Static respiratory muscle work during immersion
with positive and negative respiratory loading

NIGEL A. S. TAYLOR1 AND JAMES B. MORRISON2
1Department of Biomedical Science, University of Wollongong, Wollongong,
New South Wales 2522, Australia; and 2School of Kinesiology, Simon Fraser University,
Burnaby, British Columbia Canada, V5A 1S6

Taylor, Nigel A. S., and James B. Morrison. Static respiratory muscle work during immersion with positive and negative respiratory loading. J. Appl. Physiol. 87(4): 1397-1403, 1999.—Upright immersion imposes a pressure imbalance across the thorax. This study examined the effects of air-delivery pressure on inspiratory muscle work during upright immersion. Eight subjects performed respiratory pressure-volume relaxation maneuvers while seated in air (control) and during immersion. Hydrostatic, respiratory elastic (lung and chest wall), and resultant static respiratory muscle work components were computed. During immersion, the effects of four air-delivery pressures were evaluated: mouth pressure uncompensated; the pressure at the lung centroid (PL,c); and at PL,c ± 0.98 kPa. When breathing at pressures less than the PL,c subjects generally defended an expiratory reserve volume (ERV) greater than the immersed relaxation volume, minus residual volume, resulting in additional inspiratory muscle work. The resultant static inspiratory muscle work, computed over a 1-liter tidal volume above the ERV, increased from 0.23 J·l<sup>-1</sup> when subjects were breathing at PL,c to 0.83 J·l<sup>-1</sup> at PL,c -0.98 kPa (P < 0.05), and to 1.79 J·l<sup>-1</sup> at mouth pressure (P < 0.05). Under the control state, and during the above experimental conditions, static expiratory work was minimal. When breathing at PL,c +0.98 kPa, subjects adopted an ERV less than the immersed relaxation volume, minus residual volume, resulting in 0.36 J·l<sup>-1</sup> of expiratory muscle work. Thus static inspiratory muscle work varied with respiratory loading, whereas PL,c air supply minimized this work during upright immersion, restoring lung-tissue, chest-wall, and static muscle work to levels obtained in the control state.

breathing apparatus; lung centroid pressure; pressure breathing; pulmonary mechanics; static loading; work of breathing

HYPERBARIC IMMERSED EXERCISE elevates the work of breathing and is frequently accompanied by dyspnea, which is often beyond that which may be explained on the basis of increased gas density alone (6, 20, 23). Upright immersion imposes a pressure imbalance on the respiratory system, similar to that seen with negative-pressure breathing (1, 8, 19), which results in elevated elastic work (7, 13, 17), elevated flow-resistive work (7, 13), and increased pulmonary resistance (4, 11, 12). Each of these perturbations, on their own, may account for dyspnea during immersed hyperbaric exercise. Accordingly, it is of concern to divers using mouth-held demand regulators that such mechanical changes be reduced, permitting greater comfort and safety. This investigation focuses solely on changes in static respiratory muscle work during immersion and breathing-pressure manipulation. We sought to more precisely quantify this respiratory work, and to evaluate the efficacy of breathing-pressure manipulation in minimizing the elastic (static) work of breathing.

In healthy subjects seated and breathing air, the end-expiratory lung volume (EELV) is usually close to the respiratory relaxation volume (Vrel), the lung volume that is obtained when the glottis is open and respiratory muscles are completely relaxed. However, Taylor and Morrison (20) have shown that when subjects are immersed upright in water, Vrel is dramatically reduced, with subjects apparently defending a higher EELV, rather than permitting the thorax to relax completely. Such a volume defense would elevate respiratory muscle tone, without necessarily contributing to ventilation. This group has also found that the relationship between EELV and Vrel could be restored to nonimmersed control levels if breathing gas was supplied at lung centroid pressure (PL,c), a pressure of +1.33 kPa relative to the hydrostatic pressure at the sternal notch (18). The nonequivalence of these lung volumes indicates that, during immersion, static respiratory work cannot be computed by simply using the system Vrel, as performed by Hong et al. (7) and Sterk (17), but must be derived with an allowance for a volume-defense component. Such an allowance will incorporate discrete elastic and hydrostatic components of the static respiratory muscle work.

