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RESEARCH PAPER

Mechanical-ventilatory responses to peak and ventilation-matched upper- versus lower-body exercise in normal subjects

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Abstract

The aim of this study was to determine the extent to which the mechanical ventilatory responses to upper-body exercise are influenced by task-specific locomotor mechanics. Eight healthy men (mean \pm SD: age, 24 ± 5 years; mass, 74 ± 11 kg; and stature, 1.79 ± 0.07 m) completed two maximal exercise tests, on separate days, comprising 4 min stepwise increments of 15 W during upper-body exercise (arm-cranking) or 30 W during lower-body exercise (leg-cycling). The tests were repeated at work rates calculated to elicit 20, 40, 60, 80 and 100% of the peak ventilation achieved during arm-cranking ($\dot{V}_{E,UBE}$). Exercise measures included pulmonary ventilation and gas exchange, oesophageal pressure-derived indices of respiratory mechanics, operating lung volumes and expiratory flow limitation. Subjects exhibited normal resting pulmonary function. Arm-crank exercise elicited significantly lower peak values for work rate, O_2 uptake, CO_2 output, minute ventilation and tidal volume ($p < 0.05$). At matched ventilations, arm-crank exercise restricted tidal volume expansion relative to leg-cycling exercise at 60% $\dot{V}_{E,UBE}$ (1.74 ± 0.61 versus 2.27 ± 0.68 l, $p < 0.001$), 80% $\dot{V}_{E,UBE}$ (2.07 ± 0.70 versus 2.52 ± 0.67 l, $p < 0.001$) and 100% $\dot{V}_{E,UBE}$ (1.97 ± 0.85 versus 2.55 ± 0.72 l, $p = 0.002$). Despite minimal evidence of expiratory flow limitation, expiratory reserve volume was significantly higher during arm-cranking versus leg-cycling exercise at 100% $\dot{V}_{E,UBE}$ (39 ± 8 versus $29 \pm 8\%$ of vital capacity, $p = 0.002$). At any given ventilation, arm-cranking elicited greater inspiratory effort (oesophageal pressure) relative to thoracic displacement (tidal volume). Arm-cranking exercise is sufficient to provoke respiratory mechanical derangements (restricted tidal volume expansion, dynamic hyperinflation and neuromechanical uncoupling) in subjects with normal pulmonary function and expiratory flow reserve. These responses are likely to be attributable to task-specific locomotor mechanics (i.e. non-respiratory loading of the thorax).

KEYWORDS

airflow limitation, arm exercise, arm-crank ergometry, respiratory mechanics, upper-body exercise

1 | INTRODUCTION

During dynamic whole-body exercise (e.g. cycling, running), the increase in pulmonary ventilation is achieved, in part, by the progressive recruitment of expiratory muscles to reduce end-expiratory lung volume (EELV) below functional residual capacity (Abraham et al., 2002; Lind & Hesser, 1984). This reduction in EELV provides several mechanical advantages. First, it improves the

length-tension relationship of the diaphragm in order that more pressure can be generated for a given neural drive (Smith & Bellemare, 1987). Second, it assists inspiration by facilitating passive recoil of the chest and abdominal wall (Aliverti et al., 1997). Third, a decrease in EELV permits a substantial increase in tidal volume without encroaching on the non-linear upper portion of the pressure-volume relationship (Henke, Sharratt, Pegelow, & Dempsey, 1988). At ventilations approaching maximum, EELV may increase towards

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or above resting values; a phenomenon termed dynamic lung hyperinflation. This compensatory mechanism may alleviate limitations to expiratory flow by decreasing airway resistance (Cormier, Laviolette, Atton, & Series, 1991). Nevertheless, dynamic hyperinflation has several consequences, which include: functional inspiratory muscle weakness; increased elastic and threshold loading on the inspiratory muscles, with concomitant increases in the work and O_2 cost of breathing; mechanical restriction of tidal volume expansion; and adverse effects on cardiocirculatory function (Sheel & Romer, 2012). Moreover, there is a growing body of evidence that the resulting increase in neural respiratory drive contributes to dyspnoea and exercise intolerance (Sheel, Foster, & Romer, 2011).

Exercise primarily comprising the upper body forces the thoracic musculature to assume multiple non-respiratory functions, including stiffening the spine (Hodges, Eriksson, Shirley, & Gandevia, 2005), maintaining torso stabilization (Celli, Criner, & Rassulo, 1988) and positioning the arms (Hodges & Gandevia, 2000). Consequently, the respiratory muscle contribution to breathing may be compromised, resulting in a relative inability to reduce EELV below relaxation volume. Given that the muscles involved in moving the arms and stabilizing the trunk attach to the ribcage, upper-body exercise would also be expected to increase chest-wall impedance, constrain tidal volume and necessitate an increase in respiratory frequency to achieve a given level of ventilation (Takano, 1993). Maximal upper-body exercise has been shown to elicit dynamic hyperinflation in the absence of expiratory flow limitation in athletes with high-lesion (cervical) spinal cord injury (Taylor, West, & Romer, 2010; West, Goosey-Tolfrey, Campbell, & Romer, 2014). Notwithstanding, individuals with high-lesion spinal cord injury present with derangements in respiratory mechanics and profound weakness of the expiratory muscles (Taylor et al., 2010; West et al., 2014). As such, it is unclear whether the hyperinflation noted during upper-body exercise is attributable to the physiological consequences of spinal cord injury, the locomotor mechanics of upper-body exercise, or both. More recently, we have observed dynamic hyperinflation in healthy, able-bodied subjects performing severe-intensity upper-body (arm-cranking) exercise, but no such hyperinflation during heavy exercise (Tiller, Campbell, & Romer, 2017a). Given that ventilation was greater during severe exercise, it was not possible to discern to what extent the hyperinflation during upper-body exercise was attributable to expiratory flow limitation or task-specific locomotor mechanics (i.e. non-respiratory loading of the thorax).

To explore this further, it would be necessary to compare the mechanical ventilatory responses to upper- and lower-body exercise at the same ventilation, yet only two studies have attempted this assessment in normal subjects (i.e. those free from physical impairment or cardiorespiratory disease). Alison et al. (1998) reported a relative inability of their normal subjects to reduce EELV below resting values during arm-cranking relative to leg-cycling exercise, at peak work rates and at fixed percentages of peak ventilation. In contrast, Cerny & Ucer (2004) reported greater decreases in EELV below resting values during arm-cranking relative to leg-cycling exercise at matched levels of ventilation. Both studies assessed dynamic hyperinflation using the flow-volume technique (Johnson, Weisman, Zeballos, & Beck,

New Findings

- **What is the central question of this study?**

To what extent are the mechanical-ventilatory responses to upper-body exercise influenced by task-specific locomotor mechanics?

- **What is the main finding and its importance?**

When compared with lower-body exercise performed at similar ventilations, upper-body exercise was characterized by tidal volume constraint, dynamic lung hyperinflation and an increased propensity towards neuromechanical uncoupling of the respiratory system. Importantly, these responses were independent of respiratory dysfunction and flow limitation. Thus, the mechanical ventilatory responses to upper-body exercise are attributable, in part, to task-specific locomotor mechanics (i.e. non-respiratory loading of the thorax).

1999), which is contingent on the accurate measurement of inspiratory capacity (IC) to track operational changes in EELV (Guenette, Chin, Cory, Webb, & O'Donnell, 2013). Neither study, however, assessed peak inspiratory (oesophageal) pressure during the IC manoeuvre to ensure that submaximal inspiration did not artificially inflate EELV and thereby overestimate the extent of dynamic hyperinflation. A further concern is that neither study quantified the magnitude of expiratory flow limitation at equivalent ventilations. Thus, the mechanisms that underpin hyperinflation during upper-body exercise require clarification. Such data could have implications for patients who hyperinflate during exercise (e.g. those with obstructive lung disease) and for individuals who participate in activities involving the upper limbs (e.g. kayaking, rowing, wheelchair racing).

