original investigations

Endurance athletes' stroke volume does not plateau: major advantage is diastolic function

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ABSTRACT

GLEDHILL, N., D. COX, and R. JAMNIK. Endurance athletes' stroke volume does not plateau: major advantage is diastolic function. *Med. Sci. Sports Exerc.*, Vol. 26, No. 9, pp. 1116–1121, 1994. Left ventricular function was examined during incremental work rates to maximum using simultaneous determinations of stroke volume, left ventricular ejection time (LVET), and diastolic filling time (DT). Seven endurance trained and seven untrained young adult males were studied on a cycle ergometer at matched heart rates of 90, 120, 140, 160, 180, and 190 bpm (±1 bpm). Stroke volume of the untrained subjects reached a plateau at 120 bpm, but stroke volume of the trained subjects continued to increase to their maximum heart rate with no plateau. Throughout incremental work rates, LVET was significantly longer and DT was significantly shorter in the trained subjects. At a heart rate of 190 bpm, the corresponding rates of ventricular emptying and ventricular filling of the trained versus the untrained subjects were 20% and 71% greater, respectively ($P < 0.01$). We conclude that during incremental work rates the stroke volume of endurance trained athletes increases progressively to maximum with no plateau. In addition, although trained athletes rely on enhancements in both ventricular filling and ventricular emptying to augment stroke volume, by far their major advantage over untrained subjects is in ventricular filling.

EXERCISE, LEFT VENTRICULAR EJECTION TIME, DIASTOLIC FILLING TIME, BLOOD VOLUME, BLOOD PRESSURE, ACETYLENE REBREATHE TECHNIQUE, MALE CYCLISTS

It is widely accepted that during incremental work rates, stroke volume reaches a plateau at a submaximal heart rate. Also, the stroke volumes of endurance trained athletes are considerably larger than the stroke volumes of untrained individuals (1,2,21,27). However, there is also some generally overlooked evidence that the stroke volumes of endurance trained athletes do not plateau at submaximal work rates, but increase progressively to maximum (8,9,26). This would mean that despite their considerably larger stroke volumes, endurance trained athletes are able to augment ventricular filling and emptying even at very fast heart rates. On the contrary, it is generally believed that at high heart rates the progressively diminishing time available for diastolic filling limits stroke volume, causing it to plateau. We hypothesized, though, that in endurance trained athletes, because of an enhanced myocardial contractility, less time in the cardiac cycle would be required for ventricular emptying, so that more time would be available for filling. Thus, ventricular filling might not restrict the stroke volume of trained athletes at high heart rates.

Previous research on training-induced changes in ventricular emptying and diastolic filling times during incremental work rates is inconclusive, since the limited reported measurements were restricted to rest or confined to moderate work rates and made at varying heart rates (13,15,16,20,25,28). As well, simultaneous determinations of stroke volume and cardiac time intervals during incremental work rates to maximum have not previously been reported. Hence, the purposes of the present investigation were i) to confirm whether the stroke volume of endurance trained athletes increases progressively throughout incremental work rates to maximum, and ii) to compare the ventricular filling and emptying times of endurance trained versus untrained subjects throughout incremental work rates to maximum.

METHODS

Seven male competitive endurance cyclists and seven normally active males participated in the investigation with written informed consent in accord with the policy of the American College of Sports Medicine. The characteristics of the participants are summarized in Table 1. The trained and untrained subjects were matched for age and lean body mass. However, the trained subjects had a significantly higher blood volume than the untrained subjects; and, as dictated by the criteria for subject selection,

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TABLE 1. Characteristics of the subjects.

Subjects		Age (yr)	Height (cm)	Lean Body Mass (kg)	[Hb] ($g \cdot 100^{-1}$)	Blood Volume (ml)	Blood Volume ($ml \cdot kg^{-1}$ lean body mass)	HR_{max} (bpm)	$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$\dot{Q}_{max}$ ($l \cdot min^{-1}$)
Untrained	Mean	22.2	174	69.2	14.4	4457	64.4	192	44.1	24.5
	SEM	±4.9	±2	±4.5	±0.2	±191	±2.6	±4	±1.3	±1.0
Endurance trained	Mean	22.5	178	64.4	14.5	4994*	77.3*	190	68.6*	34.8*
	SEM	±2.1	±1	±3.5	±0.5	±199	±2.8	±2	±2.1	±1.3

* Endurance trained significantly higher than untrained.

