REVIEW

A review of the stroke volume response to upright exercise in healthy subjects

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Traditionally, it has been accepted that, during incremental exercise, stroke volume plateaus at 40% of VO_2MAX . However, recent research has documented that stroke volume progressively increases to VO_2MAX in both trained and untrained subjects. The stroke volume response to incremental exercise to VO_2MAX may be influenced by training status, age, and sex. For endurance trained subjects, the proposed mechanisms for the progressive increase in stroke volume to VO_2MAX are enhanced diastolic filling, enhanced contractility, larger blood volume, and decreased cardiac afterload. For untrained subjects, it has been proposed that continued increases in stroke volume may result from a naturally occurring high blood volume. However, additional research is needed to evaluate the importance of blood volume, or other mechanisms, that influence the stroke volume response to exercise in untrained subjects.

well trained athletes (subject sex was not stated). Unfortunately, these findings were largely ignored and it became accepted that stroke volume plateaus during exercise of increasing intensity.

More recent investigations have reported that stroke volume progressively increases in certain people.^{7–12} The mechanisms for the continual increase in stroke volume are not completely understood. Gledhill *et al*⁷ proposed that enhanced diastolic filling and subsequent enhanced contractility are responsible for the increased stroke volume in trained subjects. However, an increase in stroke volume with an increase in exercise intensity has also been reported in untrained subjects.^{8–9} Table 1 presents a summary of the past research that has quantified stroke volume during exercise.

Current research indicates that there is a range of responses in stroke volume to reflect the range of a subject's training status. In addition, training may not be the only factor affecting the stroke volume response to exercise. Four main types of stroke volume responses to exercise have been reported in the literature: plateau,^{1–4 10 13–16} plateau with a drop,^{14 15 17–19} plateau with a secondary increase,^{5 11 20} and progressive increase^{6–10 12 14 21} (fig 1). The implications of a progressive increase in stroke volume to VO_2MAX have yet to be completely understood.

This review will examine recent discussions and evidence describing the stroke volume response to increasing exercise intensity. In addition, the role of fitness level, age, and sex on the stroke volume response to incremental exercise will be clarified.

FITNESS LEVEL

Gledhill and coworkers⁷ were the first to recognise the difference in the stroke volume response between trained and untrained young men. The study compared the stroke volume response in competitive, male endurance cyclists (22.5 (2.1) years; mean (SD)) and normally active males (22.2 (4.9) years) at matched heart rates ranging from 90 to 190 beats/min during cycle ergometry. The stroke volume of the competitive endurance cyclists increased progressively throughout exercise, whereas that of the normally active males plateaued at an average heart rate of 120 beats/min or 40% of VO_2MAX . In addition, the stroke volumes of the trained subjects were significantly larger than those of the untrained subjects at all heart rates ($p < 0.001$).

There is accumulating evidence that, in some endurance trained men and women, stroke volume continues to increase to VO_2MAX . Zhou

HISTORICAL PERSPECTIVE

It is commonly accepted that, during incremental, upright exercise to maximum, stroke volume increases from rest to exercise and plateaus at 40–50% of VO_2MAX .¹ The theory of a stroke volume plateau developed from early research based on a few subjects during two or three exercise intensities,^{1–3} with the latter characteristic limiting the number of data points used to describe the stroke volume response to exercise.^{2–3} The concept of a plateau in stroke volume was attributed mainly to a decrease in the diastolic filling time that occurs during exercise of increasing intensity.⁴

Interestingly, a progressive increase in stroke volume was reported in the literature as early as 1960. In a study by Chapman and others⁵ the stroke volume responses during discontinuous treadmill exercise were evaluated in 26 normal, male subjects aged 19–63 years. The fitness level of the subjects was not noted, but the mean (SD) VO_2MAX was 3.38 (0.46) litres/min. Stroke volume increased progressively with increasing levels of exercise in most subjects, but the relation between stroke volume and VO_2 was not linear. Similarly, Ekblom and Hermansen⁶ reported that stroke volume progressively increased during treadmill exercise at workloads of 40–80% of VO_2MAX and at VO_2MAX in nine of 13

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Table 1 Summary of the literature on stroke volume response to exercise