The aim of this study, therefore, was to characterize the mechanical ventilatory responses to peak and ventilation-matched upper- versus lower-body exercise in normal subjects. Specifically, we sought to: (i) compare operating lung volumes between arm-cranking and leg-cycling exercise; and (ii) compare the prevalence and magnitude of expiratory flow limitation between the exercise modes. It was hypothesized that, compared with leg-cycling, arm-cranking exercise would elicit dynamic hyperinflation in the absence of flow limitation.

2 | METHODS

2.1 | Ethical approval

The study was approved by Brunel University London Research Ethics Committee (RE34-10) and conformed to the standards set by the *Declaration of Helsinki*, except for registration in a database. Before data collection, subjects were issued with an information document, completed a pre-test medical questionnaire and provided written, informed consent.

2.2 | Subjects

Eight healthy, non-smoking, recreationally active men volunteered to participate. Subjects abstained from intense exercise for 48 h, alcohol and caffeine for 12 h, and food for 3 h before testing.

2.3 | Experimental overview

The study followed a randomized, counterbalanced design. All subjects completed four maximal incremental exercise tests, each separated by ≥ 48 h. The tests were performed at the same time of day in stable laboratory conditions. The purpose of test 1 and test 2 was to compare mechanical ventilatory responses between peak upper-body (arm-cranking) exercise and peak lower-body (leg-cycling) exercise after increments of absolute work rate and to establish exercise intensities for the subsequent tests. The purpose of test 3 and test 4 was to compare mechanical ventilatory responses between upper- and lower-body exercise modes across ventilation-matched work rates.

2.4 | Pulmonary function tests

Forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were determined using spirometry performed at rest in the seated position (Miller et al., 2005). The test consisted of a series of forced expiratory manoeuvres performed into a low-resistance, bidirectional turbine connected to an online system (Oxycon Pro; Jaeger GmbH, Hoechberg, Germany). Maximal static inspiratory pressure ($P_{I_{max}}$) from residual volume (RV) and maximal static expiratory pressure ($P_{E_{max}}$) from total lung capacity (TLC) were measured through the side-port of a semi-occluded mouthpiece using a linear differential transducer (DP45; Validyne, Northridge, CA, USA; range, ± 229 cmH₂O) (Evans & Whitelaw, 2009). All values were expressed in absolute units and as percentages of predicted normal (Evans & Whitelaw, 2009; Quanjer et al., 2012).

2.5 | Incremental exercise tests

Upper-body exercise was performed in the upright position using an electromagnetically braked arm-crank ergometer (Angio; Lode, Groningen, The Netherlands). The ergometer was mounted to a wall and positioned so that the scapulohumeral joint and the distal end of the crank were aligned horizontally. Subjects sat in a straight-backed chair and kept their feet flat to the floor to minimize bracing. Lower-body exercise was performed in the upright position using a cycle ergometer (Excalibur; Lode). Subjects remained seated throughout all tests. After 5 min of rest, test 1 and test 2 commenced with 4 min of unloaded exercise (0 W), followed by stepwise increments of 15 W (upper-body exercise) or 30 W (lower-body exercise) every 4 min. Work rates during test 3 and test 4 were established for each subject using inter-stage linear interpolation and were equivalent to those attained at 20, 40, 60, 80 and 100% of the peak ventilation achieved during the initial upper-body exercise test ($\dot{V}_{E,UBE}$) (Figure 1). Both ergometers were set in the hyperbolic mode. Higher cadences have been shown to elicit significantly greater oxygen uptake (\dot{V}_{O_2}), cardiac frequency (f_C) and minute ventilation (\dot{V}_E) during arm-cranking

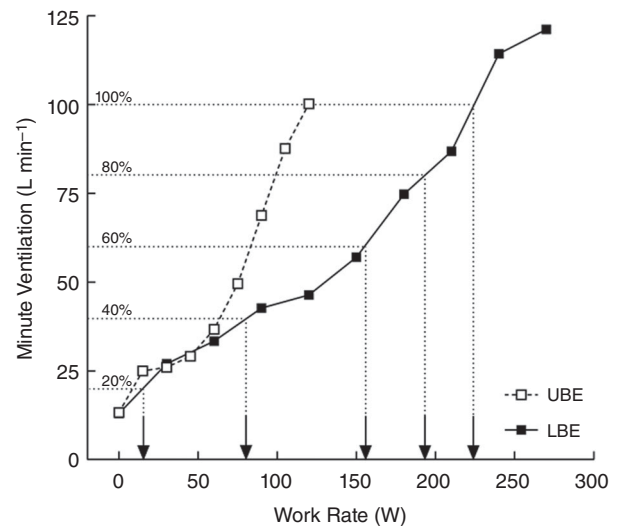


FIGURE 1 Representative plot for a single subject showing minute ventilation versus work rate during maximal, incremental upper- and lower-body exercise (test 1 and test 2). Work rates during test 3 and test 4 were established using inter-stage linear interpolation and were equivalent to those attained at 20, 40, 60, 80 and 100% of the peak ventilation achieved during the initial upper-body exercise test ($\dot{V}_{E,UBE}$)

(Tiller, Price, Campbell, & Romer, 2017b) and leg-cycling (Ettema & Loras, 2009). As such, cadence was standardized at 75–80 r.p.m. to approximate the spontaneously chosen crank rates for both exercise modes (Brisswalter, Hausswirth, Smith, Vercauysen, & Vallier, 2000; Weissland et al., 1997). Exercise was terminated when cadence fell below 65 r.p.m. for >3 s, despite verbal encouragement.

2.6 | Measurements

2.6.1 | Cardiorespiratory measurements

Pulmonary gas exchange and ventilatory indices were assessed using an online breath-by-breath system (Oxycon Pro; Jaeger GmbH), cardiac frequency using a telemetric device (Vantage NV; Polar Electro Oy, Kempele, Finland), and arterial oxygen saturation (S_{pO_2}) using a forehead pulse-oximeter (OxiMax N-560; Nellcor, Tyco Healthcare, Pleasanton, CA, USA). Data were averaged over the penultimate 30 s of each exercise stage, with the final 30 s reserved for the assessment of operating lung volumes and expiratory flow limitation (see 2.6.2). Immediately after a given test, subjects were asked their reason(s) for stopping exercise.

2.6.2 | Operating lung volumes and expiratory flow limitation

To determine the pattern of change in operating lung volumes, subjects performed duplicate IC manoeuvres from relaxation volume, at rest and during the final 30 s of each exercise stage (Guenette et al., 2013). The IC manoeuvre exhibiting the most negative oesophageal pressure (peak P_{oe}) was used to position the averaged tidal flow-volume loop within the maximal envelope. Verbal encouragement was given to ensure a maximal inspiratory effort. The manoeuvre was

considered acceptable when there was no evidence of a prior anticipatory reduction in expiratory reserve volume (ERV) and the peak P_{oe} matched that achieved at rest. Expiratory reserve volume, a surrogate for EELV, was calculated by subtracting IC from vital capacity (VC). Inspiratory reserve volume (IRV) was calculated as the sum of tidal volume (V_T) and ERV. Both ERV and IRV were expressed in absolute terms (in litres) and as a percentage of VC. To account for thoracic gas compression and exercise-induced bronchodilatation, a composite maximum expiratory flow–volume curve was created for each subject from the highest instantaneous flow achieved at any given volume during several maximal and submaximal expiratory manoeuvres (100, 20, 40, 60 and 80% of maximal effort) at resting baseline and within 2 min of exercise cessation (Guenette et al., 2010). Expiratory flow limitation was quantified as the percentage of the tidal flow–volume loop that met or exceeded the expiratory boundary of the maximum flow–volume curve (Johnson et al., 1999).