The purpose of this investigation was to first determine the effects of immersion and subsequent breathing-pressure manipulations on lung-tissue, chest-wall, and total respiratory elastic work. Second, using differences between the EELV and Vrel for these experimental conditions, we sought to fractionate static respiratory work into its elastic, hydrostatic, and muscle work components. This unique approach allowed a more complete evaluation of the role of breathing gas-delivery pressure in the amelioration of immersion-induced respiratory mechanical changes. The central focus of this evaluation surrounds the capacity of such manipulations, within an air-supply system, to modify static respiratory muscle work. Since, when this component approaches zero, the combined man-machine system approximates optimal efficiency.

METHODS

Eight nonsmoking men with normal lung-function history, aged 20–40 yr, participated as subjects. Experiments were conducted with the subjects seated upright in air (control)
and during total upright submersion in water regulated to 34.8 ± 0.5°C. Immersion trials were performed with subjects wearing a modified diving hood (Kirby Morgan band mask), which enabled the application of positive facial and pharyngeal counterpressure during immersion (13). All methods were implemented as approved by the Ethics Review Committee of Simon Fraser University, Canada.

Definitions

Pl,c. The air-supply pressure required to elicit a pressure displacement of the immersed, total respiratory relaxation volume (Vrel,i) back to the value obtained when seated in air (18).

Lung volumes. 1) EELV: total lung volume at the end of expiration including residual volume (RV); 2) expiratory reserve volume (ERV): the volume of air that may be exhaled from the end of expiration (excludes RV). In the present paper, the use of EELV implicitly includes RV, whereas ERV use implicitly excludes RV.

Relaxation volume (Vrel). The lung volume obtained after respiratory muscle relaxation (glottis open) with the airway unoccluded. When obtained during immersion, it becomes the immersed relaxation volume (Vrel,i). In this paper, both Vrel and Vrel,i were measured and reported as volumes above RV.

Subjects were trained to perform reproducible, pressure-volume relaxation maneuvers between RV and total lung capacity, both in air and during full upright immersion. Immersion trials were performed with air provided from a demand regulator positioned at the sternal notch. Each maneuver was performed as a series of inspiratory (only) steps, always commencing at RV. Five to ten relaxations against an airway opening occlusion (glottis open: Vrel,occ) were performed, each lasting 4–6 s, with the full procedure repeated over five to seven trials. Several large tidal breaths were taken between successive trials to minimize volume-history influences. Between trials, for any one subject, relaxations were performed at different lung volumes, as determined by the experimenter, to ensure maximal distribution of pressure-volume coordinates along the relaxation curve. The Vrel,occ differed from the system Vrel, since the airway opening was occluded by the experimenter before relaxation, thereby preventing the system from assuming its condition-specific Vrel.

Transrespiratory (P’trs) and transpulmonary pressures (Ptp) were measured at each relaxation pause, whereas the lung volume above RV was determined from the integration of inspiratory flow between successive pauses and summed across each trial. Transthoracic pressure (P’tth) was derived by subtraction (P’tth = P’trs – Ptp). Details of these methods have been reported elsewhere (13). The use of a prime in P’trs and P’tth indicates that these variables were not yet adjusted for the hydrostatic effects associated with submersion and a negative air-supply pressure (see below).

Respiratory airflows were measured by using a heated pneumotachograph (Fleisch) coupled to a differential pressure transducer (Validyne DP103 ±0.25 kPa). Uncorrected P’trs was obtained by using a differential pressure transducer (S.E. Laboratories SE 1150 ±6.2), as the difference between static alveolar pressure (PA, at the mouth) and body surface pressure at the sternal notch (Pst, water column; Fig. 1). Pleural pressure was approximated from esophageal pressure, by using an esophageal balloon (10 cm long, wall thickness 8.45 × 10⁻³ mm) and catheter (ID 1.35 mm) connected to one side of a second differential pressure transducer.
ducer (Validyne MP45 ± 3.92 kPa). The other side monitored PA, so that Ptp was obtained from this transducer as the absolute difference between PA and esophageal pressure. Outputs from both transducers were amplified (S.E. Laboratories SE423/LE amplifier demodulator), whereas flow signals were amplified (Daytronic LDVT, model 9130) and low-pass filtered at 5 Hz (Rockland model 432). All data were sampled at 50 Hz by using an analog-to-digital converter (Teclam Labpac) and stored by using an IBM personal computer. Lung volumes were derived by digital integration of flow across time and converted to BTPS conditions.

