CONTINUING MEDICAL EDUCATION

Competitive Sports and the Heart: Benefit or Risk?

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SUMMARY

Background: Controversy surrounds the cardiac effects of competitive sports and the athlete's heart. In this review, we present and discuss the main cardiological findings in competitive athletes.

Method: Selective review of pertinent literature retrieved by a search with the keywords "athlete's heart," "ECG," "echocardiography," "endurance exercise," "longevity," and others.

Results: Regular exercise leads to functional and structural adaptations that improve cardiac function. Athlete's heart, which develops rarely, is a typical finding in endurance athletes. This condition is characterized by physiological, harmonically eccentric hypertrophy of all cardiac chambers. The athlete's ECG can be used to distinguish physiological, training-related changes from pathological training-unrelated changes. The athlete's heart function is normal at rest and increases appropriately during exercise. The cardiac markers troponin and B-type natriuretic peptide are within the normal range in healthy athletes at rest, but can temporarily be mildly elevated after exhausting endurance-exercise, without evidence of myocardial damage. The epidemiological data suggest that participation in competitive sports increases life expectancy.

Conclusion: Competitive exercise does not induce cardiac damage in individuals with healthy hearts, but does induce physiological functional and structural cardiac adaptations which have positive effects on life expectancy.

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Physicians of the ancient and early modern world often recommended physical exercise as a means of staying healthy. Today, various medical specialty societies recommend exercise and athletic activity to maintain and preserve health, on the basis of strong evidence from numerous single studies and meta-analyses (1). On the other hand, ever since the "athlete's heart" was described more than 100 years ago (e1), there has been concern that prolonged and intense athletic activity might confer certain risks, while exercise-induced cardiac changes have been interpreted as a potential sign of damage (e2, e3). The question whether sport has a net positive or negative effect on health was already debated in 1912 at the first sports-medicine conference in Germany (e4). Among physicians, those with no special training or experience in sports medicine tend to view athlete's heart as a potentially dangerous condition and to think that the associated cardiac enlargement and ECG changes indicate an increased cardiac risk. Reindell et al. (2), and later Kindermann (3), interpreted the radiological, electrocardiographic, and hemodynamic changes seen in healthy athletes as physiological adaptations of the heart; yet the recurring reports of cardiac events, and even sudden cardiac death, among athletes (4, e5) continue to sustain the debate over the potential pathological effects of sports on the heart, rare as they may be (e6).

Learning objectives

We critically review the current evidence about the risks and benefits of athletic activity and describe the proper classification of cardiac findings in competitive athletes from the point of view of the sports cardiologist. Readers should become well informed about the following matters:
- physiological cardiovascular adaptations,
- ECG changes,

Strong evidence

Various medical specialty societies recommend exercise and athletic activity to maintain and preserve health, on the basis of strong evidence from numerous single studies and meta-analyses.

- structural and functional findings of imaging studies,
- stress-induced elevation of the concentrations of the cardiac markers troponin and BNP,
- and the life span of competitive athletes.

Methods

We selectively searched the PubMed database for articles published up to June 2012 that contained the keywords "athletes," "athlete's heart," "BNP," "ECG," "echocardiography," "electrocardiography," "endurance exercise," "magnetic resonance imaging," "MRI," "NT-proBNP," "troponin," "longevity," and others. We present their most important findings in this article. The recommendations given here regarding ECG interpretation in patients who participate in sports correspond to the current criteria of the European Society of Cardiology (5).

Physiological cardiovascular changes

Oxygen uptake rises by a factor of 10 to 12 during exercise in healthy, untrained individuals and by a factor of 20 or more in highly trained endurance athletes (e7). This rise is accounted for by increases in cardiac output and in the arteriovenous oxygen concentration gradient. The increased cardiac output is mainly due to a faster heart rate; as a rule of thumb, the maximum attainable number of beats per minute can be estimated as 220 minus the age in years. The increased heart rate is caused initially by a drop in parasympathetic activity, and then, during intermediate or intense exercise, by an increase in sympathetic activity. The stroke volume increases by only 30% to 50%.

Dynamic exercise causes a rise in systolic blood pressure that is linearly related to exercise intensity, while the diastolic pressure is only marginally changed. On the other hand, static exercise—in particular, maximal exertion—or high-intensity dynamic exercise or Valsalva maneuvers causes a much greater rise in both systolic and diastolic blood pressure (e8). The pressures in the pulmonary artery and in the heart itself rise only slightly during exercise and remain in the normal range even under exercise stress *(Figure 1)* (3, e9). In general, dynamic exercise mainly increases the heart's volume load, while static stress mainly increases its pressure work (e10).

