Effects of self-contained breathing apparatus on ventricular function during strenuous exercise

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Effects of self-contained breathing apparatus on ventricular function during strenuous exercise. J Appl Physiol 106: 395–402, 2009. First published November 13, 2008; doi:10.1152/japplphysiol.91193.2008.—The purpose of this study was to investigate left-ventricular function during strenuous exercise with the self-contained breathing apparatus (SCBA). With the use of two-dimensional echocardiography, images of the left ventricle (LV) were acquired during sustained exercise (3 × 10 min) under two conditions: 1) SCBA, or 2) low resistance breathing valve. Twenty healthy men volunteered for the study, and in each condition subjects wore fire protective equipment. Heart rate, systolic blood pressure, cavity areas during systole and diastole (ESCA and EDCA, respectively), esophageal pressure, ventilation rate, oxygen consumption, perceived physical, thermal and respiratory distress, and core temperature were measured at regular intervals. Urine specific gravity (<1.020 g/ml) and hematological variables were used to infer hydration status. All subjects began both trials in a euhydrated state. No differences were found between conditions for heart rate, systolic blood pressure, ventilation rate, oxygen consumption, perceived distress, or any hematological variables. Peak inspiratory esophageal pressure was always higher (P < 0.05), while EDCA and stroke area (SA) were significantly lower (P < 0.05) with the SCBA. ESCA, end-systolic transmural pressure (ESTMP), and LV contractility (ESTMP/ESCA) were similar between conditions. Sustained exercise with fire protective equipment resulted in significant reductions in EDCA, ESCA, and SA from the start of exercise, which was associated with a 6.3 ± 0.8% reduction in plasma volume, an increase in core temperature (37.0 ± 0.4 to 38.8 ± 0.3°C), and a significant increase in heart rate (146.9 ± 2.1 to 181.7 ± 2.4 beats/min) throughout exercise. The results from this study support research by others showing that increased intrathoracic pressure reduces LV preload (EDCA); however, the novelty of the present study is that when venous return is compromised by sustained exercise and heat stress, SA cannot be maintained.

intrathoracic pressure; left-ventricular function; aerobic exercise

A SELF-CONTAINED BREATHING apparatus (SCBA) is worn for respiratory protection in hazardous work environments. The SCBA has been shown to impose a significant expiratory resistance leading to increases in peak expiratory esophageal pressure (intrathoracic pressure) with heavy exercise (5, 6, 15). Increases in intrathoracic pressure reduce stroke volume and cardiac output at rest and during exercise (4, 19, 20, 21, 27) as a result of decreased venous return.

Dynamic exercise in a warm environment places considerable strain on the cardiovascular system. In the present study, we used fire protective equipment as a means to decrease the rate of heat exchange with the environment. Under conditions of restricted evaporative heat loss, metabolic and environmental heat stress can exceed the capacity to dissipate heat, resulting in uncompensable heat stress (9). We chose this model to study the effects of dehydration and hyperthermia, coupled with increased intrathoracic pressure (SCBA), on left ventricular (LV) function during aerobic exercise.

LV function has recently been studied (10, 29, 33) during exercise with the use of echocardiography, which makes this mode of investigation applicable to the present experiment. This laboratory (23) has also previously investigated the effects of the SCBA on LV function during brief exercise bouts. The design of that study included short, intermittent bouts of exercise that minimized heat strain and dehydration. The effect of the SCBA and fire protective equipment during sustained exercise (as occurs in firefighting) is still unknown. Moreover, the combined effects of increased intrathoracic pressure coupled with dehydration and blood volume loss on LV function are unknown. Therefore, the primary aim of this investigation was to examine the effects of SCBA vs. a low resistance breathing valve on LV systolic function under a significant heat load. Our hypothesis was that the SCBA would reduce LV systolic function secondary to a reduction in LV preload.

