Stroke volume does not plateau during graded exercise in elite male distance runners

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ABSTRACT

ZHOU, B., R. K. CONLEE, R. JENSEN, G. W. FELLINGHAM, J. D. GEORGE, and A. G. FISHER. Stroke volume does not plateau during graded exercise in elite male distance runners. *Med. Sci. Sports Exerc.*, Vol. 33, No. 11, 2001, pp. 1849–1854. Stroke volume (SV) responses during graded treadmill exercise were studied in 1) elite male distance runners ($N = 5$), 2) male university distance runners ($N = 10$), and 3) male untrained university students ($N = 10$). **Methods:** Cardiac output ($\dot{Q}$) and SV were determined by a modified acetylene rebreathing procedure. **Results:** There were no differences in SV responses among the three groups during the transition from rest to light exercise ($P > 0.05$). However, the rates of change of SV during light to maximal exercise in untrained subjects (slope = -0.1544 mL·beat^{-1}) and university distance runners (slope = 0.1041) did not change, whereas it dramatically increased ($P < 0.001$) in elite distant runners (slope = 0.6734). Moreover, the elite distance runners showed a further slope increase in SV when heart rate was above 160 bpm, which resulted in an average maximal SV of 187 ± 14 mL·beat^{-1} compared with 145 ± 8 and 128 ± 14 mL·beat^{-1} in the university runners and untrained students, respectively ($P < 0.001$). Similarly, max $\dot{Q}$ reached 33.8 ± 2.3, 26.3 ± 1.7, and 21.3 ± 1.5 L·min^{-1} in the three groups, respectively ($P < 0.001$). On the other hand, there was a nonsignificant tendency for maximal arteriovenous oxygen content difference to be lower in the elite athletes compared with the other groups. **Conclusion:** Results from university distance runners and untrained university students support the classic observation that SV plateaus at about 40% of maximal oxygen consumption despite increasing intensity of exercise. In contrast, stroke volume in the elite athletes does not plateau but increases continuously with increasing intensity of exercise over the full range of the incremental exercise test.
Key Words: CARDIAC OUTPUT, ACETYLENE REBREATHING TECHNIQUE, SLOPE ANALYSIS, INCREMENTAL EXERCISE, ARTERIOVENOUS OXYGEN DIFFERENCE.

Exercise physiologists have taught that both stroke volume (SV) and heart rate (HR) increase linearly during graded exercise until about 40% of maximal oxygen consumption ($\dot{V}O_{2max}$) (14,25). Beyond that point, increases in cardiac output ($\dot{Q}$) are dependant only on increases in HR, because SV plateaus or increases only slightly in both untrained and trained endurance athletes (1,8,13,19). The plateau in SV was thought to be related to a progressively diminishing time available for diastolic filling of the left ventricle with increasing intensity of exercise (17). However, Gledhill et al. (10), using an acetylene rebreathing technique, recently found that the SV of highly trained endurance cyclists increased progressively from rest to maximal exercise without a plateau. The work by Gledhill and coworkers (10) raised several questions regarding the classic SV theory: 1) Do all highly trained endurance athletes increase SV throughout the entire range of graded exercise, or 2) does this SV increase only occur in highly trained cyclists; and, 3) is there a difference in SV response to graded exercise in subjects of different training status? To answer these questions, we studied the SV response to increasing intensities of exercise in untrained university students, trained university distance runners, and elite distance runners.

METHODS

Subjects. Ten male untrained university students, 10 male university distance runners, and 5 male elite distance runners participated in this investigation. The untrained subjects were undergraduate and graduate physical education students. The university runners were members of the Brigham Young University cross-country team. The elite distance runners were professional runners, who could complete a 5-km race within 14 min, a 10-km race within 28 min, and/or a marathon race within 2 h and 15 min. The study was approved by the Institutional Review Boards at Brigham Young University (Provo, UT) and LDS Hospital (Salt Lake City, UT), and all subjects signed a written informed consent form.

