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 $V_{O_2}MAX$. However, recent research has documented that stroke volume progressively increases to $V_{O_2}MAX$ in both trained and untrained subjects. The stroke volume response to incremental exercise to $V_{O_2}MAX$ may be influenced by training status, age, and sex. For endurance trained subjects, the proposed mechanisms for the progressive increase in stroke volume to $V_{O_2}MAX$ 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.

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 $V_{O_2}MAX$.[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 others[5] 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) $V_{O_2}MAX$ 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 V_{O_2} was not linear. Similarly, Ekblom and Hermansen[6] reported that stroke volume progressively increased during treadmill exercise at workloads of 40–80% of $V_{O_2}MAX$ and at $V_{O_2}MAX$ in nine of 13 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 $V_{O_2}MAX$ 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 coworkers[7] 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 to190 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 $V_{O_2}MAX$. 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 $V_{O_2}MAX$. Zhou

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

Reference	Subjects (age)	Mode	$V_{O_2}MAX$	SVMAX (ml/beat)	Q	SV response
Chapman *et al*[5]	26 men (19–63)	TM	3.7 litres/min	136	DD	Bimodal ↑
Bevegard *et al*[2]	8 ET men (17–28)	Cycle	3.4 litres/min	155	HC	Plateau
Astrand *et al*[1]	11 women (20–31)	Cycle	2.6 litres/min	100	DD	Plateau
	12 men (20–31)		4.05 litres/min	134		
Grimby *et al*[3]	9 ET men (45–55)	Cycle	3.8 litres/min	163	DD	Plateau
Ekblom & 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 *et al*[23]	12 CR men & women	Cycle	–	–	RNA	Progressive ↑
	16 NCR men & women					Plateau
Hagberg *et al*[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 *et al*[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 *et al*[16]	34 men (20–70)	Cycle	31.5 ml/kg/min	–	HC	Plateau
	27 women (20–63)		28.4 ml/kg/min			
Ogawa *et al*[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 $V_{O_2}MAX$ in all
	14 SED women (23)		37 ml/kg/min	80		groups
	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 *et al*[19]	12 Men & women (25)	Cycle & TM	Before training:	Before training:	ARB	Before training: Progressive ↓
	Training study		42 ml/kg/min	106		50–100% of $V_{O_2}MAX$ After
			After training:	After training:		training: Plateau
			50 ml/kg/min	115		
Spina *et al*[15]	15 men (63)	TM	After training:	After training:	ARB	Before & after training:
	16 women (64)		2.8 litres/min	116		Progressive ↓
	Training study		1.66 litres/min	70		50–100% of $V_{O_2}MAX$
Leyk *et al*[25]	7 ET men (26)	Cycle	4.05 litres/min	–	TDE	Plateau
	2 ET women (26)					
Gledhill *et al*[7]	7 ET men (22.5)	Cycle	68.6 ml/kg/min	183	ARB	UT plateau
	7 UT men (22)		44.1 ml/kg/min	129		ET progressive ↑
McLaren *et al*[13]	10 Male cyclists (65)	Cycle	54 ml/kg/min	–	IC	Plateau
	11 Male runners (65)		48 ml/kg/min			
	10 UT men (66)		28 ml/kg/min			
Krip *et al*[8]	6 UT men (22)	Cycle	41.5 ml/kg/min	130	ARB	Progressive ↑
	6 ET men (24.8)		64 ml/kg/min	171		
Proctor *et al*[14]	8 Young ET men (24)	Cycle	56.5 ml/kg/min	–	ARB	Young men progressive ↓
	8 Older ET men (64)		39.9 ml/kg/min			Older men plateau with some
	6 Young ET women (27)		45.6 ml/kg/min			exhibiting progressive ↓
						70–90% of $V_{O_2}MAX$
						Young women plateau
						Older women progressive ↓
	8 Older ET women (61)		35.1 ml/kg/min			70–90% of $V_{O_2}MAX$
McCole *et al*[18]	11 SED women (63)	TM	–	–	ARB	Progressive ↓
	19 PA women (63)					60–100% of $V_{O_2}MAX$
	14 MA women (65)					
Wiebe *et al*[11]	23 ET women (20–63)	Cycle	40–70 ml/kg/min	104–125	ARB	Progressive ↑
Warburton *et al*[21]	9 ET men (22)	Cycle	68.9 ml/kg/min	160	ARB	Progressive ↑
Ferguson *et al*[20]	7 MT women (18–30)	Cycle	64.3 ml/kg/min	121	ARB	MT bimodal ↑
	7 ET women (18–30)		42.1 ml/kg/min	90		ET progressive ↑
Zhou *et al*[12]	10 UT men (28)	TM	48.9 ml/kg/min	128	ARB	UT plateau
	10 male DR (26)		72.1 ml/kg/min	145		DR plateau
	5 male ER (30)		84.1 ml/kg/min	187		ER progressive ↑
Martino *et al*[9]	6 UT men (19–22)	TM	43–67 ml/kg/min	89–159	ARB	Bimodal ↑
Warburton *et al*[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; CO_2, CO_2 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 $V_{O_2}MAX$, whereas in elite distance runners, it continued to increase throughout exercise to maximum. Although the distance runners' $V_{O_2}MAX$ 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