Uncorrected Ptrs-volume and Ptth-volume curves were initially constructed with the pressure axis referenced to the hydrostatic pressure at the sternal notch. By using the method of Taylor and Morrison (18), the PL,c was determined for each subject from the positive-pressure displacement of the unoccluded control respiratory Vrel during upright immersion. The Ptrs-volume Vrel was dictated by the combined influences of chest and lung elastic recoil and the hydrostatic pressure gradient between the Pst and PA. The Vrel was obtained from the volume intercept of the zero-pressure axis. Thus the reference pressure was the pressure at which breathing gas was supplied to the subject. The PL,c was defined as the pressure required to restore the unoccluded Vrel,i to the control Vrel and was deemed to be equivalent to the mean thoracic surface pressure. The PL,c was computed for each subject. Both Ptrs and Ptth could now be referenced to PL,c for each subject (13). The combined pressure-volume coordinates were calculated (Ptrs = PA − PL,c, Ptth = Ptrs − Ptp). With this correction, both the Ptrs-volume and Ptth-volume curves move leftward by a pressure equal to the PL,c of each subject. The Ptp and corrected Ptrs and Ptth coordinates were used for all subsequent calculations.

With the use of pressure-volume relationships referenced to PL,c it was possible to study the effects of changes in the gas-delivery pressure on static respiratory work components. This was justified, since it has been demonstrated that immersion produces a parallel displacement of the total system respiratory compliance curve (1, 8, 18), with minimal effects on the plethysmographically determined RV (15). In the control and immersed trials, the ERV was determined spirometrically. Because both Vrel and Vrel,i were measured above RV in the present investigation and in our previous experiments (13, 18, 20, 21), direct volume comparisons between ERV, Vrel, and Vrel,i were possible, and this approach has been adopted below, unless otherwise stated. In the immersed state, subjects breathed under steady-state conditions at each of four air-supply pressures (see Ref. 20): Pm (an uncompensated pressure simulating use of a mouth-held demand regulator, where air-supply pressure equals the hydrostatic pressure at the mouth); PL,c air supply (using a mean PL,c of +1.33 kPa relative to Pst; see Ref. 18); and at 0.98 kPa above and below PL,c. This was accomplished by altering the vertical position of the demand regulator, relative to the seated subject. Subsequently, by using the ERV and Vrel,i as starting points, the impact of these four breathing loads on static respiratory muscle work was investigated.

Control and immersion pressure-volume (compliance) curves for the total respiratory system, lung tissue, and chest wall were constructed from an average of 40 (control, SD 7.7) and 36 (immersed, SD 9.0) data points. Curves were analyzed by using least squares, best fit polynomials, with the equation providing the best fit being used for each compliance curve of any given subject (all correlations were >0.9). The coefficients of each equation were used to compute respiratory, lung-tissue, and chest-wall compliances and the components of static respiratory work. Respiratory work was computed over a standard 1-liter tidal volume by using two methods. The methods differed only on the basis of the lung volume from which the static work computations commenced. Traditionally, such computations are performed from the Vrel and the Vrel,i (14). However, in states other than seated at rest breathing air, subjects have been shown to defend lung volumes both above and below the Vrel,i (20). Thus a comparison between the static work calculations from both methods permitted an assessment of the additional static respiratory work involved in defending a lung volume other than the Vrel,i.

