The following letters are in response to Point:Counterpoint: Stroke volume does/does not decline during exercise at maximal effort in healthy individuals.

To the Editor: Disagreement between studies showing a decrease (3) or no decrease in the stroke volume (SV) of healthy humans approaching volitional exhaustion during large muscle mass exercise (6) can be explained by differences in methods for SV assessment, body position, training status, exercise mode and protocol, the subject’s motivation to cope with symptoms of fatigue, and individual differences in the behavior of heart rate (HR) and SV as peak exercise is approached (4). In most motivated, well trained athletes, maximal SV and cardiac output (Q) tend to level off during maximal efforts engaging large muscle mass. This is related to an unfavorable change in the balance between myocardial contractility, left ventricular filling (preload), and resistance to left ventricular emptying (afterload; Ref. 1). During graded exercise, the HR-workload relationship shows a negative acceleration in individuals where SV decreases, potentially attempting to defend diastolic filling time (4). Conversely, in ~20% of healthy, well trained individuals where SV increases, the HR-workload relationship remains linear up to maximal exertion (4). The reasons for this individuality of response are unclear, but may depend on the ability to promote high vascular conductance (e.g., low afterload) during incremental exercise (1); the contractility of the myocardium, which may depend on cardiac dimensions (5); and the ability of venous return and myocardial compliance to maintain/allow adequate cardiac filling in the face of a shrinking diastolic filling time (2). However, any two individuals may solve the equation of defending SV and, indeed, Q in different ways that are yet to be elucidated.

REFERENCES


Pierre-Marie Lepretre
Laboratoire de Biomécanique et de Physiologie
Institut National du Sport et de l’Education Physique

To the Editor: Despite the large quantity of data on LV performance during exercise, basic data on left ventricular (LV) performance are conflicting. Many of these inconsistencies can certainly be explained by methodological differences, measurement variability, supine versus upright exercise, athletes versus sedentary subjects, and different exercise protocols (1, 4).

We used contrast echocardiography to assess changes in left ventricular volumes during incremental upright submaximal bicycle exercise in male endurance athletes (2). Maximal oxygen uptake and oxygen pulse were measured by using cardiopulmonary exercise testing. We found an almost linear increase in stroke volume (SV; from 105 to 152 ml from rest to a heart rate of 160 beats/min) mainly explained by an almost linear increase in LV end-diastolic volume and an initial small decrease in end-systolic volume during upright exercise. No significant differences were observed between stroke volume calculated from LV volumes with contrast echocardiography
and SV calculated from oxygen pulse at heart rates of 130 and 160 beats/min. To explain the VAO2 during maximal exercise, the SV had to increase an additional 10% from submaximal to maximal exercise. Additional data suggesting an incremental increase in SV during upright exercise was that the mean transmural pressure gradient, peak transmural velocity, and LV filling rate showed a almost linear increase with exercise findings not easily explained by the decrease in LV filling time alone (3). In a hitherto unpublished paper, we showed that the increase in LV end-diastolic volume is explained by a significant augment in the inner transverse LV diameters creating a more spherical LV end-diastolic cavity during exercise.

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Egil Henriksen
M. Sundstedt
P. Hedberg
Department of Clin Physiol
Central Hospital
Västraas Sweden

To the Editor: Endurance training, resistance training, body composition, or different disease states can lead to diverse adaptations in cardiac structure and/or function. It is currently unknown if these adaptations contribute to the ability, or the inability, to maintain SV during near-maximal or maximal exercise.

Contrary to Warburton and Gledhill’s (6) statement that the SV response to exercise is “highly reliant on maximal effort,” not all endurance-trained individuals maintain SV (3, 4) and not all sedentary individuals show a drop in SV at maximal exercise (2). These responses have been demonstrated in cross-sectional and longitudinal studies (3–5).

Gonzalez-Alonso (1) correctly points out that most of the published research in this area has not measured SV at maximal exercise and, thus, SV may have been overestimated. However, we (5) measured cardiac output and SV (CO2 rebreathing) 20–30 s prior to volitional fatigue and demonstrated three different SV responses at maximal exercise: drop, plateau, and continued increase. Scientists must refrain from concluding absolute interpretations, resulting in oversimplified explanations and understanding. This is especially true in exercise physiology, where multiple control systems combine with a multitude of individual differences in numerous exercise scenarios to create varied physiological responses.

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Chantal A. Vella
Robert A. Robergs
University of Texas at El Paso

To the Editor: The parties involved in the debate (4, 6) appear to be discussing two different experimental conditions: incremental vs. constant-power cycling. On the basis of the title of the original Point:Counterpoint, we will offer our opinion on the SV response during or throughout 4–8 min of constant-power exercise that elicits VO2max. Only a few investigations have reported SV and cardiac function during several minutes at constant maximal aerobic intensity (2, 5). These investigations have shown that SV reached maximal values 2–3 minutes into the exercise bout and then decreased prior to fatigue. Cardiac output (CO) and oxygen delivery also declined prior to exhaustion, suggesting a central limitation (e.g., myocardial) to maximal aerobic power. Unfortunately these data demonstrating reduced SV after several minutes of exercise at 95–100% VO2max were compiled primarily by one laboratory using invasive techniques (2, 5). Therefore, other laboratories should consider investigating these fundamental cardiovascular responses to maximal exercise. Data pertaining to prolonged exercise at 65–75% VO2max with ensuing hyperthermia (1, 3) should not be ignored as the pattern of cardiovascular instability (reduced SV, CO, and MAP) is remarkably similar and just accelerated during high intensity exercise compared with prolonged moderate-intensity exercise. A common denominator linking high-intensity, short-term exercise and prolonged moderate-intensity exercise eliciting dehydration may be the development of hyperthermia (esophageal temperature of 39.3°C), which has been shown to reduce SV by 8% independent of dehydration and elevated skin temperature (4).

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To the Editor: This (4, 8) is the same debate as that on the occurrence of a VO$_2$ drop after the achievement of a VO$_2$ peak or VO$_2$max plateau. For incremental model (7), the stroke volume should be expressed per unit of time since the independent factor is the power output for comparing stroke volume independently of the heart rate values. For constant load exercise, we must distinguish exercise below and above the maximal lactate steady state and not consider the intensity in percentage of since catecholamine concentration influences the vascular regulation (3, 5, 6). Indeed, exercise at 80% of can be below the maximal lactate steady state in well trained endurance athletes and above for sprinters (2). The kinetics of oxygen uptake must also be taken into consideration and the stroke volume must be expressed per unit of oxygen uptake when is not stable (1). In a marathon, stroke volume increased until the 24th kilometer and then was maintained despite a decrease in speed (personal data). Consequently, the stroke volume per unit of meter ran increased while the number of heart beats per unit of meter run was maintained constant. This result could be due to a cardiovascular endurance training adaptation for avoiding the increase in heart beat (and then the myocardium oxygen consumption) as reported for incremental exercise (7). Free-pace exercise models must also be studied since sudden deaths occur and the stroke volume kinetics could be one candidate for this sudden death due to dehydration or heart pathology.

REFERENCES