyte gradients (11) were greater in hypoxia compared with normoxia, leading to diminished tongue muscle excitability and M-wave amplitude.

Physiological significance. Patients with obstructive sleep apnea (OSA) experience frequent episodes of pharyngeal airway obstruction during sleep (i.e., “apneic events”), with some episodes lasting 10 s or more. In severe cases, OSA patients can experience up to 600 apneic episodes per night (27). Significant arterial hypoxia occurs during each apneic episode (25), with the mean rate of decline in arterial oxyhemoglobin saturation ranging from 0.1 to 1.6%/s (28, 25). Termination of apneic events in OSA patients is accompanied by vigorous activation of pharyngeal muscles secondary to hypoxia and/or hypercapnia. Accordingly, Salomone and van Lunteren (23) suggest that the pharyngeal musculature may fatigue as a result of intermittent periods of intense activation during apnea-induced hypoxia. The present work establishes that the endurance capabilities of the tongue protrudor and retractor muscles are impaired by sustained hypoxia; however, the influence of intermittent hypoxia was not examined. We suggest that future investigations of tongue muscle endurance attempt to more accurately reproduce the conditions associated with OSA (e.g., by applying intermittent hypoxia and varying XII stimulus parameters). Such experiments will extend the current data and may provide information regarding the influence of tongue muscle fatigue on OSA pathology.

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