the trained subjects had a significantly higher $\dot{V}O_{2max}$ (69 $ml \cdot kg^{-1} \cdot min^{-1}$, range 63–79) than the untrained subjects (44 $ml \cdot kg^{-1} \cdot min^{-1}$, range 41–50). Prior to initiating data collection, participants practiced all experimental procedures until they were thoroughly familiar with the protocol. During the familiarization period, each subject's $\dot{V}O_{2max}$ was determined at least twice on a cycle ergometer using an incremental to maximum plus supramaximum protocol with direct gas analysis (24), and their $\dot{V}O_{2max}$ was measured again during the experimental protocol. The coefficient of variation, expressed as the standard deviation of the difference between triplicate determinations of $\dot{V}O_{2max}$, was ±2.3%. Blood volume was measured via Evans Blue dye employing the methodology described by Chien et al. (4). The coefficient of variation for repeat blood volume measurements, expressed as the standard deviation of the difference between duplicate determinations, was ±3.8%. The duration of exercise at each incremental work rate was 4 min, with $\dot{V}O_2$ and cardiac time intervals recorded toward the end of the 2nd min, followed by triplicate measurements of cardiac output in the final 2 min. This provided ample time in the first 90 s of exercise at each successive work rate for a steady state to be achieved at the new target heart rate before measurements were made. Cardiac output was determined via the acetylene rebreathe technique as described previously (23), and stroke volume was calculated by dividing the mean of three cardiac output determinations (coefficient of variation ± 5.8%, expressed as the standard deviation of the difference between triplicate determinations) by the corresponding heart rate. Repeat rebreathe maneuvers were separated by approximately 45 s to ensure elimination of acetylene as confirmed by end-tidal monitoring.

Cardiac time intervals were determined by averaging the measurements from 10 consecutive heart beats. At least 5 of each 10 beats were sufficiently clear of artifact to permit these measurements. Diastolic filling time (DT), left ventricular ejection time (LVET), and the preejection period (PEP) were measured using simultaneously recorded (Fig. 1) electrocardiograms, phonocardiograms, and ear pulse densitograms (5,19). LVET was determined from the ear pulse densitogram by measuring the time from the beginning of the upslope of the pulse wave to the junction of the incisural notch on the downslope. DT was determined by measuring the time from the second heart sound on the phonocardiogram to the subsequent Q wave on the QRS complex of the electrocardiogram. The PEP was determined by subtracting the combined LVET plus DT from the total time between subsequent Q waves. The rate of ventricular emptying (average dV/dt) was calculated by dividing the change in volume during emptying by the elapsed time during emptying (stroke volume/LVET), and the rate of ventricular filling (average dV/dt) was calculated by dividing the change in volume during filling by the elapsed time during filling (stroke volume/DT).

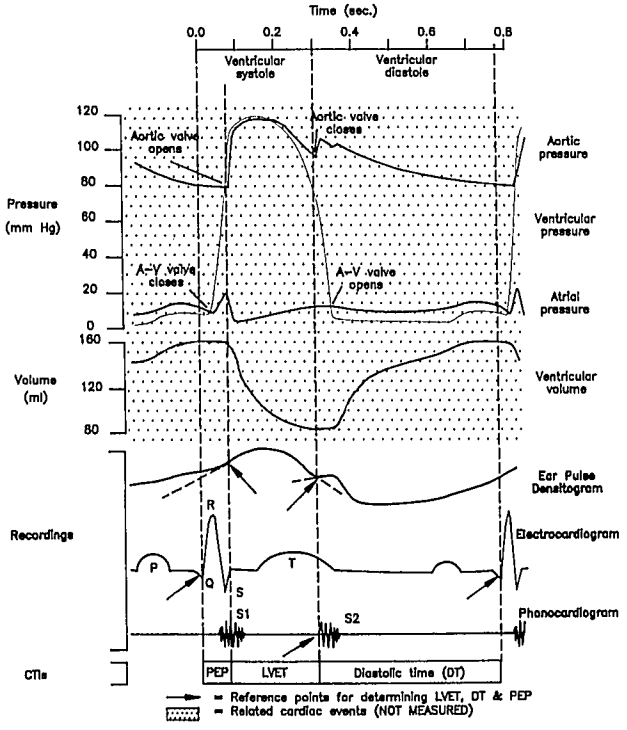

Figure 1—Determination of cardiac time intervals from the electrocardiogram, phonocardiogram, and ear densitogram, plus their relationship to events of the cardiac cycle at a heart rate of 76 bpm.