2.6.3 | Oesophageal pressure and abdominal muscle EMG

Oesophageal pressure (P_{oe}) was measured using a balloon-tipped catheter (5 Fr catheter; Ackrad Labs, Cooper Surgical, Berlin, Germany) connected to a calibrated differential pressure transducer (DP45; Validyne Engineering, Northridge, CA, USA; range ± 229 cmH₂O) and amplifier (CD280; Validyne Engineering). The catheter was passed per nasally into the stomach, filled with 1 ml of air, and withdrawn until there was a negative pressure deflection on inspiration. The balloon was then withdrawn another 10 cm until the distal end was situated in the lower one-third of the oesophagus (Benditt, 2005), with the position validated using the occlusion technique (Baydur, Behrakis, Zin, Jaeger, & Milic-Emili, 1982). The tidal inspiratory P_{oe} swing (ΔP_{oe}) was expressed in absolute terms and as a percentage of the maximum oesophageal pressure exhibited during a maximal static inspiratory manoeuvre ($P_{oe,max}$). The ratio of inspiratory effort ($\Delta P_{oe}/P_{oe,max}$) to thoracic displacement (V_T/VC) was calculated as an index of neuromechanical uncoupling (O'Donnell, Bertley, Chau, & Webb, 1997). In a subset of three subjects, electrical activity of the rectus abdominis (EMGra) was assessed using a pair of 28 mm bipolar differential skin-surface electrodes (Med Trace; Covidien/Medtronic, Minneapolis, MN, USA) attached to the main belly of the muscle, in accordance with published procedures (Ng, Kippers, & Richardson, 1998). Electrode positions were marked on the skin for consistency of placement between trials. Electromyographic signal [root-mean square (RMS)] was recorded during full tidal breaths performed over the penultimate 30 s of each stage and was normalized against the largest RMS achieved during a maximal static expulsive manoeuvre (%RMS_{max}).

2.7 | Signal acquisition

The digital signal for respiratory airflow was converted to a real-time analog signal using an external device (μ DAQ-30A16; Eagle Technology, Cape Town, South Africa). The EMGra signal was amplified (1902; Cambridge Electronic Design, Cambridge, UK), high-pass filtered at 20 Hz and notch filtered at 50 Hz. All signals were acquired using a 16-bit analog-to-digital converter (micro 1401 mkII; Cambridge

TABLE 1 Subject characteristics

Characteristic	Value	Percentage of predicted
Age (years)	24 \pm 5	–
Stature (m)	1.79 \pm 0.07	–
Mass (kg)	74 \pm 11	–
VC (l)	5.67 \pm 0.44	103 \pm 5
FEV ₁ (l)	4.34 \pm 0.41	94 \pm 7
FEV ₁ /VC (%)	77 \pm 7	91 \pm 8
$P_{I,max}$ (cmH ₂ O)	–153 \pm 19	139 \pm 19
$P_{E,max}$ (cmH ₂ O)	160 \pm 45	104 \pm 30

Values are means \pm SD, $n = 8$. Abbreviations: FEV₁, forced expiratory volume in 1 s; $P_{E,max}$, maximum static expiratory pressure; $P_{I,max}$, maximal static inspiratory pressure; and VC, vital capacity.

Electronic Design), sampled at 150 Hz (flow and pressure) or 4 kHz (EMGra) and displayed on a computer running dedicated software (Spike2 v7; Cambridge Electronic Design).

2.8 | Statistics

Descriptive and inferential statistics were calculated using dedicated software (SPSS v24; IBM Corp., Armonk, NY, USA). Cardiorespiratory responses [\dot{V}_{O_2} , CO₂ output (\dot{V}_{CO_2}), RER, \dot{V}_E , f_R , V_T , f_C and S_{pO_2}] and respiratory mechanics (ERV, IRV and ΔP_{oe}) at peak upper- versus lower-body exercise (test 1 and test 2) were compared using Student's paired *t* test. The same indices at rest and during ventilation-matched upper- versus lower-body exercise (test 3 and test 4) were compared using two-factor (mode \times ventilation) repeated-measures ANOVA. In the event of significant interactions, follow-up pairwise comparisons were performed using a Bonferroni-adjusted α -level of 0.008. Effect size (Cohen's *d*) was used to quantify the magnitude of the difference between group means (0.2 = small; 0.5 = medium; 0.8 = large; Cohen, 1977). Data are presented as means \pm SD, unless stated, and the critical α -level was set at 0.05.

3 | RESULTS

3.1 | Subject characteristics

Subject characteristics are shown in Table 1. With the exception of $P_{I,max}$, which tended to be higher than predicted, pulmonary function was within the normal range of predicted values.

3.2 | Responses at peak exercise (test 1 versus test 2)

Physiological responses at peak exercise are shown in Table 2. Compared with lower-body exercise, upper-body exercise elicited significantly lower ($p < 0.05$) work rate, \dot{V}_{O_2} (absolute and relative), \dot{V}_{CO_2} , \dot{V}_E and V_T , and significantly higher IRV. There were no between-mode differences ($p > 0.05$) in any of the other variables shown in Table 2. In addition, there was no between-mode difference in peak P_{oe} during the IC manoeuvre (-70 ± 20 versus -69 ± 27 cmH₂O; $p = 0.89$, $d = 0.05$), and peak P_{oe} during the IC manoeuvre was not

TABLE 2 Physiological responses to upper- versus lower-body exercise at peak work rates (test 1 versus test 2)

Parameter	Lower-body exercise	Upper-body exercise	P value	Cohen's <i>d</i>
Work rate (W)	251 ± 32	118 ± 33	0.000*	4.09
\dot{V}_{O_2} (l min ⁻¹)	3.12 ± 0.72	2.36 ± 0.54	0.001*	1.19
\dot{V}_{O_2} (ml kg ⁻¹ min ⁻¹)	40.7 ± 10.0	30.7 ± 6.3	0.002*	1.20
\dot{V}_{CO_2} (l min ⁻¹)	3.64 ± 0.51	2.67 ± 0.53	0.000*	1.87
RER	1.22 ± 0.30	1.14 ± 0.08	0.465	0.36
\dot{V}_E (l min ⁻¹)	127 ± 27	100 ± 25	0.006*	1.04
V_T (l)	2.60 ± 0.59	2.03 ± 0.42	0.000*	1.11
f_R (breaths min ⁻¹)	47 ± 10	48 ± 11	0.903	0.10
\dot{V}_E/\dot{V}_{O_2}	42.9 ± 15.1	42.6 ± 6.6	0.961	0.03
\dot{V}_E/\dot{V}_{CO_2}	34.8 ± 5.3	37.5 ± 5.6	0.059	0.50
t_I (s)	0.72 ± 0.12	0.70 ± 0.19	0.621	0.13
t_E (s)	0.72 ± 0.15	0.70 ± 0.17	0.685	0.12
t_{TOT} (s)	1.42 ± 0.26	1.38 ± 0.35	0.715	0.13
t_I/t_{TOT}	0.51 ± 0.03	0.50 ± 0.03	0.493	0.33
t_E/t_{TOT}	0.51 ± 0.02	0.51 ± 0.02	0.753	0.00
V_T/t_I (l s ⁻¹)	3.60 ± 0.56	3.07 ± 0.88	0.116	0.72
IRV (l)	1.27 ± 0.46	1.95 ± 0.91	0.012*	0.94
IRV (%VC)	78 ± 8	66 ± 17	0.014*	0.90
ERV (l)	1.81 ± 0.40	1.70 ± 0.91	0.642	0.16
ERV (%VC)	32 ± 8	30 ± 17	0.640	0.15
EMGra (%RMS _{max})	30 ± 16	96 ± 63	—	—
ΔP_{oe} (cmH ₂ O)	25.7 ± 5.2	26.3 ± 8.7	0.810	0.08
f_C (beats min ⁻¹)	179 ± 11	171 ± 11	0.060	0.73
S_{pO_2} (%)	95.6 ± 2.0	97.2 ± 2.1	0.226	0.78

