Point:Counterpoint: Stroke volume does/does not decline during exercise at maximal effort in healthy individuals

POINT: STROKE VOLUME DOES DECLINE DURING EXERCISE AT MAXIMAL EFFORT IN HEALTHY INDIVIDUALS

A widely held theory explaining the physiological factors limiting maximal aerobic power \( (V_{\text{O2 max}}) \) in humans postulates that the perfusion capacity of skeletal muscle greatly exceeds the capacity of the heart to pump blood into the systemic circulation (1, 2, 20, 22, 27). This theory implies that any human being would be able to recruit more motor units and produce more work during maximal exercise if cardiac output (\( Q \)) did not reach such a hypothetical upper limit. In this debate, I will argue that the decline in stroke volume (SV) underpinning the plateau or drop in \( Q \) during incremental and constant maximal exercise supports the idea of a regulatory limit of the heart. The approach is necessarily integrative to be able to support the contention that SV does decline during exercise at maximal effort in healthy individuals. It is emphasized that the question under debate is whether the SV value preceding fatigue (i.e., 10–30 s before exhaustion) is lower than the values observed at submaximal intensities during incremental exercise to exhaustion or during the initial stages of maximal constant-load exercise. It is important to note, however, that due to methodological difficulties, only a handful of studies in the literature report measures of SV and \( Q \) in the few seconds before exhaustion. The common observation that heart rate and systemic \( a-V_O2 \) difference increase progressively during exercise to maximum makes the behavior of \( Q \) the central issue of the debate.

A number of observations made with a variety of experimental techniques provide evidence that SV declines during maximal incremental exercise in humans, a response that accompanies the attenuation in the rate of rise, the plateau, or the decline in \( Q \) (9, 10, 12, 14, 16, 18, 21, 25, 26, 28, 29). In the early 1980s, Keul et al. (16) provided the first echocardiographic data supporting that SV falls during upright cycling to exhaustion. Using right heart catheterization, radionuclide angiography, and expired gas analysis, Higginbotham et al. (14) later showed a slight decline in SV at peak exercise following the “classic” plateau response from 40–50% \( V_{\text{O2 max}} \) (3), which was attributed to the effects of tachycardia on left ventricular filling. In the same year and using a dye-dilution method for determining \( Q \), Yamaguchi et al. (30) reported a decline in SV during incremental supine cycling in 31 subjects showing an attenuation or levelling off in \( Q \). Adding inconsistency to their results, however, they reported a plateau in SV in 9 subjects displaying a linear increase in \( Q \) and a conflicting levelling off in the calculated systemic \( a-V_O2 \) difference above 50% \( V_{\text{O2 max}} \) (30). In the 1990s, a number of independent research groups showed an 8–11% decline in SV at peak exercise using angiographic, acetylene rebreathing, and echocardiographic techniques (9, 25, 26). Recent experiments employing the Fick principle to estimate \( Q \) expand on these early observations by showing a significant fall in SV during incremental exercise accompanying nonlinear locomotor muscle blood flow and \( Q \) dynamics leading to a plateau in exercising limb muscle \( V_{\text{O2}} \) (19, 28, 29). Of note is the strikingly similar pattern of response in Stringer et al. (28, 29) and in our recent study during incremental exercise to exhaustion (19). Both studies found a significant decline in SV at peak exercise compared with 50% \( V_{\text{O2 max}} \), which accompanied the attenuation and subsequent plateau in \( Q \) above 90% \( V_{\text{O2 max}} \) (19, 28, 29). Because the methods available to assess heart rate, \( a-V_O2 \) difference, and \( V_{\text{O2}} \) are more sensitive than the methods used to measure SV or \( Q \), a concerted examination of components of the Fick equation (\( V_{\text{O2}} = Q \times a-V_O2 \) difference, where \( Q = SV \times \text{heart rate} \)) is warranted to ascertain the validity of the measured or estimated SV. In our study, heart rate and \( V_{\text{O2}} \) increased in a linear fashion (\( r^2 = 0.99; P < 0.001 \)) in agreement with studies in the literature. Interestingly, Stringer et al. (28, 29) showed that systemic \( a-V_O2 \) differences display a linear rather than a hyperbolic profile, thus indicating that \( Q \) as a function of \( V_{\text{O2}} \) must be curvilinear and that SV must decline during incremental exercise to exhaustion. Indeed, direct measures of blood oxygen content demonstrate that systemic and leg oxygen extraction increases continuously until exhaustion, reaching values ranging from 80 to 95%, with the extraction across the exercising legs being higher than across the systemic circulation (6, 10, 12, 23, 24). The continuously increasing \( a-V_O2 \) difference and the linear increase in heart rate during incremental exhausting exercise suggest that the fall in SV may be more accentuated in individuals showing a plateau in \( V_{\text{O2 max}} \) because \( Q \) must decline in that setting (4, 17).