METHODS

Twenty healthy, physically active men with no history of cardiac or respiratory problems volunteered for the study (Table 1), which was approved by the University of Alberta Health Research Ethics Board. Each subject completed five exercise sessions separated by at least 24 h. During all the exercise sessions, subjects wore properly fitting fire protective equipment and carried the SCBA (for description see Ref. 5); however, depending on the experimental condition, they may or may not have breathed from the SCBA. The fire protective jacket was modified to allow access to the right arm (blood pressure) and chest (echocardiography) as required for data collection.

Graded Exercise Test

On the first visit, subjects completed a baseline pulmonary function test (Medgraphics, St. Paul, MN) in accordance with standard procedures (1). During the first two sessions, the subjects also completed graded exercise tests on a motorized treadmill. In randomized order, subjects either completed the graded exercise test with the SCBA or with a low resistance breathing valve (Hans Rudolph 2700, Kansas City, MO) designated hereafter as RV. Measurements of respiratory

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by 5 min of active recovery (walking at 0% incline, 54 m/min). The ventilatory threshold was determined. Each work bout was separated grade less than the stage of the graded exercise challenge where each subject then completed three, 10-min bouts of exercise at 2% of maximal aerobic power previously reported with the SCBA (12, 15).

**Experimental Protocol**

Before the start of each experimental protocol, a standard 5-min warm-up (walking for 3 min at 0% incline, followed by 2 min at 2% incline), the treadmill grade was increased by 2% every 2 min until ventilatory threshold; thereafter, the grade was increased by 2% every min until volitional exhaustion. Ventilatory threshold was defined as a systematic rise in $\dot{V}O_2/V_{O_2}$, while $\dot{V}O_2$ remained constant or declined slightly (30). Gas samples were collected continuously and recorded every 20 s; heart rate was monitored using a Polar heart rate monitor (FS1 receiver and T-31 transmitter; Polar Electro Canada, Lachine, QC, Canada). Analysis of expired respiratory gases was performed (FS1 receiver and T-31 transmitter; Polar Electro Canada, Lachine, QC, Canada). Gas exchanges were measured using a TrueOne metabolic cart, LV function (2-dimensional echocardiography), blood pressure (sphygmomanometer), heart rate, rating of perceived exertion (3), breathing distress (14), and thermal stress were measured using visual-analog scales. The thermal distress scale was modified from previously described versions of psychophysical scales (14, 25), where odd numbers are rated as follows: 1 = “My body temperature is comfortable,” 3 = “I am starting to get hot,” 5 = “I am hot,” 7 = “I am very hot” and 9 = “The heat is unbearable.”

**Esophageal Balloon**

Esophageal pressure was measured by an esophageal balloon catheter (Ackrad Laboratories, Cranford, NJ) placed at a depth ~45 cm from the nostril. The esophageal balloon was inserted through one naris with local anesthetic [12 mg/metered dose of Xylocaine (lido- caine hydrochloride)]. With the head in a neutral or slightly forward flexed position, the catheter was advanced into the stomach (verified by a positive pressure on inspiration) before being slightly withdrawn and positioned in the lower third of the esophagus. The subject then performed a brief Valsalva maneuver while the catheter was open to the atmosphere to empty the balloon. After this, 1.0 ml of air was administered into the balloon using a syringe (24). Esophageal pressures were measured using a differential pressure transducer (Valdyne MP45), which was calibrated before each test using a water filled manometer. The pressure was amplified (model MC1–3; Validyne) and recorded with a digital chart recorder (PowerLab/SSP; ADInstruments, Castle Hill, Australia).

