The rowing stroke elicits large fluctuations in cardiac stroke volume

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Abstract

Preload to the heart is likely limited during rowing as not only blood pressure but also central venous pressure increases markedly when force is applied on the handle of an ergometer. Considering that only the recovery phase of the rowing stroke allows for unhindered venous return, it was hypothesized that rowing induces large fluctuations in stroke volume (SV). Eight nationally competitive male rowers (mean ± SD: age 21 ± 2 years, height 190 ± 9 cm, and weight 90 ± 10 kg) exercised on a rowing ergometer at a targeted heart rate of 130 and 160 beats per minute. SV was derived from arterial pressure waveform by pulse contour analysis, while ventilation and force on the handle were registered. The study confirmed a marked increase in blood pressure during the stroke (to 173 ± 17 mmHg), while SV did not change significantly from seated rest on the ergometer (106 ± 10 ml) to exercise (111 ± 5 and 113 ± 12 ml, P = 0.613 and P = 0.432, respectively). Yet, during one rowing stroke SV was 116 ± 26 ml but decreased 39% (45 ml) in the catch and increased 34% (40 ml) in the recovery, independent of the breathing pattern. These observations indicate that during rowing cardiac output depends critically on SV during the recovery phase of the stroke.

Keywords: breathing pattern, cardiac output, stroke volume, ergometer rowing, submaximal exercise

INTRODUCTION

In contrast to many other types of exercise, during rowing arterial pressure fluctuates, i.e. there is an abrupt increase in blood pressure at the onset of each stroke (Clifford *et al.,* 1994; Pott *et al.*, 1997). Initiation of the drive is carried out with a Valsalva-like maneuver, as illustrated by a large increase in central venous pressure (Pott *et al.*, 1997) in order to stabilize the spine. Consequently, blood pressure fluctuations are synchronized with the rowing cycle rather than with contraction of the heart. A Valsalva-like maneuver represents an increase in intrathoracic pressure (Taggart *et al.*, 1992) that could diminish cardiac preload during rowing (Rosiello *et al.*, 1987). Similarly, the cramped position in the catch of the stroke (i.e. legs eventually pressing on the chest) may affect pleural and intra-abdominal pressure (Rosiello *et al.,* 1987; Manning *et al.*, 2000).

According to the Frank-Starling mechanism (Patterson & Starling, 1914) decreasing venous return/preload to the heart reduces stroke volume (SV). However, even though preload may be impeded during part of the stroke, rowers are capable of achieving a high maximal oxygen uptake (VO_{2max}; Secher *et al.*, 1983; Volianitis & Secher, 2009) indicating a high cardiac output (CO) and, presumably, SV. The oxygen pulse, an index of SV, is larger during rowing than during running at a matched work intensity (Yoshiga & Higuchi, 2003) suggesting that compared to running, the seated position and/or the involvement of almost all muscles during rowing facilitates venous return. Thus, immediately after exercise at a similar heart rate (HR), the increase in SV was larger for rowing than for cycling during submaximal exercise (Horn *et al.* 2015), while Rosiello *et al.,* (1987) found a lower SV during rowing when compared with cycling. Considering that the seated position of rowing facilitates cardiac preload, as evaluated by plasma arterial natriuretic peptide, compared with running at matched workloads (Yoshiga *et al.,* 2019), determination of SV during rowing in relation to the rowing stroke could elucidate the how CO increases during rowing.

This study evaluated SV, derived from arterial pulse contour analysis during ergometer rowing while the breathing pattern and force developed were registered. Considering that only the recovery (i.e. while moving forward for the next stroke) allows for unhindered venous return to the heart, it was hypothesized that rowing induces large fluctuation in SV.