Values are means ± SD, $n = 8$ (EMGra, $n = 3$). Abbreviations: EMGra, rectus abdominis EMG; ERV, expiratory reserve volume; f_C , cardiac frequency; f_R , respiratory frequency; IRV, inspiratory reserve volume; ΔP_{oe} , tidal inspiratory oesophageal pressure swing; RER, respiratory exchange ratio; S_{pO_2} , arterial oxygen saturation; t_E , expiratory time; t_I , inspiratory time; t_{TOT} , total respiratory time; \dot{V}_{CO_2} , CO₂ output; \dot{V}_{O_2} , O₂ uptake; \dot{V}_E , minute ventilation; and V_T , tidal volume. *Significant difference ($p < 0.05$).

different from that recorded at rest for either upper-body exercise (-69 ± 27 versus -64 ± 22 cmH₂O; $p = 0.64$, $d = 0.20$) or lower-body exercise (-70 ± 20 versus -68 ± 22 cmH₂O; $p = 0.55$, $d = 0.09$). Despite lower peak \dot{V}_E during upper-body exercise, EMGra RMS was more than threefold greater.

3.3 | Responses at ventilation-matched work rates (test 3 versus test 4)

Physiological responses were compared at fixed percentages of the peak ventilation attained during the initial upper-body exercise test. The ventilations were slightly lower during upper-body exercise at work rates $\geq 60\%$ $\dot{V}_{E,UBE}$ (Figure 2); however, there was no significant main effect for mode [$F(1, 7) = 4.59$, $p = 0.069$] and no significant mode \times ventilation interaction [$F(1.73, 12.12) = 1.73$, $p = 0.220$].

3.3.1 | Cardiorespiratory responses

Cardiorespiratory responses to upper- versus lower-body exercise at ventilation-matched work rates are summarized in Table 3. Statistically significant effects are reported below.

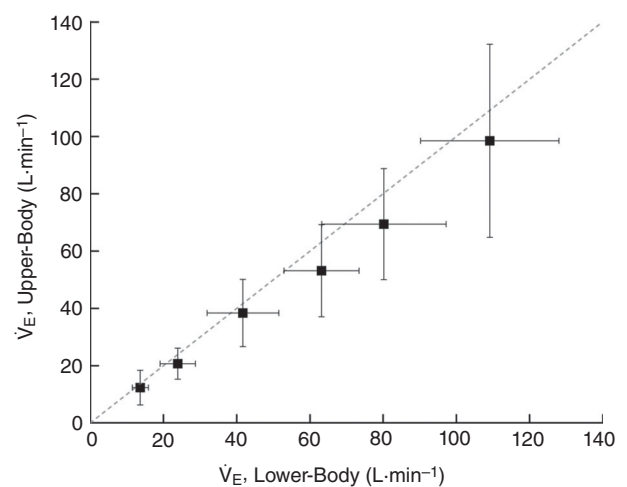


FIGURE 2 Minute ventilation during incremental, ventilation-matched upper- versus lower-body exercise (test 3 versus test 4). Values are means ± SD, $n = 8$. Dotted line: $x = y$

O₂ uptake

There were main effects for mode, showing lower absolute (and relative) values during upper-body exercise [$F(1, 7) = 39.11$, $p < 0.001$]

TABLE 3 Cardiorespiratory responses to upper- versus lower-body exercise at ventilation-matched work rates (test 3 versus test 4)