Cardiovascular adaptation differs depending on the type of physical exercise and on the extent and intensity of training. Functional adaptations appear within a few

FIGURE 1

Pulmonary-artery pressure (mmHg)

PA$_s$
PA$_m$
PA$_d$

50±5   HR (min^{-1})   173±8

At rest   300 Watts
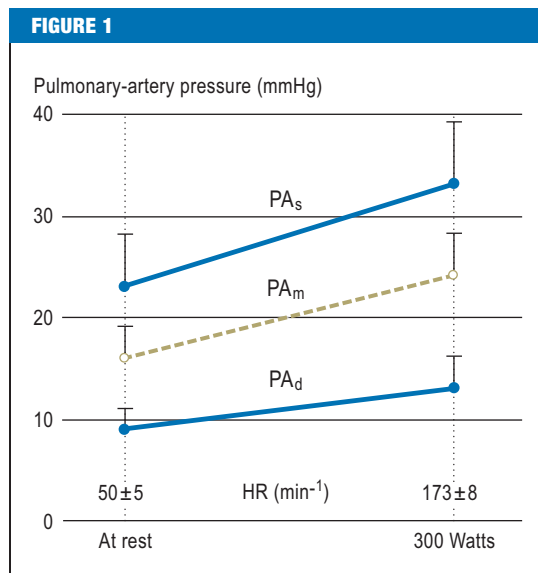
Pulmonary-arterial pressure in athlete's heart
Measurements, by Swan-Ganz catheterization, of pulmonary-arterial pressures (PA$_s$ = systolic; PA$_d$ = diastolic; PA$_m$ = mean) and heart rate (HR) at rest and during maximal bicycle ergometry in nine endurance athletes (age. 21 ± 3 years) with athlete's heart (heart volume 1148 ± 133 mL, 15.1 ± 1.5 mL/kg). From Reference (3).

weeks, requiring an additional energy consumption of at least 500 to 1000 kcal per week (6, e11, e12), which corresponds, for example, to brisk walking for one hour two to three times a week. Aerobic training lowers the heart rate and increases stroke volume without changing cardiac output at rest or for a given exercise intensity representing an economization of cardiac function (e13). Maximal cardiac output rises because the heart-rate reserve capacity increases while the maximal heart rate remains constant. The stroke volume is greater because of improved filling dynamics of the left ventricle, including greater compliance (e9, e14) and reduced peripheral vascular resistance (e15), so that the end-diastolic volume rises and the end-systolic volume falls. Increased shear stress during exercise improves endothelial function, with secretion of vasodilatory substances (e16).

Training beyond a certain level (which differs from one individual to another) leads to structural adaptations as well. These dimensional changes of the heart are known as athlete's heart; they generally arise only

Oxygen uptake in untrained persons and in highly trained endurance athletes

Oxygen uptake rises by a factor of 10 to 12 during exercise in healthy, untrained persons and by a factor of 20 or more in highly trained endurance athletes.
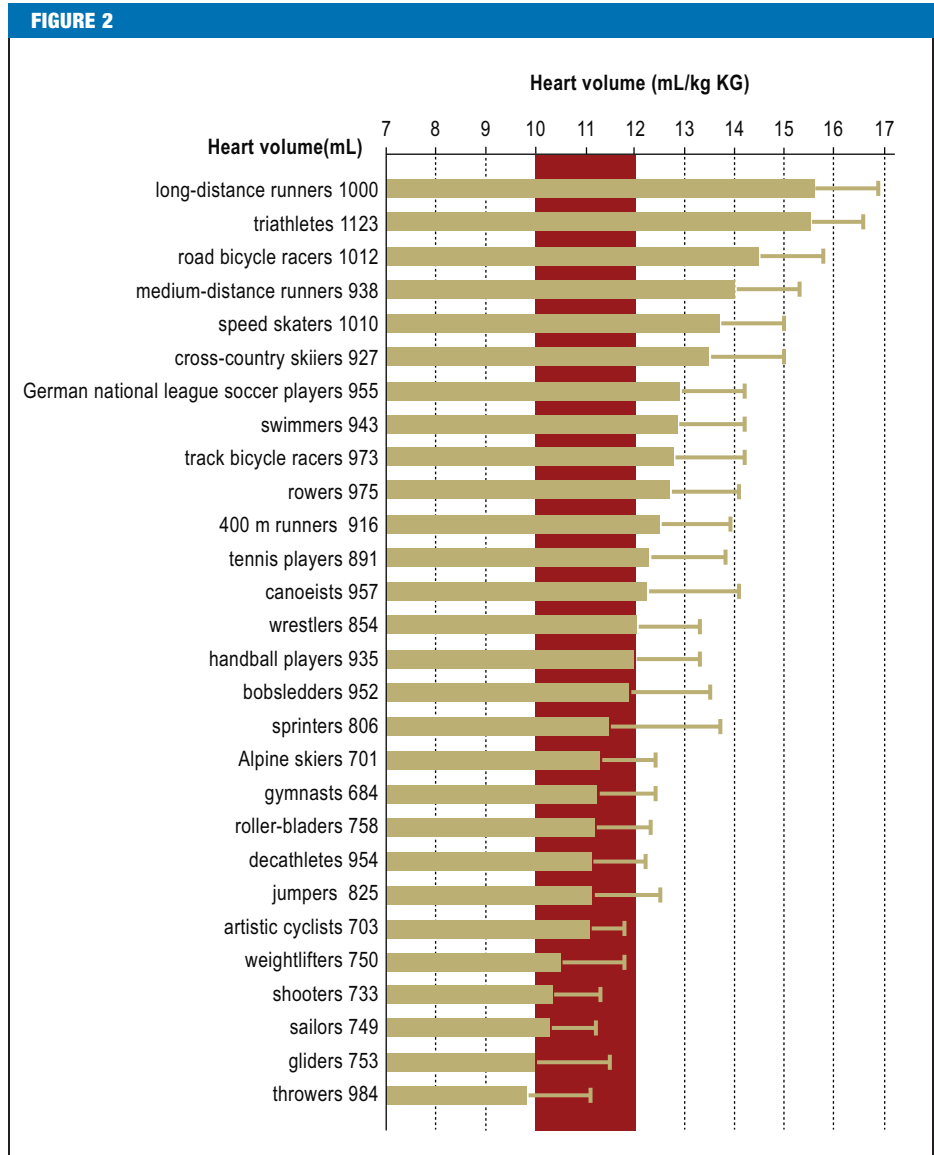
The response of blood pressure to dynamic stress

Dynamic stress causes a rise in systolic blood pressure that is linearly related to the intensity of stress, while the diastolic pressure is only marginally changed.