Since the cardiac time intervals become shorter as heart rate increases, it is essential that comparisons between subjects be made at the same heart rates. Therefore, employing an electromagnetically braked cycle ergometer, work rates were carefully set so that the participants were studied at matched heart rates of 90, 120, 140, 160, 180, and 190 bpm (±1 bpm). The cycle ergometer settings required to elicit the target heart rates were established prior to the onset of the study; and, if required, minor adjustments were made during the initial

90 s of each increment in work rate to achieve the target heart rate ± 1 bpm. At each heart rate, blood pressure was measured using a sphygmomanometer and stethoscope. Statistical analyses were conducted employing linear regression analysis and two-way mixed design repeated measures analysis of variance with post hoc paired group *t*-tests using GB-Stat with a minimum acceptable level of significance of $P \leq 0.05$.

RESULTS

The $\dot{V}O_2$ of both the trained and untrained subjects increased significantly ($P > 0.05$) with each increment in work rate to achieve the target heart rates (Table 2), and predictably, the $\dot{V}O_2$ requirements to elicit heart rates of 120–190 bpm were significantly higher in the trained subjects. Analysis of variance indicated highly significant ($P < 0.0001$) differences between and within groups during exercise for both cardiac output (Table 2) and stroke volume (Fig. 2). *Post-hoc* analysis revealed that while the cardiac output of the untrained subjects increased progressively throughout incremental work rates ($\dot{Q} = 6.62 + 5.1 \times \dot{V}O_2$ l·min^{-1}), their stroke volume reached a plateau at a heart rate of 120 bpm. The cardiac outputs of the trained subjects were significantly ($P < 0.01$) larger than those of the untrained subjects and

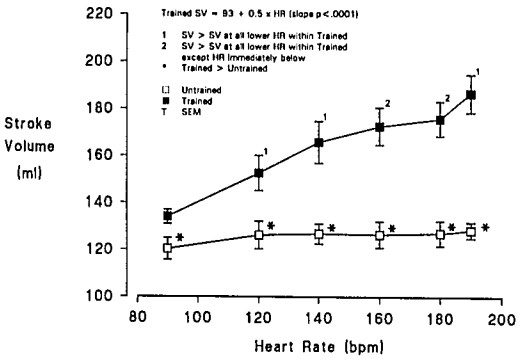

Figure 2—Stroke volume (ml) at each target heart rate during incremental work rates.

increased progressively ($P < 0.05$) throughout incremental work rates ($\dot{Q} = 7.2 + 5.9 \times \dot{V}O_2$ l·min^{-1}). This relationship between $\dot{Q}$ and $\dot{V}O_2$ is remarkably similar to that reported previously by Ekelund and Holmgren (10). In addition, the stroke volumes of the trained subjects were significantly ($P < 0.001$) larger than those of the untrained subjects at all heart rates, and increased progressively ($P < 0.05$) throughout incremental work rates to maximum with no plateau. Further, linear regression analysis indicated that the stroke volume of the untrained subjects reached a plateau (slope = 0.02; not significant)

TABLE 2. $\dot{V}O_2$, cardiac output, left ventricular ejection time (LVET), diastolic filling time (DT), preejection period (PEP), and blood pressure corresponding to each heart rate.

