Estimating Hemodynamic Responses to the Wingate Test Using Thoracic Impedance

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Abstract
Techniques including direct Fick and Doppler echocardiography are frequently used to assess hemodynamic responses to exercise. Thoracic impedance has been shown to be a noninvasive alternative to these methods for assessing these responses during graded exercise to exhaustion, yet its feasibility during supramaximal bouts of exercise is relatively unknown. We used thoracic impedance to estimate stroke volume (SV) and cardiac output (CO) during the Wingate test (WAnT) and compared these values to those from graded exercise testing (GXT). Active men (n = 9) and women (n = 7) (mean age = 24.8 ± 5.9 yr) completed two Wingate tests and two graded exercise tests on a cycle ergometer. During exercise, heart rate (HR), SV, and CO were continuously estimated using thoracic impedance. Repeated measures analysis of variance was used to identify potential differences in hemodynamic responses across protocols. Results: Maximal SV (138.6 ± 37.4 mL vs. 135.6 ± 26.9 mL) and CO (24.5 ± 6.1 L·min⁻¹ vs. 23.7 ± 5.1 L·min⁻¹) were similar (p > 0.05) between repeated Wingate tests. Mean maximal HR was higher (p < 0.01) for GXT (185 ± 7 b·min⁻¹) versus WAnT (177 ± 11 b·min⁻¹), and mean SV was higher in response to WAnT (137.1 ± 32.1 mL) versus GXT (123.0 ± 32.0 mL), leading to similar maximal cardiac output between WAnT and GXT (23.9 ± 5.6 L·min⁻¹ vs. 22.5 ± 6.0 L·min⁻¹). Our data show no difference in hemodynamic responses in response to repeated administrations of the Wingate test. In addition, the Wingate test elicited similar cardiac output compared to progressive cycling to VO₂max.

Key words: Stroke volume, cardiac output, cycle ergometer, maximal oxygen uptake, supramaximal exercise.

Introduction
Techniques available to measure stroke volume (SV) and cardiac output (CO) include single breath acetylene uptake, thermodilution, dye dilution, echocardiography, the direct Fick method, and CO₂ rebreathing. The direct Fick method is considered to be the most reliable method of measuring hemodynamic function (Charloux et al., 2000). However, these methods can be invasive, require an experienced operator, and do not allow continuous measurements during exercise. Alternatively, thoracic impedance has been used as a noninvasive means to assess hemodynamics during exercise. Early devices using this technology were based on the equations of Kubicek et al. (1966) and were shown to be affected by sweating and poor electrical contact, limiting its reproducibility (Warburton et al., 1999). Nevertheless, current versions of this device (Physioflow™) do not rely on measurement of baseline impedance unlike that of the Kubicek et al. (1966) equations. This device was shown to elicit similar determinations of CO during progressive exercise compared to the direct Fick method and CO₂ rebreathing (Charloux et al., 2000; Tordi et al., 2004). More recently, thoracic impedance has been used in obese women (Vella et al., 2012), healthy adults (Astorino et al., 2015), and in marathon runners (Billat et al. 2012) to classify changes in SV/CO in response to various forms of exercise.

Nevertheless, most data describing changes in hemodynamic function are derived from submaximal and/or graded exercise protocols. Less is known about hemodynamic responses to supramaximal exercise, which is applied during incorporation of short-term interval training (Astorino et al., 2011; Burgomaster et al., 2008) as well as to verify attainment of VO₂max (Midgley et al., 2006). In sport science, the Wingate test (WAnT) is a widely-administered protocol which despite its short duration, evokes substantial cardiac work by requiring peak power outputs up to 300 % maximal workload (Wmax). Previously, Sagiv et al. (2000) used echocardiography to compare SV and CO between a graded exercise test (GXT) and WAnT. Despite similar maximal heart rate (HR), maximal SV and CO were significantly lower after the WAnT versus the GXT. However, participants’ backs were strapped to the wall during exercise, limiting power generation and potentially cardiac work. Using the inert gas rebreathing technique, Fontana et al. (2011) reported higher maximal SV but similar CO in response to WAnT compared to GXT. Nevertheless, maximal HR (149 b·min⁻¹) and peak power (~ 6 W·kg⁻¹) for the WAnT were quite low versus previously-reported values for active men (Astorino and Cottrell, 2011), so it is uncertain if effort was truly supramaximal during the Wingate test. These equivocal findings, dissimilar methods of CO assessment across studies, and significant limitations of each study merit further examination of hemodynamic function during intense exercise. Moreover, no study has used thoracic impedance to assess CO during supramaximal exercise.