Parameter	Rest		20% $\dot{V}_{E,UBE}$		40% $\dot{V}_{E,UBE}$		60% $\dot{V}_{E,UBE}$		80% $\dot{V}_{E,UBE}$		100% $\dot{V}_{E,UBE}$	
	LBE	UBE	LBE	UBE	LBE	UBE	LBE	UBE	LBE	UBE	LBE	UBE
Work rate (W) [†]	0 ± 0	0 ± 0	26 ± 19	18 ± 12	111 ± 39	63 ± 24*	168 ± 37	84 ± 25*	199 ± 37	97 ± 29*	221 ± 39	113 ± 30*
Work rate (% maximum)	0 ± 0	0 ± 0	10 ± 7	15 ± 10	44 ± 11	55 ± 13*	67 ± 9	74 ± 7*	80 ± 9	86 ± 6*	89 ± 9	100 ± 0*
\dot{V}_{O_2} (l min ⁻¹) [†]	0.51 ± 0.27	0.43 ± 0.08	0.99 ± 0.28	0.76 ± 0.21	1.72 ± 0.51	1.26 ± 0.35*	2.35 ± 0.53	1.69 ± 0.39*	2.76 ± 0.55	2.04 ± 0.52*	3.07 ± 0.62	2.38 ± 0.54*
\dot{V}_{O_2} (ml kg ⁻¹ min ⁻¹) [†]	6.6 ± 3.4	5.7 ± 1.4	12.9 ± 3.3	10.1 ± 3.1	22.3 ± 5.6	16.3 ± 3.7*	30.6 ± 6.1	21.9 ± 4.0*	36.1 ± 6.5	26.4 ± 5.8*	39.7 ± 5.5	31.0 ± 6.5*
\dot{V}_{CO_2} (l min ⁻¹) [†]	0.45 ± 0.16	0.42 ± 0.09	0.91 ± 0.23	0.76 ± 0.15	1.68 ± 0.45	1.37 ± 0.34	2.43 ± 0.53	1.89 ± 0.34*	2.96 ± 0.60	2.25 ± 0.51*	3.42 ± 0.76	2.69 ± 0.55*
RER [†]	0.86 ± 0.39	0.98 ± 0.20	0.92 ± 0.09	0.99 ± 0.12	0.98 ± 0.06	1.10 ± 0.07	1.04 ± 0.04	1.14 ± 0.10*	1.07 ± 0.04	1.11 ± 0.08*	1.11 ± 0.05	1.13 ± 0.07*
\dot{V}_E (l min ⁻¹)	13.7 ± 6.1	12.3 ± 2.1	23.9 ± 5.5	20.7 ± 4.8	41.7 ± 11.7	38.4 ± 9.8	63.2 ± 16.1	53.1 ± 10.3	80.2 ± 19.5	69.4 ± 17.0	109.1 ± 33.8	98.5 ± 18.9
V_T (l) [†]	0.87 ± 0.30	0.61 ± 0.25*	1.08 ± 0.40	0.92 ± 0.26	1.75 ± 0.64	1.50 ± 0.66	2.27 ± 0.68	1.74 ± 0.61*	2.52 ± 0.67	2.07 ± 0.70*	2.55 ± 0.72	1.97 ± 0.85*
f_R (breaths min ⁻¹)	14.9 ± 3.8	15.2 ± 2.4	20.5 ± 4.2	18.8 ± 3.4	22.9 ± 2.4	24.4 ± 6.0	26.6 ± 3.3	27.2 ± 5.7	31.2 ± 5.2	34.3 ± 8.7	42.3 ± 11.4	48.4 ± 13.3
\dot{V}_E/\dot{V}_{CO_2} [†]	27.9 ± 15.0	28.8 ± 3.8	24.6 ± 4.4	27.9 ± 4.9	24.5 ± 2.8	30.8 ± 2.6	26.8 ± 2.4	31.8 ± 2.8	28.8 ± 2.5	34.3 ± 4.0	35.0 ± 4.5	41.8 ± 4.8
t_I (s) [†]	30.3 ± 10.7	30.0 ± 6.2	26.7 ± 4.1	27.0 ± 2.6	24.8 ± 2.5	28.1 ± 3.2	25.8 ± 2.3	28.1 ± 2.4	27.0 ± 2.5	30.9 ± 3.4	31.5 ± 4.0	36.9 ± 4.2
t_E (s)	1.75 ± 0.58	1.47 ± 0.28	1.21 ± 0.23	1.20 ± 0.20	1.16 ± 0.24	1.14 ± 0.26	1.04 ± 0.21	0.99 ± 0.23	0.94 ± 0.23	0.94 ± 0.24	0.78 ± 0.20	0.70 ± 0.23
t_{TOT} (s)	2.14 ± 0.79	1.91 ± 0.28	1.69 ± 0.32	1.66 ± 0.24	1.50 ± 0.36	1.49 ± 0.33	1.24 ± 0.27	1.19 ± 0.26	1.08 ± 0.26	1.06 ± 0.33	0.83 ± 0.24	0.69 ± 0.23
t_I/t_{TOT}	3.88 ± 1.29	3.38 ± 0.50	2.89 ± 0.53	2.86 ± 0.40	2.66 ± 0.59	2.62 ± 0.58	2.28 ± 0.47	2.18 ± 0.48	2.02 ± 0.49	2.00 ± 0.56	1.61 ± 0.44	1.39 ± 0.45
t_I/t_{TOT}	0.45 ± 0.06	0.43 ± 0.04	0.42 ± 0.03	0.42 ± 0.03	0.44 ± 0.01	0.43 ± 0.02	0.46 ± 0.01	0.45 ± 0.02	0.47 ± 0.01	0.47 ± 0.03	0.48 ± 0.01	0.50 ± 0.03
t_E/t_{TOT}	0.55 ± 0.06	0.57 ± 0.04	0.58 ± 0.03	0.58 ± 0.03	0.56 ± 0.01	0.57 ± 0.02	0.54 ± 0.01	0.55 ± 0.02	0.53 ± 0.01	0.53 ± 0.03	0.52 ± 0.01	0.50 ± 0.03
V_T/t_I (l s ⁻¹) [†]	0.53 ± 0.19	0.42 ± 0.16	0.89 ± 0.22	0.77 ± 0.21	1.49 ± 0.35	1.31 ± 0.37	2.20 ± 0.51	1.76 ± 0.40*	2.72 ± 0.60	2.23 ± 0.53	3.38 ± 0.87	2.85 ± 0.64*
f_C (beats min ⁻¹)	64 ± 10	63 ± 6	84 ± 7	75 ± 9	110 ± 13	104 ± 13	135 ± 16	133 ± 19	156 ± 14	150 ± 20	169 ± 12	167 ± 14
S_{pO_2} (%) [†]	98 ± 2	99 ± 2	97 ± 3	99 ± 1	96 ± 4	98 ± 3	97 ± 3	98 ± 3	97 ± 2	98 ± 2	95 ± 3	98 ± 2

Values are means ± SD, n = 8. Abbreviations: f_C , cardiac frequency; f_R , respiratory frequency; LBE, lower-body exercise; RER, respiratory exchange ratio; S_{pO_2} , arterial O₂ saturation; t_E , expiratory time; t_I , inspiratory time; t_{TOT} , total respiratory time; UBE, upper-body exercise; V_{CO_2} , CO₂ output; V_{O_2} , O₂ uptake; V_E , minute ventilation; and V_T , tidal volume. *Significant difference versus lower-body exercise ($p < 0.008$). † Significant main effect for exercise mode ($p < 0.05$).

and a mode \times ventilation interaction [$F(3, 35) = 10.73, p < 0.001$]. Pairwise comparisons revealed that O_2 uptake was lower during upper-body exercise at 40% ($p = 0.004, d = 1.05$), 60% ($p < 0.001, d = 1.75$), 80% ($p < 0.001, d = 1.36$) and 100% $\dot{V}_{E,UBE}$ ($p = 0.001, d = 1.17$).

Tidal volume

There was a main effect for mode, showing lower values during upper-body exercise [$F(1, 7) = 131.06, p < 0.001$] and a mode \times ventilation interaction [$F(5, 35) = 4.83, p = 0.002$]. Pairwise comparisons revealed that tidal volume was lower during upper-body exercise at rest ($p < 0.001, d = 0.94$), 60% ($p < 0.001, d = 0.82$), 80% ($p < 0.001, d = 0.66$) and 100% $\dot{V}_{E,UBE}$ ($p = 0.002, d = 0.74$).

Respiratory frequency

There was no main effect for mode [$F(1, 7) = 65.01, p = 0.203$], but there was a mode \times ventilation interaction [$F(5, 35) = 29.44, p = 0.028$]. Pairwise comparisons revealed no differences at any level of ventilation.

Ventilatory equivalent for CO_2

There was a main effect for mode, showing higher values during upper-body exercise [$F(1, 7) = 11.33, p = 0.012$], but no mode \times ventilation interaction [$F(1.52, 10.66) = 1.37, p = 0.283$].

Mean inspiratory flow (V_T/t_i)

There was a main effect for mode, showing lower values during upper-body exercise [$F(1, 7) = 29.86, p = 0.001$], and a mode \times ventilation interaction [$F(5, 35) = 6.38, p < 0.001$]. Pairwise comparisons revealed lower values during upper-body exercise at 60% ($p < 0.001, d = 0.88$) and 100% $\dot{V}_{E,UBE}$ ($p = 0.006, d = 0.78$).

3.3.2 | Operating lung volumes and expiratory flow limitation

Operating lung volumes during upper- and lower-body exercise at ventilation-matched work rates are illustrated in Figure 3. During lower-body exercise, ERV decreased below rest at 20% $\dot{V}_{E,UBE}$ and remained below rest through to end-exercise (rest, $36 \pm 10\%$ VC; 20% $\dot{V}_{E,UBE}$, $31 \pm 9\%$ VC; and 100% $\dot{V}_{E,UBE}$, $29 \pm 8\%$ VC), with only two of eight subjects elevating ERV above rest. In contrast, upper-body exercise elicited an initial decrease in ERV, followed by an increase back towards rest at increasing percentages of $\dot{V}_{E,UBE}$, and finally increasing above rest at peak exercise (rest, $33 \pm 12\%$ VC; 20% $\dot{V}_{E,UBE}$, $27 \pm 11\%$ VC; 100% $\dot{V}_{E,UBE}$, $39 \pm 9\%$ VC), with six of eight subjects elevating ERV above rest.

With respect to ERV (as a percentage of vital capacity), there was no main effect for mode [$F(1, 7) = 1.33, p = 0.287$], but there was a mode \times ventilation interaction [$F(5, 35) = 2.61, p = 0.041$]. Pairwise comparisons revealed that ERV was higher during upper-body exercise at 100% $\dot{V}_{E,UBE}$ (39 ± 9 versus $29 \pm 8\%$ VC; $p = 0.002, d = 1.17$).

