Increased ventilation does not impair maximal voluntary contractions of the elbow flexors

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Increased ventilation does not impair maximal voluntary contractions of the elbow flexors. J Appl Physiol 104: 1674–1682, 2008. First published April 10, 2008; doi:10.1152/japplphysiol.01358.2007.—Exercise performance is impaired by increased respiratory work, yet the mechanism for this is unclear. This experiment assessed whether neural drive to an exercising muscle was affected by cortically driven increases in ventilation. On each of 5 days, eight subjects completed a 2-min maximal voluntary contraction (MVC) of the elbow flexor muscles, followed by 4 min of recovery, while transtracical magnetic stimulation tested for suboptimal neural drive to the muscle. On 1 day, subjects breathed without instructions under normocapnia. During the 2-min MVC, ventilation was ~3.5 times that at rest. On another day, subjects breathed without instruction under hypercapnia. During the 2-min MVC, ventilation was ~1.5 times that on the normocapnic day. On another 2 days under normocapnia, subjects voluntarily matched their breathing to the uninstructed breathing under normocapnia and hypercapnia using target feedback of the rate and inspiratory volume. On a fifth day under normocapnia, the volume feedback was set to each subject’s vital capacity. On this day, ventilation during the 2-min MVC was approximately twice that on the uninstructed normocapnic day (or ~7 times rest). The experimental manipulations succeeded in producing voluntary and involuntary hyperpnea. However, maximal voluntary force, fatigue and voluntary activation of the elbow flexor muscles were unaffected by cortically or chemically driven increases in ventilation. Results suggest that any effects of increased respiratory work on limb exercise performance are not due to a failure to drive both muscle groups optimally.

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brain stem by reflex-mediated chemical drive. Typically, voluntary activation falls progressively during a sustained maximal contraction. Subjects show central fatigue and are unable to maintain their initial level of drive to the muscle (15). Thus the performance of a sustained maximal contraction should be sensitive to any added impairments of drive related to simultaneous performance of respiratory tasks. Our hypothesis was that high levels of voluntary ventilation would impair subjects’ ability to drive the elbow flexor muscles maximally during a fatiguing contraction.

**METHODS**

**Participants.** Eight healthy adult subjects (aged 23–47 yr, 5 women; 1.63–1.89 m; 61–85 kg) completed the experiment. Subjects varied from sedentary to exercising 4 times/wk. All subjects gave their written informed consent, and all experimental procedures were approved by the local ethics committee and were conducted according to the Declaration of Helsinki.

**Setup.** Subjects sat with the right arm flexed to 90° in an isometric myograph that measured flexion torque at the elbow [transducer linear to 2 kN, X-tran, Melbourne, Australia; (2)]. The forearm was vertical and supinated, and it was strapped to the myograph just proximal to the wrist. Feedback of flexion torque was provided to the subject by a light-emitting diode (LED) display.

Subjects were connected via a mouthpiece to a partial rebreathing circuit for controlling end-tidal CO2. A reservoir bag was placed in the circuit, as well as a manually operated valve, which allowed fresh (room) air to enter the circuit and which was controlled by an experimenter monitoring the end-tidal CO2 (Normocap CO2 monitor, Datex, Helsinki, Finland). O2 saturation was monitored with a pulse oximetry probe (Biox 3740 pulse oximeter, Ohmeda, Louisville, CO) on the left middle finger, and remained >95% throughout in all subjects. A second LED display provided target and feedback information on ventilation. The mouthpiece was connected via a two-way valve with the inspiratory port connected to a pneumotachometer (Hans Rudolph, Kansas City, MO). The flow signal was integrated to give inspired volume.

Surface electromyograms (EMG) were recorded with electrodes (Ag-AgCl, 10-mm diameter) fixed to the skin (cleaned with alcohol and mild abrasion) overlying the muscle bellies of biceps brachii, brachioradialis, and triceps brachii. Surface signals were amplified (300–1,000 times) and filtered (16–1,000 Hz; CED 1902 amplifiers, Cambridge Electronic Design, Cambridge, UK). Force and EMG signals were sampled at 2,000 Hz through a laboratory interface for offline analysis (CED 1401 interface, Spike 2 software, Cambridge Electronic Design).