Reference	Subjects (age)	Mode	VO ₂ MAX	SVMAX (ml/beat)	Q	SV response
Chapman <i>et al</i> ⁵	26 men (19–63)	TM	3.7 litres/min	136	DD	Bimodal ↑
Bevegard <i>et al</i> ²	8 ET men (17–28)	Cycle	3.4 litres/min	155	HC	Plateau
Astrand <i>et al</i> ¹	11 women (20–31)	Cycle	2.6 litres/min	100	DD	Plateau
	12 men (20–31)		4.05 litres/min	134		
Grimby <i>et al</i> ³	9 ET men (45–55)	Cycle	3.8 litres/min	163	DD	Plateau
Eklblom & Hermansen ⁶	13 athletes (22–34)	TM	73.9 ml/kg/min	189	DD	9 progressive ↑ 4 plateau
Vanfraechem ²²	17 ET men (21)	Cycle	4.55 litres/min	122	IC	Progressive ↑
Crawford <i>et al</i> ²³	12 CR men & women	Cycle	–	–	RNA	Progressive ↑
	16 NCR men & women					Plateau
Hagberg <i>et al</i> ²⁴	8 MA men (56)	TM	56.6 ml/kg/min	133	CO ₂	Plateau
	8 Matched to MA (25)		62.2 ml/kg/min	131		Plateau
	8 CR men (26)		70.3 ml/kg/min	133		Plateau
	15 SED men (58)		29.7 ml/kg/min	109		Plateau
Higginbotham ⁴	12 UT men (20–50)	Cycle	2.0–3.8 litres/min	–	DD	Plateau
	12 ET men (20–50)					
Rivera <i>et al</i> ¹⁰	11 ET men (27–39)	Cycle	70.4 ml/kg/min	137	ARB	ET plateau
	11 MA men (59–81)		45 ml/kg/min	117		5/11 MA plateau 6/11 MA progressive ↑
Sullivan <i>et al</i> ¹⁶	34 men (20–70)	Cycle	31.5 ml/kg/min	–	HC	Plateau
	27 women (20–63)		28.4 ml/kg/min			
Ogawa <i>et al</i> ¹⁷	14 SED men (27)	TM	45.9 ml/kg/min	115	ARB	Progressive ↓
	13 SED men (63)		27.2 ml/kg/min	101		50–100% of VO ₂ MAX in all groups
	14 SED women (23)		37 ml/kg/min	80		
	14 SED women (64)		22.2 ml/kg/min	74		
	15 ET men (28)		63.3 ml/kg/min	154		
	14 ET men (63)		47.6 ml/kg/min	124		
	13 ET women (26)		52.1 ml/kg/min	102		
	13 ET women (57)		35.3 ml/kg/min	85		
Spina <i>et al</i> ¹⁹	12 Men & women (25) Training study	Cycle & TM	Before training: 42 ml/kg/min After training: 50 ml/kg/min	Before training: 106 After training: 115	ARB	Before training: Progressive ↓ 50–100% of VO ₂ MAX After training: Plateau
Spina <i>et al</i> ¹⁵	15 men (63) 16 women (64) Training study	TM	After training: 2.8 litres/min 1.66 litres/min	After training: 116 70	ARB	Before & after training: Progressive ↓ 50–100% of VO ₂ MAX
Leyk <i>et al</i> ²⁵	7 ET men (26) 2 ET women (26)	Cycle	4.05 litres/min	–	TDE	Plateau
Gledhill <i>et al</i> ⁷	7 ET men (22.5) 7 UT men (22)	Cycle	68.6 ml/kg/min 44.1 ml/kg/min	183 129	ARB	UT plateau ET progressive ↑
McLaren <i>et al</i> ¹³	10 Male cyclists (65) 11 Male runners (65) 10 UT men (66)	Cycle	54 ml/kg/min 48 ml/kg/min 28 ml/kg/min	–	IC	Plateau
Krip <i>et al</i> ⁸	6 UT men (22) 6 ET men (24.8)	Cycle	41.5 ml/kg/min 64 ml/kg/min	130 171	ARB	Progressive ↑
Proctor <i>et al</i> ¹⁴	8 Young ET men (24) 8 Older ET men (64) 6 Young ET women (27)	Cycle	56.5 ml/kg/min 39.9 ml/kg/min 45.6 ml/kg/min	–	ARB	Young men progressive ↓ Older men plateau with some exhibiting progressive ↓ 70–90% of VO ₂ MAX Young women plateau Older women progressive ↓ 70–90% of VO ₂ MAX
McCole <i>et al</i> ¹⁸	8 Older ET women (61) 11 SED women (63) 19 PA women (63) 14 MA women (65)	TM	35.1 ml/kg/min –	–	ARB	Progressive ↓ 60–100% of VO ₂ MAX
Wiebe <i>et al</i> ¹¹	23 ET women (20–63)	Cycle	40–70 ml/kg/min	104–125	ARB	Progressive ↑
Warburton <i>et al</i> ²¹	9 ET men (22)	Cycle	68.9 ml/kg/min	160	ARB	Progressive ↑
Ferguson <i>et al</i> ²⁰	7 MT women (18–30) 7 ET women (18–30)	Cycle	64.3 ml/kg/min 42.1 ml/kg/min	121 90	ARB	MT bimodal ↑ ET progressive ↑
Zhou <i>et al</i> ¹²	10 UT men (28) 10 male DR (26) 5 male ER (30)	TM	48.9 ml/kg/min 72.1 ml/kg/min 84.1 ml/kg/min	128 145 187	ARB	UT plateau DR plateau ER progressive ↑
Martino <i>et al</i> ⁹	6 UT men (19–22)	TM	43–67 ml/kg/min	89–159	ARB	Bimodal ↑
Warburton <i>et al</i> ²⁶	10 ET men (18–30)	Cycle	67.8 ml/kg/min	–	RNV	Progressive ↑

Data are presented as the mean or range.

SV, Stroke volume; ET, endurance trained; UT, untrained; PA, physically active; MT, moderately trained; MA, master athlete; DR, distance runners; ER, elite runners; CR, competitive runners; NCR, non-competitive runners; SED, sedentary; TM, Treadmill; DD, dye dilution; ARB, acetylene rebreathing; TDE, two dimensional echo; HC, heart catheter; IC, impedance cardiography; CO₂, CO₂ rebreathing; RNA, radionuclide angiography; RNV, radionuclide ventriculography.

*et al*¹² compared the stroke volume response in untrained men (28.1 (7.5) years), male distance runners (25.5 (4.3) years), and elite male runners (29.8 (2.5) years) during incremental exercise to exhaustion on the treadmill. The stroke volume of the untrained and distance runners plateaued at about 40% of VO₂MAX, whereas in elite distance runners, it continued to increase throughout exercise to maximum. Although

the distance runners' VO₂MAX averaged 72.1 ml/kg/min, 12 ml/kg/min lower than that of the elite runners, they still exhibited a plateau in stroke volume, indicating that endurance training may not be the only factor influencing the stroke volume response to exercise.