Before testing, all subjects were familiarized with the treadmill and the acetylene rebreathing protocol. The subjects then underwent a graded exercise test (GXT) during which all performance variables were assessed.

0195-9131/01/3311-1849/$3.00/0
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Submitted for publication September 2000.
Accepted for publication January 2001.

The treadmill graded exercise test. The first stage of the GXT involved three-min of warm up at approximately 40% of the participant's maximal oxygen consumption. This first stage is defined as light exercise. The second stage was a self-selected, comfortable running/jogging pace (5–12 mph) for 3 min. The work rate of each subsequent stage was increased by raising the treadmill grade 2–3% every 3 min until exhaustion.

Cardiac output ($\dot{Q}$), stroke volume (SV), and heart rate (HR) measurement. $\dot{Q}$ was measured at rest and during the last 30 s of each stage of the GXT by using the method initiated by Triebwasser et al. (21) and redeveloped into the computerized rebreathing system by Jensen et al. (12). The estimated coefficient of variation of the technique for repeated measurements in a population of 86 healthy subjects (51 men and 35 women) was 6.8% (12). The correlation coefficients of the test-retest for resting and maximal values in our lab were 0.99 ($P < 0.01$) and 0.93 ($P < 0.05$) respectively. A modification of the equipment and technique was made so that both $\dot{Q}$ and $\dot{V}O_{2max}$ could be measured during the test (7). A pneumatic switch kept the bag system closed for the first 2 min of each stage of the GXT to allow subjects to breathe room air. After 2 min, subjects were instructed to open the pneumatic valve at end expiration. The mixture of gas in the bag was 1% acetylene, 9.18% helium, 40% oxygen, and 48.82% nitrogen. This mixture was breathed for eight complete breathes at rest and six breathes during each stage of exercise. The volume of the gas in the bag varied according to the work rate, body mass, vital capacity, and training level of the participants.

The changes in concentration of acetylene and helium during rebreathing were measured by a mass spectrometer and digitized at 100 Hz by a computer (Marquette 1100, St. Louis, MO) for storage and processing of the data. Typically, the concentration of helium did not change after the second breath, but the concentration of acetylene continued to decline after each sequential expiration due to the uptake of acetylene by the capillary blood. Thus, $\dot{Q}$ was calculated from the exponential disappearance rate of acetylene relative to helium during the rebreathing procedure. The slope of the acetylene concentration curve relative to the helium concentration curve is proportional to the rate of blood flow through the lungs or $\dot{Q}$. Blood flow, in turn, depends on the work rate; thus the steeper the $[C_2H_2]$ slope, the greater the $\dot{Q}$ as calculated by the equations used by Cander and Forster (2).

An electronic HR monitor was used to record the HR at rest and during each stage of exercise while $\dot{Q}$ was measured. Stroke volume of each subject at rest and during each stage of the GXT was obtained by the equation: $SV = \dot{Q} \div HR$.

Maximal oxygen consumption ($\dot{V}O_{2\ max}$) measurement. Maximal oxygen consumption was determined using a standard open-circuit system in which expired volumes were measured by a Fleish Pneumotach (Hans Rudolph, Model 3813, Kansas City, MO), and the concentrations of oxygen and carbon dioxide were quantified by a medical gas analyzer (Marquette 1100 Mass Spectrometer).

TABLE 1. Subject characteristics (mean ± SEM).