The primary aim of this study was to elucidate the potential of thoracic impedance to estimate SV and CO during the Wingate test, which is unknown, and to compare these responses to those obtained from graded exercise testing, which should elicit maximal values for these variables. To strengthen our results, participants repeated each exercise to allow assessment of the test-retest reproducibility of SV and CO obtained from thoracic impedance. It was hypothesized that estimates of SV and CO would not differ between repeated administrations of the WAnT, and despite its brief duration, the WAnT would...
eliciting greater SV and CO versus GXT.

**Methods**

**Participants**

Active, healthy participants (9 men and 7 women, mean age = 24.8 ± 5.9 yr) participated in the study. Their physical characteristics are presented in Table 1. Subjects completed a minimum of 4 h·wk⁻¹ of exercise including aerobic and resistance training as well as recreational sports in the last year, and were familiar with laboratory tests as completed in the present study. Subjects filled out a health history and physical activity questionnaire to ensure that they were non-smokers and free of joint issues or known disease, which may preclude their participation in the study. Participants provided written informed consent before participating in this study, whose protocol was approved by the University Institutional Review Board.

**Table 1. Participant (n = 16) physical characteristics.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>24.8 ± 5.9</td>
<td>18 - 40</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.78 ± 0.11</td>
<td>1.60 – 2.01</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>77.8 ± 15.9</td>
<td>53.1 – 104.1</td>
</tr>
<tr>
<td>PA* (h/wk)</td>
<td>7.2 ± 2.6</td>
<td>4 - 15</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>17.0 ± 5.7</td>
<td>6.8 – 27.1</td>
</tr>
<tr>
<td>VO₂max (mL·kg⁻¹·min⁻¹)</td>
<td>48.7 ± 7.8</td>
<td>38.5 – 69.3</td>
</tr>
</tbody>
</table>

* = current level of physical activity

**Experimental design**

Each subject reported to the laboratory (temperature = 21 – 23°C, relative humidity = 30 – 50 %) four times at the same time of day within subjects across all trials. Before each trial, participants were instructed to refrain from intense exercise for 24 h, caffeine intake for 12 h, and arrive to the lab at least 3 h post-absorptive. Trials were separated by a minimum of 48 h and completed within a 2 wk period. Initially, body composition was calculated using the Jackson and Pollock (1978) and Jackson et al. (1980) skinfold paradigm (chest, abdomen, and thigh for men and triceps, abdomen and thigh for women). Each subject completed two VO₂max tests and two Wingate tests, during which strong verbal encouragement was provided. Individual adjustments on the cycle ergometer including seat height/angle, foot position, and handlebar height were adjusted for each participant during the initial trial and maintained for subsequent trials. Trial order for each subject consisted of VO₂max testing on day 1 and Wingate testing on day 2, followed by randomized order for the subsequent two sessions.