**Brachial plexus stimulation.** Single electrical stimuli were delivered to the brachial plexus via a cathode in the supraclavicular fossa (Erb’s point) and an anode on the acromion (100-µs duration, constant current, DS7AH, Digitimer, Welwyn Garden City, UK). In each experimental session, stimulus intensity was gradually increased until no further increase was observed in the resting compound muscle action potential (M wave) of biceps brachii, brachioradialis, and triceps brachii muscles. Stimulus intensity was set at 50% above this action potential (M wave) of biceps brachii, brachioradialis, and no further increase was observed in the resting compound muscle potential (M wave) of biceps brachii, brachioradialis, and triceps brachii muscles. Stimulator output (47–75% of maximum) was set during brief maximal voluntary contractions (MVCs) to obtain a large MEP in the biceps brachii (>60% of Mmax) and a small MEP in the triceps (<20% of Mmax) (45). Stimulus intensity was set in the first experimental session for each subject and remained constant throughout the study.

**Experimental protocol.** Subjects completed similar experimental protocols on 5 separate days at least 3 days apart. On each day, subjects initially performed six sets of 3 brief contractions. Sets comprised a brief (2–3 s) MVC of the elbow flexor muscles, with motor cortical and brachial plexus stimulation delivered during the MVC (see Fig. 1A), followed at 8-s intervals by contractions to 75 and 50% MVC, with motor cortical stimulation during each contraction (these submaximal contractions were used to obtain an estimate of the resting twitch, see below). During the first three control sets of contractions, subjects breathed room air, while for the second three sets subjects breathed through the mouthpiece according to their assigned ventilation protocol (see below). Sets of contractions were separated by intervals of at least 1 min to minimize fatigue. Subjects then performed a 2-min MVC, with motor cortical and brachial plexus stimulation delivered 5 s apart, every 20 s. Brief MVCs with motor cortical and brachial plexus stimulation were performed at 30 s and 1, 2, 3, and 4 min after the end of the 2-min MVC. Each of these MVCs was followed by contractions to 75 and 50% MVC with cortical stimulation. During the fatiguing contraction and the recovery period, subjects continued to breathe through the mouthpiece according to their assigned ventilation protocol.

Subjects followed different ventilation protocols on each of the 5 days. On day 1 (“Control”), subjects were allowed to breathe normally (i.e., with no breathing instructions, other than not to hold their breath), and the valve on the breathing circuit was fully open to allow fresh room air to enter. CO2 was not experimentally manipulated (see Fig. 1B).

On the second day (“Matched Control”), subjects matched their breathing to that from the first day. Targets of inspiratory volume, inspiratory time and expiratory time were provided through a LED display which showed volume as increasing lights during inspiration. Three separate targets were set during the experiment. These were derived from each subject’s mean data measured separately during the control period, the 2-min MVC and the recovery period on the Control day. CO2 was maintained at levels similarly calculated for each period. Thus, mimicking the Control day, CO2 was allowed to fall during the 2-min MVC.

On the third day (“CO2-driven Hyperpnea”), subjects again breathed under no instructions, but end-tidal PCO2 was maintained at a high level. This level (41–54 Torr) was set for each subject by allowing end-tidal CO2 to rise by rebreathing until ventilation was increased to two to three times their ventilation during the day 1 control period. The same level of end-tidal CO2 was maintained throughout the control MVCs, 2-min MVC, and recovery.

On the fourth day (“Voluntary Hyperpnea”), subjects matched their breathing to the CO2-driven Hyperpnea day with targets provided for inspiratory volume, inspiratory time, and expiratory time but with end-tidal CO2 matched to Control day levels. Ventilation and CO2 were separately matched for control MVCs, 2-min MVC, and during recovery.