Groups	University Students ($N = 10$)	University Distance Runners ($N = 10$)	Elite Distance Runners ($N = 5$)
Age (yr)	28.1 ± 7.5	25.5 ± 4.3	29.8 ± 5.2
Height (cm)	178 ± 7.3	179 ± 4.3	179 ± 6.5
Body Mass (kg)	76.5 ± 11.5*	66.8 ± 3.7	63.6 ± 1.5
HR_{max} (beats·min^{-1})	186 ± 8.0	187 ± 8.6	180 ± 4.0
$\dot{V}O_{2max}$ (L·min^{-1})	3.76 ± 0.4*	4.81 ± 0.3*	5.35 ± 0.1*
$\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1})	48.9 ± 5.2*	72.1 ± 6.3*	84.1 ± 0.9*
$\dot{Q}_{max}$ (L·min^{-1})	21.3 ± 1.5*	26.3 ± 1.7*	33.8 ± 2.3*
SV_{max} (mL·beat^{-1})	128 ± 14.0*	145 ± 7.7*	187 ± 14.5*

* Denotes significantly different from other groups ($P < 0.001$).
HR_{max}, maximal heart rate; $\dot{V}O_{2max}$, maximal oxygen consumption; $\dot{Q}_{max}$, maximal cardiac output; and SV_{max}, maximal stroke volume.

Data from the pneumotachometer and mass spectrometer were transferred to a computer that calculated $\dot{V}O_2$ by using software developed by Consensus Technologies (Salt Lake City, UT). Maximal oxygen consumption was considered to be reached when two of three criteria were satisfied: $\dot{V}O_2$ leveling off despite an increase in work rate, heart rate greater than 90% of the age-predicted maximal value (220 − age), and a respiratory exchange ratio (RER) greater than 1.10 (4).

Statistical analysis. The differences of age, height, body mass, HR_{max}, $\dot{V}O_{2max}$, $\dot{Q}_{max}$, and SV_{max} among the three groups were tested using ANOVA (Proc GLM in SAS). The general linear models procedure was used to determine whether there were significant treatment effects. If there was a treatment effect, then Duncan's multiple range test was employed to determine significant differences between groups. A random coefficient growth curve model (Proc Mixed in SAS) was utilized to detect if the differences in the slopes describing the changes in $\dot{Q}$ and SV during the GXT among the three groups of subjects were significant. Statistical significance was set at the level of $P < 0.05$.

RESULTS

The subject characteristics are presented in Table 1. There were no significant differences in age and height among the three groups. However, both university and elite distance runners had a significantly lower body mass compared with the untrained university students ($P < 0.001$). The maximal heart rate (HR_{max}) was slightly, but not significantly ($P > 0.05$), lower in elite runners than in either of the other two groups.

When compared with the other groups, the elite runners exhibited the highest maximal oxygen consumption ($\dot{V}O_{2max}$), maximal cardiac output ($\dot{Q}_{max}$), and maximal stroke volume (SV_{max}) during maximal exercise ($P < 0.001$; Table 1). The values for $\dot{V}O_{2max}$, $\dot{Q}_{max}$, and SV_{max} were also higher in university distance runners compared with untrained university students ($P < 0.001$).

$\dot{Q}$ increased progressively from rest to maximal exercise in all three groups of subjects. When compared with the other groups, $\dot{Q}$ of the elite distance runners increased at a greater rate from light to maximal exercise (Fig. 1). Differences in the rates of increase of $\dot{Q}$ from light to maximal exercise among the three groups were statistically signifi-

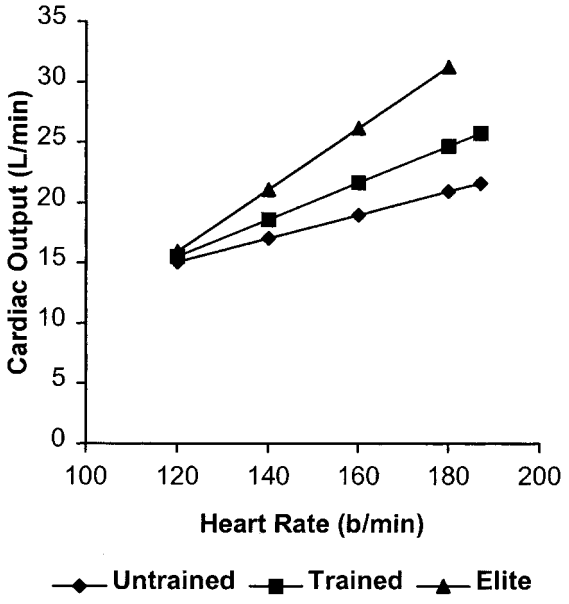

FIGURE 1—Regression lines of cardiac output from light to maximal exercise in three groups of subjects.