Heart volumes in different types of competitive athletes
Means and standard deviations of relative heart volumes in male athletes in different sports. The mean absolute heart volumes are given at the left edge of the figure.
Red: normal range. Athlete's heart is defined, in men, as a heart volume ≥ 13 mL/kg body weight.
Gray zone: 12–13 mL/kg body weight

FIGURE 2

Heart volume (mL/kg KG)

Heart volume(mL)

- long-distance runners 1000
- triathletes 1123
- road bicycle racers 1012
- medium-distance runners 938
- speed skaters 1010
- cross-country skiiers 927
- German national league soccer players 955
- swimmers 943
- track bicycle racers 973
- rowers 975
- 400 m runners 916
- tennis players 891
- canoeists 957
- wrestlers 854
- handball players 935
- bobsledders 952
- sprinters 806
- Alpine skiers 701
- gymnasts 684
- roller-bladers 758
- decathletes 954
- jumpers 825
- artistic cyclists 703
- weightlifters 750
- shooters 733
- sailors 749
- gliders 753
- throwers 984

after endurance training in an amount that is generally only undertaken by competitive athletes. All chambers of the heart become dilated and hypertrophic. This eccentric hypertrophy is a harmonic enlargement of the heart (7) in which the heart mass does not exceed the critical value of 7.5 g/kg, corresponding on average to 500 g. In some cases, an athlete's heart can be nearly twice as big as that of an untrained, healthy person (e17, e18).

Athlete's heart is rarer than generally thought and is not a prerequisite for a beneficial effect of training on health. At least five hours of endurance training per week, and more in many cases, are necessary for the volume load to result in dimensional changes (e19).

Blood pressure during static exercise
Static exercise causes a much greater rise in both systolic and diastolic blood pressure.

Athlete's heart
Athlete's heart is rarer than generally thought. It typically affects endurance athletes and is characterized by harmonic, biventricular, eccentric hypertrophy. Strength and speed athletes generally do not develop athlete's heart.

The amount is highly variable: Running 60–70 km per week leads to the development of athlete's heart in some persons, while others do not develop it even if they run 100 km per week. Athlete's heart appears most prominent in long-distance runners, road bicycle racers, cross-country skiers, and triathletes. Athlete's heart may also develop in older endurance athletes. On the other hand, strength and speed athletes, such as weightlifters, gymnasts, sprinters, high-jumpers, discus- and javelin-throwers, or Alpine skiers, generally do not develop athlete's heart *(Figure 2)*. The so-called strength athlete's heart, which—in contrast with the eccentric hypertrophy characteristic of endurance athlete's heart—shows concentric hypertrophy (8, e20), is discussed in the literature, not primarily in connection with exercise-related cardiac load, but more frequently in connection with the abuse of anabolic steroids and other performance-enhancing drugs (9, 10, e21). Pulmonary-arterial pressure and intracardiac pressure is not elevated in athlete's heart *(Figure 1)* (3).

When training ceases, an athlete's heart size reduces again (e22, e23), with individually variable rates of regression. Immobilization (e.g., bed rest) leads to rapid regression of heart size (2). Athlete's heart often regresses incompletely, however, with persistent left ventricular enlargement despite normalization of chamber thickness (e13, e23). Incomplete regression is thought to be due to genetic factors combined with continued athletic activity at a lower level than before. An important point for clinical practice is that, as long as there is still some degree of cardiac enlargement from athlete's heart, the individual will still have a higher than average ergometric performance capacity for age.

ECG changes in athletes

Various authors have reported ECG changes in athletes and have attempted to distinguish them from pathological findings (2, 5, 11, 12, e25–e27). There is, however, a gray zone between physiological and pathological changes. Depending on the particular study and method of classification used, 5% to 40% of athletes are found to have an abnormal or moderately to distinctly abnormal ECG (e25, e28, e29). Even higher percentages of abnormal findings have been found in selected groups of highly trained athletes, and only a small fraction of these persons (ca. 5%) have structural heart disease unrelated to their athletic activity (e25). The necessary distinction between common, training-related ECG changes and uncommon, training-

unrelated ones that may be pathological should be drawn according to the current criteria and recommendations *(Box)* (5, e27). Hence, false-positive findings can be markedly reduced, and specificity increased without loss of sensitivity (11, e28), compared to earlier recommendations (12). Uncommon ECG changes are more frequent in men than in women, and they are also more frequent in black athletes, and require further evaluation (13, e29, e30).