**LV Imaging**

Assessment of LV function was performed using two-dimensional transthoracic echocardiography using a commercially available ultrasound instrument (Sonos 5500; Andover, MA) with a 3.5-MHz transducer. Images were obtained from the parasternal short-axis view at the level of the midpapillary muscles. End-diastolic (largest endocardial area) and end-systolic (smallest endocardial area) cavity areas (EDCA and...
measure differences between condition (SCBA vs. ), and time and interaction (condition × time). If main condition effects were found, Tukey’s post hoc tests were run to define differences; i-tests were used to detect differences between body weight changes and urine specific gravity. The level of significance was set at P < 0.05. All analysis was completed using the statistical package Statistica 7.0 (Tulsa, OK).

RESULTS

All subjects completed both experimental trials. Five subjects were unable to tolerate the esophageal balloon catheter; therefore, esophageal data are presented as n = 15. Upon image analysis (offline), five subjects were found to have poor quality images; therefore, echocardiography data are reported as n = 15. All subjects arrived to the laboratory in a euhydration state on both conditions; urine specific gravity was 1.014 ± 0.009 and 1.011 ± 0.007 g/ml for SCBA and RV, respectively.

Effect of Condition (SCBA vs. RV) on Cardiorespiratory Function

Heart rate was not different between conditions (Fig. 2). The SCBA was associated with an increase in peak inspiratory pressure (+3.1 ± 0.8 cmH2O), a decrease in peak inspiratory pressure (−2.2 ± 0.9 cmH2O), and an increase in esophageal pressure swing (+5.2 ± 1.4 cmH2O) over RV (P < 0.05). EDCA and stroke area were reduced, while peak expiratory esophageal pressure increased with the SCBA (Fig. 2). End-systolic transmural pressure, LV area ejection fraction, and ESTMP/ESCA were not different between conditions (Fig. 2). The similar mean esophageal pressure between conditions (−4.9 ± 0.6 and −5.4 ± 0.5 for SCBA and RV, respectively) can be explained by the significant difference between conditions in peak expiratory and inspiratory esophageal pressure, with the SCBA increasing and decreasing peak esophageal pressures, respectively (Fig. 4).

Respiratory rate, V̇E, and V̇O2 were not significantly different between conditions (Table 4). V̇E/V̇O2 was higher (Table 4), and expiratory time was longer with the SCBA (Texp/Ttot: 0.40 ± 0.04 and 0.36 ± 0.02 in the SCBA and RV, respectively). There was no difference between conditions in core temperature (rest: 37.0 ± 0.4, 39 min: 38.8 ± 0.3°C), body mass loss (1,592.1 ± 175.7 g), or the degree of dehydration (Table 3). Subjects rated their perceived exertion and perception of thermal stress similarly in both trials; however, perceived respiratory distress was significantly higher with SCBA (P < 0.05).

Main time effect on cardiorespiratory function

The main time effect for hemodynamic and cardiovascular variables is presented in Fig. 3. Compared with rest, sustained exercise was associated with a significant increase in heart rate, end-systolic blood pressure, and esophageal pressure. Stroke area increased from rest in the first 9 min, by an increase in EDCA and decreased ESCA (P < 0.05). Then, EDCA and stroke area approached baseline after the first 9 min of exercise, while ESCA continued to decrease from baseline throughout the exercise challenge (Fig. 3). In turn, ESTMP, ESTMP/ESCA, and EFarea significantly increased with exercise (Fig. 3). Esophageal pressure swing increased as a result of a significant increase in peak

### Table 2. Intrarater and test-retest reliability for left-ventricular two-dimensional echocardiography

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Intrarater Reliability by Coefficient of Variation, %</th>
<th>Test-Retest by Coefficient of Variation, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDCAcm²</td>
<td>2.8±0.8</td>
<td>7.1±2.4</td>
</tr>
<tr>
<td>ESCA cm²</td>
<td>4.9±0.7</td>
<td>8.1±2.1</td>
</tr>
</tbody>
</table>

Values are means ± SD. Intrarater reliability was assessed with 35 random stages. Intrarater reliability was assessed over 4 different days (n = 3). Coefficient of variations were calculated by the SD differences between each measure and dividing by the mean. EDCA and ESCA, end-diastolic and end-systolic cavity areas, respectively.