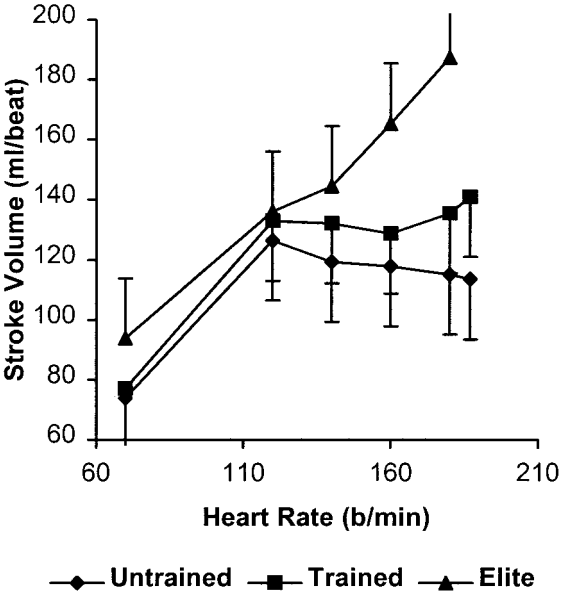

FIGURE 2—Stroke volume responses from rest to maximal exercise of three experimental groups (see Table 2 for statement of significance).

cant ($P < 0.001$; Table 2), with the elite runners having the greatest rate of increase and university runners the second greatest rate of increase of the three measured.

It is interesting that SV responses from rest to light exercise were the same ($P > 0.05$) in all three groups (Fig. 2, Table 2). However, from light to maximal exercise, the rate of change of SV showed a downward trend in untrained university students, an upward trend in university distance runners, and a steep rate increase in elite distance runners, where the rate of change of SV is equal to the change in the number of mL·beat^{-1} for each beat·min^{-1} change of heart rate (Fig. 2). The differences of the slopes among the three groups were significant ($P < 0.01$; Table 2). A change point analysis of each slope revealed no statistically significant change from zero in the slope of SV with increased HR for either the untrained university students or the university distance runners. However, the change point analysis showed a significant ($P < 0.05$) change in slope for the elite distance runners, which occurred at 89% of $HR_{max.}$ (The change point analysis requires that the independent variable be on the same scale for each individual, so % HR_{max} rather than absolute HR was used.) The slope changed from 49.5 slope units to 357 when % HR_{max} went above 89% (see Fig. 3).

TABLE 2. Comparisons of the slopes of $\dot{Q}$ and SV from rest to light or from light to maximal exercise (mean ± SEM).

	University Students (N = 10)	University Distance Runners (N = 10)	Elite Distance Runners (N = 5)
Slope of $\dot{Q}$ from rest to light exercise	0.2032 ± 0.014	0.2015 ± 0.013	0.1772 ± 0.018
Slope of $\dot{Q}$ from light to max exercise	0.0968 ± 0.009**	0.1515 ± 0.010**	0.2538 ± 0.012**
Slope of SV from rest to light exercise	0.9622 ± 0.156	0.9087 ± 0.142	0.7175 ± 0.200
Slope of SV from light to max exercise	−0.1544 ± 0.069*	0.1041 ± 0.069*	0.6734 ± 0.088*

* Denotes significantly different from other groups ($P < 0.01$).
** Denotes significantly different from other groups ($P < 0.001$).
$\dot{Q}$, cardiac output and SV, stroke volume; slopes are expressed as unit change of stroke volume (mL·beat^{-}) or cardiac output (L·min^{-1}) per unit change of heart rate (beat·min^{-1}).

