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Strain Imaging: Key to the Specific Left Ventricular Diastolic Properties in Endurance Trained Athletes

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Background: To increase the performance capacity of endurance trained athletes (ETA), a variety of adaptations are imperative. The heart is the central and the most important limiting factor. The structural heart adaptations in ETA have important repercussions on cardiac function. The left ventricular diastole shows specific characteristics that determine the performance capacity.

Methods: 40 male ETA were compared with 31 active male controls and with 112 coronary patients. All subjects underwent tissue Doppler and strain imaging in equal basal conditions.

Results: Compared with normal controls we noted similar signs of both eccentric and concentric left ventricular hypertrophy in ETA as well as in patients with severe ischaemic heart disease. In contrast with the disturbed diastolic function in the coronary patients, the left ventricular diastolic function in the ETA was perfectly normal and even different and better than in the healthy control group. Excluding influences of preload, the values of the pulsed Doppler tissue imaging peak atrial systolic velocities (A), peak early diastolic velocities (E) and E/A ratios clearly indicate that the ETA have a supernormal left ventricular function compared with the nonathletic men. Extremely striking were the significant differences between the three groups concerning the strain values at the basal and the mid septum in the longitudinal axis by aortic valve closure and by mitral valve opening. Fascinating were the values of the enddiastolic strain at the end of the A-wave: negative in ETA, near zero in normal controls and markedly positive in patients with severe ischaemic heart disease.

Conclusions: In spite of signs of combined eccentric and concentric left ventricular hypertrophy, the left ventricular systolic and diastolic functions remain normal and even supernormal in ETA. Arguments in support of this thesis were found by two dimensional echocardiographic Doppler examinations, by pulsed Doppler tissue imaging and by strain imaging. Interpreting our results obtained by strain imaging, we try to explain the specific left ventricular diastolic properties in ETA. *J Clin Basic Cardiol 2003; 6: 35–40.*

Key words: strain, diastolic function, endurance sports, tissue Doppler, ischaemic heart disease

Triathlon is a competition, which consists of 3.9 km of swimming, 180 km of cycling and 42 km of running, an extreme endurance sport, practised under aerobic conditions [1]. To finish a triathlon successfully, triathletes or endurance trained athletes (ETAs) have to make sure that only minimal changes occur in the homeostasis of their cardiovascular, haemodynamic and metabolic functions over a long span of time [2]. To increase the performance capacity of the endurance trained athlete (ETA), a variety of central and peripheral adaptations are necessary. The heart is the central and the most important limiting factor [3]. It is generally accepted that increased physical activity and training cause structural and functional heart adaptations with important haemodynamic implications. In particular, left ventricular diastole in ETAs shows specific characteristics which affect and determine performance capacity to a large extent [4]. On the other hand, it was also proved that not all ETAs showed an enlarged internal diameter of the left ventricle or a manifest wall hypertrophy. It was apparently not necessary to have an "athletic heart" to become an ETA or to win a triathlon [5]. This variable physiological response of the heart to training indicates that other and very likely genetic factors affect the structural heart adaptations in ETAs. We suspect the heart of the ETA and in particular the diastolic left ventricular function of the ETAs disposes of specific properties, irrespective of training, nutrition and lifestyle.

Patients and Methods

This study enclosed 183 male subjects. A group of 40 male ETAs were compared with a group of 31 active male controls and with a group of 112 patients with ischaemic heart disease.

All ETAs were engaged in competition. The 31 control subjects were healthy and active men, they did not participate in competition or recreational sports. The 112 patients with ischaemic heart disease all underwent surgical myocardial revascularisation because of a severe two or three vessel coronary artery disease. All tested subjects were in sinus rhythm. Digitalis, anti-arrhythmic medication and beta-blockers were stopped one month before cardiac echodoppler investigation with tissue Doppler imaging and strain imaging.

A Vingmed System Five apparatus (GE Medical Systems, New Orleans, USA) was used for the 2-dimensional Doppler examination and for the pulsed Doppler tissue imaging and for the strain imaging. All Doppler echocardiographic measurements were performed for 5 consecutive heartbeats. The average value of those 5 measurements was used for statistical analysis [6]. To study cardiac function, we measured the left ventricular systolic and diastolic functions. We were particularly interested in the left ventricular diastolic function and we measured, among others, the deceleration time, the E/A ratio, the isovolumic relaxation time and the ASEAC value [7]. ASEAC stands for the amplitude of excursion of the interventricular septal endocardium at the end of left ventricular diastole, that occurs just after atrial contraction. The ASEAC value is expressed in millimetres and is measured on the M-mode of the echocardiographic image of the left ventricle [8].

From the parasternal long axis of the bidimensional echocardiographic image, a switch was made to the M-mode image of the left ventricle through a cross-section just distal to the mitral leaflets (Fig. 1).