ECG changes are most commonly seen in endurance athletes (e25). Sinus arrhythmia and sinus bradycardia are very common : on Holter monitoring, the heart rate may be found to dip as low as 30 beats per minute, or rarely even lower, usually at night. First-degree AV block (asymptomatic; resolves under

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BOX

ECG changes

- **Common, training-related ECG changes**
  - sinus bradycardia
  - 1st-degree AV block, 2nd-degree AV block of Wenckebach type
  - incomplete right bundle branch block
  - early repolarization
  - isolated QRS voltage criteria for left heart hypertrophy

- **Uncommon, training-unrelated ECG changes**
  - T wave inversion in at least two adjacent leads
  - epsilon wave[1]
  - ST segment depression
  - pathological Q waves
  - left atrial enlargement
  - left anterior hemiblock, left axis deviation
  - left posterior hemiblock, right axis deviation
  - right ventricular hypertrophy
  - ventricular preexcitation syndrome (Wolff-Parkinson-White syndrome)
  - complete left or right bundle branch block
  - long or short QT intervall (long or short QT syndrome)
  - Brugada-like early repolarization[2]

Common ECG changes that require no further evaluation in healthy, asymptomatic athletes, versus uncommon ECG changes unrelated to athletic training that require evaluation in athletes; modified from Corrado et al. (5).
[1] Post-excitation with a small wave in the ST segment in leads V1 to V3 as evidence of an arrhythmogenic right ventricular cardiomyopathy (ARVC)
[2] Brugada-syndrome: an ion-channel disease characterized by ST elevation in leads V1 through V3, with an elevated risk of sudden cardiac death

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The evaluation of an athlete's ECG

A distinction must be drawn between physiological training-related ECG changes and training-unrelated changes which may be pathological.

Atrial fibrillation

Atrial fibrillation is more common among middle-aged and older endurance athletes who have been training for many years than among athletically inactive persons of the same age.

TABLE 1

Echocardiographic upper limits for athlete's heart

	Men	Women
Heart volume (mL/kg)	20	19
Heart mass (g/kg)	7.5	7
LV myocardial mass (g/m^2) – Devereux (e76) – Teichholz (e77) – Dickhuth (e34)	165–170 135 137	130
LV EDD (mm)	63 (–67*[1])	60 (–63*[1])
LV EDD (mm/m^2 BSA)	32	33
LV wall thickness (mm)	13 (–15*[2])	12
Left atrium (mm)	45 (– 50)	45
RV EDD (mm)	32	
RV EDD (mm/m^2 BSA)	17	

LV, left ventricle; RV, right ventricle; EDD, end-diastolic diameter;
BSA, body surface area
*[1]upper limit for individuals with large body dimensions
*[2]gray zone: 13–15 mm

stress) and second-degree AV block of Mobitz type I (Wenckebach block) are often seen in athletes, but second-degree AV block of Mobitz type II and third-degree AV block are atypical and require further evaluation.

Occasional ventricular and supraventricular extrasystoles are seen in athletes as well and are of no significance as long as they are asymptomatic. Frequent extrasystoles (more than 2000 in 24 hours on Holter monitoring [e31]) and paroxysmal supraventricular tachycardia require evaluation. Any arrhythmia that worsens during exercise must be evaluated; in athletes with a very low basal heart rate, extrasystoles usually disappear during exercise as sympathetic activity increases. In unclear cases, ambulatory ECG monitoring—ideally including the performance of usual athletic activities—should be performed to rule out significant arrhythmias.

Atrial fibrillation is more common among middle-aged and older endurance athletes who have been training for many years than it is among athletically inactive persons of the same age (for elderly persons, 23% versus 12.5%) (14). The proposed pathophysiological mechanisms involve altered autonomic regulation due to ath-

letic training, leading to a more intense vagal reaction, often at night; a lesser degree of sympathetic stimulation; and atrial remodeling. Endurance athletes aged 20 to 30 do not have atrial fibrillation any more commonly than non-athletes (e32). On the other hand, it has been found that moderate endurance training, as performed in preventive-fitness programs, may actually lower the risk of atrial fibrillation in old age (e33).

Alterations in the QRS complex and during repolarization are also more commonly seen in athletes and are usually physiological. 35% to 50% of athletes have an incomplete right bundle branch block; endurance athletes are the most likely to exhibit this finding (5). In contrast, complete right or left bundle branch block is not due to athletic activity and requires further evaluation. Isolated QRS voltage elevations should not be taken as an indicator of hypertrophy among athletes.

50% to 80% of all highly-trained athletes present with early repolarization, which is reflected in the ECG by elevation of the beginning of the ST segment (by at least 0.1 mV at the J point), usually seen in leads V2 through V4. The ST segment is then either concave (the typical finding in white athletes) or convex (the typical finding in black athletes). Moreover, as many as 25% of healthy black athletes with ST-segment elevation may also have negative T waves (5). The latter repolarization abnormality, which is more common in both male and female black athletes than in white athletes (13, e29, e30), may represent an ethnic variant of athlete's heart (e31). Negative T waves that are at least 2 mm deep and are seen in two or more adjacent leads (frequency among athletes, ca. 3%) and ST-segment depressions require further evaluation (5, e25).

The QT interval is generally somewhat longer in athletes because of their lower heart rate. When correcting the QT interval for the lower rate, one must bear in mind that the correction is imprecise for rates under 40 beats per minute or over 80–100 beats per minute. A QTc of 500 ms or above is considered unequivocally pathological, while the range from 440 to 500 ms (in men) or from 460 to 500 ms (in women) is considered a gray zone (5).