On the fifth day (“Maximal Voluntary Hyperpnea”), subjects matched their breathing to feedback which used their vital capacity as the target inspiratory volume and the inspiratory and expiratory times derived from CO2-driven Hyperpnea. End-tidal CO2 was matched to Control day levels. Subjects were instructed that if they could not match this feedback, they were to focus on achieving the tidal volume as fast as possible, rather than matching the rate.

**Data extraction.** Mean elbow flexion torque was calculated over 200 ms before each motor cortical stimulus and normalized to the mouthpiece control MVCs on each day. Increments in force evoked by motor cortical stimulation during all contractions were measured and normalized as a percentage of the ongoing MVC force. Voluntary activation during the brief control MVCs was calculated.
using the following formula: voluntary activation = [1 – (superimposed twitch/estimated resting twitch)] × 100. The y-intercept of a linear regression between superimposed twitch amplitude and voluntary force for each set of three contractions (50, 75, and 100% MVC) was used to estimate the resting twitch (45, 46). An estimated resting twitch was used because motor cortical neurons and motoneurons are more excitable during voluntary contraction. Thus cortical stimulation during rest does not activate the same motor units as cortical stimulation during contraction and does not produce a comparable twitch (45). For each muscle, the size of MEPs following motor cortical stimulation and the M_max evoked by brachial plexus stimulation were measured as the area under the curve between set cursors which encompassed each potential. The area of the MEP in each muscle was measured as the area under the curve between set cursors which encompassed each potential. The area of the MEP in each muscle was measured as the area under the curve between set cursors, targeting each potential.

Statistical analysis. Minute ventilation measures were entered into a repeated-measures two-way ANOVA with the within-subject factors day and time. Only main effects are reported here. Separate analyses considered the 2-min MVC and the recovery periods, with polynomial contrasts on the time factor (although only linear and quadratic trends are considered in this paper). Four single degree of freedom planned contrasts on the day factor were performed. To assess whether subjects adequately matched their target feedback, the Control day was compared with the Matched Control day, and the CO_2-driven Hyperpnea day (day 3) was compared with the Voluntary Hyperpnea day (day 4). To assess whether ventilation was significantly increased under the hyperpnea conditions, the mean of the control days was compared with the mean of the hyperpnea days (days 3 and 4). Last, to assess whether ventilation was further increased under the maximal ventilation condition, CO_2-driven Hyperpnea was compared with Maximal Voluntary Hyperpnea.

Similarly, MVC force, superimposed twitch, and the EMG-derived contrasts, with only main effects reported. Additionally, a two-way ANOVA was performed comparing the values off and on the mouthpiece during the control periods. Identical contrasts were performed on the day factor to assess whether the changes in breathing significantly affected MVC performance.

CO_2 was also entered into a two-way (day × time) ANOVA, with linear and quadratic trends on the time factor. However, a different approach to the day factor was used: because the end-tidal CO_2 was expected to be the same except for during the CO_2-driven Hyperpnea, repeated contrasts were used. That is, each normocapnic day was
compared with the subsequent normocapnic day (the Control day was compared with the Matched Control day, Matched Control with the Voluntary Hyperpnea, and the Voluntary Hyperpnea with the Maximal Voluntary Hyperpnea day), and the CO$_2$-driven Hyperpnea day was compared with the Control day.

All contrasts have (1, 7) degrees of freedom. As the contrasts were planned and there were no more of them than the degrees of freedom for effect, no Bonferroni-type adjustment to alpha was necessary (41).

**RESULTS**

We first consider the success of our experimental manipulations on producing different levels of voluntary and involuntary ventilation, and then we consider the effect this had on performance of the 2-min MVC of the elbow flexors and associated EMG measures.