**Assessment of VO₂max**

Each subject performed two GXT on an electrically-braked cycle ergometer (Velotron DynaFit Pro, RacerMate, Seattle, WA) using a ramp protocol. For women, the initial work rate was 50 or 60 W for 2 min followed by a 25 – 30 W·min⁻¹ increase in power output until volitional exhaustion. Men began cycling at 60 – 80 W with a 30 – 40 W/min increase in power rate until fatigue. During exercise, subjects expired through a plastic mouthpiece and low resistance valve into tubing connected to a mixing chamber. Measures of ventilation and expired fractions of oxygen and carbon dioxide were obtained throughout exercise by a metabolic cart (ParvoMedics True One, Sandy, UT), which was calibrated before exercise with gases of known concentration (16 %O₂ and 4 %CO₂) as well as to room air (20.93 %O₂ and 0.03 %CO₂). Furthermore, a 3-liter syringe was used to calibrate volume. Gas exchange data including VO₂, VCO₂, and ventilation (Ve) were time-averaged every 15 s, and test duration ranged from 8 - 12 min (Astorino et al., 2004). VO₂max attainment was confirmed by a plateau in VO₂ at VO₂max with further increase in workload at VO₂max (ΔVO₂ < 2.1 mL·kg⁻¹·min⁻¹) as well as maximal values of respiratory exchange ratio (RER) > 1.15 and heart rate (HR) ± 10 b·min⁻¹ within 220 – age (Midgley et al., 2006). Gas exchange data were obtained during GXT and not WAnT. For all participants, the work rate increment was identical across both trials.

**Wingate test**

A 5.5 min warm-up at 75 – 100 W was initially completed on the cycle ergometer (Velotron DynaFit Pro, RacerMate, Seattle, WA) followed by a 6 s acceleration phase, during which participants were instructed to pedal as fast as possible to attain peak cadence. Immediately following the acceleration phase, the load was electronically applied to the flywheel and subjects pedaled ‘all-out’ for 30 s at a load (in kg) equal to 0.085 and 0.075 kg/kg body weight for men and women, respectively. Peak power (PP), mean power (MP), and minimum power (Pmin = the lowest recorded power output) were recorded for all trials and expressed in W·kg⁻¹. Fatigue index (expressed in % and equal to [(PP – Pmin)/PP] X 100) was also recorded. These procedures were identical for both trials across all participants.

**Assessment of hemodynamic function**

Participants entered the laboratory and were required to sit quietly for approximately 5 min. Subsequently, seated blood pressure was measured twice at the antecubital space using a blood pressure cuff and sphygmomanometer (Omron Health Care, Vernon Hills, OH). An alcohol swab was used to clean the neck, right chest, trunk at V6, and spine, and then an electrode gel (NuPrep, Weaver and Company, Aurora, CO) was rubbed into these areas and the skin was further cleaned with a paper towel. Two sets of electrodes (Skintact ECG electrodes, Leonhard Lang GmbH, Innsbruck, Austria), one electrode transmitting and the other sensing, were applied above the supraclavicular fossa at the left base of the neck and at the height of the xiphoid on the spine. Another pair of electrodes (one placed on the right chest and another at V6) was used to monitor the ECG trace. Once applied, these leads were taped to the skin to minimize movement. The participant was seated on the cycle ergometer and told not to talk and remain motionless, and the thoracic impedance device (Physioflow Enduro, Manatec, Strasbourg, France) was calibrated following a 30-beat procedure using the baseline BP value, which was averaged. Although initial devices using impedance cardiography were sensitive to motion artifact (Warburton et al., 1999), this device em-
plays a high performance triggered adaptive filter to minimize this signal noise. During data acquisition, variations in these parameters are analyzed and compared with those obtained during calibration. Resting SV index (SVical) is calculated according to the following formula:

\[ \text{SVical} = k \times \left[ \frac{(dZ/dt \text{ max})}{(Z \text{ max} - Z \text{ min})} \right] \times W(TFITcal) \]

where \( k \) is an empirically adjusted constant, \( dZ/dt\text{ max} \) is the contractility index, \( TFI \) is the thoracic flow inversion time in ms, Z is impedance, and W is a proprietary correction algorithm. Each displayed SV represents the mean value over a 5 s, artifact-free period. The device calculates CO (in L·min\(^{-1}\)) according to the following formula:

\[ \text{CO} = \text{HR} \times \text{SV} \times \text{BSA}, \]

where \( \text{HR} \) is based on the R-R interval in the first derivative of the ECG signal (dECQ/t, which provides a more stable signal than the ECG itself), \( \text{SV} \) is determined as above in line 197, and the body surface area (BSA) is calculated according to Haycock et al.'s (1978) equation:

\[ \text{BSA} = 0.024265 \times \text{mass in kg}^{0.5378} \times \text{height in cm}^{0.3964}. \]