METHODS

Eight healthy nationally competitive male rowers with minimum 2 years of regular training ($\geq$ 4 $d \cdot wk^{-1}$) were recruited (mean ± SD: age 21 ± 2 years, height 190 ± 9 cm, and weight 90 ± 10 kg). The participants had minimal alcohol intake and were non-smokers. The study was approved by the local ethics committee (H-18029147) in accordance with the Declaration of Helsinki and all participants provided written and oral informed consent before the study. The participants were asked to refrain from caffeinated beverages for 12 h, be fasting for at least 4 h, and avoid high-intensity exercise for 24 h prior to the study.

The participants rowed for ~5 min on a wind-braked ergometer on slides (Concept II D, Morrisville, VT, USA) and aimed at a work rate corresponding to a HR of 130 and 160 bpm. Stroke rate targeted 20 and 26 min^{-1} for the two work rates with a drag factor of 115. The protocol included instrumentation, ~15 min of rest, 5 min baseline measurement seated on the ergometer, and 5 min of exercise at each of the two work rates with ~1 min recovery between trials.

Arterial pressure was determined via a catheter in the radial artery of the non-dominant arm connected to a transducer (TruWave, Edwards Lifesciences, CA, USA) placed on the back of the participant at the assumed level of the heart and signals were transferred to a monitor (Dialogue-2000, IBC-Danica Electronic, Copenhagen, Denmark). The monitor was connected to a Nexfin monitor (BMEYE B.V., Amsterdam, The Netherlands) that derives SV, HR, and thus CO by pulse contour analysis (Monnet *et al.*, 2012). The systolic area of the arterial pressure waveform is

divided by the aortic input impedance established according to a three-element Windkessel model (Westerhof *et al.*, 2009; Bogert *et al.*, 2010). The three-element model includes characteristic impedance, total arterial compliance, and total peripheral resistance, which incorporates the effect of a non-linear arterial pressure taking influence of sex, age, height, and weight into account (Westerhof *et al.*, 2009). HR was derived from the blood pressure waveform and averaged over 5 beats. SV variation (SVV) was calculated as $SSV = \frac{SV_{max} - SV_{min}}{SV_{mean}}$, where SV_{max} is the maximal value, SV_{min} the smallest, and SV_{mean} the mean of the last minute. Tidal volume (VT), respiratory frequency (Rf), ventilation (VE), and oxygen uptake (VO_2) were collected breath-by-breath (Quark CPET, Cosmed, Rome, Italy). Gas analyzers were calibrated using certified standard gases and room air, and a 3-L syringe calibrated flow according to the manufacturer's manual. Force applied to the handle of the ergometer was measured with a piezoelectric transducer (Kistler 9311A) mounted with hooks between the chain and the handle and connected to a line amplifier. The transducer generates an electrical output proportional to the force applied when exposed to compression or as here stretch.

Force and hemodynamic variables were measured continuously using a PowerLab data-acquisition device (AD Instruments, Dunedin, New Zealand). The hemodynamic and force data were synchronized to the ventilatory variables by a mark in the data recordings and data were sampled at 100 Hz.

Statistical analysis

Descriptive data are presented as mean ± SD for the last minute of baseline and exercise, and an average for 3 rowing cycles was used. The arterial waveform was inspected, and artifacts removed. Data were calculated using Microsoft Excel 2016 (Microsoft, Redmond, Wash, USA). A Shapiro-Wilk test was used to evaluate data distribution. Significance between baseline and the two work rates was tested by a one-way ANOVA or Kruskal-Wallis test, depending on data distribution.

Differences were identified with Tukey post hoc and Mann-Whitney tests and statistical significance was set at $P < 0.05$ using SPSS statistics 25 (IBM, Armonk, New York, USA).

Results

One participant did not complete the 160 bpm work rate because the radial artery catheter fell out and thus data from 7 participants are presented for that work rate. During the last minute of exercise, the participants had a HR of 134 ± 3 and 157 ± 6 bpm with a stroke rate of 20 ± 1 and 26 ± 1 min^{-1}. From sitting rest to exercise, HR, CO, MAP, VT, Rf, VE, and VO_2 increased (Table 1; $P < 0.05$), while SV did not change significantly ($P = 0.613$ and $P = 0.432$, respectively) as SV fluctuated indicated by increased SVV ($P = 0.001$).