**Core Temperature**

Core temperature was monitored using a biocompatible ingestible telemetry pill (Mini Mitter, Bend, OR) signaling an external receiver (VitalSense physiological monitor; Mini Mitter). The capsule was swallowed at least 60 min before the beginning of each experiment to allow it to pass from the stomach to the small intestine.

**Fluid Analysis**

Upon arrival to the laboratory, each subject provided a urine sample that was analyzed for urine specific gravity to ensure subjects were arriving in a hydrated state. Each subject was instrumented with an 18-gauge venous catheter in a radial or antecubital vein. Once the catheter was in place, each subject stood for 20 min before a baseline blood sample was collected. Immediately after warm-up and each exercise bout, a blood sample was collected. A two-syringe technique was used to collect blood samples from an indwelling venous catheter, which was kept patent with saline, and blood from a second syringe (5 ml) was used for the analysis of hematocrit, hemoglobin, serum total protein, serum sodium concentration, and serum osmolality. After withdrawal of the second blood sample, 3.0 ml of normal saline (0.9% NaCl) were reinjected to keep the catheter patent. Blood samples were obtained before exercise (after 20 min of standing rest), immediately after the 5-min warm-up, and immediately after each 10-min work bout. Plasma volume changes were calculated based on changes in Hb and Hct from the initial resting (baseline) sample (11). The limitations of such calculations have previously been analyzed offline, and the coefficient of variation was calculated for EDCA and ESCA (Table 2).

**Statistical Analysis**

All descriptive values are means ± SD; data used for inferential analysis are means ± SE. A repeated measures ANOVA was used to
esophageal pressure expiration and decrease in peak esophageal pressure inspiration over time (Fig. 4).

Sustained exercise significantly increased core temperature (+1.8 ± 0.4°C) and dehydration (Table 3) in each work bout. As a result, perceived exertion, thermal distress, and respiratory distress increased throughout exercise. V̇E, V̇E/V̇O₂, and RR increased from baseline, and continued to increase throughout exercise (Table 4).

![Fig. 2. Mean (± SE) cardiovascular and pulmonary response to breathing with the self-contained breathing apparatus (SCBA) at rest and during exercise. Data were collapsed across all time points to show main condition effects. EDCA, end-diastolic cavity area; ESCA, end-systolic cavity area; SA, stroke area; ESTMP, end-systolic transmural pressure; EF<ASub>, area ejection fraction; ESTMP/ESCA, end-systolic transmural pressure divided by end-systolic cavity area; Peak Pes, peak expiratory esophageal pressure. *Significant difference (P < 0.05) between SCBA and low-resistance breathing valve (RV).](http://jap.physiology.org/)

Table 3. Summary of hematological responses to the three 10-min work bouts under uncompensable heat stress