Findings of imaging studies
Echocardiography
The most important routine method for differentiating physiological from pathological cardiac hypertrophy is echocardiography, which can also be used to determine cardiac volume (e34, e35). The normal cardiac

Repolarization changes in an athlete's ECG
50% to 80% of all highly-trained athletes have early repolarization, which is reflected in the ECG by elevation of the beginning of the ST segment.

ECG changes in black athletes
Repolarization changes with negative T waves are more common in black athletes than in white athletes.

volume depends on body weight and lies in the range of 10–12 mL/kg in men and 9–11 mL/kg in women *(Figures 1 and 2)* (e36). Athlete's heart is defined as a heart volume ≥12 mL/kg body weight in women and 13 mL/kg body weight in men, up to a maximum of 19 and 20 mL/kg, respectively. The end-diastolic diameter of the left ventricle is enlarged in an athlete's heart (60 mm or more in 15% of athletes), while the left ventricular wall thicknesses are normal or in the upper portion of the normal range (13–15 mm in 2% to 4% of athletes) *(Table 1)* (15, 16). The left ventricular wall may be 1 or 2 mm thicker in black athletes (15). The clinical relevance of these findings remains unclear. The relative wall thickness, defined as the ratio of left ventricular thickness to end-diastolic inner diameter, normally does not exceed 42–43% in athletes *(Figure 3)* (10, e37). The left artrium is enlarged in 20% of athletes, with maximum values of 50 mm (men) and 45 mm (women) (e32) *(Table 1)*. Physiological atrial remodeling is closely related to left ventricular dilatation; thus, left atrial enlargement is mainly seen in endurance athletes and strength-endurance athletes (e.g., rowers, canoeists) (e32).

In healthy athletes, systolic function is normal at rest. For those with athlete's heart, it may be in the low-normal range or even slightly below it, while the stroke volume remains normal (7, 10, e24, e36). Stress echocardiography reveals a normal increase in systolic function (10). Diastolic function is normal or in the high-normal range (10, e38). Depending on the type of study and the particular variables measured, regional systolic and diastolic left and right ventricular myocardial function, which can be visualized on the basis of myocardial deformation with tissue Doppler and with two- and three-dimensional speckle tracking *(Figure 4)*, are found to lie in the low- to high-normal range; the same is true of left and right atrial function (e38–e49).

Echocardiography has often been performed before and immediately after exhausting endurance exercise, or endurance competitions, to determine whether there has been any acute exercise-induced damage to the heart (e50–e52). Such studies generally reveal a mild reduction of diastolic function, and sometimes of systolic function as well, which some authors have called "cardiac fatigue" (e50–e53). However, the validity of such comparisons is limited by the different physiological conditions that prevail before and after exercise (e.g., differences in plasma volume, heart rate, or blood pressure). Prolonged endurance exercise may acutely impair

FIGURE 3

Relative wall thickness
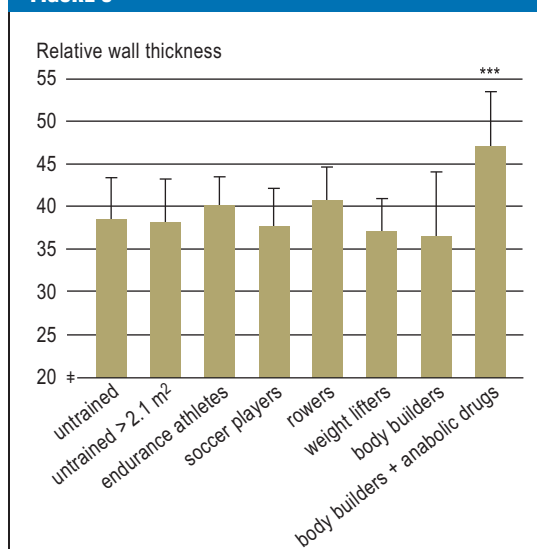
Relative wall thickness (mean and standard deviation) in healthy, athletically untrained persons of varying body size, endurance athletes, soccer players, strength-endurance athletes (rowers), and strength athletes with and without anabolic drug abuse (total, 230 subjects). Only body-builders taking anabolic drugs had concentric cardiac hypertrophy with a significantly elevated relative wall thickness ($p < 0.001$). m², body surface area in square meters. Reprinted from (10) with the kind permission of Springer publishers.

right ventricular function more than left ventricular function (17, 18, e54). In general, the echocardiographically demonstrable acute exercise-induced functional changes in healthy athletes are only transient; unlike those seen in persons with heart disease, they are mild and apparently clinically insignificant (e51, e52, e55).

Cardiac magnetic resonance imaging (MRI)

The biventricular, eccentric cardiac hypertrophy characteristic of athlete's heart, which Reindell originally described on the basis of conventional diagnostic x-rays (2), has been confirmed by cardiac magnetic imaging (MRI) and shown to consist of balanced hypertrophy of the left and right ventricle (7). In strength athletes who do not take anabolic drugs, MRI (like echocardiography) does not reveal concentric hypertrophy of either ventricle (e56, e57). The reported figures for left and right ventricular volume and myocardial mass in athletes cover a wide range because of the widely differing

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Relative wall thickness in athlete's heart

The echocardiographically measured relative wall thickness, defined as the ratio of left ventricular thickness to end-diastolic inner diameter, normally does not exceed 42–43% in athletes.