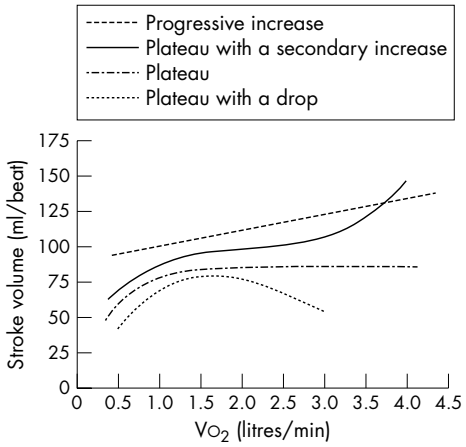

Figure 1 The four types of stroke volume response with increasing exercise intensity.

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.

Vanfraechem[22] studied healthy, male soccer players (21 (0.2) years) during cycle exercise at 25, 50, and 75% of $V_{O_2}MAX$ and reported significant increases in stroke volume at each workload. Although stroke volume was not measured at an intensity greater than 75% of $V_{O_2}MAX$, this study suggested that it may not plateau in young, healthy male soccer players.

Warburton and coworkers[21] studied the stroke volume response in male endurance trained cyclists during incremental exercise to $V_{O_2}MAX$ (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 $V_{O_2}MAX$. 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 $V_{O_2}MAX$. In both supine and upright exercise, stroke volume increased progressively to $V_{O_2}MAX$.

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 $V_{O_2}MAX$. 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 $V_{O_2}MAX$. The stroke volume of six of the master athletes continued to increase to 85% of $V_{O_2}MAX$, but the increase in stroke volume from 30% to 85% of $V_{O_2}MAX$ 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 $V_{O_2}MAX$. We tested 21 endurance trained men and women (29.4 (6.1) years) during cycle exercise to $V_{O_2}MAX$. Eighteen subjects reached a plateau in stroke volume at 37% of $V_{O_2}MAX$, with one subject unable to maintain maximal stroke volume as exercise intensity increased to $V_{O_2}MAX$. Only three subjects showed a progressive increase in stroke volume to $V_{O_2}MAX$.