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Warm-up</th>
<th>10 Min</th>
<th>20 Min</th>
<th>30 Min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb, g/l</td>
<td>152.6 ± 1.6</td>
<td>152.6 ± 1.4♣</td>
<td>154.9 ± 1.3♣</td>
<td>155.9 ± 1.4♣♫</td>
<td>157.4 ± 1.4♣♫</td>
</tr>
<tr>
<td>Hct, %</td>
<td>0.44 ± 0.01</td>
<td>0.45 ± 0.01♣</td>
<td>0.45 ± 0.01♣</td>
<td>0.46 ± 0.01♣♫</td>
<td>0.46 ± 0.01♣♫</td>
</tr>
<tr>
<td>ΔPV, %</td>
<td>—</td>
<td>-1.4 ± 0.5</td>
<td>-3.5 ± 0.7♣♫</td>
<td>-5.1 ± 0.8♣♫</td>
<td>-6.3 ± 0.8♣♫</td>
</tr>
<tr>
<td>ΔBV, %</td>
<td>—</td>
<td>-0.4 ± 0.3</td>
<td>-1.8 ± 0.4♣♫</td>
<td>-2.5 ± 0.5♣♫</td>
<td>-3.4 ± 0.5♣♫</td>
</tr>
<tr>
<td>Sodium, mmol/l</td>
<td>139.0 ± 0.3</td>
<td>139.3 ± 0.3</td>
<td>139.5 ± 0.3</td>
<td>139.9 ± 0.3♣</td>
<td>140.4 ± 0.3♣</td>
</tr>
<tr>
<td>Osmolality, mosmol/kgH₂O</td>
<td>293.8 ± 0.9</td>
<td>291.9 ± 1.0</td>
<td>293.4 ± 1.0</td>
<td>295.8 ± 0.7♣</td>
<td>297.3 ± 0.9♣</td>
</tr>
<tr>
<td>Total protein, g/l</td>
<td>72.3 ± 0.6</td>
<td>71.8 ± 0.7</td>
<td>73.8 ± 0.7♣♫</td>
<td>75.5 ± 0.6♣♫</td>
<td>75.8 ± 0.6♣♫</td>
</tr>
</tbody>
</table>

No differences between trials; data were combined to present the average response to the physical challenge (n = 36). *Significantly different than baseline; ♣significantly different than warm-up; ♫significantly different than 10 min; ♬significantly different than 20 min (P < 0.05). Plasma (PV) and blood volumes (BV) were calculated using formula by Dill and Costill (11). Data are means ± SE.

**Effect of Condition (SCBA vs. RV) by Time on Cardiorespiratory Function**

Esophageal pressure was higher with the SCBA throughout the exercise challenge (Fig. 4). EDCA and stroke area were lower with the SCBA compared with the RV (Fig. 5). During the first 9 min of exercise, when dehydration and hyperthermia were minimal (Table 3), ESCA was lower with the SCBA (Fig. 80 70 40 70 60 50 40 80 SCBA SCBA RV RV

Fig. 2. Mean (± SE) cardiovascular and pulmonary response to breathing with the self-contained breathing apparatus (SCBA) at rest and during exercise. Data were collapsed across all time points to show main condition effects. EDCA, end-diastolic cavity area; ESCA, end-systolic cavity area; SA, stroke area; ESTMP, end-systolic transmural pressure; EF<ASub>, area ejection fraction; ESTMP/ESCA, end-systolic transmural pressure divided by end-systolic cavity area; Peak Pes, peak expiratory esophageal pressure. *Significant difference (P < 0.05) between SCBA and low-resistance breathing valve (RV).
5). No significant interaction effect was found for ESTMP ($P = 0.133$), despite mean esophageal pressure significantly increasing ($P < 0.01$) with the SCBA in the final work bout. This change in esophageal pressure corresponded with a rise in $\dot{V}E/\dot{V}O_{2}$ with the SCBA at the end of exercise (33 and 39 min). Esophageal pressure swing was also higher with the SCBA throughout exercise (min 3–39; Fig. 4).

DISCUSSION

The major novel finding of this study is that sustained exercise with fire protective equipment results in a decrease in LV preload and stroke area and that SCBA amplifies these responses.

**Effects of SCBA on LV Systolic Function**

Increased intrathoracic pressure has a detrimental effect on stroke volume and cardiac output during moderate intensity exercise (4, 27). Our results complement this finding by demonstrating that the SCBA-mediated increase in intrathoracic pressure decreases EDCA and stroke area during sustained exercise. This outcome can be explained by reductions in venous return, as shown previously (4, 19). Increased afterload cannot explain the present results, as there were no differences between conditions for LV end-systolic transmural pressure. This was unexpected, as end-systolic transmural pressure was expected to decrease as a result of the increased intrathoracic pressure generated with the SCBA. We attribute this finding to the following: 1) the similar mean esophageal pressure between conditions, and/or 2) LV preload dependency.