Data analysis

Data are reported as mean ± standard deviation and were analyzed using SPSS Version 20.0 (Chicago, IL). All data were tested for normal distribution using the Shapiro-Wilk test. Data analysis was performed with repeated measures of the WAnT 1 and 2 analysis of variance with repeated measurement of the WAnT. Between GXT and WAnT, maximal (p = 0.27) and end-exercise (p = 0.18) determinations of CO were similar; however, SV (p = 0.03, \( \eta^2 = 0.19 \)) and \( p = 0.02, \eta^2 = 0.20 \)) was greater in WAnT1 and WAnT2 compared to GXT1 and GXT2 by 10 - 15 mL·beat (Table 3). HRmax and HRend values were significantly higher (p < 0.01, \( \eta^2 = 0.49 \)) and (p < 0.01, \( \eta^2 = 0.47 \)) for GXT1 and GXT2 versus WAnT1 and WAnT2 (Table 3). SV declined (p = 0.01) from maximal values to end-exercise in both protocols (Table 3).

**Table 2.** Alterations in resting and warmup indices of hemodynamic function in response to repeated bouts of graded exercise and the Wingate test. Data are means (±SD).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>GXT1 (b·min(^{-1}))</th>
<th>GXT2 (b·min(^{-1}))</th>
<th>WAnT1 (b·min(^{-1}))</th>
<th>WAnT2 (b·min(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRrest</td>
<td>75 (10)</td>
<td>70 (10)</td>
<td>72 (10)</td>
<td>70 (9)</td>
</tr>
<tr>
<td>HRwarmup</td>
<td>109 (9) *</td>
<td>107 (9) *</td>
<td>118 (13)</td>
<td>120 (11)</td>
</tr>
<tr>
<td>SVrest (mL)</td>
<td>87.8 (23.8)</td>
<td>93.8 (27.9)</td>
<td>87.8 (19.8)</td>
<td>89.9 (20.6)</td>
</tr>
<tr>
<td>SVwarmup (mL)</td>
<td>100.7 (27.2)</td>
<td>105.6 (25.6)</td>
<td>104.9 (21.0)</td>
<td>105.1 (18.2)</td>
</tr>
<tr>
<td>COrest (L·min(^{-1}))</td>
<td>6.5 (1.3)</td>
<td>6.4 (1.2)</td>
<td>6.3 (1.1)</td>
<td>6.3 (1.0)</td>
</tr>
<tr>
<td>COWarmup (L·min(^{-1}))</td>
<td>11.1 (3.0)</td>
<td>11.5 (2.7) *</td>
<td>12.6 (1.8)</td>
<td>13.0 (2.0)</td>
</tr>
</tbody>
</table>

GXT = graded exercise test; WAnT = Wingate test; HR = heart rate; SV = stroke volume; CO = cardiac output; * = p < 0.05 from WAnT

**Table 3.** Alterations in maximal indices of hemodynamic function in response to repeated bouts of graded exercise and the Wingate test. Data are means (±SD).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>GXT1 (mL·min(^{-1}))</th>
<th>GXT2 (mL·min(^{-1}))</th>
<th>WAnT1 (mL·min(^{-1}))</th>
<th>WAnT2 (mL·min(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRmax</td>
<td>186 (7) *</td>
<td>185 (7) *</td>
<td>178 (10)</td>
<td>176 (11)</td>
</tr>
<tr>
<td>HRend</td>
<td>185 (8) *</td>
<td>185 (7) *</td>
<td>177 (8)</td>
<td>178 (12)</td>
</tr>
<tr>
<td>SVmax (mL)</td>
<td>123.8 (35.0) *</td>
<td>122.3 (30.0) *</td>
<td>138.6 (37.4)</td>
<td>135.6 (26.9)</td>
</tr>
<tr>
<td>SVend (mL)</td>
<td>110.3 (31.9) *</td>
<td>112.5 (28.1) *</td>
<td>125.1 (29.6) *</td>
<td>126.4 (26.4) *</td>
</tr>
<tr>
<td>COrmax (L·min(^{-1}))</td>
<td>22.7 (6.4)</td>
<td>22.4 (5.7)</td>
<td>24.5 (6.1)</td>
<td>23.7 (5.1)</td>
</tr>
<tr>
<td>COnend (L·min(^{-1}))</td>
<td>20.3 (5.9)</td>
<td>20.8 (5.3)</td>
<td>22.3 (5.1)</td>
<td>22.4 (4.5)</td>
</tr>
</tbody>
</table>