Measurement	Subjects		Heart Rate (bpm) 90	120	140	160	180	190
$\dot{V}O_2$ (l·min^{-1})								
	Untrained	Mean	0.85	1.66†	2.17†	2.60†	3.15†	3.56†
		SEM	±0.03	±0.17	±0.16	±0.15	±0.13	±0.17
	Endurance trained	Mean	0.87	1.92*,†	2.65*,†	3.35*,†	4.05*,†	4.80*,†
		SEM	±0.07	±0.15	±0.14	±0.13	±0.11	±0.15
Cardiac output (l·min^{-1})								
	Untrained	Mean	10.8	15.1†	17.8†	20.2†	22.8†	24.5†
		SEM	±0.4	±0.4	±0.6	±0.9	±1.0	±1.0
	Endurance trained	Mean	12.1	18.2*,†	23.2*,†	27.6*,†	31.6*,†	34.8*,†
		SEM	±1.0	±0.9	±1.3	±1.3	±1.7	±1.3
Cardiac time intervals (ms)								
LVET	Untrained	Mean	212	208†	198†	175†	166†	155†
		SEM	±11	±4	±2	±3	±4	±4
	Endurance trained	Mean	262*	253*,†	230*,†	207*,†	192*,†	185*,†
		SEM	±5	±8	±5	±4	±3	±3
DT	Untrained	Mean	342	204†	185†	168†	138†	117†
		SEM	±55	±9	±4	±4	±3	±9
PEP	Endurance trained	Mean	267*	173*,†	157*,†	138*,†	113*,†	99*,†
		SEM	±22	±6	±3	±3	±4	±6
	Untrained	Mean	122	87†	68†	57	47	44
		SEM	±14	±8	±3	±4	±4	±3
	Endurance trained	Mean	107	74†	63†	55	45	41
		SEM	±28	±7	±3	±3	±4	±3
Blood pressure (mm Hg)								
Diastolic	Untrained	Mean	80	79	81	82	84	84
		SEM	±2	±3	±2	±3	±3	±2
	Endurance trained	Mean	66*	64*	67*	68*	68*	67*
		SEM	±2	±2	±1	±1	±1	±1
Systolic	Untrained	Mean	135	143†	158†	172†	184†	209†
		SEM	±3	±2	±4	±3	±3	±3
	Endurance trained	Mean	117*	125*,†	141*,†	158*,†	174*,†	190*,†
		SEM	±4	±3	±4	±5	±4	±3

* Endurance trained significantly different from untrained ($P < 0.05$).
† Significantly different from previous lower heart rate ($P < 0.05$).

at a heart rate of 120 bpm, while the stroke volume of the trained subjects increased progressively at a slope of 0.5 ($P < 0.0001$) throughout incremental work rates to maximum (Fig. 2).

The mean maximum heart rate of the trained subjects did not differ significantly from that of the untrained subjects (190 and 192 bpm, respectively; Table 1). It is apparent in Table 2 that at all heart rates, the trained subjects had a significantly longer ventricular emptying time and a significantly shorter ventricular filling time ($P > 0.01$) than the untrained subjects, but there were no significant differences in the PEP of the trained versus the untrained subjects. Also, the LVET and DT of both the trained and untrained subjects shortened progressively ($P < 0.05$) with each increase in heart rate. The mean blood volume of the trained subjects (4994 ml; 77.3 ml·kg^{-1} lean body mass) was significantly larger ($P < 0.01$) than the mean blood volume of the untrained subjects (4457 ml; 64.4 ml·kg^{-1} lean body mass). As well, at all heart rates, the systolic and diastolic blood pressure of the trained subjects were significantly lower (Table 2). For both the trained and untrained subjects, systolic blood pressure increased progressively ($P < 0.01$) with each increment in heart rate from 90 to 190 bpm. However, there were no significant within-group differences in diastolic blood pressure for either the trained or untrained subjects.

DISCUSSION

During incremental work rates to maximum, alterations in the stroke volume of the untrained subjects conformed to the classic literature description of a plateau at a heart rate of 120 bpm (1,2,21,27), which corresponded to a workload of approximately 40% $\dot{V}O_{2max}$ (Fig. 2). As has occasionally been apparent in previous reports (8,9,26), but generally overlooked, however, the stroke volume of the trained subjects increased progressively throughout incremental work rates to maximum. Hence, the trained subjects accomplished the augmentations in ventricular filling and emptying associated with their considerably larger stroke volumes even at very high heart rates.

Although the LVET and DT data are contrary to our hypothesis, the findings are comprehensible when the rates of ventricular emptying and filling are examined. At all exercise heart rates, the ventricular emptying time of the trained subjects was significantly ($P < 0.01$) longer than that of the untrained subjects (Table 2). At first glance, the fact that it took the trained subjects a longer time for ejection implies that they may not rely on an enhanced ventricular emptying. In the trained subjects, however, the rate of ventricular emptying increased progressively ($P < 0.01$) throughout exercise (Fig. 3); and at heart rates of 160, 180, and 190 bpm, it was significantly ($P < 0.01$) greater than that of the untrained subjects,

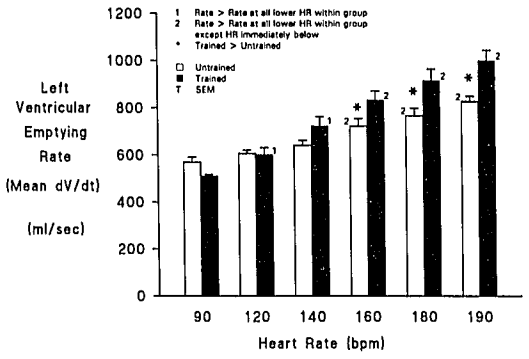

Figure 3—Ventricular emptying rate (ml·s^{-1}) at each target heart rate during incremental work rates.

indicating that the trained subjects do indeed rely on enhancements in ventricular emptying.