Crawford *et al*²³ studied the stroke volume response in male and female competitive marathon runners and

Figure 1 The four types of stroke volume response with increasing exercise intensity.

non-competitive runners. The stroke volume of the non-competitive runners plateaued at 70% of maximal heart rate, whereas that of the competitive marathon runners progressively increased to maximal heart rate. Although the stroke volume of the latter group progressively increased to maximal heart rate, the authors noted that the increase in stroke volume from 70% to 100% of maximal heart rate was not significant.

Vanfraechem²² studied healthy, male soccer players (21 (0.2) years) during cycle exercise at 25, 50, and 75% of $\text{VO}_{2\text{MAX}}$ and reported significant increases in stroke volume at each workload. Although stroke volume was not measured at an intensity greater than 75% of $\text{VO}_{2\text{MAX}}$, this study suggested that it may not plateau in young, healthy male soccer players.

Warburton and coworkers²¹ studied the stroke volume response in male endurance trained cyclists during incremental exercise to $\text{VO}_{2\text{MAX}}$ (22 (1) years) under two conditions: a 500 ml plasma volume expansion and control. In both the plasma volume expansion and control trials, stroke volume increased progressively throughout incremental cycle exercise to $\text{VO}_{2\text{MAX}}$. In addition, stroke volume and cardiac output were elevated after plasma volume expansion, suggesting that blood volume has a significant influence on the stroke volume response to exercise. Warburton *et al*²⁶ reported similar findings in highly trained male cyclists (18–30 years) during supine and upright cycle exercise to $\text{VO}_{2\text{MAX}}$. In both supine and upright exercise, stroke volume increased progressively to $\text{VO}_{2\text{MAX}}$.

In contrast, Spina *et al*¹⁹ reported a plateau in stroke volume after 12 weeks of endurance training in healthy sedentary men and women (25 (1) years). Before endurance training, stroke volume fell significantly in all subjects when exercise intensity was increased from 50% to 100% of $\text{VO}_{2\text{MAX}}$. The authors concluded that endurance training attenuated the decrease in stroke volume at maximal exercise.

Rivera *et al*¹⁰ also reported a plateau in stroke volume in endurance trained younger and older men. All of the young athletes (27–39 years) and five of 11 master athletes (59–81 years) attained a plateau in stroke volume at 30% of $\text{VO}_{2\text{MAX}}$. The stroke volume of six of the master athletes continued to increase to 85% of $\text{VO}_{2\text{MAX}}$, but the increase in stroke volume from 30% to 85% of $\text{VO}_{2\text{MAX}}$ was not significant.

Similarly, unpublished data from our laboratory suggested that not all endurance trained subjects exhibit a progressive increase in stroke volume during incremental exercise to $\text{VO}_{2\text{MAX}}$. We tested 21 endurance trained men and women (29.4 (6.1) years) during cycle exercise to $\text{VO}_{2\text{MAX}}$. Eighteen subjects reached a plateau in stroke volume at 37% of $\text{VO}_{2\text{MAX}}$,

with one subject unable to maintain maximal stroke volume as exercise intensity increased to $\text{VO}_{2\text{MAX}}$. Only three subjects showed a progressive increase in stroke volume to $\text{VO}_{2\text{MAX}}$.

Although most of the available data on the stroke volume response to exercise are from male subjects, there is also evidence to support a progressive increase in stroke volume in female subjects. Ferguson *et al*²⁰ compared the stroke volume response in moderately active and endurance trained women (18–30 years) during incremental cycle exercise to exhaustion. Stroke volume increased progressively to $\text{VO}_{2\text{MAX}}$ in both groups. The stroke volume was significantly greater in endurance trained women at all workloads. In moderately trained women, stroke volume increased from rest to exercise, plateaued, and then showed a secondary increase to maximum. Wiebe and colleagues¹¹ reported similar results in women 20–63 years of age. This type of rise in stroke volume is consistent with data from others.^{5–9,11}

Three studies to date provide evidence that stroke volume does not plateau in untrained subjects.^{5–9} Martino and colleagues⁹ studied healthy, young men (19–22 years) with no history of training, but unusually high $\text{VO}_{2\text{MAX}}$. Untrained subjects with a high $\text{VO}_{2\text{MAX}}$ (65.3 (0.6) ml/kg/min; HI group) were matched with similar subjects who had a normal $\text{VO}_{2\text{MAX}}$ (46.2 (0.9) ml/kg/min; LO group). Subjects were chosen from a group of 1900 healthy, young men who underwent prior $\text{VO}_{2\text{MAX}}$ testing. Despite no history of training, the $\text{VO}_{2\text{MAX}}$ and mean blood volume of the HI group were comparable with that of an endurance trained athlete. In both groups, stroke volume increased progressively from rest to 50% of $\text{VO}_{2\text{MAX}}$, plateaued from 50% to 75% of $\text{VO}_{2\text{MAX}}$, then increased from 75% to 100% of $\text{VO}_{2\text{MAX}}$. Martino and colleagues concluded that the high $\text{VO}_{2\text{MAX}}$ observed in the untrained subjects was due to a naturally occurring high blood volume that contributes to a high maximal stroke volume and cardiac output. Krip and colleagues⁸ reported similar results in endurance trained male cyclists and untrained male control subjects (18–30 years) during cycle exercise to exhaustion. The stroke volume of all of the subjects increased progressively throughout exercise to $\text{VO}_{2\text{MAX}}$, although the trained subjects had higher blood volumes, faster diastolic filling rates, and faster systolic emptying rates than the untrained controls.

Current research supports a continual increase in stroke volume during exercise of increasing intensity in some trained and untrained subjects. The physiological characteristics that promote this progressive increase are not yet fully understood. Data support endurance training as a mechanism of improving the stroke volume response to exercise. However, the data of Martino *et al*⁹ show that blood volume, independent of training, may be a major determinant of the stroke volume response to exercise.