GXT = graded exercise test; WAnT = Wingate test; HR = heart rate; SV = stroke volume; CO = cardiac output; * = p < 0.05 from WAnT; * = p < 0.05 from maximal values
Table 4. Reliability data for various parameters in response to repeated bouts of graded exercise and the Wingate test.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ICC</th>
<th>95 % CI</th>
<th>SEM</th>
<th>MD</th>
</tr>
</thead>
<tbody>
<tr>
<td>GXT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COmax</td>
<td>.95</td>
<td>.88 – .98</td>
<td>1.8</td>
<td>3.7</td>
</tr>
<tr>
<td>SVmax</td>
<td>.95</td>
<td>.88 – .98</td>
<td>7.2</td>
<td>19.9</td>
</tr>
<tr>
<td>HRmax</td>
<td>.97</td>
<td>.91 – .99</td>
<td>1.3</td>
<td>3.5</td>
</tr>
<tr>
<td>WAnT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COmax</td>
<td>.90</td>
<td>.72 – .96</td>
<td>1.8</td>
<td>4.9</td>
</tr>
<tr>
<td>SVmax</td>
<td>.90</td>
<td>.72 – .96</td>
<td>11.6</td>
<td>32.1</td>
</tr>
</tbody>
</table>

ICC = intraclass correlation; CI = confidence interval; SEM = standard error of the mean; MD = minimum difference; WAnT = Wingate anaerobic test; CO = cardiac output; SV = stroke volume; HR = heart rate; GXT = graded exercise test

Reliability of hemodynamic measures

Coefficient of variation of maximal CO from repeated Wingate tests and GXT was equal to 12.9 % and 10.4 %, respectively. For the Wingate test, repeated determinations of HR, SV, and CO were not different and significantly related (p < 0.001), with ICC values greater than 0.80 for all variables (Table 4, Figure 1). For the GXT, maximal and end-exercise ICC for HR, SV, and CO were above 0.90, respectively (Table 4, Figure 2).

Values for SEM were consistently higher in response to WAnT versus GXT for maximal HR, SV, and CO (Table 4). The minimum difference (MD) was calculated from SEM values computed for each hemodynamic variable obtained from both protocols. Compared to GXT, Wingate-derived MD for HR, SV, and CO was higher (Table 4).

Gas exchange data
Mean gas exchange data were similar (p > 0.75) for all parameters between GXT1 and GXT2. Maximal values of VO\textsubscript{2}, V\text{E}, RER, and power output across bouts were equal to 48.7 ± 7.8 mL·kg\textsuperscript{-1}·min\textsuperscript{-1}, 152.4 ± 49.6 L·min\textsuperscript{-1} 1.25 ± 0.06, and 329.7 ± 83.1 Watt, respectively. The VO\textsubscript{2}max values classify these subjects as recreationally-active.

**Discussion**

Cardiac output is a key determinant of maximal oxygen uptake and is frequently measured to assess adaptation to various exercise regimes, although its measurement can be invasive and potentially difficult for clinicians and scientists to perform. Using Physioflow, results revealed no difference in repeated estimates of SV and CO from the Wingate test which were shown to be highly reliable across tests. In addition, higher SV, lower HR, and similar CO were shown in response to the WAnT versus GXT. Overall, thoracic impedance as administered in the present study provides similar estimates of SV and CO in response to repeated bouts of the WAnT.