Fig. 2. Partitioning static work of the respiratory system during upright immersion of subjects, when air is supplied at negative (A, C) and positive (B) air-supply pressures relative to lung centroid pressure (PL,c; see text). Work was computed over a 1-liter tidal volume commencing at expiratory reserve volume (Vrel,i = Vrel). All volumes exclude residual volume. ERV, expiratory reserve volume.
In the first method (method A), static work computations commenced from each subject’s ERV (point V1 in Fig. 2A). That is, the ERV was not assumed to equal Vrel,i and could fall at any point (T) along the transrespiratory-pressure-volume curve. Figure 2A corresponds with the defense of an ERV greater than the predicted Vrel,i (20). To permit comparisons across breathing pressures, work components were calculated from a common reference pressure, taken to be the P1,c (zero-pressure ordinate in Fig. 2), rather than the pressure at the Vrel,i (denoted by line WZ). The point X designates the intercept of the Ptrs-volume curve and the pressure at the Vrel,i (denoted by line WZ). The point X measured in air (Fig. 3. Representative static pressure-volume curves below). Total respiratory, lung-tissue, and chest-wall elastic work were derived as defined above. The hydrostatic work performed by the demand regulator was V1TQV2. Static inspiratory work (area TSQ) was computed over a 1-liter tidal volume, commencing from the Vrel,i (V1), for each breathing pressure and subject.

Statistical analyses were based on a repeated-measures experimental design, with one within-subjects factor (air-supply pressure). Multivariate analyses of variance and t-tests were used, with the a priori significance set at the 0.05 level. Following a significant F statistic, post hoc multiple comparisons (Tukey honestly significant difference) were used to isolate sources of significant variance. All data are reported as means ± SE, unless otherwise stated (SD).

RESULTS

Under dry (control) conditions, the mean compliance of the lung, chest wall, and total respiratory system were 3.15 ± 0.63, 3.66 ± 0.57, and 1.60 ± 0.16 l/kPa, respectively. Isovolume compliances, computed over a 1-liter volume from the control Vrel, did not change significantly during immersion, with respective means of 3.65 ± 0.25, 5.35 ± 0.19, and 2.05 ± 0.19 l/kPa; P > 0.05. Representative pressure-volume data for control (Fig. 3, A and C) and immersed trials (Fig. 3, B and D) for two subjects are given in Fig. 3. The transrespiratory and transthoracic curves during immersion moved leftward by 2.02 kPa (Fig. 3B) and 1.25 kPa (Fig. 3D), when pressures were adjusted to P1,c (see METHODS).

During control trials, lung-tissue elastic work was 0.66 ± 0.12 J·l⁻¹. Typically, chest-wall elastic work is negative, since people breathe below the Vrel of the thorax. Accordingly, chest-wall elastic work averaged −0.39 ± 0.06 J·l⁻¹. Total system elastic work contained primarily positive, but also some negative, elastic components: 0.27 ± 0.07 and −0.01 ± 0.01 J·l⁻¹, respectively. Energy to perform this positive work was pro-
vided by the inspiratory muscles, since the mean control \( V_{rel} \) was equivalent to the control ERV (2.19 ± 0.11 and 2.13 ± 0.11 liters BTPS, respectively; \( P > 0.05 \)).

Elastic work partitions of the lung and chest wall (method A: areas \( V_1LUV_2 \) and \( V_1RCV_2 \)), for the control and immersed pressure breathing conditions, are summarized in Fig. 4. All components were computed from ERV and represent the change in tissue energy storage over a 1-liter tidal volume. Lung-tissue work was always positive, whereas chest-wall work was negative. Under the immersed state, moving from the most negative (Pm) to positive-pressure breathing (Pl,c + 0.98 kPa), the lung tissue gained elastic energy with each breathing pressure increment, whereas the chest wall, as part of this reciprocating system, lost stored elastic energy.

The total-respiratory elastic work (method A: area \( V_1TSV_2 \)), the work performed by the demand regulator on the subject (area \( V_1WZV_2 \)), and the resultant static respiratory muscle work computed from ERV (areas WTX and XSZ) are summarized in Fig. 5. Table 1 contains the ERV for each air-supply pressure. When air was supplied at Pm during immersion, the elastic work performed on the lung tissue, chest wall, and total respiratory system was significantly different from that obtained under control conditions (\( P < 0.05 \)).