Table 1 Hemodynamic and ventilatory variables for the last minute seated on the ergometer (Baseline) and during rowing at a targeted HR of 130 and 160 bpm.

	Baseline	**130 bpm**	**160 bpm**
MAP (mmHg)	94 ± 9	111 ± 9*	113 ± 9*†
HR (bpm)	63 ± 12	134 ± 3*	157 ± 6*†
SV (ml)	106 ± 10	111 ± 5	113 ± 12
CO (l/min)	6.7 ± 1.3	14.9 ± 0.6*	17.9 ± 2.0*†
SVV (%)	46 ± 14	125 ± 18*	152 ± 3*
VT (l/breath)	0.9 ± 0.2	2.1 ± 0.3*	2.7 ± 0.6*†
Rf (breath/min)	15 ± 3	43 ± 6 *	48 ± 10*
VE (l/min)	12.8 ± 2.7	78.4 ± 4.2*	121.5 ± 21.7*†
VO_2 (l/min)	0.6 ± 0.1	3.4 ± 0.5*	4.5 ± 0.6*†

Values are mean ± SD for 8 participants (7 during 160 bpm). MAP, arterial pressure; HR, heart rate; SV, stroke volume; CO, cardiac output; SVV, stroke volume variation; VT, tidal volume; Rf, respiratory frequency; VE, ventilation; VO_2, oxygen uptake; O_2 pulse, oxygen pulse
*Different from baseline ($p < 0.05$). †Difference between 130 and 160 bpm ($p < 0.05$).