Although the hemodynamic response to submaximal and/or graded exercise to volitional fatigue is relatively well-documented, less is known about this response to intense exercise such as the Wingate test, which despite its brief duration, elicits energy expenditure approaching 90 %VO\textsubscript{2}max (Smith and Hill, 1991) and average power outputs of approximately 150 %VO\textsubscript{2}max. Previously, lower (Sagiv et al., 2000) and similar (Fontana et al., 2011) maximal CO was demonstrated between the WAnT and GXT, with these discordant results potentially attributed to methodological differences between studies. For example, peak pedaling cadence (130 rev/min) and load (7.5 %BW) employed in a previous study (Fontana et al. 2011) were lower than those used in the present study, which may have attributed to the lower HR and power output values exhibited. A few studies have documented hemodynamic responses to exercise at or slightly above work rates equivalent to VO\textsubscript{2}max. In young men, Tordi et al. (2010) demonstrated greater mean and maximal CO during interval versus continuous exercise although similar HR across modes. In a study by Lepretre et al. (2004), maximal SV and CO derived from thoracic impedance were higher (p < 0.05) in response to incremental cycling exercise compared to constant-load exercise at 100 % or 88 %Wmax despite no difference in VO\textsubscript{2}max across protocols. These results were explained by a greater quadriceps muscle mass involved during cycling at maximal versus submaximal workloads which would augment blood flow and hence stroke volume. Further study is merited to elucidate hemodynamic changes in response to near-maximal to supramaximal exercise bouts differing in intensity, duration, and modality.

The rapid increase in SV and CO seen in response to WAnT to values comparable to GXT can be explained by recent findings (Adami et al., 2011). In this study, active men performed graded cycling to exhaustion and in addition, completed bouts at 80 %VT and 120 %Wmax, during which a gas rebreathing method was used to assess CO. Despite lower VO\textsubscript{2} at 120 %Wmax compared to VO\textsubscript{2}max, similar CO was reported across intensities, and the time constant for CO at the supramaximal workload was faster than that of VO\textsubscript{2} kinetics. Our data revealed that CO peaked approximately 15 - 20 s into the WAnT despite submaximal HR values. In a study by Elliot et al. (2015), trained men completed incremental cycling as well as supramaximal cycling at 110 %Wmax during which pulse contour analysis was used to record continuous hemodynamics. Results showed similar peak and end-exercise HR, SV, and CO across trials. Despite different patterns of change in power output during supramaximal exercise such as the WAnT, the circulatory system rapidly adjusts to the unique demands to optimize cardiac output to the exercising legs, allowing attainment of maximal CO values as demonstrated in the current study.

Despite the high test-retest reliability shown for maximal hemodynamic variables from the WAnT, ICC was lower and SEM and MD were higher versus GXT. Inexperience with this test was not a factor, as almost 80 % of participants had previously performed a Wingate test. Furthermore, there was no difference in any power output measure across trials so the performance data were relatively stable. It may be that the dynamic nature of the WAnT, specifically with its need for greater upper-body movement to apply force at such high pedaling loads, alters signal acquisition more so than during GXT, in which peak workloads are dramatically lower than the WAnT. Another potential factor is the rapid changes in physiology evoked during such a brief and dynamic exercise bout. From warm-up to end-exercise, HR increases approximately 60 – 70 b·min\textsuperscript{-1}, and CO is approximately doubled during the 30 s Wingate test (Tables 2 and 3), yet the magnitude of these changes during any similar-duration portion of the GXT is minimal. To allow these responses, immediate and dramatic changes in contractility and end-diastolic volume occur to improve oxygen delivery, and the overall magnitude of these responses may elicit the variability seen in our results. Further study is merited to confirm this explanation.

The reliability of thoracic impedance to measure changes in SV and CO during exercise was previously reviewed by Warburton et al. (1999), who reported that thoracic impedance yielded similar coefficients of variation as other techniques. Nevertheless, Warburton et al. (1999) emphasized that thoracic impedance should not be used during maximal exercise due to the effect of movement artefact on data accuracy. In contrast, high reliability (r = 0.99) for CO obtained using thoracic impedance throughout exercise to volitional exhaustion was demonstrated by Richard et al. (2001). Also using thoracic impedance, Tordi et al. (2004) reported small differences in CO (0.09 ± 0.70 L·min\textsuperscript{-1} and 0.42 ± 1.12 L·min\textsuperscript{-1}) which was continually estimated during steady-state cycling at 120 and 140 b·min\textsuperscript{-1}. Overall, our results show that repeated estimations of hemodynamic variables using thoracic impedance are not statistically different and highly reliable in a heterogeneous sample of men and women completing the WAnT. Although obtained in a different population and using thoracic impedance, the reliability data reported herein for both exercise modes (Results and Table 4) are similar to those reported from maximal exer-
exercise testing in adults using direct Fick (5 – 10 %), C2H2 rebreathing (5 – 8 %), and Doppler echocardiography (5 – 8 %) (Warburton et al., 1999). Comparison studies using all of these techniques in the same population are needed to further test the utility of thoracic impedance for determining hemodynamic responses to exercise.