Maximal arteriovenous oxygen differences [$C(a\text{-}\bar{v})O_2$] in the three groups of subjects were calculated from the equation $\dot{V}O_{2max} = (\dot{Q}_{max}) \times [C(a\text{-}\bar{v})O_2]$. The means of the [$C(a\text{-}\bar{v})O_2$] were 174.8 ± 21.4 mL·L^{-1} in university students, 183.5 ± 20.6 mL·L^{-1} in university distance runners, and 159.2 ± 14.5 mL·L^{-1} in elite distance runners. Although the trend for a lower value in the elite group is apparent, the differences among the three groups of participants were not statistically significant ($P > 0.10$).

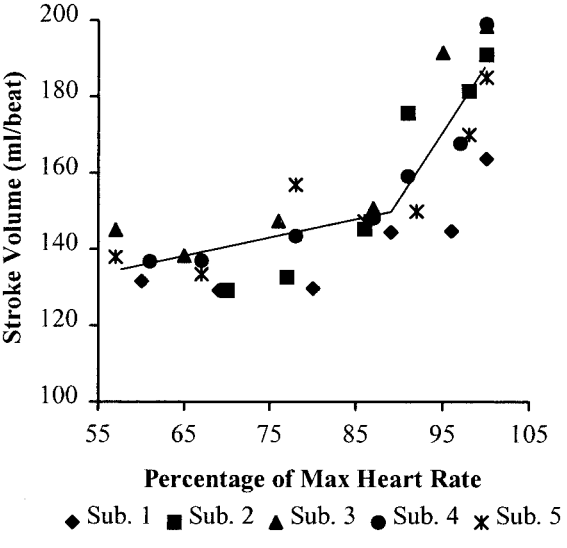

FIGURE 3—Individual stroke volume responses from rest to maximal exercise of world-class male distance runners.

DISCUSSION

It is widely accepted that stroke volume (SV) increases progressively and in a similar magnitude from rest to light exercise in all individuals, regardless of training state (1,18). That concept is supported by the results of this study. It has also been widely accepted that increasing intensity of exercise above 40% of maximal oxygen consumption results in no further appreciable increase in SV (1,6,14,25). For instance, Åstrand et al. (1), using a dye-dilution technique to measure $\dot{Q}$, suggested that maximal SV in active physical education students was essentially reached at a workload of 40–50% $\dot{V}O_{2max}$. Using the acetylene rebreathing method, Spina and colleagues (20) showed that SV of 12 sedentary adults declined or plateaued, whereas work load was above 50% of maximal oxygen uptake before and after 12 wk of endurance training. In addition, the results of Christie et al. (3), who used the methods of both Doppler echocardiography and thermodilution, showed that maximal SV of 17 healthy adult men was reached at the first stage of the graded treadmill exercise. The results from the untrained and trained university students in the present study support those previous findings. That is, SV in those groups did not increase appreciably with increasing intensity of exercise above the values recorded at light exercise.

The unique finding of the present study was that during the graded exercise test, the stroke volume of elite distance runners continued to increase without a plateau (Figs. 2 and 3). As shown in Figure 2, SV from light to maximal exercise increased about 52 mL in elite distance runners compared with an increase of only about 12 mL in university distance runners and a decrease of 15 mL in untrained university students. Our findings in elite runners are similar to the responses of elite cyclists reported by Gledhill et al. (10). Using the acetylene rebreathing technique similar to that employed in the present study, those authors showed for the first time that SV continued to rise with increasing intensity of exercise in elite bicyclists. Based on the observations of Gledhill and colleagues (10) and those in the present study, we conclude that the continuous increase in stroke volume during exercise of increasing intensity is not limited to elite bicyclists but can occur in elite runners as well. We have quantified this phenomenon by performing a change point analysis on the three groups. There is no indication of a change in slope of SV with increased HR for either the untrained university students or the university distance runners. However, the analysis showed a significant ($P < 0.05$) change in slope for the elite distance runners (Fig. 3), which occurred at 89% of $HR_{max.}$ This observation has not been reported previously.