Concurrently, the ventricular filling time of the trained subjects was significantly ($P < 0.01$) shorter than that of the untrained subjects (Table 2). The fact that it took the trained subjects a shorter time to fill a greater volume implies that they rely on considerable enhancements in ventricular filling to accomplish the augmentation in stroke volume. Further, at each heart rate from 90 to 190 bpm, the rate of diastolic filling increased progressively ($P < 0.01$) and was significantly greater in the trained subjects (Fig. 4), which further supports their reliance on an enhanced ventricular filling. In fact, at heart rates of 160–190 bpm, the rate of ventricular emptying was only 15–20% greater in the trained subjects, whereas the corresponding rate of ventricular filling was 66–71% greater in the trained subjects.

Therefore, although these findings support the contention that to increase stroke volume endurance trained athletes rely on augmentations in ventricular emptying, it appears that they depend far more heavily on enhancements in ventricular filling. Moreover, at a heart rate of 190 bpm, the rate of ventricular filling for the trained subjects was 86% greater than their rate of ventricular emptying (1880 ml·s^{-1} vs 1010 ml·s^{-1}), whereas the rate of ventricular filling for the untrained subjects was only

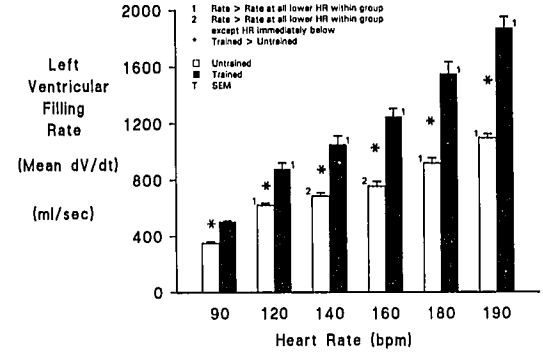

Figure 4—Ventricular filling rate (ml·s^{-1}) at each target heart rate during incremental work rates.

32% greater than their rate of ventricular emptying (1100 $ml \cdot s^{-1}$ vs 830 $ml \cdot s^{-1}$). When diastolic filling is expressed in this manner it is somewhat startling to consider that during maximal exercise the ventricle of the endurance trained athletes was refilled with each 188 ml stroke volume in 0.1 s, or a rate of 1880 $ml \cdot s^{-1}$! This finding implies that the ventricular preload and/or compliance of endurance trained athletes is considerably enhanced.

The functional definition employed in this paper for the onset of the period of diastole is the beginning of the second heart sound, and therefore, the resultant "filling" phase also includes the period of isovolumetric relaxation prior to the opening of the atrioventricular valve. Thus, the time in which filling (diastole) actually occurs is shorter than the reported DT, and the true rate of ventricular filling is even higher than calculated. Hence, the enormity of the difference between the rates of ventricular filling and emptying is even greater than described above.

The cardiac time intervals of endurance trained athletes have been studied previously, but the findings are inconclusive. Many of the measurements in earlier investigations were restricted to rest, and exercise measurements were often made shortly after rather than during exercise or only up to moderate work rates and frequently with comparisons made between cardiac time intervals determined at different heart rates. Hence, endurance trained athletes have been reported to have a prolonged LVET and a shortened DT (13,16,20) or an unchanged LVET and DT (15,25,28) relative to normally active individuals during exercise. Further, since simultaneous measurements of stroke volume and cardiac time intervals have not previously been conducted during maximal exercise, the corresponding rates of ventricular emptying and filling (average dV/dt) have not been completely examined prior to this investigation. Hence, in prior investigations the relative magnitude of changes in the LVET and DT of endurance trained athletes has not been illustrated as graphically as in the present study.