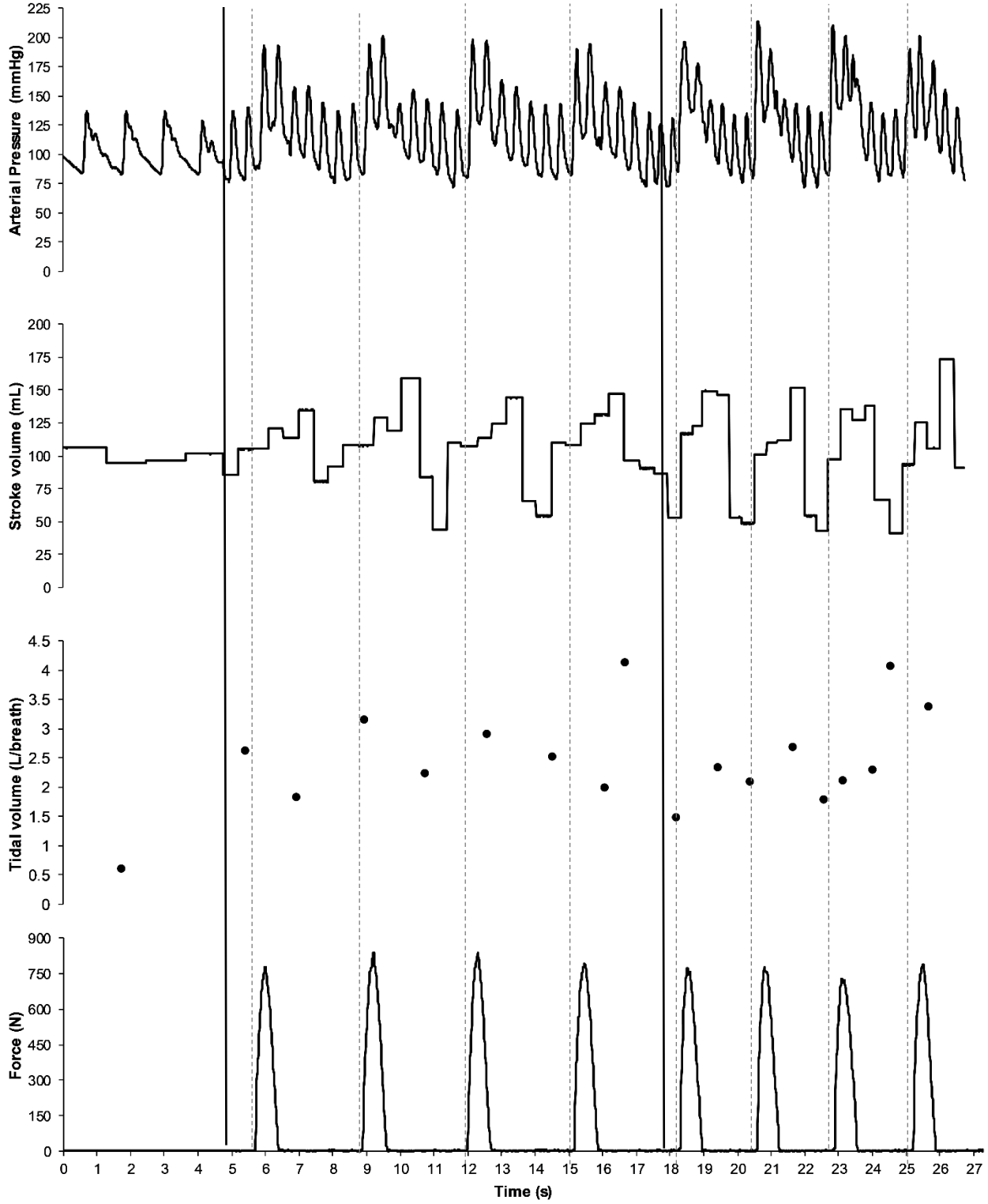

Figure 1. Cardiorespiratory variables at baseline and for 4 strokes at a targeted 130 and 160 bpm work rate for one participant. Thick lines indicate separation of baseline, 130 bpm, and 160 bpm. Dotted lines beginning of the drive where force is applied on the rowing handle. Arterial pressure and force raw data; tidal and stroke volume estimated.

Arterial pressure and SV showed distinctive fluctuation during rowing (Fig. 1). When the arterial pressure tracing is related to the force applied on the handle, an abrupt increase is revealed at the onset of each stroke. Fluctuations in SV demonstrated a peak during the recovery phase where no force was applied, and SV was lowest at the catch.

The rowing stroke at 130 bpm lasted 3.0 ± 0.1 s (P = 0.69) with force applied for 0.7 ± 0.1 s (P < 0.001) corresponding to a stroke rate of 20 min^{-1}. Arterial pressure was largest (168 ± 21 mmHg) during the drive when force was applied on the handle and decreased to 103 ± 18 mmHg at the catch (Fig. 2 left panel). Conversely, SV increased during the drive but peaked thereafter (122 ± 8 ml, P = 0.17) and decreased to 73 ± 19 ml (P = 0.015) at the end of the recovery phase. The mean SV was 105 ± 18 ml and increased 16% (17 ml) throughout the recovery but decreased 31% (50 ml) at the catch just prior to the next propulsive phase and SVV was 49%.

The participants used approximately 2 breaths per stroke (1.8 ± 0.5). The first breath had a volume of 2.5 ± 0.7 L and lasted for 1.9 ± 0.5 s with inspiration (0.9 ± 0.3 s) accounting for 49% of the duration. The second breath had a smaller volume (1.6 ± 0.4 L, P < 0.001) in accordance with a shorter total (1.3 ± 0.3 s, P = 0.002) and inspiratory duration (0.5 ± 0.2 s, P = 0.001) or 41% of the total duration.

At 160 bpm the rowing stroke lasted 2.3 ± 0.1 s (P = 0.94) with force applied to the handle for 0.7 ± 0.0 s (P < 0.001) corresponding to a stroke rate of 26 min^{-1}. Arterial pressure was highest (173 ± 17 mmHg) during the drive and decreased to 102 ± 15 mmHg at the catch (Fig. 2 right panel).