**Minute ventilation.** Minute ventilation increased to ~3.5 times control levels as soon as the 2-min MVC began (see Fig. 2A; Table 1), mainly due to a rise in breathing rate, but showed no further reliable increase over the sustained contraction (linear $F < 1$, $P = 0.631$). Minute ventilation was well matched between the Control day and the Matched Control day ($F < 1$, $P = 0.856$). The increase from rest to during the sustained MVC was from 12.6 ± 3.0 to 45.5 ± 12.2 l/min (mean ± SD) on the Control day and from 12.6 ± 2.3 to 46.3 ± 13.2 l/min on the Matched Control day. Ventilation was significantly increased for hyperpnea days compared with control days (~1.5 times during the 2-min MVC), mainly due to an increase in inspiratory volume ($F = 15.8$, $P = 0.005$), but it was lower for the Voluntary Hyperpnea compared with CO$_2$-driven Hyperpnea ($F = 14.2$, $P = 0.007$), due to the failure of subjects to reach the target volume during Voluntary Hyperpnea. With CO$_2$-driven Hyperpnea, minute ventilation went from 27.3 ± 7.0 l/min at rest to 64.8 ± 13.3 l/min during the 2-min MVC, whereas with Voluntary Hyperpnea it went from 25.4 ± 7.0 to 57.6 ± 11.2 l/min. As intended, ventilation was further increased for Maximal Voluntary Hyperpnea (55.6 ± 14.5 l/min at rest and 87.6 ± 13.0 l/min during the 2-min MVC) compared with CO$_2$-driven Hyperpnea ($F = 12.9$, $P = 0.009$), with a slightly slower rate (88 ± 12%) but much higher tidal volume (158 ± 34%). Hence, during Maximal Voluntary Hyperpnea, ventilation during the 2-min MVC was approximately double that on the control day (that is, ~7 times normal breathing at rest).

Ventilation declined toward control levels with the end of the sustained contraction, and then it remained stable at approximately the level of the control periods (linear $F = 14.0$, $P = 0.007$; quadratic $F = 12.1$, $P = 0.010$). Because constant target levels were set for the duration of the recovery period, matching of ventilation was poor initially (Control vs. Matched Control: $F = 153$, $P = 0.006$; CO$_2$-driven Hyperpnea vs. Voluntary Hyperpnea: $F = 101.9$, $P < 0.001$). However, ventilation was increased for the hyperpnea compared with control days ($F = 24.8$, $P = 0.002$), and it further increased for Maximal Voluntary Hyperpnea ($F = 27.1$, $P = 0.001$).

CO$_2$. To assess the chemical drive leading to hyperpnea, it was necessary to consider CO$_2$ levels during the experiment. These are displayed in Fig. 2B. Although end-tidal CO$_2$ dropped as soon as the 2-min MVC began, there were no significant changes in expired-CO$_2$ levels over the 2-min contraction (linear and quadratic $F$ both < 1). As intended, expired CO$_2$ levels were significantly increased for CO$_2$-driven Hyperpnea compared with the Control day ($F = 157.1$, $P < 0.001$), but there were no significant differences between the normocapnic days (all $P > 0.05$).

A similar pattern of results was observed in recovery. There were no reliable changes in CO$_2$ over time (linear and quadratic $F$ both < 1). As planned, CO$_2$ levels were significantly higher on the CO$_2$-driven Hyperpnea day ($F = 95.6$, $P <$
ELBOW FLEXOR FORCE UNAFFECTED BY INCREASED VENTILATION