AGE

Studies on the effects of age on the stroke volume response to exercise have produced conflicting results. Whereas most studies have reported a plateau or a fall in stroke volume,^{14–15,17,18} two have reported a progressive increase in stroke volume in older men and women.^{10,11} The age related effects on the stroke volume response to exercise may be due to increases in end systolic volume, decreases in end diastolic volume, or a combination of both.

The results of Wiebe *et al*¹¹ and Rivera *et al*¹⁰ provide evidence of a progressive increase in stroke volume in older subjects. Wiebe and others¹¹ examined the stroke volume response during incremental cycle exercise to $\text{VO}_{2\text{MAX}}$ in endurance trained women (20–63 years). The subjects were matched for lean body mass, then divided into four age groups: 20–29, 40–45, 49–54, and 58–63 years. In all groups, stroke volume increased progressively throughout

incremental exercise to $\dot{V}_{O_2\text{MAX}}$. Maximal stroke volume was lower in endurance trained women aged 40–63 years than in those aged 20–29 years. The authors hypothesised that the age related differences in maximal stroke volume may be due to decreases in end diastolic volume or end systolic volume. These data suggest that older subjects have the ability to progressively increase stroke volume during exercise of increasing intensity.

Rivera and colleagues¹⁰ reported similar results when comparing the stroke volume response of master athletes (59–81 years) and young athletes (27–39 years) during incremental treadmill exercise to 85% of $\dot{V}_{O_2\text{MAX}}$. The stroke volume of all of the young runners and five of 11 master runners plateaued during exercise, whereas the stroke volume of the other six master runners progressively increased to 85% of $\dot{V}_{O_2\text{MAX}}$. The authors noted that, in all groups, the average stroke volume did not significantly increase after 30% of $\dot{V}_{O_2\text{MAX}}$; however, visual inspection of individual data revealed that stroke volume progressively increased in six of 11 master runners.

In contrast, several authors have reported a plateau or fall in stroke volume during exercise in older subjects.^{13 14 17 18} Proctor and others¹⁴ compared younger (20–31 years) and older (51–72 years) endurance trained men and women during cycle exercise at 40%, 70%, and 90% of $\dot{V}_{O_2\text{MAX}}$. The stroke volume in the young men continued to increase progressively throughout exercise to 90% of $\dot{V}_{O_2\text{MAX}}$, whereas the stroke volume of older men and younger women exhibited a plateau which was maintained throughout exercise. The older women showed an impaired ability to maintain stroke volume when intensity was increased from 70% to 90% of $\dot{V}_{O_2\text{MAX}}$. Several of the oldest men also showed a modest decline in stroke volume at exercise intensities above 70% of $\dot{V}_{O_2\text{MAX}}$. The authors noted that the stroke volume response in both the older men and women was related to age ($r = -0.50$), with the oldest subjects having the largest decrease in stroke volume.

McCole *et al*¹⁸ reported similar results in postmenopausal competitive master athletes, physically active women, and sedentary women (63 (5) years) during treadmill exercise to $\dot{V}_{O_2\text{MAX}}$. In all groups, stroke volume reached maximal values at 40–60% of $\dot{V}_{O_2\text{MAX}}$ and decreased significantly as exercise intensity increased from 60% to 100% of $\dot{V}_{O_2\text{MAX}}$. The decrease in stroke volume was similar across all physical activity groups.

Ogawa *et al*¹⁷ studied healthy younger (18–31 years) and older (51–72 years) sedentary and endurance trained men and women. The subjects were divided into eight groups based on age (younger versus older), training status (sedentary versus trained), and sex. In all groups, the highest stroke volume was attained at 50% of $\dot{V}_{O_2\text{MAX}}$, and a fall in stroke volume occurred between 50% and 100% of $\dot{V}_{O_2\text{MAX}}$.

Hagberg *et al*¹⁴ compared master athletes (56 (5) years), young runners matched in training and performance to the master athletes (25 (3) years), competitive young runners (26 (3) years), and healthy older sedentary subjects (58 (5) years) during treadmill exercise at 50%, 60%, and 70% of $\dot{V}_{O_2\text{MAX}}$. In all groups, stroke volume plateaued at 50% of $\dot{V}_{O_2\text{MAX}}$ and was maintained up to 70% of $\dot{V}_{O_2\text{MAX}}$. Although the stroke volume of the older subjects did not decline, stroke volume measurements were only obtained up to 70% of $\dot{V}_{O_2\text{MAX}}$. Several studies have reported a fall in stroke volume at intensities above 70% of $\dot{V}_{O_2\text{MAX}}$ in older subjects,^{14 17 18} therefore it is difficult to ascertain if the plateau in stroke volume in older subjects would have been maintained up to $\dot{V}_{O_2\text{MAX}}$.

McLaren and colleagues¹³ reported a stroke volume plateau in trained older male cyclists (65 (2.1) years), trained older male runners (65 (3.5) years), and healthy but untrained

older male controls (66 (1.2) years). The stroke volume of the older trained runners plateaued at about 40% of $\dot{V}_{O_2\text{MAX}}$, whereas the stroke volume of the older trained cyclists and controls plateaued at an average of 30% of $\dot{V}_{O_2\text{MAX}}$.

In a training study by Spina *et al*,¹⁵ the stroke volume of older men and women decreased as exercise intensity increased from 50% to 100% of $\dot{V}_{O_2\text{MAX}}$ before and after 9–12 months of endurance training, suggesting that the adaptability in the stroke volume response to exercise may be influenced by age.