Mean esophageal pressure significantly increased in the present study over time, with the SCBA increasing mean esophageal pressure above the RV in the final work bout ($P < 0.01$). While no main condition effect was observed, we suggest that mean esophageal pressure was acting on end-systolic blood pressure to reduce end-systolic transmural pressure in the final work bout. Our finding that stroke area was still compromised despite a reduction in transmural pressure is consistent with previous work, which imposed greater expiratory resistances than those used in the present study (27). We therefore contend that preload is far more important in healthy subjects than small changes in afterload.

The present results are different than Mayne et al. (23) who found that stroke area was maintained by increases in myocardial contractility, as ESTMP/ESCA was not different in the present investigation. Importantly, LV ESTMP/ESCA did tend
to increase throughout exercise between conditions \( (P < 0.01) \), with a significant interaction being observed \( (P < 0.05) \). This explains the decrease in ESCA with the SCBA in the first 9 min of exercise, when dehydration and hyperthermia were minimal \(( -3.5 \pm 0.7\% \) plasma volume and \( +0.6 \pm 0.3^\circ C \), respectively). Thus there appears to be a compensatory mechanism early in the exercise challenge that is negated after sustained exercise under heat stress. It is unclear what effect lower core body temperature and/or euhydration would have on myocardial contractility during sustained exercise; presumably, we would find an increase in contractility similar to that found in the first work bout in an effort to maintain stroke area \( (23) \). Further investigation is warranted.

### Effects of Sustained Exercise with Fire Protective Equipment on LV Function

Along with a progressive decrease in EDCA and stroke area in both conditions throughout the exercise protocol, ESCA also decreased over time (Fig. 3). These results support previous work \( (7, 8) \) that described the effects of uncompensable heat stress on exercise performance while wearing personal protective equipment. Furthermore, the combination of heat stress and dehydration during upright exercise was associated with decreases in cardiac output, secondary to reductions in stroke volume \( (16, 17) \). As shown in our present results, heart rate increases to compensate for reductions in stroke volume in an