Tissue velocity imaging has enabled echocardiographic assessment of diastolic function as it appears to be less limited

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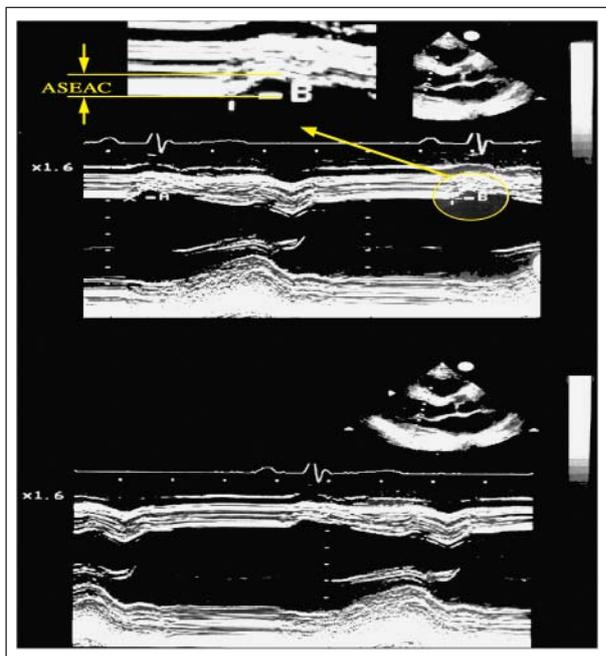


Figure 1. Method to determine and quantify the ASEAC value

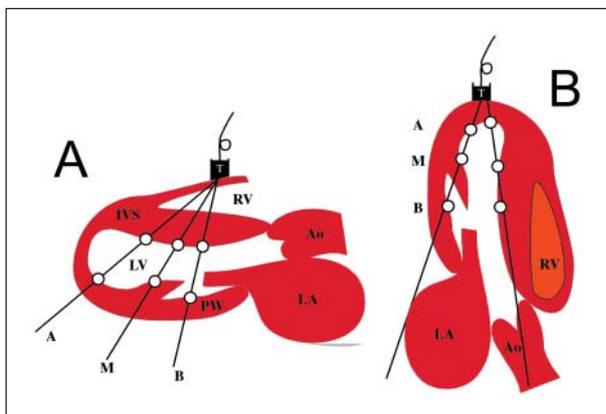


Figure 2. Parasternal long axis view (A), and longitudinal or apical long axis view (B) (T = transducer, B = basal site, M = middle site, A = apical site, IVS = interventricular septum, PW = posterior wall, LV = left ventricle, RV = right ventricle, LA = left atrium, Ao = aorta)

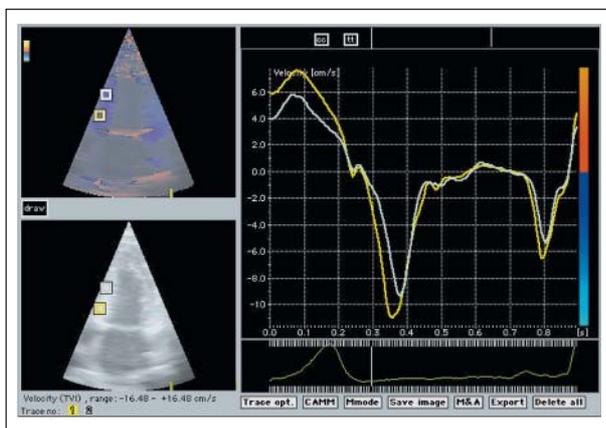


Figure 3. Tissue Doppler imaging; measurements of peak systolic velocity, peak E- and peak A-velocities in the apical long axis

by the compensatory changes in loading conditions which impair measurement of diastolic function by conventional echocardiographic methods [9–12]. In the parasternal long-axis view and in the apical long-axis view of the left ventricle the sample volume was set at the basal and middle sites of the interventricular septum and at the basal and middle sites of the left ventricular posterior wall (Fig. 2). From the obtained patterns, the peak atrial systolic (A) velocities and the peak early diastolic (E) velocities were measured, permitting us to define E/A ratios. At the basal site of the septum and at the basal site of the posterior wall the peak systolic velocities were also determined in the apical long axis (Fig. 3).

Strain is defined as the deformation of an object, normalised to its original shape. In a one-dimensional object, the only possible deformation of the object is lengthening or shortening. Since it is the change in length relative to its initial length, strain is a dimensionless quantity, often expressed in percent. Strain rate is the instantaneous difference in velocity between two points separated by a distance (d) (Fig. 4). Since only the velocity component along the ultrasound beam is available, only the strain rate in the beam direction is estimated. When imaging from the apical view only the strain rate in the longitudinal direction is measured, but the longitudinal direction may contain the main information [13]. Negative strain rate means that the segment is becoming shorter, while positive strain rate means that the segment is becoming longer. Strain Doppler echocardiography permits us to quantify the intrinsic deformation of the myocardium, irrespective of global motion of the heart. Strain rate and strain images were obtained by frame rates around 148 [14].