The LVET and DT findings are supported by related measurements in the present study. For example, the observed larger blood volumes of the trained subjects would likely have helped maintain an adequate filling pressure and end diastolic volume in the presence of a shorter DT even at high heart rates. In fact, as has been proposed previously (14), we feel that the higher blood volume of endurance trained athletes is a major contributor to their enhanced ventricular function. Also, since the trained subjects had lower systolic and diastolic blood pressures, cardiac afterload would have been reduced, thereby facilitating ventricular emptying. In addition, the accompanying lower total peripheral resistance (approximately 40% less in the trained subjects), together with their increased venomotor tone would have augmented venous return. As well, venous return would have been further enhanced by an increase in the effective transmural filling pressure owing to the decreased intrathoracic pressure accompanying the higher ventilation of the trained subjects (165 vs 130 $l \cdot min^{-1}$ for the untrained subjects at the maximum work rate).

It is important to point out that the diastolic phase of the cardiac cycle is of primary importance for the supply of blood and oxygen to the cardiac muscle. Hence, the curtailment of diastolic time during exercise is possibly counterproductive to the accompanying need for increased myocardial oxygenation in endurance trained athletes. However, since the product of heart rate and systolic blood pressure provides an indication of myocardial oxygen demand, the lower systolic blood pressure of the trained subjects at all exercise heart rates indicates that although the corresponding values for cardiac output and $\dot{V}O_2$ were higher, the myocardial O_2 demand of the trained subjects was lower throughout exercise. Hence, the possibility of myocardial ischemia compromising ventricular function during incremental work rates to maximum is lower in trained subjects.

Providing support for the observations in the present study is the echocardiographic finding that diastolic function in endurance trained athletes is improved such that ventricular relaxation is significantly faster (7,12,18). In addition, left ventricular distensibility in trained cyclists is unaltered (11) or perhaps even increased (17) so that the dilation of the ventricle during exercise (6,8,18) would cause a distension of the atrioventricular aperture. As well, the sucking effect created when the compressed (elastic) myocardium recoils from a lower end systolic volume in endurance trained individuals (3,22), together with a more effective atrial contraction, would have assisted cardiac filling (18). As reported previously by Crawford et al. (8), the net result of an enhanced ventricular filling is that endurance trained individuals are able to make greater use of the more energy efficient Frank-Starling mechanism to accomplish the high stroke volumes during maximal exercise.

We conclude that during incremental work rates the stroke volume of endurance trained athletes increases progressively to maximum with no plateau. In addition, to augment stroke volume even at high heart rates, endurance trained athletes rely on enhancements in both ventricular filling and ventricular emptying. They rely to a far greater degree on augmentations in ventricular filling, however, possibly due to a greater ventricular preload and/or compliance. It is important to point out, though, that a limitation of the cross-sectional research approach employed in the present investigation is that the "endurance trained" participants could have been genetically endowed with the observed enhancement in ventricular function and excelled in endurance cycling due to natural selection. Hence, it is not possible to state with certainty, based on these findings, that the difference in cardiac function between endurance trained athletes and normally active individuals is an adaptation to endurance training, a consequence of genetics, or a combination of

these influences. We speculate, however, that a major portion of the difference is due to the training-induced higher blood volume of the endurance athletes, and this is a topic for further investigation.