From our study, it is unclear whether the continued increase in SV with increasing intensity of exercise in elite runners is an adaptation to training, a consequence of genetics, or a combination of both influences. Because this was a cross-sectional study, we could not quantify the differences in training, other than to conclude that the university students were not engaged in any training. Because the other two groups had trained for years and we still observed differences in the stroke volume responses of the two groups, we assume that both genetics and training contributed to the differences. Regardless of whether the differences are due to genetics or training, the mechanisms responsible for the different SV responses from light to maximal exercise are not apparent from the present study. However, it was suggested by Gledhill et al. (10) that at a heart rate of 190 bpm, the corresponding rate of ventricular emptying and the rate of ventricular filling of highly trained endurance cyclists versus untrained subjects were 20% and 71% greater, respectively. In contrast, in the study by Ginzton et al. (9), the authors found that end-diastolic filling and stroke volume actually decreased at maximal exercise in nonelite endurance athletes and sedentary controls. Therefore, although endurance-trained athletes appear to rely on enhancements in both ventricular filling and emptying to augment SV, by far their major advantage over untrained subjects according to Gledhill and associates is in ventricular filling (10). Why athletes can achieve a greater ventricular filling and a subsequent stroke volume is unknown, but it may be a function of several factors (16). An increased ventricular filling could be related to an increased blood volume seen in trained individuals (11). However, the increased blood volume should be present in both the elite subjects and the well-trained university runners, because it is a general adaptation to training; therefore, that mechanism would not explain the increase in SV in the elite group. Another factor proposed by Rowell and colleagues (16) is a reduction in pericardial resistance, which would allow the ventricle to stretch more during periods of rapid filling, thus providing greater contractility (Frank-Starling mechanism) of the stretched myocardium and a subsequent increase in ejection fraction (16). There are no reports of reduced pericardial resistance in elite athletes, so that explanation awaits confirmation. Finally, another mechanism that could possibly explain the increased stroke volume in the elite runner could be increased heart size. Chronic and prolonged training could lead to increases in the thickness of the posterior and septal walls of the left ventricle resulting in greater force of contraction at high intensities of exercise providing greater ejection fractions and higher stroke volumes. Such adaptations have been observed in endurance trained subjects (22).

As we expected, $\dot{Q}$ increased progressively from rest to maximal exercise in all three groups of participants. The rate of increase in $\dot{Q}$ was similar from rest to light exercise but significantly different during light to maximal exercise in the three groups ($P < 0.001$; Table 2). Our values for $\dot{Q}_{max}$ in both untrained university students and trained distance runners are similar to the results of previous studies in which different methodologies were used to measure $\dot{Q}$ in different populations (Table 3). This suggests that the acetylene rebreathing technique can be used with confidence to assess $\dot{Q}$ throughout incremental exercise, including maximal work rate (23).

It is interesting to note the trend for a lower $C(a\text{-}\bar{v})O_2$ in the elite subjects at the exercise intensity that elicited $\dot{V}O_{2max}$ compared with the other groups (paragraph 5 of

TABLE 3. Summary from the literature of $\dot{Q}$ and SV measurements during exercise.