With respect to IRV (as a percentage of vital capacity), the main effect came close to statistical significance, showing lower values

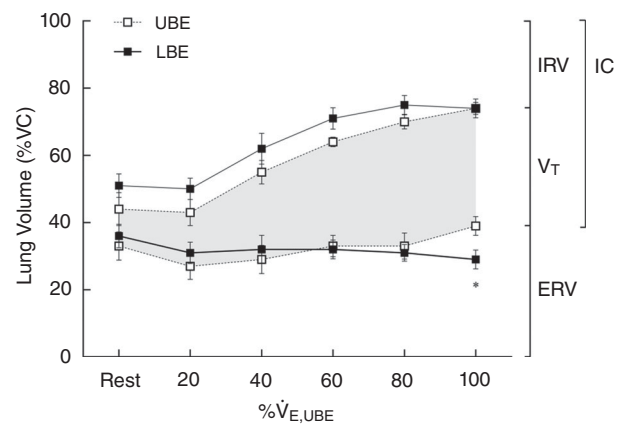


FIGURE 3 Operating lung volumes during incremental, ventilation-matched upper- versus lower-body exercise (test 3 versus test 4). Values are means \pm SEM, $n = 8$. Abbreviations: ERV, expiratory reserve volume; IC, inspiratory capacity; IRV, inspiratory reserve volume; VC, vital capacity; $\dot{V}_{E,UBE}$, peak ventilation achieved during the initial upper-body exercise test; and V_T , tidal volume. *ERV significantly different from lower-body exercise ($p < 0.05$)

during upper-body exercise [$F(1, 7) = 5.58, p = 0.050$], but there was no mode \times ventilation interaction [$F(5, 35) = 0.73, p = 0.603$].

Comparing peak P_{oe} during the IC manoeuvres, there was a main effect for mode, showing less negative values during upper-body exercise [$F(1, 7) = 20.75, p = 0.003$ (-73 ± 19 versus -65 ± 17 cmH $_2$ O)], but no mode \times ventilation interaction [$F(5, 35) = 0.61, p = 0.691$]. There was also no difference in peak P_{oe} during the IC manoeuvres during exercise compared with rest for either mode ($p > 0.05$), suggesting consistently maximal efforts.

During lower-body exercise, two of eight subjects exhibited expiratory flow limitation and, in these subjects, the magnitude of the tidal flow-volume loop that encroached on the expiratory portion of the maximal flow-volume loop was 51 and 80%. The same two subjects exhibited expiratory flow limitation during upper-body exercise, albeit to a lesser extent (15 and 56%, respectively). When the two subjects were removed from the group mean analysis ($n = 6$), the difference in ERV between upper- versus lower-body exercise at 100% $\dot{V}_{E,UBE}$ was of a similar magnitude ($\sim 10\%$) and remained statistically significant (41 ± 7 versus $32 \pm 8\%$ VC for upper- versus lower-body exercise, respectively; $p = 0.008$), suggesting that the increase in ERV during upper-body exercise was independent of expiratory flow limitation.

3.3.3 | Oesophageal pressure and abdominal muscle EMG

Tidal inspiratory oesophageal pressure, expressed as a percentage of maximum static inspiratory oesophageal pressure ($\Delta P_{oe}/P_{oe,max}$), is illustrated in Figure 4. At ventilation-matched work rates, ΔP_{oe} tended to be higher during upper-body exercise. Indeed, there was a main effect for mode [$F(1, 7) = 8.718, p = 0.021$], showing higher values during upper-body exercise, but no mode \times ventilation interaction [$F(5, 35) = 1.214, p = 0.323$].

With respect to the ratio of tidal volume to vital capacity (V_T/VC), there was no main effect for mode [$F(1, 7) = 1.537, p = 0.255$] and no mode \times ventilation interaction [$F(5, 35) = 1.675, p = 0.167$].

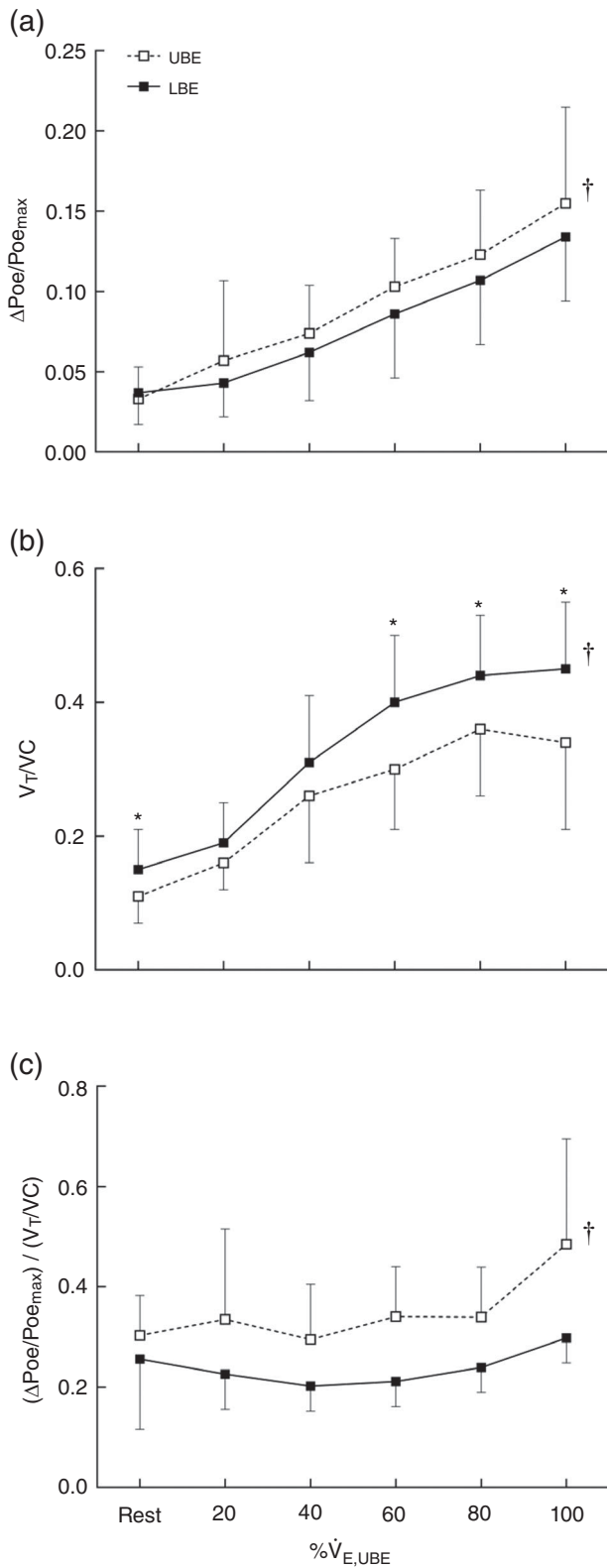


FIGURE 4 Tidal inspiratory oesophageal pressure relative to maximal static inspiratory pressure (a), tidal volume relative to vital capacity (b) and the effort/displacement ratio (c), during incremental, ventilation-matched upper- versus lower-body exercise (test 3 versus test 4). Abbreviations: $P_{oe,max}$, maximal tidal inspiratory oesophageal pressure; ΔP_{oe} , tidal inspiratory oesophageal pressure swing; VC, vital capacity; $\dot{V}_{E,UBE}$, peak ventilation achieved during the initial upper-body exercise test; and V_T , tidal volume. Values are means \pm SD, $n = 8$. * Significant difference versus lower-body exercise ($p < 0.008$). † Significant main effect for exercise mode ($p < 0.05$)