Function of the athlete's heart

In healthy athletes, systolic function is normal at rest. For those with athlete's heart, it may be in the low-normal range or even slightly below it, while the stroke volume remains normal. Diastolic function is normal or in the high-normal range.
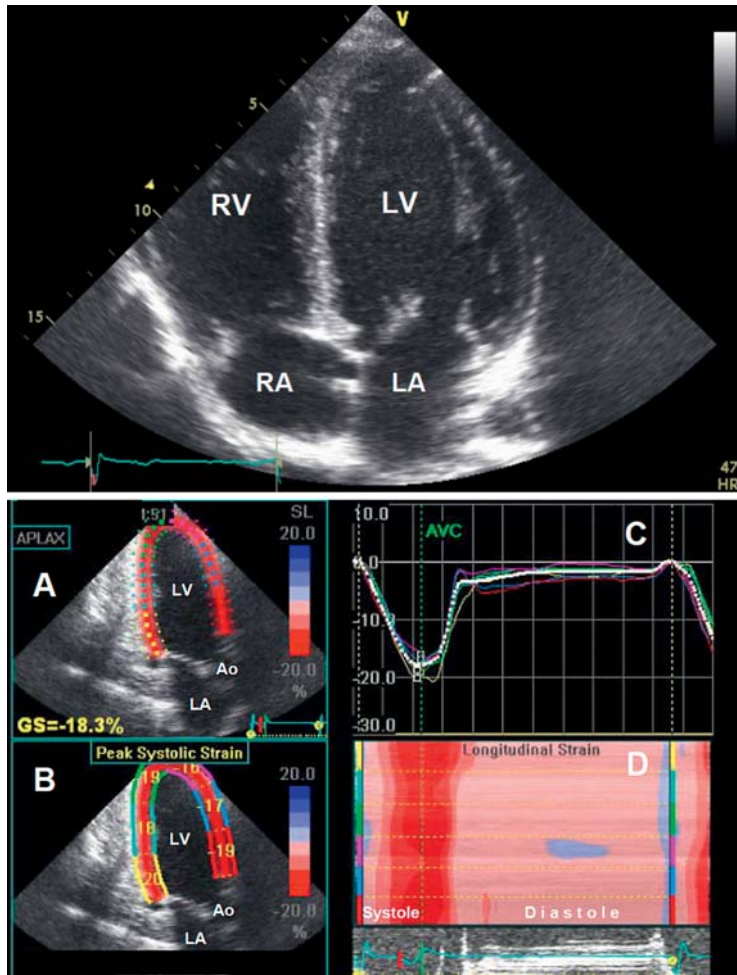
Figure 4:
Above: Echocardiography (four-chamber view) of an athlete's heart in a male world-class endurance athlete with a heart volume of 19.0 mL/kg body weight. There is a harmonic relationship between the left and right ventricle (LV and RV) and between the left and right atrium (LA and RA).
Below: Echocardiography of an athlete's heart in a healthy, female, nationally top-ranked endurance athlete with a heart rate of 29 beats per minute at rest.
A: Three-chamber view with wall-motion analysis by 2D speckle tracking.
B: Display of the maximal relative deformation (strain) and shortening of the myocardial segments in systole.
C: The course of relative deformation of the myocardial segments over the cardiac cycle.
D: Display of all analyzed myocardial segments in a three-chamber view (y-axis with color labeling of the segments from B) with synchronous short contraction (red) of all myocardial segments, followed by a long period of relaxation and filling in diastole (pink). LV, left ventricle; LA, left atrium; Ao, aorta.

groups of individuals studied and measuring techniques used. These volumes and masses are, however, well correlated with maximal oxygen uptake (VO_2max) (7, e58); spiroergometry is, therefore, recommended in unclear cases for an objective assessment of endurance capacity. In this context, it has to be remembered that MRI generally yields higher values than echocardiography for the atrial and ventricular dimensions and lower values for wall thicknesses and muscle masses (19).

45% is considered the lower reference limit of normal for the left and right ventricular systolic ejection fraction in athletes, as measured by MRI (e59). Lower values may be physiological in some cases, particularly in endurance athletes with athlete's heart.

Cardiac biomarkers

Exercise-induced increases in the concentrations of the cardiac biomarkers troponin I and T (TnI, TnT) and B-type natriuretic peptide (BNP and N-terminal proBNP [NT-proBNP]) have been extensively studied in the last decade and a half (20, 21). In non-athletes, these biomarkers are found at pathologically high levels only after myocardial infarction or in congestive heart failure.

Troponin

A meta-analysis of studies involving a total of more than 1000 endurance athletes showed that 47% had an elevation of third-generation TnT concentration beyond the upper reference limit of normal after exhausting endurance exercise (e.g., a marathon or triathlon) (e60). In studies that showed a percentage of TnT-positive athletes in this range, the percentage of TnI-positive athletes was even higher, ca. 75% to 80% (e61–e63). More recent studies employing high-sensitivity troponin tests have revealed comparable or even higher percentages of troponin-positive athletes after exhausting endurance exercise (e62, e63). Presumably, nearly all athletes engaging in such activities have a transient, reversible increase in troponin concentration.

Right and left ventricular function on MRI

45% is considered the lower reference limit for the left and right ventricular systolic ejection fraction in athletes, as measured by MRI.