The central focus of this project was an evaluation of the effect of breathing-pressure manipulation on static respiratory muscle work. This is seen in Fig. 5C. Positive (inspiratory) and negative (expiratory) components existed in all conditions, with the latter being minimal for all but the Pl,c + 0.98 kPa breathing pressure. During immersion, when air was provided at Pm, static inspiratory muscle work was elevated more than sixfold relative to control (\( P < 0.05 \)), thereafter decreasing sequentially with each breathing-pressure increment. Only at the Pl,c air supply did the inspiratory muscle work approximate that obtained in the control state (\( P > 0.05 \)). Static expiratory muscle work was performed to defend ERV against the positive-pressure bias of the demand regulator. This was only observed to a significant extent during Pl,c + 0.98 kPa pressure breathing. In this state, the ERV was defended at 1.2 liters lower than the corresponding Vrel,i (3.18 ± 0.29 vs. 4.39 ± 0.21 liters BTPS, respectively; \( P < 0.05 \)), resulting in a significant elevation in expiratory muscle work over the control level (\( P < 0.05 \)).

Static muscle work was also computed from the control \( V_{rel} \), and the \( V_{rel} \) of the immersed lung-chest wall.
Table 1. ERV and Vrel during control and immersed states

<table>
<thead>
<tr>
<th>Volume</th>
<th>Control</th>
<th>Pm, kPa</th>
<th>PL,c - 0.98, kPa</th>
<th>PL,c, kPa</th>
<th>PL,c + 0.98, kPa</th>
</tr>
</thead>
<tbody>
<tr>
<td>ERV, liters</td>
<td>2.13 ± 0.1 0.94 ± 0.2* 1.42 ± 0.2 2.14 ± 0.2 3.18 ± 0.3*</td>
<td>1.42 ± 0.2 2.14 ± 0.2 3.18 ± 0.3*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vrel,i</td>
<td>2.19 ± 0.1 0.26 ± 0.1* 0.92 ± 0.1 2.20 ± 0.2 4.39 ± 0.2*</td>
<td>0.92 ± 0.1 2.20 ± 0.2 4.39 ± 0.2*</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE. Subjects were breathing at mouth pressure (Pm) and 3 air-supply pressures relative to lung centroid pressure (PL,c). ERV, expiratory reserve volume; Vrel,i, relaxation volume; *Significant difference between lung volumes, P < 0.05.

The analyses reported herein provide a unique and novel treatment of the effects of pressure breathing on static respiratory work during upright immersion. We have identified three forms of work that must be considered when evaluating the static work of pressure breathing: elastic work performed on or by tissues; hydrostatic work (relative to PL,c); and static respiratory muscle work, which may be separated into inspiratory and expiratory components. Of particular concern to the immersed diver is the muscular effort required during tidal breathing, for it is this additional respiratory work that may predispose the diver to dyspnea (6, 19, 22), elevating the work-related risks associated with hyperbaric immersion. Our observations have demonstrated the capacity of breathing pressure manipulations (i.e., gas-delivery pressure) to modify the static respiratory work of the immersed diver.

Uncompensated immersion (Pm) significantly elevated the static muscle work (Fig. 5C) due to two effects: decreased respiratory compliance at lower lung volumes; and the pressure difference between the air-supply pressure (Pm) and respiratory recoil pressure at end expiration, attributed to active EELV (ERV + RV) defense during immersion. Increments in gas-delivery pressure sequentially reduced static muscle work, with values obtained at PL,c most closely matching those observed under control nonimmersed conditions. It must be noted that PL,c air supply represents a positive pressure at the mouth but a neutral pressure at the midl thoracic alveoli. Thus, unless the facial regions are also supplied with an equivalent positive pressure (Fig. 1), subjects will experience oropharyngeal distension, resulting in both altered respiratory sensations and some level of discomfort (23).