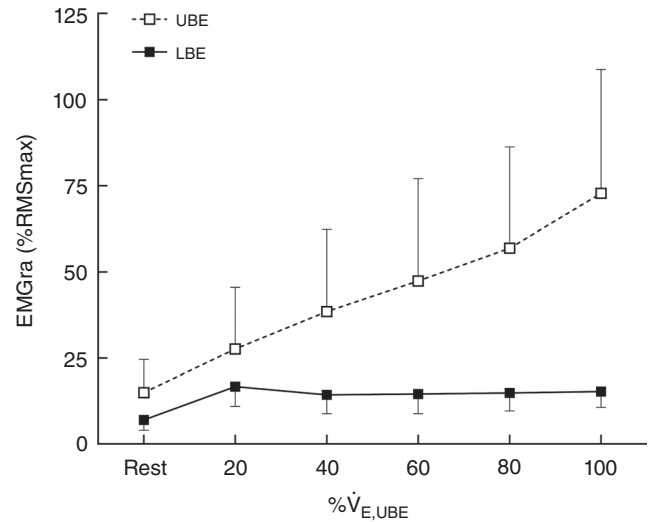


FIGURE 5 Electromyographic activity of the rectus abdominis (EMGra) during incremental, ventilation-matched upper- versus lower-body exercise (test 3 versus test 4). Values are means \pm SEM, $n = 3$

When pressure responses were expressed relative to changes in tidal volume [$(\Delta P_{oe}/P_{oe,max})/(V_T/VC)$; an index of neuromechanical (un)coupling], there was a main effect for mode, suggestive of greater uncoupling during upper-body exercise [$F(1, 7) = 13.696$, $p = 0.008$], but no mode \times ventilation interaction [$F(1.572, 11.006) = 1.216$, $p = 0.321$].

As shown in Figure 5, EMGra was greater during upper- versus lower-body exercise at all ventilation-matched work rates (20% $\dot{V}_{E,UBE}$, 28 versus 18% RMS_{max} ; 40% $\dot{V}_{E,UBE}$, 36 versus 14% RMS_{max} ; 60% $\dot{V}_{E,UBE}$, 47 versus 15% RMS_{max} ; 80% $\dot{V}_{E,UBE}$, 57 versus 15% RMS_{max} ; and 100% $\dot{V}_{E,UBE}$, 73 versus 15% RMS_{max}).

4 | DISCUSSION

The principal aim of this study was to characterize the mechanical ventilatory responses to peak and ventilation-matched upper-body (arm-cranking) versus lower-body (leg cycling) exercise in normal subjects. Upper-body exercise was associated with a relative inability to reduce ERV at peak work rates, with an overt dynamic hyperinflation at 100% $\dot{V}_{E,UBE}$, in agreement with our hypothesis. Moreover, upper-body exercise evoked an increased inspiratory effort (oesophageal pressure) relative to thoracic displacement (tidal volume) when compared with ventilation-matched lower-body exercise. The finding that the responses occurred in healthy subjects with normal pulmonary function and minimal evidence of flow limitation suggests that these characteristic responses to upper-body exercise are attributable, in part, to task-specific locomotor mechanics.

4.1 | Mechanical ventilatory responses

Ventilation during exercise is typically achieved via a progressive reduction in ERV to expand tidal volume (see *Introduction*). In

accordance with previous studies on upper-body exercise (Takano, 1993), we found that upper-body exercise performed at peak and ventilation-matched work rates resulted in restricted expansion of tidal volume, thereby necessitating an increase in respiratory frequency to meet ventilatory demands. At peak exercise, tidal volume was constrained by a decrease in IRV (%VC), whereas at peak ventilation-matched exercise, ERV was significantly elevated during upper- compared with lower-body exercise (39 versus 29% VC; Figure 3), with a large observed effect ($d = 1.21$). This observation of upper-body exercise-mediated dynamic hyperinflation is congruent with our previous observations during constant-load arm-cranking exercise (Tiller et al., 2017a). In the present study, subjects exhibited normal pulmonary function (Table 1) and, after correcting the maximal flow-volume envelope for the effects of thoracic gas compression and exercise-induced bronchodilatation (Guenette et al., 2010), only two of eight subjects exhibited expiratory flow limitation during upper-body exercise compared with six of eight who showed dynamic hyperinflation. Although, in general, there was substantial expiratory reserve during upper-body exercise, dynamic airway compression can occur at expiratory flows below maximal capacity (Mead, Turner, Macklem, & Little, 1967). Thus, the increase in ERV might have occurred in an anticipatory manner as subjects approached their mechanical expiratory flow-generating capacity. It is worth noting, however, that when the two subjects who exhibited frank expiratory flow limitation were removed from the group mean analysis, the difference in ERV between upper- and lower-body exercise remained. Collectively, these findings suggest that the inability to reduce ERV during upper-body exercise in normal subjects is not mechanistically linked with ventilatory demand or flow limitation.

Other mechanisms are likely to underpin the mechanical ventilatory responses to upper-body exercise. Neural activation of the rectus abdominis, assessed indirectly via surface EMG, was substantially elevated during upper- versus lower-body exercise at any given level of ventilation (Figure 5). Unlike the diaphragm, the activation of which is modulated to prioritize pulmonary ventilation during prolonged exercise (Hodges, Heijnen, & Gandevia, 2001), the rectus abdominis contracts to reduce ERV during dynamic expiration (Henke et al., 1988) and to flex/rotate the vertebral column (Cresswell, Grundstrom, & Thorstensson, 1992). Hence, the abdominal muscles undergo additional loading during upper-body exercise to carry out a series of respiratory and non-respiratory tasks. Arm-cranking intensities that approach maximum require the subject to exert a substantial force in overcoming increased external resistances on the flywheel, and the contribution of the abdominal muscles to locomotion is thereby increased. In the present study, EMG_{ra} increased at a faster rate during upper- relative to lower-body exercise (Figure 5), reinforcing the notion that the abdominal muscle contribution to locomotion is a function of work rate (Abraham et al., 2002). Accordingly, the competing roles for the abdominal muscles during upper-body exercise are likely to impede the capacity of these muscles to reduce ERV below relaxation volume. Thus, in accordance with research in patients with chronic obstructive pulmonary disease (COPD) (Gigliotti et al., 2005), we explain the hyperinflation in healthy subjects via mode-specific locomotor mechanics, restriction of normal tidal volume expansion and

the discordance in respiratory pattern between upper- and lower-body exercise.

Alongside dynamic hyperinflation, there was evidence of neuro-mechanical uncoupling of the respiratory system during upper-body exercise; that is, an apparent dissociation between inspiratory effort (tidal swing of oesophageal pressure relative to maximal inspiratory pressure) and subsequent thoracic displacement (tidal volume relative to vital capacity). In patients with COPD, elevated inspiratory effort has been attributed to the consequences of expiratory flow limitation caused by respiratory bronchiole thickening, excess mucous production, and airways that collapse when exposed to modest thoracic pressures (Hogg & Timens, 2009). Presently, the greater effort-displacement ratio during upper-body exercise cannot be explained by airway disease or expiratory flow limitation; instead, a more likely cause was the ventilation-mediated increase in inspiratory oesophageal pressure and comparatively low tidal volume (Figure 4). The thoracic muscles attach to the ribcage and serve an important role in maintaining posture (Celli, 1988). Consequently, elevated thoracic loads will increase chest wall impedance and impose a mechanical constraint on ribcage expansion. There is a strong correlation between the effort/displacement ratio and dyspnoea in patients with COPD (O'Donnell et al., 1997). As such, a potential interaction between neuromechanical uncoupling and dyspnoea warrants prospective study.