#### Table 4. Ventilatory responses to the repeated exercise bouts while breathing from SCBA or RV

<table>
<thead>
<tr>
<th></th>
<th>3 Min</th>
<th>9 Min</th>
<th>18 Min</th>
<th>24 Min</th>
<th>33 Min</th>
<th>39 Min</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_{\text{E}}, \text{l/min} ) ( \text{SCBA} )</td>
<td>68.1±2.3</td>
<td>79.4±2.5</td>
<td>77.0±2.9</td>
<td>89.8±3.4</td>
<td>86.2±2.9</td>
<td>100.1±3.3</td>
</tr>
<tr>
<td>( V_{\text{O}_2}, \text{ml·kg}^{-1}·\text{min}^{-1} ) ( \text{SCBA} )</td>
<td>32.4±1.4</td>
<td>34.4±1.4</td>
<td>33.8±1.4</td>
<td>35.2±1.6</td>
<td>34.2±1.3</td>
<td>35.5±1.4</td>
</tr>
<tr>
<td>( V_{\text{E}}/V_{\text{O}_2}, \text{l/min} ) ( \text{SCBA} )</td>
<td>29.2±1.3</td>
<td>33.1±1.7</td>
<td>32.6±1.7</td>
<td>38.2±2.0</td>
<td>37.8±2.0</td>
<td>42.4±2.5</td>
</tr>
<tr>
<td>( \text{RR, breaths/min} ) ( \text{SCBA} )</td>
<td>28.6±1.6</td>
<td>33.8±2.0</td>
<td>35.2±1.8</td>
<td>38.8±1.6</td>
<td>39.2±1.6</td>
<td>44.5±1.8</td>
</tr>
<tr>
<td>( V_{\text{E}}, \text{l/min} ) ( \text{RV} )</td>
<td>69.5±1.9</td>
<td>80.8±2.3</td>
<td>76.5±1.8</td>
<td>90.0±2.8</td>
<td>83.8±2.0</td>
<td>97.4±2.6</td>
</tr>
<tr>
<td>( V_{\text{O}_2}, \text{ml·kg}^{-1}·\text{min}^{-1} ) ( \text{RV} )</td>
<td>33.6±1.4</td>
<td>35.5±1.3</td>
<td>34.1±1.2</td>
<td>36.4±1.3</td>
<td>34.9±1.1</td>
<td>36.6±1.2</td>
</tr>
<tr>
<td>( V_{\text{E}}/V_{\text{O}_2}, \text{l/min} ) ( \text{RV} )</td>
<td>25.7±0.6</td>
<td>28.2±0.7</td>
<td>27.8±0.6</td>
<td>30.6±0.8</td>
<td>30.0±0.7</td>
<td>33.2±0.8</td>
</tr>
<tr>
<td>( \text{RR, breaths/min} ) ( \text{RV} )</td>
<td>29.2±1.3</td>
<td>33.1±1.7</td>
<td>32.6±1.7</td>
<td>38.2±2.0</td>
<td>37.8±2.0</td>
<td>42.4±2.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. \( V_{\text{E}} \), minute ventilation (BTPS); \( V_{\text{O}_2} \), oxygen consumption; \( V_{\text{E}}/V_{\text{O}_2} \), ratio between ventilation and oxygen consumption; RR, respiration rate. Condition effect: data were collapsed over all time points to show main condition effects. *Significant difference between conditions; †significant interaction (condition × time \( (P < 0.05) \).
effort to maintain cardiac output (30). The combination of upright exercise, uncompensable heat stress, and significant plasma volume reduction contributed to reduce central blood volume in the present study, leading to a decline in LV preload and stroke volume over time.

Practical Implications

Our findings are important for those occupations using SCBA in combination with uncompensable heat stress and/or significant dehydration. We have previously shown that brief exercise with the SCBA, without a significant change in core temperature or plasma volume, has little effect on stroke area (23). These findings were explained by compensatory increases in contractility. We demonstrate in the present investigation that exercise lasting over 9 min, coupled with uncompensable heat stress and dehydration, leads to an impairment of stroke area. Our results suggest that heat stress and exercise with the SCBA should be followed by rest, cooling, and/or rehydration to maintain optimal LV function.

Limitations

A limitation of this investigation is that two-dimensional echocardiography is subject to movement and respiratory artifacts. However, our laboratory has demonstrated low test-retest variability (Table 2). Moreover, our results agree with previous work (19) done at rest, which assessed LV cavity area during positive pressure breathing, as well as previous investigations (10, 29, 33) of the left ventricle during sustained exercise. Another limitation is that esophageal pressure was measured as a surrogate for intrathoracic pressure. Esophageal pressure has been shown to underestimate pericardial pressure (22), and therefore, our calculation of LV end-systolic transmural pressure may be elevated compared with actual values. Thus our afterload may actually be lower than our reported values. However, the pressure in the lower one-third of the esophagus is believed to closely approximate the pressure in the adjacent pleura so long as the subject is upright (24). Finally, gastrointestinal pill temperature is acknowledged to be a poor reflection of core body temperature when cool fluids are regularly ingested (32). However, in the present study, fluid ingestion was restricted, so we are therefore confident that our core temperature data would be in agreement with rectal temperature or esophageal temperature, as previously reported (26).

Conclusions

This study investigated the effect of the SCBA regulator on LV function during repeated exercise bouts. LV preload was found to be lower with the SCBA, secondary to increased peak expiratory esophageal pressure. The heat stress in the present study resulted in a significant decrease in plasma volume and significant sweat loss. The combined effects of sustained exercise with fire protective equipment and increased intrathoracic pressure led to significant reductions in stroke area without compensatory changes in LV systolic function.

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REFERENCES


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