Table 1. Breathing variables on each day

<table>
<thead>
<tr>
<th></th>
<th>Day 1 (Control)</th>
<th>Day 2 (Matched Control)</th>
<th>Day 3 (CO2-driven Hyperpnea)</th>
<th>Day 4 (Voluntary Hyperpnea)</th>
<th>Day 5 (Maximal Voluntary Hyperpnea)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ventilation</strong></td>
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<td></td>
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<tr>
<td>l/min</td>
<td>12.6 ± 3.0</td>
<td>12.6 ± 2.3</td>
<td>27.3 ± 7.0</td>
<td>25.4 ± 6.9</td>
<td>55.6 ± 14.5</td>
</tr>
<tr>
<td>%MVV</td>
<td>11 ± 3</td>
<td>11 ± 2</td>
<td>24 ± 8</td>
<td>23 ± 7</td>
<td>49 ± 13</td>
</tr>
<tr>
<td><strong>Vt</strong></td>
<td></td>
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<tr>
<td>liters</td>
<td>0.92 ± 0.30</td>
<td>0.93 ± 0.29</td>
<td>1.68 ± 0.34</td>
<td>1.56 ± 0.33</td>
<td>3.24 ± 0.65</td>
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<tr>
<td>%VC</td>
<td>23 ± 6</td>
<td>24 ± 5</td>
<td>44 ± 10</td>
<td>40 ± 9</td>
<td>82 ± 6</td>
</tr>
<tr>
<td><strong>Ti, s</strong></td>
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<td></td>
<td>1.52 ± 0.37</td>
<td>1.39 ± 0.32</td>
<td>1.55 ± 0.30</td>
<td>1.45 ± 0.26</td>
<td>1.46 ± 0.31</td>
</tr>
<tr>
<td><strong>Te, s</strong></td>
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<tr>
<td></td>
<td>2.95 ± 0.66</td>
<td>3.05 ± 0.77</td>
<td>2.29 ± 0.64</td>
<td>2.38 ± 0.73</td>
<td>2.20 ± 0.79</td>
</tr>
<tr>
<td><strong>Frequency</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>breaths/min</td>
<td>14.3 ± 3.0</td>
<td>14.2 ± 3.2</td>
<td>16.5 ± 4.1</td>
<td>16.5 ± 4.1</td>
<td>17.5 ± 4.4</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Data are for ventilation expressed also as a percentage of maximum voluntary ventilation (%MVV), tidal volume (Vt) expressed also as a percentage of vital capacity (%VC), inspiratory time (Ti), expiratory time (Te), and frequency of breathing, measured at rest and across the 2-min maximal contraction of elbow flexors on each experimental day (days 1–5) where ventilation was varied. Ventilation was significantly higher on days 1 and 2 (P = 0.005), on day 3 compared with day 4 (P = 0.007), and on day 5 compared with day 3 (P = 0.009).

During rest between brief control contractions of elbow flexor muscles on each day

During the 2-min sustained maximal contraction of elbow flexor muscles on each day

0.001), and they were not significantly different between the normocapnic days (all P > 0.05).

MVC force. MVC force (as a percentage of control MVCs on each day) is displayed in Fig. 3A. There were no significant differences between MVCs performed off and on the mouthpiece (F = 3.9, P = 0.089), and this result was the same over all days (all P > 0.101). MVC force decreased quickly over the first minute and then more slowly in the second minute of the sustained MVC (linear F = 526.1, P < 0.001; quadratic F = 164.839, P < 0.001). There were no differences between days (all P > 0.170).

During recovery, the MVC force increased steadily (linear F = 80.8, P < 0.001) and in a similar way on most days, although recovery forces were significantly higher on the Matched Control day compared with the Control day (F = 6.9, P = 0.034).
Superimposed twitches. The amplitude of the superimposed twitches evoked by cortical stimulation during MVCs throughout the experimental protocol are displayed in Fig. 3B for the group and in Fig. 4 for a single subject for days 1 and 5 (where ventilation was most disparate). There were no differences in the superimposed twitches or voluntary activation during brief control elbow flexor MVCs when subjects were breathing with or without a mouthpiece. Overall, voluntary activation in the control MVCs during normal breathing was 91 ± 2.9% (mean ± SD) and when breathing through the mouthpiece as dictated by the various protocols, 91 ± 3.0%. The superimposed twitch increased over the sustained maximal contraction (linear \( F = 29.9, P = 0.001 \); quadratic \( F = 30.3, P = 0.001 \)). There were no significant main effects for the different days (all \( P > 0.336 \)).

The superimposed twitch recovered almost to baseline levels within 30 s of the end of the 2-min MVC, and it continued to decline thereafter (linear \( F = 9.9, P = 0.016 \)). No main effects for the days factor reached significance (all \( P > 0.116 \)).

**EMG measures.** The EMG measures assessed during the protocols changed as expected during the 2-min MVC and recovery period (42, 44). Some isolated statistically significant differences between breathing conditions were found for some measures.