Exercise-induced rises in troponin concentration

In healthy athletes, elevated troponin levels induced by endurance exercise usually decline markedly within 24 hours and are back in the normal range within 24 to 48 hours (at most, 72 hours).

TABLE 2

Illustrative findings from studies on the longevity of elite endurance athletes

Author / type of sporting activity	Number of athletes	Control group	Athletes	Effect
Grimsmo (e71) Cross-country skiing	122	40% dead	31% dead	9% lower mortality at the time of the study
Karvonen (e72) Cross-country skiing	396	Finnish general population, no data about age	73 years mean life span	95% CI: life prolonged by 2.8 to 4.3 years
Sarna (25) Cross-country skiing, long-distance running	303 (2613 athletes total)	age attained: 69.9 years 95% CI: 69.0–70.9 (1712 persons)	age attained: 75.6 years 95% CI: 73.6–77.5 (303 persons)	life prolonged by a mean of 5.7 years
Sanchis-Gomar (24) Bicycle racing (Tour de France)	834	50% dead by the age of 73.5 years	50% dead by the age of 81.5 years	17% prolongation of mean life span

CI: confidence interval

In myocardial infarction, the troponin concentration increases when bound troponin is released from the tropomyosin complex of necrotic cardiomyocytes. In contrast, the elevation of troponin concentration in athletes is attributed to the release of unbound troponin from the cytoplasmic pool by membrane vesicles, without cell necrosis (20, 21, e64). How this comes about is currently unknown. The putative mechanisms include changes of intracellular metabolism, changes of intracellular calcium concentration that activate intracellular proteases, free-radical effects, and exercise-induced ischemia (20, 21, e51, e55). Further experimental studies are needed to clarify the matter.

BNP and NT-proBNP

BNP and NT-proBNP also rise, particularly after exhausting endurance activities. The percentage of athletes with elevated concentrations of these biomarkers after such activity is in the same range as that mentioned above for troponin. Well-trained marathon runners or those with high training volumes seem to have lower increases of BNP and NT-proBNP concentrations, as well as lower increases of troponin concentration, than less well-trained marathon runners do (17). The elevation of BNP-/NT-proBNP, unlike that of troponin, is affected by the duration of exercise, not just by its intensity (e37).

Compared to healthy, but athletically untrained persons of like age, athletes with or without athlete's heart do not have elevated BNP-/NT-proBNP concentrations at rest (20, e66–e68), nor do these concentrations rise to any greater degree than in untrained persons in response to standardized exercise, despite greater ergometric and cardiac performance (e69). The recently reported moderate association between exercise-induced elevation of the BNP or TnI concentration amd transient right ventricular dysfunction after prolonged endurance stress may indicate that endurance exercise has a stronger effect on the right ventricle (17, 18).

The important point for clinical practice with respect to exercise-induced elevation of troponin and BNP-/NT-proBNP concentrations is that a majority of athletes may have a transient elevation after exhausting endurance exercise in the absence of any pathological abnormality. In healthy athletes, the troponin values usually decline markedly within 24 hours and reach normal limits within 24 to 48 hours (at most, 72 hours). Doubtful cases with unclear findings require further cardiological evaluation.

The life expectancy of competitive athletes

The benefits and risks of competitive sports can also be judged in terms of life expectancy . Although there is good evidence for a higher life expectancy among persons who regularly participate in recreational sports (1), there has always been concern that the much more intense, exhausting physical activity demanded of competitive and high-performance athletes might actually shorten their lives (22, e70). A recent review article (23) and editorial (e70) addressed this question by summarizing the findings of 14 (resp. 15) published studies: it was concluded that endurance

BNP/NT-proBNP

Compared to healthy, but athletically untrained individuals, athletes with athlete's heart do not have elevated BNP-/NT-proBNP concentrations at rest.

Life expectancy

The epidemiological data imply that endurance sports prolong life even when they are pursued competitively.

athletes who achieve national and international success in sports such as long-distance running, cross-country skiing, and bicycle racing, as well as athletes in mixed types of sport, such as soccer, basketball, and ice hockey, have a longer life span than the general population. This, in turn, is largely due to lower cardiovascular mortality, particularly among athletes who continue endurance training even after they stop competing. Strength-based sports, on the other hand, seem not to prolong life (23).

Because of the recent debate over potential danger to the heart from extreme endurance sports, studies on elite endurance athletes whose extensive training and high performance make them likely to have an athlete's heart (i.e., physiological cardiac remodeling) are presented separately in Table 2 (24, 25, e71, e72). One such study, recently published, is a retrospective assessment of 834 cyclists who took part in the Tour de France bicycle race from 1930 to 1964: 50% of them were still alive at age 82, while 50% of the general population born from 1892 to 1942 was already dead by age 74 (24). It remains an open question, however, whether endurance and high-performance exercise can still be as beneficial as this in the modern era, in which athletes' life expectancy may be shortened by the cardiovascular and other side effects of doping drugs such as anabolic steroids, growth hormone, and erythropoietin (9, e73).

With respect to amateur endurance sports performed on a competitive level, a study of more than 73 000 participants in the Swedish Vasa cross-country ski race (over 30 and 90 km) from 1989 to 1998 revealed that only 410 of them had died by 1999, a much lower number than the 851 deaths that would have been expected in the general population, after adjustment for age (standardized mortality ratio [SMR] 0.48) (e74). The relative reduction of cardiovascular mortality among participants was 57% (SMR 0.43). In a Dutch study of 2129 men who skated in the single-day Eleven Cities Ice-Skating Tour (with a course of more than 200 km), the observed number of deaths in athletes from any cause over 32 years of observation was 24% lower than would have been expected for the general population (SMR 0.76) (e75).