Current evidence indicates that older subjects (men and women) have an impaired ability to maintain stroke volume at near maximal exercise. Although there is evidence of a progressive increase in stroke volume in older subjects, additional longitudinal research is needed to evaluate the effects of age on the stroke volume response to exercise.

SEX DIFFERENCES

The studies on sex differences in the stroke volume response to exercise have produced conflicting results. Sullivan and others¹⁶ investigated the effects of sex on stroke volume control during cycle exercise to $\dot{V}_{O_2\text{MAX}}$ in healthy men (20–70 years) and women (20–63 years). In both men and women, stroke volume reached its maximum at 50% of $\dot{V}_{O_2\text{MAX}}$ and remained unchanged through maximal exercise. There were no differences in stroke volume index (ml/m^2) at rest or during exercise between groups. In addition, the increase in stroke volume index from rest to exercise was similar in men and women. These authors concluded that, in healthy subjects, matched for body size and fitness level, sex is not an important determinant of the stroke volume response to exercise.

In contrast, although Ogawa *et al*¹⁷ reported similar stroke volume responses in healthy younger (18–31 years) and older (52–72 years) sedentary and endurance trained men and women, the stroke volume of the women was lower at all workloads compared with the men, even after normalisation to body weight. After normalisation of stroke volume to fat free mass, the sex difference was eliminated in sedentary subjects, but only reduced in the endurance trained subjects. The authors concluded that the sex difference in the stroke volume of the endurance trained subjects was due to a greater percentage of body fat in women.

Proctor and others¹⁴ examined the influence of age and sex on cardiovascular responses to exercise. The stroke volume response of younger (20–31 years) and older (51–72 years) endurance trained men and women were compared during cycle exercise at 40%, 70%, and 90% of $\dot{V}_{O_2\text{MAX}}$. The stroke volume in the young men had not yet reached a plateau at 90% of $\dot{V}_{O_2\text{MAX}}$, whereas the stroke volume of older men and younger women reached a plateau at 40% of $\dot{V}_{O_2\text{MAX}}$ which was maintained throughout exercise. The older women showed an impaired ability to maintain stroke volume at exercise intensities above 90% of $\dot{V}_{O_2\text{MAX}}$, which was also evident, to a lesser degree, in the oldest men. Spina and coworkers¹⁵ studied sex differences in the cardiovascular responses before and after training in older men and women (64 (3) years). Before and after training, stroke volume decreased as exercise intensity was increased from 50% to 100% of $\dot{V}_{O_2\text{MAX}}$ in both sexes. However, the decrease in stroke volume was greater in women than in men.

Current evidence suggests that the stroke volume response to exercise may differ between men and women. However, sex differences cannot be elucidated at this time because of conflicting results and a lack of relevant studies.

MECHANISMS OF ENHANCED STROKE VOLUME

With increasing exercise intensity, diastolic filling time, as well as systolic ejection time, decreases.⁷ This decrease is

Figure 2 Comparison of ventricular ejection time between trained and untrained subjects. Adapted from Gledhill *et al.*⁷

Figure 3 Comparison of ventricular diastolic filling time between trained and untrained subjects. Adapted from Gledhill *et al.*⁷

thought to lead to a plateau in stroke volume with increasing exercise intensity. However, current research is suggesting that, in young endurance trained subjects, diastolic filling, as well as ventricular emptying, is enhanced, leading to a progressive increase in stroke volume during exercise to $\text{VO}_{2\text{MAX}}$.^{7, 8, 20} Interestingly, research has shown that endurance trained subjects have significantly longer ventricular ejection times,^{7, 8, 27} greater myocardial contractility,^{16, 22} greater left ventricular diameter and mass,^{28–31} and significantly shorter diastolic filling times^{7, 8, 27} than untrained subjects.

Gledhill *et al.*⁷ reported that, in endurance trained men, ventricular ejection times were longer, and diastolic filling times were shorter, than in untrained men (figs 2 and 3). If athletes are able to increase end diastolic volume in less time than untrained subjects, this suggests that the rates of ventricular filling are dramatically increased in trained subjects. What allows for this increased rate of filling? Gledhill and coworkers⁷ suggested that the higher blood volumes in the trained subjects maintained an adequate ventricular filling pressure during exercise, thereby enhancing diastolic filling. The longer ejection times and enhanced diastolic emptying reported by Gledhill *et al.*⁷ were attributed to lower systolic and diastolic blood pressures in trained subjects, which reduced cardiac afterload and facilitated ventricular emptying. Further support for enhanced diastolic filling in athletes was reported by Vinereanu *et al.*³¹ Using tissue Doppler echocardiography, they reported augmented left ventricular diastolic filling velocities in endurance trained subjects compared with strength trained and control subjects.

Wolfe and others²⁷ reported similar findings in trained male athletes. The athletes tested had shorter pre-ejection periods, longer ejection times, and faster mean systolic ejection rates at the same exercise heart rates than subjects who were moderately trained and untrained. The authors hypothesised that an enhanced end diastolic volume and ejection fraction were involved in the increased stroke volume in athletes.

Ferguson and colleagues²⁰ suggested that, in endurance trained women, the increase in stroke volume at higher exercise intensities was due to an enhanced ventricular preload, not myocardial contractility. In contrast, Jensen-Ustad and colleagues³² reported that training induced increases in myocardial contractility, and possibly a decreased afterload, were the main contributing factors to the increase in stroke volume during incremental exercise in elite male runners. Similarly, Vanfraechem²² reported that left ventricular ejection times decreased at each workload in male soccer players. The author hypothesised that the continued increase in stroke volume, despite the decrease in ventricular ejection time, may be due to an increase in ejection fraction during exercise of increasing intensity.