In contrast, SV kept increasing throughout the recovery phase and peaked at 156 ± 13 ml (P < 0.001) and then abruptly decreased to 98 ± 22 ml (P = 0.001) at the catch. The mean SV during the rowing stroke was 116 ± 26 ml decreased 39% (45 ml) at the catch and peaked during recovery with a 34% increase (41 ml), i.e. an SVV of 73%.

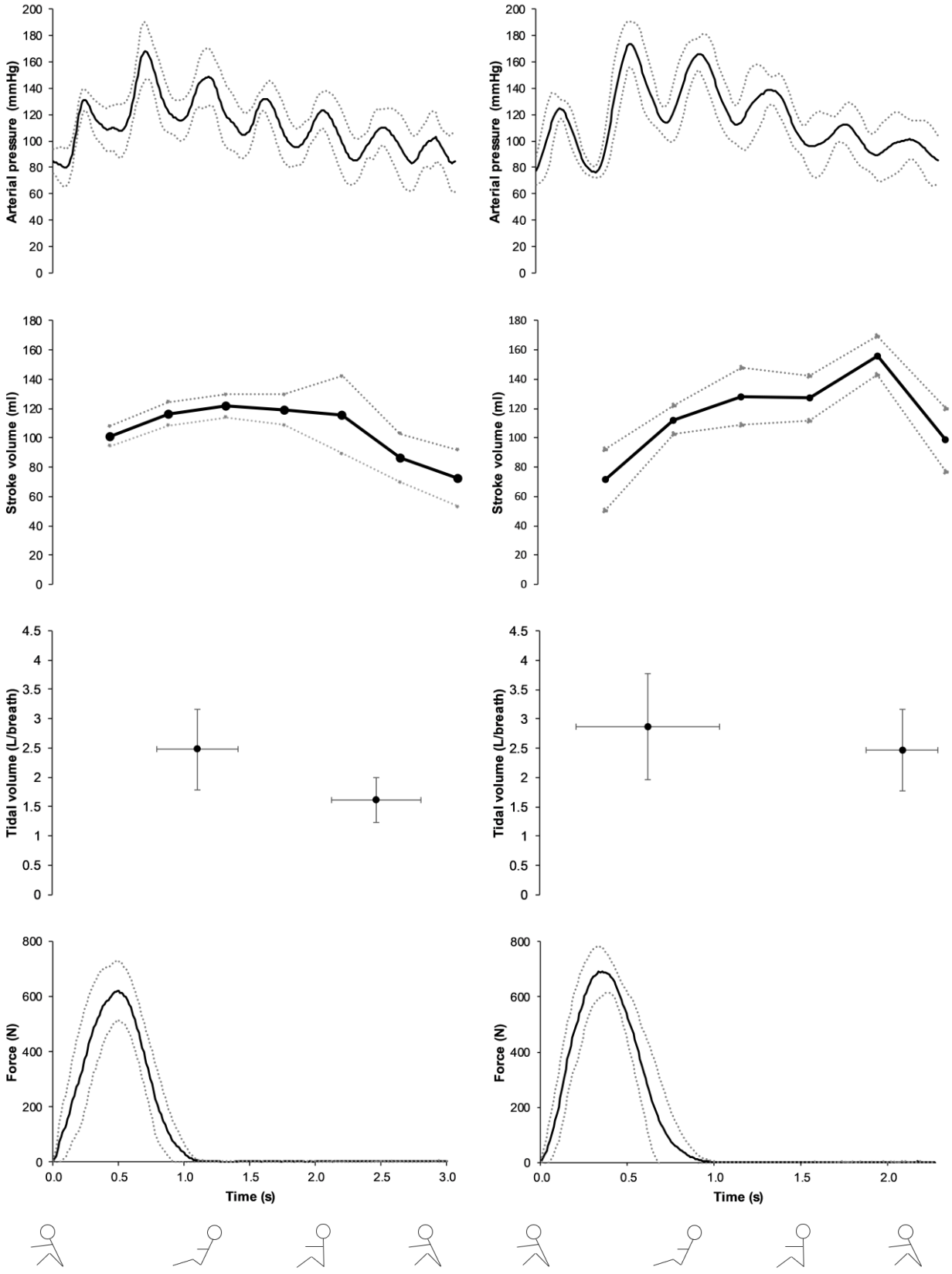

Figure 2 Values for one rowing cycle. Arterial pressure, stroke volume, tidal volume, and force for 8 participants at a work rate targeting a HR of 130 bpm (left) and 160 bpm (right, n = 5). Stroke volume is depicted as beat-to-beat. Values are given as mean (——) ± SD (- - - -).

Similarly, to the 130 bpm work rate, the participants breathed twice per stroke (1.8 ± 0.4) (Fig. 2). However, there was no difference between the volume of the two breaths (2.9 ± 0.9 *vs.* 2.5 ± 0.7 L, P = 0.23); inspiratory time was 0.7 ± 0.3 *vs.* 0.6 ± 0.2 s (P = 0.48) and total duration (1.6 ± 0.5 *vs.* 1.4 ± 0.4 s, P = 0.25).

DISCUSSION

This study in young male rowers is the first to describes variation in SV during the rowing stroke in relation to the force applied on the ergometer handle and the breathing pattern. At both work rates SV changed markedly during the stroke with deviations being most clear at the higher work rate. On an average, SV did not increase from seated rest on the ergometer to the two work rates, but during one rowing stroke the catch of the stroke was associated with a 38% decrease followed by a 36% increase in SV during in the recovery when the rower was mowing forward to the next stroke and no force was applied on the handle. These fluctuations in SV were unrelated to the breathing pattern, which was entrained to the rowing stroke with one breath during the stroke and another breath when the rowers were preparing for the next stroke (Steinacker *et al.