Taken together, the foregoing epidemiological data imply that endurance sports prolong life even when they are pursued competitively. The athletes' healthier lifestyle, with regular physical activity even when their sporting careers are over, is presumably decisive.

Conclusion

Regular athletic activity brings about functional adaptations that improve cardiac function. Athlete's heart, which develops only after intensive training with a high endurance component, is not a pathological condition and is much rarer than commonly thought. An athlete's ECG may well reveal certain common, training-related changes which must be distinguished from uncommon, training-unrelated ECG-changes that require further evaluation. The variables measured in cardiac imaging studies exceed the normal range for untrained individuals only in persons who have undergone competitive endurance training. Cardiac function in athlete's heart is normal at rest and increases appropriately during exercise. The concentrations of the cardiac biomarkers troponin and BNP/NT-proBNP may rise transiently in healthy athletes in response to endurance exercise; this is not currently thought to represent exercise-induced damage to the heart. Epidemiological data show that endurance sports prolong life even when they are pursued at the competitive level.

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The solutions to the following questions will be published in issue 9/2013. The CME unit "The Treatment of Hallux Valgus" (issue 49/2012) can be accessed until 18 January 2013.

For issue 5/2013, we plan to offer the topic "Postoperative Care and Follow up After Coronary Stenting."

Solutions to the CME questionnaire in issue 45/2012:5

Kaufmann L, Aster v M: The Diagnosis and Management of Dyscalculia.
Answers: 1c, 2a, 3d, 4d, 5b, 6a, 7e, 8b, 9e, 10c

Please answer the following questions to participate in our certified Continuing Medical Education program. Only one answer is possible per question. Please select the answer that is most appropriate.

Question 1
Which of the following is a typical feature of athlete's heart in a male athlete?
a) heart volume between 10 and 12 mL/kg body weight
b) septum thickness 17 mm
c) end-diastolic left ventricular diameter 61 mm
d) right ventricle not enlarged
e) diastolic function in low-normal range

Question 2
What ECG change is frequently due to training?
a) first-degree AV block
b) left atrial hemiblock
c) complete right bundle branch block
d) prolonged QT interval
e) pre-excitation syndrome

Question 3
What type of arrhythmia is more common among middle-aged and older endurance athletes than in athletically inactive persons of the same age?
a) supraventricular extrasystoles
b) ventricular extrasystoles
c) atrial fibrillation
d) ventricular tachycardia
e) third-degree AV block

Question 4
A leisure-time jogger (45 minutes, three times a week) presents for cardiovascular assessment. Which of the following findings requires further evaluation?
a) incomplete right bundle branch block
b) end-diastolic left ventricular diameter 52 mm
c) septum thickness 16 mm
d) ejection fraction 60%
e) Sinus bradycardia with a resting heart rate of 55 beats per minute

Question 5
How does the blood pressure change during dynamic exercise?
a) systolic and diastolic pressures rise as an exponential function of stress intensity
b) systolic pressure rises exponentially, diastolic pressure rises linearly
c) diastolic pressure rises exponentially, systolic pressure rises linearly
d) diastolic pressure rises linearly, systolic pressure barely changes
e) systolic pressure rises linearly, diastolic pressure barely changes

Question 6
What cardiovascular adaptation is physiological in endurance athletes?
a) concentric hypertrophy of the left ventricle
b) increased end-systolic volume of the left ventricle
c) an up to 20-fold increase of oxygen uptake during excercise
d) elevated pulmonary-arterial pressure at rest
e) elevated peripheral vascular resistance at rest

Question 7
Which of the following is true of the blood levels of cardiac biomarkers in healthy endurance athletes with athlete's heart?
a) Cardiac troponin levels are elevated.
b) BNP/NT-proBNP levels are elevated at rest.
c) Increases in cardiac troponin levels are unlikely in a competitive endurance event.
d) Well trained endurance athletes have higher exercise-induced elevations of BNP and NT-proBNP.
e) Exercise-induced elevation of troponin usually reverts to the normal range within 24 to 48 hours.

Question 8
Which of the following findings on echocardiography or MRI would be unusual for an endurance athlete?
a) low-normal systolic left ventricular function at rest
b) high-normal diastolic left ventricular function at rest
c) only slight rise of systolic function in stress echocardiography
d) harmonic biventricular eccentric hypertrophy on MRI
e) atrial diameter of 45 mm with athlete's heart

Question 9
Which of the following ECG findings is attributable to training in an asymptomatic competitive endurance athlete?
a) evidence of left atrial enlargement
b) incomplete right bundle branch block
c) Wolff-Parkinson-White syndrome
d) long QT syndrome
e) short QT syndrome

Question 10
There is epidemiological evidence that competitive and high-performance athletes live longer than the population at large. What is the most probable reason for this?
a) persistently prominent athlete's heart
b) regular strength training
c) regular medical check-ups
d) regular endurance training even after retirement from competition
e) moderate nicotine consumption

Competitive Sports and the Heart: Benefit or Risk?

Jürgen Scharhag, Herbert Löllgen, Wilfried Kindermann

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