The amplitude of RMS EMG in biceps and brachioradialis decreased during the sustained MVCs and increased in the recovery period. Two comparisons between days of RMS EMG during the 2-min MVC were significant for individual muscles (Control > Matched Control for biceps, \( F = 15.6, P = 0.006 \); \( CO_2 \)-driven Hyperpnea > Voluntary Hyperpnea in brachioradialis, \( F = 6.9, P = 0.034 \)). As these differences each occurred in only one of the elbow flexors and did not result in changes in force, their physiological significance is doubtful.

MEPs in biceps and brachioradialis showed no significant differences between the five breathing protocols. Both \( M_{max} \) and the MEP increased during the 2-min MVC (all linear and quadratic \( P < 0.05 \)). At the end of the sustained contraction the area of \( M_{max} \) in biceps and brachioradialis averaged 156 ± 17 and 142 ± 22% of control values, respectively. However, the increase in the MEP (biceps to 178 ± 42% and brachioradialis to 172 ± 40% of control) was greater, such that when the MEP was normalized to \( M_{max} \) elicited close in time, the MEP still grew significantly during the sustained effort (biceps, quadratic \( F = 8.5, P = 0.022 \); brachioradialis, quadratic \( F = 13.0, P = 0.009 \)). The MEPs in both muscles recovered to baseline levels within 30 s of the end of the sustained contraction. The silent period lengthened during the 2-min contraction by an average of 47 ± 30 ms in biceps (linear \( F = 7.8, P = 0.027 \); quadratic \( F = 6.0, P = 0.044 \)) and 54 ± 33 ms in brachioradialis (\( F = 1.4, P > 0.05 \)), and it recovered quickly in both muscles. The increase in duration of the silent period in biceps but not brachioradialis was greater on the Control than Matched Control day (\( F = 7.3, P = 0.031 \)). There were no other significant effects of day.

**DISCUSSION**

This study shows that performance of a 2-min sustained isometric maximal contraction of the elbow flexors is not affected by increased voluntary activity of the respiratory muscles. In each experiment, during the 2-min sustained MVC of the elbow flexors voluntary force fell, indicating the development of fatigue. At the same time, the superimposed twitch evoked by cortical stimulation increased in amplitude, indicating a progressive failure of voluntary activation that can be attributed to suboptimal output from the motor cortex (15, 45). By definition, the fall in voluntary activation demonstrates the development of central fatigue (14). However, neither voluntary force nor voluntary activation of the elbow flexors was further reduced when subjects made high respiratory efforts. Peripheral and central fatigue of the elbow flexors developed in a similar way whether subjects breathed normally or hyperventilated, and whether subjects were normocapnic or hypercapnic. Our results indicate that high levels of voluntary or involuntary drive to the respiratory muscles do not impair drive to the limb muscles. This is in contradiction to our hypothesis.
that high levels of voluntary drive to the respiratory muscles would impair arm muscle performance because subjects would be unable to direct sufficient motor cortical drive to both muscle groups simultaneously, as has been described with bilateral contractions of limb muscles (26, 34, 48).

Subjects performed four different ventilation protocols to test various possible mechanisms that might have impaired limb muscle performance compared with performance when breathing was not controlled. First, subjects were required to use visual feedback to follow targets of respiratory volume and timing. Here, we expected that attention would be required to follow the feedback and that the origin of the neural drive to the respiratory muscles would be altered from largely automatic to voluntary, and thus this would require additional output from the motor cortex (9, 12, 28, 31). Second, subjects were exposed to high CO₂ but given no breathing instructions. The resulting chemically driven increase in ventilation should occur through drive to the motoneurons of the respiratory muscles from centers in the medulla (39). It should not require attention or output from the motor cortex to drive respiration (9, 28, 31), although sensations related to breathing will increase. Third, subjects used feedback to match the hypercapnic hyperpnea while end-tidal CO₂ was held at a normal level. This task required attention and high levels of output from the motor cortex to respiratory muscles. We expected that this would result in a deficit in drive from the motor cortex to the arm muscles. Finally, when no deficits in arm performance were seen with the matched voluntary hyperpnea, subjects were set ventilatory targets with high rates and a volume equivalent to their vital capacity. Thus this breathing task required close to maximal efforts in inspiration and expiration. A slight decline in minute ventilation over the 2-min MVC was due to a steady decrease in inspiratory volume, and this may indicate some fatigue of the respiratory muscles (7, 17, 18). Indeed, subjects complained of sore abdominal (expiratory) muscles at the conclusion of the experiment. However, the combination of near-maximal respiratory efforts with a fatiguing sustained contractile effort would impair arm muscle performance because they can be driven by both the cortex and/or the brain stem.