*, 1993; Siegmund *et al.*, 1999). This study did not address why SV fluctuates during the rowing stroke, but the catch has been associated with a Valsalva-like maneuver by demonstrating a large increase in central venous pressure and the associated increase in blood pressure (Clifford *et al*, 1994; Pott *et al.*, 1997) was confirmed and likely explains the three-fold increased SVV.

Interest in SVV during rowing was inspired by the extremely large heart found in rowers (Spirito *et al.*, 1994). In principal two “sports hearts” are described (Pluim *et al.*, 2000). Athletes involved in sports with a high dynamic component (e.g. running) develop predominantly enlarged left ventricular chamber size surrounded by a myocardium of normal thickness. In contrast, sports involving mainly static or isometric/strength exercise (e.g. weightlifting) develop increased left

ventricular wall thickness while the volume of the ventricles are maintained. However for rowers, an intermediate form of hypertrophy develops as both internal diameters of the heart and thickness of the myocardium are enlarged (Spirito *et al.*, 1994).

As confirmed here, rowing may be seen as continued weightlifting, explaining why the myocardium of rowers become enlarged. Furthermore, with VO_{2max} exceeding 6 l/min, the CO of rowers would be expected to exceed ~36 l/min (Ekblom & Hermansen, 1968). To these considerations the present results add that SV during the recovery phase of rowing becomes 36% larger than the average value, whereas the maximal obtained SV for the participants represented a ~70% (84 ml) increase and the deviation in SV would likely be larger during maximal exercise.