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REFERENCES

1. ASTRAND, P. O., T. E. CUDDY, B. SALTIN, and J. STENBERG. Cardiac output during submaximal and maximal work. *J. Appl. Physiol.* 19:268–274, 1964.
2. BEVEGARD, S., A. HOLMGREN, and B. JONSSON. Circulatory studies in well trained athletes at rest and during heavy exercise, with special reference to stroke volume and the influence of body position. *Acta Physiol. Scand.* 57:26–50, 1963.
3. BRECHTER, G. A. and A. T. KISSEN. Relation of negative ventricular pressure to intraventricular volume. *Circ. Res.* 5:157–162, 1957.
4. CHIEN, S., U. SHUNICHI, R. L. SIMMONS, F. F. MCALLISTER, and M. I. GREGERSEN. Blood volume and age: repeated measurements on normal men after 17 years. *J. Appl. Physiol.* 21:583–588, 1966.
5. CHIRFIE, R. and D. H. SPODICK. Densitography: a new method for evaluation of cardiac performance at rest and during exercise. *Am. Heart J.* 83:493–503, 1972.
6. COHEN, J. L. and K. R. SEGAL. Left ventricular hypertrophy in athletes: an exercise-echocardiographic study. *Med. Sci. Sports Exerc.* 17:695–700, 1985.
7. COLAN, S. D., S. P. SANDERS, D. MACPHERSON, and K. M. Borow. Left ventricular diastolic function in elite athletes with physiologic cardiac hypertrophy. *J. Am. Coll. Cardiol.* 6:545–549, 1985.
8. CRAWFORD, M. H., M. A. PETRU, and C. RABINOWITZ. Effect of isotonic exercise training on left ventricular volume during upright exercise. *Circulation* 72:1237–1243, 1986.
9. EKBLOM, B. and L. HERMANSON. Cardiac output in athletes. *J. Appl. Physiol.* 25:619–625, 1968.
10. EKELUND, L. G. and A. HOLMGREN. Central hemodynamics during exercise. *Circ. Res.* 20–21(Suppl. I):33–43, 1967.
11. FAGARD, R., C. VAN DEN BROEKE, E. BIELEN, et al. Assessment of stiffness of the hypertrophied left ventricle of bicyclists using left ventricular Doppler velocimetry. *J. Am. Coll. Cardiol.* 9:1250, 1987.
12. FAGARD, R., C. VAN DEN BROEKE, and A. AMERY. Left ventricular dynamics during exercise in elite marathon runners. *J. Am. Coll. Cardiol.* 14:112, 1989.
13. FARDY, P. S. The influence of physical activity on selected cardiac time components. *J. Sports Med. Phys. Fitness* 11:227–233, 1971.
14. HOPPER, M. K., A. R. COGGAN, E. E. COYLE. Exercise stroke volume relative to plasma-volume expansion. *J. Appl. Physiol.* 64:404–408, 1988.
15. KRZEMINSKI, K., W. NIEWIADOMSKI, and K. NAZAR. Dynamics of changes in the cardiovascular response to submaximal exercise during low intensity endurance training with particular reference to the systolic time intervals. *J. Appl. Physiol.* 59:377–384, 1989.
16. LAMONT, L. Effects of training on echocardiographic dimensions and systolic time intervals in women swimmers. *J. Sports Med.* 20:397–404, 1980.
17. LEVINE, B. D., L. D. LANE, J. C. BUCKEY, D. B. FRIEDMAN, and C. G. BLOMQUIST. Left ventricular pressure-volume and Frank-Starling relations in endurance athletes. *Circulation* 84:1016–1023, 1991.
18. MATSUDA, M., Y. SUGISHITA, S. KOSEKI, et al. Effect of exercise on left ventricular diastolic filling in athletes and non-athletes. *J. Appl. Physiol.* 55:323–338, 1983.
19. QUARRAY-PIGOTT, V., R. CHIRFIE and D. H. SPODICK. Clinical and physiological applications. *Circulation* 43:239–246, 1973.
20. RAAB, W. Aims and perspectives: training, physical inactivity, and the cardiac dynamic cycle. *J. Sports Med. Phys. Fitness* 6:38–47, 1966.
21. ROWELL, L. B. Circulation. *Med. Sci. Sports* 1:15–22, 1969.
22. SABBAH, H. N. and P. D. STEIN. Pressure-diameter relations during early diastole in dogs. Incompatibility with the concept of passive left ventricular diastolic filling. *Circ. Res.* 48:357–365, 1981.
23. SMYTH, R. J., N. GLEDHILL, A. B. FROESE, and V. JAMNIK. Validation of non-invasive maximal cardiac output measurement. *Med. Sci. Sports Exerc.* 16:512–515, 1984.
24. THODEN, J. S. Testing aerobic power. In: *Physiological Testing of the High Performance Athlete*, D. MacDougall, et al. (Eds.). Champaign, IL: Human Kinetics, 1990, pp. 107–173.
25. UNDERWOOD, R. H. and J. L. SCHWADE. Noninvasive analysis of cardiac function of elite endurance runners-echocardiography, vectorography, and cardiac intervals. *Ann. N. Y. Acad. Sci.* 301: 297–309, 1977.
26. VANFRAECHEM, J. H. P. Stroke volume and systolic time interval adjustments during bicycle exercise. *J. Appl. Physiol.* 46:588–592, 1979.
27. WANG, Y., R. J. MARSHALL, and J. T. SHEPHERD. The effect of changes in posture and of graded exercise on stroke volume in man. *J. Clin. Invest.* 39:1051–1061. 1960.
28. ZONERAICH, S., O. ZONERAICH, J. J. RHEE, D. JORDAN, and J. APPEL. Evaluating endurance athlete's heart. *Circulation* 52:455–458, 1975.