Another approach to determine whether SV declines during exercise at maximal effort is to examine the hemodynamic responses to short duration (3–10 min) constant-load exercise. An advantage of this protocol is that it assesses the capacity of the body to sustain \( V_{\text{O2 max}} \) and the capacity of the cardiovascular system to sustain maximal systemic and locomotor muscle blood flow and \( O_2 \) delivery. In three studies involving 29 subjects, we consistently observed significant reductions in SV and \( Q \) prior to exhaustion in both the presence and absence of heat stress (10, 12, 19). These responses were associated with high core temperature, catecholamines, and plasma ATP, near-maximal heart rate, altered or stable central venous, and mean arterial pressures and reduced locomotor muscle blood flow. This suggests that the fall in SV might have resulted from the interaction of several factors transiently depressing preload and/or left ventricular function. The similar cardiovascular instability during exhausting incremental and constant-load exercise points toward an upper limit in cardiovascular regulation, which might implicate both central and peripheral factors (4–6, 10, 12, 19). Interestingly, the rate-pressure product of heart rate and mean arterial pressure increases until exhaustion, indicating that myocardial oxygen demand is rising when SV declines during constant and incremental maximal exercise. Under these conditions, an increase in myocardial \( V_{\text{O2}} \) can only occur by an increase in \( O_2 \) delivery provided by augmented coronary blood flow because the \( O_2 \) extraction reserve is minimal. The blunted \( Q \) raises the possibility that impaired coronary circulation sets a limit to cardiac function (19), an inference already advanced by Hill and Lupton (15) 84 years ago.
The cardiovascular instability described here is not unique to maximal exercise. A similar drop in SV occurs during prolonged submaximal exercise as part of the classical phenomenon termed “cardiovascular drift” (7, 8) or the cardiovascular strain evoked by severe dehydration and hyperthermia, which also features reductions in Q and exercising muscle blood flow (11, 13). In conclusion, comprehensive studies measuring Q and other components of the Fick principle during both submaximal and maximal exercise also feature reductions in Q and exercising muscle blood flow evoked by severe dehydration and hyperthermia, which is termed “cardiovascular drift” (7, 8) or the cardiovascular strain evoked by severe dehydration and hyperthermia. These observations are consistent with the idea of a central limitation to VO_2max.

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COUNTERPOINT: STROKE VOLUME DOES NOT DECLINE DURING EXERCISE AT MAXIMAL EFFORT IN HEALTHY INDIVIDUALS

Lack of agreement regarding the stroke volume (SV) response to exercise is not new; in fact it dates back to the mid 1900s. However, in 1994 Gledhill et al. (9) provided clear evidence that the SV of endurance athletes increases throughout incremental to maximal exercise while the SV of untrained individuals plateaus early in progressive exercise. Considerable previous and subsequent findings strongly support the ability of the healthy human heart to maintain and even increase SV during short-term, incremental upright exercise. Nevertheless, the evidence that SV does not decline but is maintained and even increases during maximal exercise is still questioned, as reflected by this Point:Counterpoint debate. Fol-