In addition, left ventricular eccentric hypertrophy may be related to enhanced diastolic filling and stroke volume. Data indicate that endurance trained athletes develop an increase in both left ventricular diameter and wall thickness (eccentric hypertrophy), as the heart adapts to both volume and pressure loads.^{28–31} Longitudinal data from Levy *et al.*³⁰ indicate that endurance training significantly increases left ventricular mass and is associated with improvements in early diastolic filling rates at rest and during exercise. Similarly, Hoogsteen *et al.*²⁹ reported greater left ventricular end diastolic diameter and early filling rates in endurance trained subjects compared with previously established normal values. However, data are conflicting in this area.^{28, 30}

The above findings indicate that endurance trained subjects may have enhanced diastolic filling, greater left ventricular diameter and mass, greater ventricular compliance, greater myocardial contractility, and may depend more on the Frank Starling mechanism (preload) as exercise intensity increases, compared with untrained subjects. Although research suggests that untrained subjects with a naturally occurring high blood volume may have enhanced diastolic filling,^{8, 9} there is currently no evidence to suggest that untrained people have enhanced ventricular compliance, greater left ventricular diameter and mass, or myocardial contractility, as seen in trained subjects.

What is already known on this topic

Research data on the stroke volume response to incremental exercise are conflicting. Early research supports a plateau in stroke volume in healthy untrained and trained subjects. Recent research has documented that stroke volume progressively increases to $\text{VO}_{2\text{MAX}}$ in both trained and untrained subjects, but this finding has not been consistently reported.

What this study adds

This is the first review of stroke volume responses to exercise in healthy subjects. This study adds to the understanding of the various stroke volume responses to increasing exercise intensity, the effects of endurance training, sex, and age on the stroke volume response to exercise, and the mechanisms responsible for a progressive increase in stroke volume during exercise.

CONCLUSIONS

Current findings indicate that the stroke volume response to exercise may depend on many factors, including age, fitness level, sex, and genetics. Those with a high blood volume may be more likely to exhibit a progressive increase in stroke volume during exercise of increasing intensity. The progressive increase in stroke volume with endurance training has some training implications and clinical relevance. In terms of myocardial oxygen demand, increasing stroke volume is much more efficient than increasing heart rate during exercise. In athletes, increasing stroke volume for a given heart rate may increase work output and performance. The physiological mechanisms for an enhanced stroke volume during exercise may include enhanced diastolic filling due to increases in blood volume, left ventricular diameter, and ventricular compliance, enhanced systolic emptying due to increases in myocardial contractility and decreases in ventricular afterload, or both.

Future investigations should evaluate the type and amount of endurance training needed to facilitate a progressive increase in stroke volume during incremental exercise and determine if the adaptability in the stroke volume response to exercise is influenced by age, sex, type of training, and training status.

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PostScript

LETTERS

Month by month analysis of the number of athletic training injuries: a prospective one year study on 2701 athletes

Various studies have focused on sport injuries.^{1,2} In a previous report the incidence of injuries in athletes in a one year study was analysed using a sample size that was less than 150 subjects.¹ It was hypothesised that subjects who were more involved in sport before injury (eight hours or more a week of sport and exercise) would exhibit a greater emotional response to injury and perceive their recovery to be less.²

In view of this we analysed a large sample of athletes (2701) who trained a minimum of 10 hours a week to see in which months of the year athletic training injuries were most common.

In a one year prospective study (from January 2004 to December 2004) we recorded the number of training injuries sustained in a month by month fashion. The mean (SD) age of the subjects was 39.62 (12.98) (range 14-63).

We recorded a total of 450 athletic training injuries. The greatest number of injuries were recorded in January (n = 71; 15.8%) followed by February (n = 64; 14.2%), March (n = 56; 12.4%), May (n = 54; 12.0%), April (n = 47; 10.4%), December (n = 37; 8.2%), June (n = 34; 7.5%), September (n = 29; 6.4%), July (n = 27; 6.0%), November (n = 22; 4.9%), August (n = 7; 1.5%), and October (n = 2; 0.4%).

A total of 191 injuries (42.4%) were recorded between January and March, 135 (30.0%) between April and June, 63 (14.0%) between July and September, and 61 (13.5%) between October and December. The total recorded between January and June was 326 (72.4%) compared with 124 (27.5%) between July and December.

In summary, our study has shown that the number of athletic training injuries was higher during the first semester of the year than the second.

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Competing interests: none declared

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Effective measures to improve driver safety

In a recent article in this journal by Leonard and colleagues,¹ it was concluded that changing the configuration of motor racing circuits by introducing chicanes may significantly decrease the risk of severe injury to drivers. We believe that this evidence is reliable up to a certain point. There are several other measures that could be implemented to improve the overall safety of drivers; making circuits slower is not necessarily the only or the best strategy. From a spectator point of view, the introduction of numerous slow chicanes over the past decade, disrupting the original configuration of some legendary circuits such as Hockenheim, Imola, and Monza, has contributed to making motor racing less spectacular. From a scientific perspective, driver safety may be improved by the adoption of alternative measures that will not substantially affect the attractiveness of motor racing. Some of these are much more readily and economically applied, and may even turn out to be safer. The foremost of these measures is to increase the weight of the car and reduce engine power, which may encourage manufacturers to build more robust cockpits and slower competition vehicles.² Next, we propose the obligatory use of efficient protective measures for the most commonly and severely traumatised body parts (head, limb, and legs), such as the recently designed HANS carbon fibre collar, thereby improving safety without affecting the spectacle.³ Finally, more efficient protective measures on circuits, such as replacing tyre barriers and metal rails with temporary crash protection barriers made of steel tubes and pads of hard foam, may absorb some of the crash energy, reducing the loading to both head and neck during dramatic decelerations up to 100 g.⁴ As technological advances in competition are usually translated to production vehicles, these strategies may also be effective in preventing or limiting the severity of injuries from road traffic accidents outside the racing circuits.