One methodological consideration is that voluntary activation of the elbow flexor muscles was measured using cortical stimulation rather than peripheral nerve stimulation (43, 45). Using cortical stimulation to measure voluntary activation reveals whether, at the moment of stimulation, there is motor cortical output that is untapped by voluntary effort but available for recruitment by the stimulus and that can produce more force from the muscle. If all available cortical output were engaged voluntarily but this were not enough to drive the muscle maximally, then voluntary activation could be high as measured by cortical stimulation but low if measured by peripheral nerve stimulation (a test of whether the muscle is driven fully). Thus it would be possible for drive to the muscle to be impaired despite unimpaired voluntary activation as measured with cortical stimulation. However, it is unlikely that this occurred in the present study because, like voluntary activation, subjects’ maximal voluntary force was not altered by any of the breathing tasks.

A second consideration is whether the voluntary hyperpnea was truly voluntary. A large increase in ventilation occurred at the start of the 2-min MVC in every condition, mostly due to a tripling of breathing rate. It is under debate whether this exercise hyperpnea is due to a central feed-forward command to the cardiorespiratory centers in parallel with the descending command to the motoneurons, or toafferent feedback, for example, from metabolites and changes in muscle tension (e.g., Refs. 19, 51). The instantaneous onset of the exercise hyperpnea favors the former interpretation, although both central feed-forward and afferent feedback play a role in ongoing exercise. On the first day when subjects’ breathing was not controlled, ventilation increased approximately threefold at the start of the 2-min MVC. A similar absolute increase occurred when subjects were hypercapnic but could otherwise breathe freely. Thus, when subjects were required to hyperventilate voluntarily, it is likely that this was assisted during the 2-min MVC by an “automatic,” exercise-related drive. This was reflected in reports from some subjects that matching the hyperpnea targets became less effortful when the 2-min MVC started, despite the target ventilation itself also increasing. However, subjects’ ventilation when attempting target volumes of vital capacity was approximately twice that of uncontrolled breathing so that, at least for this condition, a large proportion of drive to the respiratory muscles remained subject to volition.

Our results differ from studies that show impaired exercise performance following periods of increased respiratory work (e.g., Refs. 8, 10, 20, 21, 37). These studies suggest that impaired performance results from decreased blood flow to the exercising muscle and that blood flow is decreased by a sympathetic reflex response generated by fatigue of the diaphragm (8, 10, 20, 21, 37). Fatigue of the diaphragm was not an aim of our study, and the short period of respiratory work probably did not fatigue this resistant muscle (16, 32). In addition, the type of limb exercise performed in our study
differed from the experiments reporting a large effect of respiratory work. Most previous studies have looked at dynamic exercise in the legs (cycling or running), in contrast to a static upper limb contraction. The effect of respiratory work on dynamic arm exercise is unclear. Volianitis et al. (50) have reported improved rowing performance after a period of inspiratory muscle training, whereas van Houtte et al. (49) reported no change in the endurance time of arm cranking exercise following a period of isocapnic hyperventilation. Furthermore, if the limit to performance is caused by blood flow interactions, the maximal isometric contraction of our study will have minimized any effect because intramuscular pressure makes such sustained contractions virtually ischemic (36, 53).

In summary, the study aimed to show that an impairment of exercise performance arose due to an inability to direct neural drive to the exercising muscles and the respiratory muscles simultaneously. In contrast to expectations, cortically driven breathing had no effect on MVC force or voluntary activation at low, medium, and near-maximal levels of respiratory work.

GRANTS
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REFERENCES
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