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 flow (11, 13). In conclusion, comprehensive studies measuring Q and other components of the Fick principle during both flow (11, 13).

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GRANTS

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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-
lowing is the chronologically ordered evidence to support our "point."

Research on humans in 1950s and 1960s illustrated that there were varied SV responses to exercise, but in general it supported the contention that SV could increase during incremental exercise with no decline during maximal exercise (2). For example, Chapman et al. (2) reported that although there was variability among individuals, the mean SV of 26 healthy men increased progressively during incremental exercise. Often overlooked from the landmark work of Astrand et al. (1) is the fact that 11 of the 23 (48%) participants reached their highest SV during maximal exercise. It is important to point out that the average fitness levels of these participants suggests that they were normally to moderately active (females = 41.4 and males = 54 ml·kg⁻¹·min⁻¹).

Grimby et al. (11) reported that four of nine (44%) endurance-trained masters level athletes (mean \(\bar{V}_O_{2}\max = 51.5\) ml·kg⁻¹·min⁻¹) attained their highest SV during maximal exercise. Similarly, Ekbloem and Hermansen (5) reported a progressive increase in SV with increasing exercise intensity (between 40 and 80% \(\bar{V}_O_{2}\max\)) in eight elite (\(\bar{V}_O_{2}\max = 74.6\) ml·kg⁻¹·min⁻¹) and five regional level (\(\bar{V}_O_{2}\max = 66.0\) ml·kg⁻¹·min⁻¹) endurance athletes. Nine of these athletes (69%) achieved their highest SV during maximal exercise.

In the 1970s and 1980s, a number of investigators using both non-invasive and invasive measurement techniques supported the ability of humans (in particular endurance athletes) to increase SV during incremental exercise (3, 17, 18). For example, Spriet et al. (18) using dye dilution, found that elite endurance runners increased their SV while progressing from 91 to 100% \(\bar{V}_O_{2}\max\).

Recent literature has provided even more compelling evidence that endurance-trained individuals increase their SV throughout incremental to maximal exercise (9, 13, 25, 26, 28, 29). This SV response has been reported by several international laboratories using various (invasive and noninvasive) techniques in a variety of populations: young men (4, 9, 13, 14, 16, 19, 29, 30), young women (8, 16, 29), older women (28), and even patients with heart disease (12, 27). An analysis of individual SV responses to exercise indicates that many untrained or moderately trained individuals achieve their highest SV during maximal exercise (1, 8, 9, 13) and close to 100% of endurance athletes achieve their highest SV during maximal exercise (8, 9, 13, 19, 25; Fig. 1).

Collectively, the evidence is overwhelming that the SV of endurance-trained athletes is maintained and generally increased throughout incremental to maximal exercise. The only question concerns the prevalence of this response in untrained and moderately trained individuals. Investigators reported that the SV of untrained individuals plateaus at submaximal exercise levels and may decline at near maximal exercise (16, 30), and others have reported a small increase in SV during maximal exercise (2, 8, 9, 13, 15, 22). It is important to point out that, generally, when a supramaximal exercise protocol was employed, untrained individuals exhibited a small, secondary increase in SV during maximal exercise (8, 9, 13, 15). Additionally, untrained individuals with high blood volumes increased their SV during maximal exercise (15).

As discussed in the early 1970s (21), the "possibility exists that the intensity of exercise induced by previous studies in which left ventricular dimensions were measured in experimental animals or man was not maximal and that the Frank-Starling mechanism does play a role in the response to severe exertion." This is a very important point, as SV often shows smaller increases at submaximal exercise intensities (when multiple exercise stages are employed) and then reaches its highest level at maximal exercise (9, 13, 25, 28, 29). This point also raises the necessity of using SV measurement techniques that are valid and reliable during maximal exercise (23, 24).

Certainly there is variability among individuals in the SV response to exercise that appears to be highly reliant on fitness level. For example, similar to Janicki and coworkers (12) in patients with chronic heart failure, we identified three major SV patterns during exercise. Most endurance-trained athletes exhibit an increase in SV throughout incremental to maximal exercise (9, 13, 19, 26). However, there is considerably more variability in the response of untrained and moderately trained individuals. Some (like the endurance-trained individuals) are able to progressively increase their SV during strenuous exercise, some exhibit a plateau at submaximal levels and then maintain their SV at or near this level throughout incremental exercise, and some display a reduction in SV at higher exercise intensities. In our experience, only the least fit exhibit a decrease in SV at higher intensities, a pattern that is consistent with reduced myocardial compliance and marked pericardial constraint (12, 27).

Body position clearly affects the SV response to exercise and likely explains some of the discrepancies in the literature (26). In the supine position, the myocardium appears to approach its limits for diastolic filling (i.e., a reduced diastolic reserve capacity) and although endurance athletes are still able to increase their end-diastolic volume and SV during maximal exercise in the supine position, the relative changes are significantly smaller than that observed during upright exercise (26). Thus the SV response to exercise is highly dependent on the loading conditions of the heart and the interplay of intra- and extramyocardial factors (6, 7).

There are several well-described mechanisms that collectively maintain (or increase) SV at high heart rates in healthy humans, including increased venous return (via the abdomino-
thoracic and skeletal muscle pump and venoconstriction), increased atrial and ventricular inotropy, and enhanced lusitropy (ventricular relaxation; Ref. 10). Moreover, there are several training-induced changes in diastolic and systolic function that allow endurance-trained athletes to increase their SV throughout progressive exercise. Diastolic filling, in particular, appears to be enhanced throughout incremental to maximal exercise (despite a reduced time for diastolic filling) (9, 13, 20, 26). Several training-related adaptations are thought to enhance the capacity for diastolic filling, including increased myocardial compliance, reduced diastolic ventricular interaction, increased left ventricular internal cavity dimensions, increased early filling (i.e., E/A ratio), increased transmitial pressure gradient and flow velocity (e.g., enhanced diastolic suction), training-induced hypervolemia, increased rate of LV pressure decline (−dP/dt), and/or increased rate of calcium uptake within the sarcoplasmic reticulum (6, 10, 20).

In summary, the evidence is compelling that the human heart is able to maintain and even increase SV during maximal exercise. The only question in our opinion concerns the prevalence of this response in untrained and moderately trained individuals.

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REBUTTAL FROM DR. GONZÁLEZ-ALONSO

Plato’s Myth of the Cave came to mind when reading my opponents’ argument: “... the SV of endurance-trained athletes is maintained and generally increased throughout incre-