Ref. No.	Investigator	Subject Status	HR_{max} (beats·min^{-1})	$\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1})	$\dot{Q}_{max}$ (L·min^{-1})	SV_{max} (mL·beat^{-1})	Plateau in SV
	Present	Untrained	186	48.3	21.3	127.8	Yes
		Trained	187	71.5	26.3	144.9	Yes
		Elite	180	83.5	33.8	187.4	No
1	Åstrand	Active	186	54.1	24.1	132.0	Yes
3	Christie	Untrained	180	NA	21.4	121.0	Yes
		Untrained	180	NA	18.2	101.0	Yes
6	Ekblom	Elite	190	73.9	36.0	189.0	NA
10	Gledhill	Trained	NA	68.8	31.0	NA	NA
		Untrained	192	44.1	24.5	127.6	Yes
		Elite	190	68.6	34.8	183.0	No
13	Moon	Active	187	53.1	30.1	161.0	Yes
20	Spina	Pretrain	185	46.0	21.0	113.5	Yes
		Posttrain	180	55.0	23.0	127.8	Yes

Ref. No., reference number; HR_{max}, maximal heart rate; $\dot{Q}_{max}$, maximal cardiac output; and SV_{max}, maximal stroke volume.

Results). Whereas these differences did not reach statistical significance, that may be the result of the small numbers of subjects in the elite group. This trend is contrary to the accepted notion that $C(a\text{-}\bar{v})O_2$ at $\dot{V}O_{2max}$ actually increases with training (5,14,18,25). However, close inspection of the data from the study of Gledhill and associates (10) on elite bicyclists shows the same trend (i.e., lower $C(a\text{-}\bar{v})O_2$ in the elite subjects). If elite athletes exercising at near $\dot{V}O_{2max}$ do have a reduced $C(a\text{-}\bar{v})O_2$, contrary to traditional belief, we must ask how this could be. One explanation may be that, as a result of the very high blood flow through active muscle due to the high cardiac outputs achieved in these athletes, the blood is passing by the active tissues faster than they are able to extract the oxygen. Unfortunately, there is no evidence for such a supposition. Another explanation is that, as a result of training and the concomitant higher cardiac output, more blood flow is going to inactive tissue (i.e., liver, kidneys, gastrointestinal track, etc.). This could be logical based on the conclusion of Rowell et al. (16), who state that resistance to blood flow is at a maximum in inactive tissue at $\dot{V}O_{2\ max}$ and is unchanged with training. If resistance is unchanged in the trained organism and blood flow is higher, than more blood flow could be going to unused areas. Because those unused areas extract little oxygen, then more blood is returning to the heart still oxygenated. Whether this could explain the reduction in $C(a\text{-}\bar{v})O_2$ observed in this study is unknown. This supposition assumes that blood pressure at $\dot{V}O_{2max}$ would also be higher in the trained person, which would be the impetus for increased blood flow to inactive tissue with similar resistance in the system, but systolic blood pressure at $\dot{V}O_{2max}$ seems to be unaffected by training (25). A third explanation for the reduction in $C(a\text{-}\bar{v})O_2$ is that the arterial blood could be carrying less oxygen as a result of poor oxyhemoglobin saturation as it passes through the lung. This phenomenon has been seen as a limiting factor in highly trained athletes working at near maximum workloads (15). In those conditions cardiac outputs may be so high that the rate of blood flow past the alveoli exceeds the time necessary to allow full saturation of the hemoglobin with oxygen (24). Whatever the explanation, the possibility of a reduced $C(a\text{-}\bar{v})O_2$ in elite athletes is intriguing and needs verification.

One potential limitation of the present study was that seven of the university distance runners were tested 2 wk after the close of their competitive season. If they had been totally sedentary during that time, this may have resulted in a slight detraining effect on $\dot{Q}_{max}$, SV_{max}, and $\dot{V}O_{2max,}$. However, because they remained active, even though not formally training, the effect should have been minimal.

In conclusion, our results show that, contrary to the SV response in untrained university students and trained university distance runners, there is no plateau in the SV response of elite male distance runners from rest to maximal exercise. Instead, SV continued to increase throughout incremental exercise. This concurs with what had been previously reported in elite bicyclists (10). The explanation for this observation remains to be elucidated.

This study was supported by a Graduate study Fellowship award from Brigham Young University.

We express thanks to Troy Chinevere, Rachel Evans, and Keary Zhou for their technical assistance.

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