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 $V_{O_2}MAX$ 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 colleagues[11] 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 8 9] Martino and colleagues[9] studied healthy, young men (19–22 years) with no history of training, but unusually high $V_{O_2}MAX$. Untrained subjects with a high $V_{O_2}MAX$ (65.3 (0.6) ml/kg/min; HI group) were matched with similar subjects who had a normal $V_{O_2}MAX$ (46.2 (0.9) ml/kg/min; LO group). Subjects were chosen from a group of 1900 healthy, young men who underwent prior $V_{O_2}MAX$ testing. Despite no history of training, the $V_{O_2}MAX$ 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 $V_{O_2}MAX$, plateaued from 50% to 75% of $V_{O_2}MAX$, then increased from 75% to 100% of $V_{O_2}MAX$. Martino and colleagues concluded that the high $V_{O_2}MAX$ 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 colleagues[8] 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 $V_{O_2}MAX$, 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 others[11] examined the stroke volume response during incremental cycle exercise to $V_{O_2}MAX$ 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 $V_{O_2}MAX$. 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 colleagues[10] 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 $V_{O_2}MAX$. 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 $V_{O_2}MAX$. The authors noted that, in all groups, the average stroke volume did not significantly increase after 30% of $V_{O_2}MAX$; 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 others[14] compared younger (20–31 years) and older (51–72 years) endurance trained men and women during cycle exercise at 40%, 70%, and 90% of $V_{O_2}MAX$. The stroke volume in the young men continued to increase progressively throughout exercise to 90% of $V_{O_2}MAX$, 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 $V_{O_2}MAX$. Several of the oldest men also showed a modest decline in stroke volume at exercise intensities above 70% of $V_{O_2}MAX$. 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 $V_{O_2}MAX$. In all groups, stroke volume reached maximal values at 40–60% of $V_{O_2}MAX$ and decreased significantly as exercise intensity increased from 60% to 100% of $V_{O_2}MAX$. 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 $V_{O_2}MAX$, and a fall in stroke volume occurred between 50% and 100% of $V_{O_2}MAX$.

Hagberg *et al*[24]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 $V_{O_2}MAX$. In all groups, stroke volume plateaued at 50% of $V_{O_2}MAX$ and was maintained up to 70% of $V_{O_2}MAX$. Although the stroke volume of the older subjects did not decline, stroke volume measurements were only obtained up to 70% of $V_{O_2}MAX$. Several studies have reported a fall in stroke volume at intensities above 70% of $V_{O_2}MAX$ 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 $V_{O_2}MAX$.

McLaren and colleagues[13] 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 $V_{O_2}MAX$, whereas the stroke volume of the older trained cyclists and controls plateaued at an average of 30% of $V_{O_2}MAX$.

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 $V_{O_2}MAX$ 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 others[16] investigated the effects of sex on stroke volume control during cycle exercise to $V_{O_2}MAX$ in healthy men (20–70 years) and women (20–63 years). In both men and women, stroke volume reached its maximum at 50% of $V_{O_2}MAX$ 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 (52–27 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 others[14] 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 $V_{O_2}MAX$. The stroke volume in the young men had not yet reached a plateau at 90% of $V_{O_2}MAX$, whereas the stroke volume of older men and younger women reached a plateau at 40% of $V_{O_2}MAX$ which was maintained throughout exercise. The older women showed an impaired ability to maintain stroke volume at exercise intensities above 90% of $V_{O_2}MAX$, which was also evident, to a lesser degree, in the oldest men. Spina and coworkers[15] 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 $V_{O_2}MAX$ 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

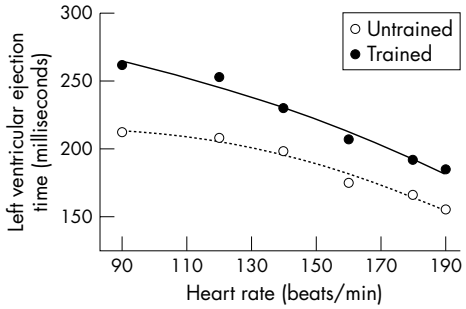

Figure 2 Comparison of ventricular ejection time between trained and untrained subjects. Adapted from Gledhill *et al*.[7]

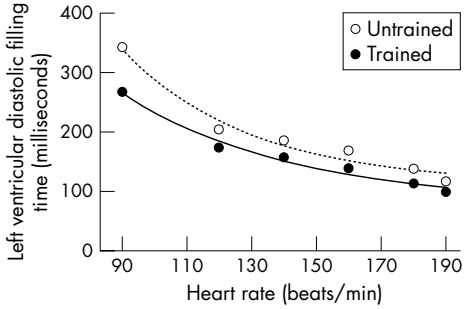

Figure 3 Comparison of ventricular diastolic filling time between trained and untrained subjects. Adapted from Gledhill *et al*.[7]

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 $V_{O_2}MAX$.[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 coworkers[7] 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 others[27] 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 colleagues[20] 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-Urstad and colleagues[32] 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, Vanfraechem[22] 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 $V_{O_2}MAX$ 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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