4.2 | Cardiorespiratory responses

In accordance with previous literature (Sawka, 1986), \dot{V}_{O_2} at peak intensity and for any given level of ventilation was significantly lower during upper- compared with lower-body exercise. Two interrelated mechanisms might underpin these observations. First, the absolute volume of active muscle mass recruited was probably smaller during upper-body exercise. It is reasonable to suppose, therefore, that the legs and gluteals would provoke greater O_2 demand than the arms, chest, back and shoulders (Bergh, Kanstrup, & Ekblom, 1976). Second, there was probably a mode-specific disparity in O_2 kinetics. Indeed, the upper limbs contain a greater percentage of type II muscle fibres (Jennekens, Tomlinson, & Walton, 1971) which, in turn, have a longer O_2 time constant compared with type I fibres (Kushmerick, Meyer, & Brown, 1992). Greater and/or earlier recruitment of type II fibres during upper-body exercise might explain the slower adjustment of pulmonary \dot{V}_{O_2} to arm-cranking ergometry (Koppo, Bouckaert, & Jones, 2002). This mechanism might also explain, at least in part, why the ventilatory response was slightly, but not significantly, lower during the ventilation-matched upper-body exercise trial.

4.3 | Critique of methods

Several considerations should predicate the interpretation of our findings. The IC manoeuvre used in the assessment of operating lung volumes is effort dependent and must, therefore, be truly maximal to ensure the attainment of TLC (see *Introduction*). Although peak P_{oe} during the IC manoeuvre tended to be less negative during upper-body exercise, there were no significant differences across

exercise intensities for either mode. A further assumption of the IC method is that TLC does not change during exercise. Tonic activation of abdominal and chest wall muscles, especially during upper-body exercise, might change chest wall compliance sufficiently to reduce TLC. Nevertheless, the peak P_{oe} achieved during the IC manoeuvre during exercise was not significantly different from the values achieved reproducibly at rest. Moreover, had TLC been underestimated, this would probably have been evident earlier in the ventilation-matched protocol (i.e. at submaximal intensities), rather than only manifesting at 100% $\dot{V}_{E,UBE}$. We are confident, therefore, that subjects gave consistently maximal inspiratory efforts and that the differences in ERV and IRV between upper- and lower-body exercise were not the result of an inability to attain TLC. Furthermore, we took care to monitor the respiratory pattern used by subjects immediately before the IC manoeuvre and, on occasions when anticipation of the IC manifested as an artificial decrease in ERV, both the IC and the anomalous tidal breath were excluded from analysis.

We noted that EMG activity of the rectus abdominis was higher when resting data were collected immediately before upper- versus lower-body exercise. Resting data for arm-cranking exercise were collected while subjects sat upright in a chair with their hands resting at the sides. In contrast, resting data for leg-cycling exercise were collected while subjects sat upright on the ergometer with their hands resting on the upper handlebars. This latter body position may have permitted offloading of the abdominal muscles, thereby requiring less neural activation for postural support, leading to the noted differences in resting values. It is unlikely, however, that these discrepancies were sufficient to explain the more substantial exercise-mediated differences in function. Finally, we made an effort to compare operating lung volumes at ventilation-matched work rates. Although minute ventilations were not significantly different between exercise modes, there was a tendency towards lower values during upper-body exercise (8–14%). Thus, if anything, the degree of hyperinflation during upper-body exercise might have been underestimated.

4.4 | Implications

Owing to the critical role of the upper-limbs in executing activities of daily living (Tangri & Woolf, 1973), the ventilatory responses to upper-body exercise have been widely studied. Arm-cranking ergometry, specifically, features in pulmonary rehabilitation programmes and is considered the most appropriate method for assessing supported arm-exercise capacity in patients with COPD (Janaudis-Ferreira, Beauchamp, Goldstein, & Brooks, 2012). It should be noted, however, that supported arm ergometry is not consistent with the unsupported nature of activities of daily living. Indeed, unsupported arm exercise does not appear to elicit dynamic hyperinflation in patients in whom chest wall expansion is proportional to ventilation (Romagnoli et al., 2011). Notwithstanding, we used arm-cranking exercise because the repetitive, cyclical nature of the task makes it comparable with stationary leg-cycling exercise. In addition, both modes induce substantial physiological stress without the need for technical coaching.

To limit dyspnoea, patients with COPD often minimize use of the arms during activities such as housework, carrying groceries and self-grooming (Tangri & Woolf, 1973). This suggests that tasks involving the upper limbs might provoke changes in mechanical ventilatory function, resulting in respiratory distress. Although we observed evidence of significant neuromechanical uncoupling during upper-body exercise, it seems unlikely that this would induce substantial respiratory distress in healthy individuals. Indeed, when asked their principal reason for terminating upper-body exercise, all our subjects cited symptoms of arm fatigue rather than dyspnoea. In patients with COPD, however, a poor effort/displacement ratio during upper-body exercise might form the basis of distressing respiratory sensations and elevated perceptions of dyspnoea (O'Donnell et al., 1997). Given these considerations, our data bring into question the appropriateness of dynamic upper-body exercise for use in certain patient populations (e.g. those undergoing pulmonary rehabilitation), and careful consideration should be given to individual patients before engaging them in exercises that provoke neuromechanical uncoupling or exacerbate dynamic hyperinflation. Nonetheless, training programmes that include unsupported arm exercise and training with an arm ergometer have been shown to attenuate hyperinflation during arm-cranking ergometry in patients with COPD (Gigliotti et al., 2005). Clearly, further research is needed to determine the influence of upper-body exercise-mediated hyperinflation and neuromechanical uncoupling on exertional dyspnoea in health and disease. The literature pertaining to obstructive lung disease suggests that the unfavourable respiratory mechanics of upper-body exercise are related to both disease pathology and mechanical constraints (Alison et al., 1998; Gigliotti et al., 2005; Hannink, Van Helvoort, Dekhuijzen, & Heijdra, 2011). The present findings expand current understanding by showing that changes in respiratory mechanics (tidal volume restriction, dynamic hyperinflation and neuromechanical uncoupling) occur independently of pulmonary dysfunction and flow limitation and are likely to depend on task-specific locomotor mechanics. The importance of these findings also extends to athletes engaged in upper-body-dependent sports (e.g. kayaking, rowing, wheelchair racing), for whom arm-cranking ergometry is an essential training and/or profiling tool.

In conclusion, the present study presents new data showing a marked reduction in mechanical ventilatory function during upper-body (arm-cranking) exercise relative to lower-body (leg-cycling) exercise at peak and ventilation-matched work rates in normal subjects. Relative to ventilation-matched lower-body exercise, high-intensity upper-body exercise was characterized by tidal volume constraint and increases in ERV towards or above resting values (i.e. dynamic hyperinflation), which were statistically significant at peak intensities. Furthermore, there was a greater propensity towards neuromechanical uncoupling of the respiratory system during upper-body exercise. Importantly, these observations were independent of respiratory dysfunction and expiratory flow limitation. We propose, therefore, that the aforementioned responses are characteristic of upper-body exercise and are likely to be attributable to the competing respiratory and non-respiratory functions of thoracic muscles.

AUTHOR CONTRIBUTIONS

The experiments were performed at Brunel University London. All authors conceived and designed the study. N.B.T. performed data collection and analysis. N.B.T. and L.M.R. interpreted results and drafted the work. All authors revised the work critically for important intellectual content, approved the final version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

COMPETING INTERESTS

None declared.

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