Rowers tend to entrain breathing during the rowing stroke at either 1:1 or 2:1 frequency ratio (Steinacker *et al.*, 1993; Siegmund *et al.*, 1999), i.e., locomotion drives respiration, which is developed with training (Mahler *et al.*, 1991). Entrainment has dual effects as it both maintains ventilation and stabilizes the upper body, assisting the propulsive force generation by increasing intra-abdominal pressure. The participants applied a 2:1 entrainment pattern at the two work rates with one breath during the drive and another during the recovery. During the drive the arterial pressure, and thereby afterload, is increased by the Valsalva-like maneuver (Pott *et al.*, 1997). At the same time, the effect of breathing is added with distinct increases in intra-abdominal pressure associated with the knees-to-chest position in the beginning of the drive and at the finish by contraction the abdominal muscles to decelerate the upper body (Manning *et al.*, 2000). In contrast, during the recovery phase no force is exerted, and intrathoracic pressure is decreased (Pott *et al.*, 1997) thereby widening the venous return gradient and SV is able to increase as depicted in this study. During the low work rate changes in SV were less apparent in accordance with the lower VT during recovery resulted in less swing in intrathoracic pressure (Lalande *et al.*, 2012). A more likely explanation, however, is that low intensity exercise is associated with low intra-abdominal pressure

in the drive (Manning *et al.*, 2000). During the 130 bpm work rate the intensity of the abdominal muscles contraction might have been different in relation to how demanding the force of the stroke was relative to the maximal capacity of the participants. Inspection of SV variation showed fluctuations in SV as indicated by the increased SVV, but the peaks occurred differently in the recovery, perhaps in relation to the relative demand of the work rate.

Similar for both work rates is the decrease in SV at the catch prior to the next stroke illustrating a biomechanical limitation. Manning et al. (2000) found that even when inhaling during the drive, i.e., the lungs are deflated when at the catch and therefore low intra-abdominal pressure would be expected, there is an increased intra-abdominal pressure, presumably due to legs compressing the chest. Conversely, exhaling during the drive, i.e. the lungs are inflated at the catch and the intra-abdominal pressure is further increased (Manning *et al.*, 2000).

Limitations to the study include the lack of a preload measure. However, the diastolic filling of the heart cannot be determined (e.g. by echocardiography) during whole-body exercise such as rowing. Preload index such as central venous pressure was intended for this study but due to inadequate data it was excluded from the analysis. The use of central venous pressure as index of preload is debated (Marik & Cavallazzi, 2013; Sondergaard *et al.*, 2015) and may indicate increases in intra-thoracic pressure rather than preload during exercise. A measure of intrathoracic or intra-abdominal pressure could elucidate the possible mechanical constraint of preload during the rowing stroke. Also, the use of Nexfin might have some limitations during exercise and not all data can be deemed plausible (Siebenmann *et al.*, 2015). In the present study CO data were compared with VO_2. Assuming a near linear relationship with ~5 l CO to a 1 l increase in VO_2, CO would be expected to be ~23 l/min during 160 bpm. Yet, this study focused on changes, i.e. investigated SV deviation. Data were inspected and artifacts were removed as Nexfin still have the advantage of continuous measure whereas more conventional methods (e.g. thermodilution) are intermittent. As the participants

performed submaximal exercise, the conclusion cannot be extrapolated to maximal exercise. However, as intra-abdominal and intrathoracic pressure increases with exercise intensity (Clifford, Hanel and Secher, 1994; Manning *et al.*, 2000) by more vigorous contraction of the abdominal muscles and less filling time with increased HR, it is assumed that maximal rowing would elicit larger SV fluctuations.

In summary, this study demonstrated significant fluctuations in SV during rowing, which increased with increasing work rate. It is assumed that these fluctuations are due to increased intrathoracic pressure mainly during the catch and thereby impeding the preload due to reduced venous pressure gradient. These observations indicate that the ability of rowers to develop a large CO to a large extent depends on SV in the recovery phase of the stroke. Thus, in accordance with the hypothesis, these results illustrate that SV fluctuates during the rowing stroke peaking in the recovery and is lowest during the catch presumably due to increased intrathoracic pressure limits preload.

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