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Competing interests: none declared

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BOOK REVIEWS

Sports ethics: an anthology

Edited by Jan Boxill. Published by Blackwell Publishers, 2002, £60.00 (hardcover), £17.99 (paperback). ISBN 0631216960

This is a collection of 35 papers, 10 of which were written specifically for this anthology. It presents a wide range of material, spanning specific topics, such as racial issues or drugs in sport, and also more abstract areas, such as the quality of sportsmanship. Most of the writing is by philosophers, but there are pieces representing sportspeople, physical education specialists, sports psychologists, sport scientists, journalists, a lawyer, and a basketball coach. For this reason, the book is stylistically very diverse. Fittingly, the volume closes with a 1999 piece by Rick Reilly, a writer for *Sports Illustrated*, written from the perspective of an 8 year old sports fan who is getting the wrong message from televised sport.

The theme of sport's corruption seems to run strongly in this collection. There seems to be an undercurrent of moral outrage at sport's degradation through commercialisation, new technology, and competition taken too far.

Editor Jan Boxill's introductory piece on the moral significance of sport, for example, defines sport in terms of four "paradigmatic" properties, all of which seem prone to "perversion". Sport, Boxill explains, can be degraded when we make a living from it, when we break rules, or when we view our sporting success as our opponent's loss. These paradigmatic qualities, Boxill says, also explain the importance of sport: it is the "single most available and the single most participated in means" for attaining self development, self expression, and self respect. "Sport is the art of the people", she claims, which sounds wonderful—yet we wonder if perhaps this amounts to a devaluation of sports fans' appreciation of sport's baser, less artistic, merits: Beckham's glamour, McEnroe's tantrums, or Mohammed Ali's propensity to verbally raise the stakes of victory. Some of the "best" sportspeople on Boxill's definition, such as Pete Sampras, are the least loved. And if sports are really "of the people", then surely the popularity of a sport is a good metric of whether or not it is headed in the right direction?

We are not so worried about the perversion of sport as most of these authors are. Boxill, like many of the authors of this volume, is an avid amateur sportsperson as well as a philosopher, and it shows in her conception of sport—a conception that is more "of the sportspeople" than "of the people". Sport evolves in ways that the sportspeople themselves are often slowest to accept, because they are most strongly affected by change. But from the point of view a sports appreciator,

commercial sponsorship did not eliminate sportsmanship from cricket, graphite racquets failed to eliminate skill from tennis, and professionalism did not ruin the Olympics. As these changes have rolled in, participation and audience involvement have ballooned—"the people" have loved it. Nonetheless, in this book Peter Wenz's article on "Human equality in sports" decries professionalism, Kathleen Pearson presents an indictment of deliberate fouls, and all three papers on performance enhancing drugs conclude that banning drug use in sport is justified.

Whether we agree with the authors' sporting politics or not, the quality of the work selected is often very good. One of the pieces specifically written for this book is Laura Morgan's "Enhancing performance in sports: what is morally permissible", in which she looks for a new argument supporting her intuition that performance enhancements "do not belong" in sport. Her novel argument is that the use of drug enhancements is harmful to the sport, meaning that it worsens the nature of sport. The difficulty for many commentators on this issue is that they want to prohibit even harmless drugs on the basis that they make a sport unfair, but have no answer to the objection that sport is already a genetic contest which is intrinsically unbalanced and unfair. Morgan avoids this issue by placing the emphasis instead on matching contestants to produce competitive contests which will challenge every competitor. Harmless drugs, she argues, would worsen the nature of sport because they inhibit this matchmaking, and thus would undermine the "mutual quest to achieve excellence" which is the ideal goal of sport. This argument entails the rather radical conclusion that harmless drugs would be permissible in solitary, non-competitive sports. We have argued that far from perverting the spirit of sport, performance enhancement embodies the human drive to be better. To be human is to be better. Performance enhancement, we have argued, embodies the spirit of human sport.¹

This is a volume that questions much of the status quo concerning how we ought to play sport, and how we ought to appreciate it, but only rarely challenges this conservative conception of what sport is all about. Those who share the authors' stance on what is valuable about sport are likely to be fully satisfied by this anthology, but for some of us, perhaps a few more challenging papers could have been presented. Boxill's introductory piece is intended to motivate an academic discourse on the nature of sport, which is an excellent goal, and the breadth of the material presented here gives this goal impressive support. This is an intriguing and comprehensively interdisciplinary collection of writing.

Rating

● Presentation	17/20
● Comprehensiveness	15/20
● Readability	19/20
● Relevance	15/20
● Evidence basis	14/20
● Total	80/100

J Savulescu, B Foddy

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Performance enhancing substances in sport and exercise

Edited by Michael S Bahrke, Charles E Yesalis. Published by Human Kinetics, 2002, £43.00 (hardcover), pp 384. ISBN 0736036792

This is a worthwhile addition to the library of all who work in sport and exercise medicine whether as physician, physiotherapist, or sports scientist. It extends to over 350 pages, is straightforward to read, well arranged, and with a useful index.

The initial chapter on the history of performance enhancing substances (PES) contains considerable information on early attempts at performance enhancement within sport, with much that was new to the reviewer. It even manages a brief reference to the current Governor of California under the subject of body building and anabolic agents!

The first 300 pages consider ergogenic agents in specific groups—identifying the mode of action, likely performance gains, relevant clinical studies, potential problems resulting from use, and ending with a brief overall conclusion. The statements made are generally referenced, and the sources for these are listed extensively at the end of each chapter.