When air was supplied at PL,c + 0.98 kPa (positive-pressure breathing), subjects maintained an ERV less than the Vrel,i. In this condition, expiratory (negative) muscle work was performed against the positive-pressure bias of the demand regulator (Fig. 5C) to actively defend the ERV at a volume below the system Vrel,i. Taylor and Morrison (20) reported disparities between the ERV and the unocccluded Vrel,i during immersion, when the air was not provided at the PL,c. It was postulated that elevated muscle tone accompanying this active lung volume defense could invalidate static respiratory work calculations, if computed from the Vrel,i (Fig. 2C) rather than from the ERV. When the present data were analyzed from both the ERV (method A) and Vrel,i (method B), during both positive-pressure and negative-pressure breathing (relative to PL,c), static respiratory muscle work differed significantly. During negative-pressure breathing (Pm and PL,c - 0.98 kPa), the latter method underestimated respiratory muscle work. The additional work, derived when calculations were performed from the ERV, was attributed to the hydrostatic load, resulting from the pressure difference between the air-supply pressure and the static respiratory recoil pressure at end expiration. This load is shown as area TSQ of Fig. 2A, where V1 is the ERV.

It is unclear why subjects chose a tidal volume excursion commencing at a volume other than the Vrel,i. In this instance, inspiratory muscle work would have been minimized during negative-pressure breathing, while expiratory work would have been minimized in all instances. It is possible that the EELV is defended in response to a volume-dependent reflex or due to changes in respiratory comfort (perhaps via proprioceptive feedback) encountered at high and low lung volumes. At low lung volumes, expiratory airway resistance is substantially elevated (4, 11, 13), and the choice of the EELV may represent a compromise that attempts to minimize total respiratory work, in the face of conflicting static and flow-resistive respiratory work changes (13) encountered during tidal breathing at low lung volumes.

Alternatively, the EELV may be influenced by dynamic airway closure during negative-pressure breathing. Sterk (16), Dahlbäck et al. (3), and Taylor and Morrison (21) have each shown that dynamic lung compliance is reduced at low lung volumes. As this effect is not seen in the static lung compliance, it is unlikely that it represents an actual change in lung-tissue elasticity. It is possible that such changes in
dynamic lung compliance represent either a measurement artifact (21) or result from airway closure during expiration, as demonstrated by Dahlbäck and Lundgren (5). Because the ERV differs systematically from the Vrel,i in response to both positive and negative respiratory loading (14, 20), it is unlikely that airway closure is the sole determinant. Instead, it is probable that the EELV is determined by some combination of the above factors.

The air-supply pressures used in this study were physically analogous to positive- and negative-pressure breathing, relative to the neutral supply at PPl,c. Previously, Rahn et al. (14) found that, when subjects were breathing at neutral and negative pressures, elastic work was inspiratory, whereas during positive-pressure breathing inspiratory work decreased as expiratory work developed. The present data show a similar pattern (Fig. 5C), with positive-pressure breathing introducing a substantial component of expiratory work, which does not exist in normal (control) respiration. Whereas positive-pressure breathing has clear and well-established clinical applications, it eliminates static inspiratory muscle work, it is suggested that positive-pressure breathing should be approached with caution during immersion. Not only does it introduce significant expiratory work but it has been shown to produce pharyngeal discomfort (unless appropriately countered; see Ref. 23), to modify cardiac output (24), to increase pulmonary shunting (10), and to precipitate dyspnea (9) and respiratory fatigue (2).

The results of this study not only highlight errors that may be introduced when respiratory static work components are derived without due consideration for the actual end-expiratory lung volume but they further emphasize the importance of providing breathing-pressure compensation for workers during upright immersion. Thus breathing air at a pressure equal to the PPl,c most closely reproduced the control static inspiratory work. Air supply at PPl,c has previously been shown to best replicate control pulmonary resistance (12), to minimize dyspnea during hyperbaric exercise (19), and to reproduce lung volume dimensions that exist in air (20). Hence, PPl,c is recommended as the preferred air-supply pressure during upright immersion, provided that the breathing apparatus design also accommodates transpharyngeal and facial counterpressures.

The authors thank Drs. D. R. Stirling and D. Hedges; E. A. Taylor, V. Stobbs, and G. Morariu for technical assistance; and the subjects for their patient cooperation. This study was conducted at the Environmental Physiology Unit, Simon Fraser University, Burnaby, BC, Canada, and was supported by the Natural Sciences and Engineering Research Council of Canada (Strategic Grant number G 0872).

REFERENCES