At the basal and mid septum in the longitudinal axis strain values were measured by aortic valve closure, by mitral valve opening and enddiastolic by the end of the A-wave. On the velocity curve we localised the end of the A-wave and we determined the time at the end of the A-wave, for that time we read off on the strain curve the value of the strain (Fig. 5). At the basal site of the left ventricular posterior wall in the longitudinal apical axis the peak systolic strain rate (s^{-1}) and the E/A ratio strain rate were determined (Fig. 6).

Statistical Analysis

An essential step in the process of testing statistical hypotheses involves evaluating the extent to which the analyzed parameters meet the assumptions of the tests being considered. A first important characteristic is the normality of the distribution. Comparison of the histograms with a normal bell-shaped curve showed that several parameters deviate

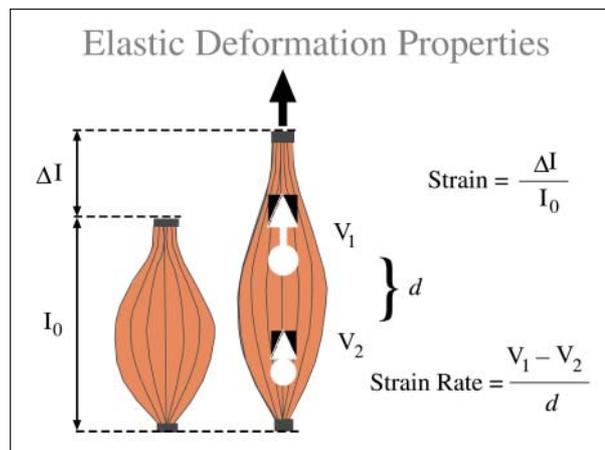


Figure 4. Definition of strain and strain rate

seriously from normal distribution. These observations are supported by calculating the skewness and kurtosis of the distribution, and by performing the Kolmogorov-Smirnov statistical-test for normality. Creating boxplots identified outliers and extreme values, which can have a major impact on the mean of a given variable in a given population. In view of our current choice for non-parametric, rank-ordered statistics, which tend to be insensitive towards these atypical datapoints, no attempt was made to remedy them.

In our study, the homogeneity of variance among different groups was tested using the Levene-test, which is fairly independent on the assumption of normality. We concluded from these tests that not all variances can be assumed equal.

Since our data do not meet the assumptions for the traditional parametric tests, such as t-tests and ANOVA, the Mann-Whitney-U-test is used instead, being the non-parametric alternative for testing differences between two or more independent samples [15].

Results

Roughly, the anthropometric data and the general physical parameters of the different groups were similar. There were no significant differences between the three groups concerning length, weight, body surface area and body mass index. Concerning the cardiac structure, there were significant differences between ETAs and the coronary patients on the one hand and the normal controls on the other hand. The maximal enddiastolic diameter of the left ventricle was markedly larger and statistically significantly different in the ETAs and in the coronary patients, compared with the normal controls. The same statistically significant differences occurred in the diastolic interventricular septum thickness and in the diastolic left ventricular posterior wall thickness (Tab. 1).

The left ventricular systolic function was completely normal in the three groups. We still noted differences between the ETAs and the normal controls on the one hand and the coronary patients on the other hand, but even in the coronary patients' group the systolic left ventricular function remained within normal limits (Tab. 2).

In contrast with the systolic left ventricular function, we registered significant differences between the three groups in the diastolic left ventricular function (Tab. 3).

In spite of an underlying left ventricular hypertrophy, the left ventricle of the ETAs behaves in the diastole perfectly normally and even better than normal. The E-point velocity was distinctly higher ($p < 0.05$) and the A-point velocity distinctly lower ($p < 0.05$) in the ETAs compared with the normal controls. The E/A ratio was clearly higher in the ETAs. The isovolumetric relaxation time (IVRT) was distinctly shorter in the ETAs than in the control group, and was prolonged in patients with an ischaemic heart disease. The deceleration time was within normal limits in the three groups, but markedly higher in the coronary patients. There are statistically significant differences between the three groups in the ASEAC value. The ASEAC value was much higher and significantly very different in the ETAs than in the control group and in the control group much higher and significantly very different compared with the coronary patients.