The book would appear to have been initially published some three years ago—my review copy was dated 2002. As the world of PES changes very rapidly, the book inevitably predates some very significant incidents, publications, and changes in regulatory activity. There is no reference to therapeutic use exemption (TUE) certificates, nor to the 2002 IOC report on supplement contaminants. It follows that the 2004 whistle blowing on "designer drug" use by elite USA athletes is also omitted.

I attempted to use the book to find specific information on a drug that had recently been brought before the UK TUE committee for consideration. There was good information on the group to which it belonged, but only a passing reference to the drug itself. That said, the chapter-end discussion of potential benefits and disadvantages was helpful and evidence based.

There is an inevitable North American slant to the text, and some of the examples cited refer to American sports that are not universal in popularity. I noted reference to a local regulatory control—the *US Dietary and Supplement Health and Education Act 1994*; the need for regulation of non-drug supplements is clearly in harmony with current European thought on the matter.

In the chapter on anabolics, there is what seems to be an unusual statement (p 33) "from the late 30s to the mid 80s, anabolic steroids were used successfully to treat depression, melancholia and involutional psychoses". This certainly hasn't been my experience of conventional UK management of any of these conditions.

B-agonists are dealt with in some depth, Clenbuterol, which is not available in the UK or the USA but is obtainable in Europe, being the most closely scrutinised. This revisited the

few unpleasant memories I have of the Barcelona Olympics, where its use by two athletes caused significant problems to GB team officials. However, I could find nothing in the chapter to justify the considerable effort many of us have made in implementing recent IOC regulations in respect of permitted B-agonist inhaler use.

One area of discussion struck a chord with me, the difference in perception of doctors and athletes—or to be precise some athletes. Doctors and sports scientists put their faith in scientific studies. Athletes, however, often place much greater importance on testimonials and internet advertorials and treat research with suspicion if not contempt.

Which raises the question: how valid are clinical studies of PES in athletes? The point is well made that there may be numerous confounders at play, and this is demonstrated in the often conflicting study results contained in many chapters. Athletes rarely use ergogenic aids in the framework that would be demanded of a clinical study. PES are not used in isolation, but rather within "cocktails" where the dosage consumed and the frequency of dosage may have little to do with their use in clinical indications and which would be rejected out of hand if forwarded to an ethics committee for consideration.

The later part of the book looks to possible future developments in doping techniques and deals with some of the more difficult areas of drug testing: its problems and limitations. In the final chapter, the legal context of PES is also considered. I'll leave the saddest quotation in the book to the end: "To be a great athlete today you need a great coach, a great chemist and a great lawyer".

I really do wish that didn't ring any bells with me.

Rating

● Presentation	17/20
● Comprehension	16/20
● Readability	15/20
● Relevance	13/20
● Evidence basis	16/20
● Total	77/100

C Jarvis

CALENDER OF EVENTS

Osteosynthese International 2005

15–17 September 2005, Curiohaus, Hamburg
Congress-Chairman: Johannes M. Rueger, M.D., Professor and Chair
Topics:

- Innovations in intramedullary osteosynthesis
- New frontiers in osteoporosis and fracture treatment
- Current trauma research
- Special topic: Recent development in pelvic and acetabular fractures

Abstract submission deadline: 31 March 2005

Further details: INTERCONGRESS GmbH, Martin Berndt, Düsseldorf Str. 101, 40545 Düsseldorf-Germany. Tel: +49 211 585897-80; Fax: +49 211 585897-99; Email: martin.berndt@intercongress.de; Website: www.osteoint2005.de

4th European Sports Medicine Congress

13–15 October 2005, Lemesos, Cyprus

Further details: Email: pyrgos.com@cytanet.com.cy

BASEM Conference 2005

10–12 November 2005, Edinburgh, Scotland

Further details: Email: BASEMinfo@aol.com; Website: www.basem.co.uk

8th International Congress of the Society for Tennis Medicine and Science

14–15 January 2006, Melbourne, Australia

To be held immediately prior to the 2006 Australian Open tennis championships, the congress will combine presentations from international and Australian experts, including Professors Tim Noakes, Bruce Elliott, and Mark Hargreaves to stimulate discussion on topical tennis science and medicine issues.

Registrations are now open via the congress website.

Further details: Email: stms2006@meeting-planners.com.au; Website: www.stms2006.com.au

13th Commonwealth International Sport Conference (CISC2006)

9–12 March 2006, Melbourne, Australia

Further details: Email: enquiries@cisc2006.com; Website: www.cisc2006.com

BASEM Conference 2006

5–7 October 2006, Oxford, UK

Further details: Email: BASEMinfo@aol.com; Website: www.basem.co.uk

CORRECTION

doi: 10.1136/bjsm.2004.013037corr1

C A Vella, R A Robergs. A review of the stroke volume response to upright exercise in healthy subjects (*Br J Sports Med* 2005;**39**: 190–5).

A mistake in the second sentence of the first paragraph of "Historical Perspective" has been noted:

"The theory of a stroke volume plateau developed from early research based on a few subjects during two or three exercise intensities..." should read "two to five exercise intensities".

In addition, there is a misprint in table 1. Under Ferguson *et al* the values in the $\text{VO}_{2\text{MAX}}$ column and the SV_{MAX} (ml/beat) are incorrect and should be:

7 MT women (18–30): $\text{VO}_{2\text{MAX}}$ 42.1 ml/kg/min, SV_{MAX} (ml/beat) 90

9 ET women (18–30): $\text{VO}_{2\text{MAX}}$ 64.3 ml/kg/min, SV_{MAX} (ml/beat) 121