This study faces a few limitations. Participants included active young individuals, so data cannot be applied to older untrained populations or those with chronic disease. Gender differences in SV and CO have been reported (Vella and Robergs, 2005) showing that men maintain greater maximal stroke volume which is attributed to greater heart size and blood volume. However, the repeated measures nature of the design and lack of significant gender interaction across protocol suggests that gender did not alter the observed changes in SV and CO to exercise, although use of this heterogeneous sample potentially elicited high values for SEM and thus MD. Despite our data showing similar determinations of CO in response to WAnT and GXT, we can only speculate as to which mechanisms, such as changes in contractility and/or end diastolic volume, elicit these responses. Completion of an additional trial of each modality may better portray the reliability of thoracic impedance (Welsman et al., 2005); however, in this study, insignificant differences were revealed between trials 2 and 3. In addition, subject position on the ergometer was not considered, and it was evident that some participants preferred to exercise in a relatively upright position yet others chose to bend forward and be oriented more over the pedals at higher intensities. Previous data (Warburton et al., 2002) revealed that supine cycling elicited higher SV (p < 0.05) versus upright cycling in trained men. Despite these limitations, determinations of SV and CO were highly correlated across repeated tests and there was no difference in VO2peak/Wmax or any Wingate-derived variable across repeated testing, which strengthens the reliability of our findings. Although our results show that thoracic-impedance-derived CO is highly reliable in response to GXT, recent findings may question if these values are truly valid. In healthy men, CO obtained from thoracic impedance was significantly higher than obtained from the Fick method as well as other techniques including inert gas rebreathing and pulse contour analysis (Siebenmann et al., 2015). Nevertheless, the authors did not comprehensively describe the specific procedures used to prepare subjects and/or calibrate the device before exercise, so it is unclear if they followed recommended procedures established by the manufacturer to ensure optimal signal quality and data acquisition.

Conclusion

Data show no differences in repeated estimates of SV and CO obtained during the Wingate test from thoracic impedance. In addition, CO was similar although HR and SV varied between WAnT and GXT despite these bouts dramatically differing in intensity and duration, which opposes some existing data (Sagiv et al., 2000). This lack of agreement in SV and CO responses to intense exercise merits further studies investigating hemodynamic changes to acute exercise using different techniques as well as in response to chronic aerobic and interval training regimens in various populations.

Acknowledgements

The authors acknowledge our subjects for their dedication in the completion of these exercise protocols and confirm that experiments comply with the current laws of the country in which they were performed. No funding for this work was received from NIH or other entities. There is no conflict of interest in the execution of this study.

References


Midgley, A.W., McNaughton, L.R. and Carroll, S. (2006) Verification of agreement in SV and CO responses to intense exercise using different techniques as well as in response to chronic aerobic and interval training regimens in various populations.

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Cardiac output during the Wingate test

Key points

- Measurement of cardiac output (CO), the rate of oxygen transport delivered by the heart to skeletal muscle, is not widely-employed in Exercise Physiology due to the level of difficulty and invasiveness characteristic of most techniques used to measure this variable.
- Nevertheless, thoracic impedance has been shown to provide a noninvasive and simpler approach to continuously measure CO at rest and during exercise.
- Results show that measurements of CO are not different and highly reliable in response to repeated administrations of the Wingate test.
- Despite vastly different intensities and durations, maximal CO was similar between the Wingate test and graded exercise to VO2max.

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