Pulsed Doppler tissue imaging, with the sample

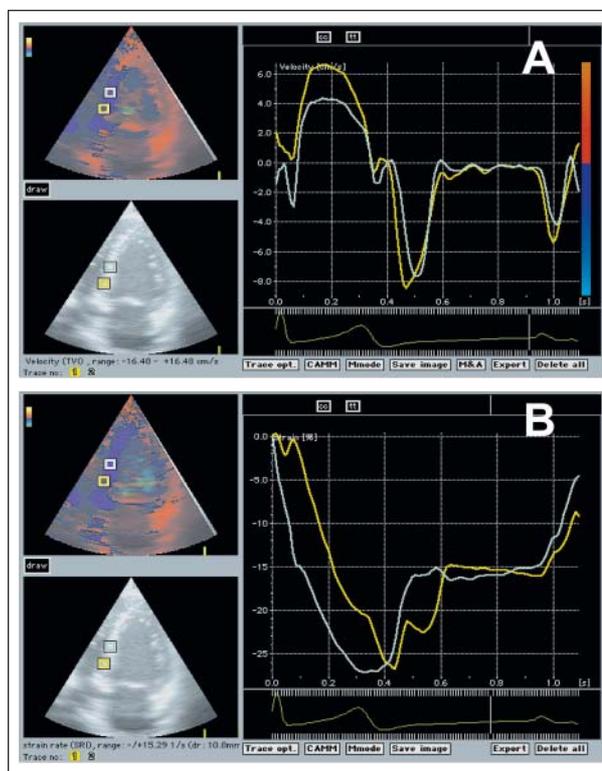


Figure 5. A: Velocity curve (tissue Doppler imaging): localisation of the end of the A-wave and determination the time on the end of the A-wave at the basal and middle septum in the long apical axis; B: Strain curve: measurement of the strain value at the time of the end of the A-wave at the basal and middle septum in the long apical axis

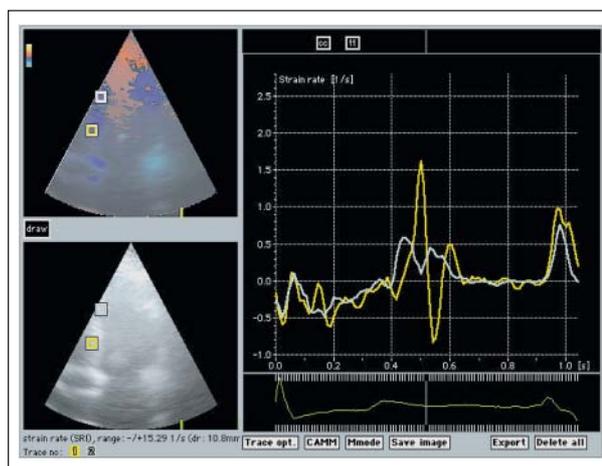


Figure 6. Measurement of the peak systolic strain rate and the E/A ratio strain rate in the longitudinal or apical long axis

Table 1. Cardiac structure

	Group I (triathletes)	Group II (normal controls)	Group III (ischaemic heart disease)	Mann-Whitney-U (p-value)		
				gr. 1 vs. gr. 2	gr. 1 vs. gr. 3	gr. 2 vs. gr. 3
Septum thickness diastole (cm)	1.19	0.92	1.04	p < 0.01	p < 0.05	p < 0.05
Posterior wall thickness diastole (cm)	0.93	0.88	0.96	p < 0.05	nsd	p < 0.01
Enddiastolic diameter left ventricle (cm)	6.02	5.24	5.84	p < 0.01	p < 0.05	p < 0.05

volume at the basal site in the transverse axis and also in the longitudinal axis, demonstrated the peak atrial systolic motion velocities of the interventricular septum and the left ventricular posterior wall were significantly higher in the control group, compared with the ETAs. Regarding the peak early diastolic motion velocities we also noted statistically significant differences between the three groups; the peak early diastolic motion velocity at the basal site of the interventricu-

lar septum and of the left ventricular posterior wall was much higher in the ETAs than in the normal controls and was much higher in the normal controls compared with the coronary patients. When the pulsed Doppler tissue imaging E/A ratios were calculated, there were statistically significant differences between the three groups. In all axes and at each site, the pulsed Doppler tissue imaging E/A ratios were significantly higher in the ETAs than in the control group. By calculating the E/A ratios at the basal septum in the longitudinal apical axis we noted striking and statistically very significant differences between the ETAs and the normal controls but also between the control group and the patients with ischaemic heart disease (Tab. 4).

Strain means the amount of local deformation caused by an applied force. The temporal derivative of strain, the strain rate, is a measure of the rate of deformation. Strain rate in the three groups was always negative throughout systole. The peak systolic strain rate was significantly different between the ETAs and the normal controls on the one hand and

patients with ischaemic heart disease on the other. In competitive ETAs the E/A ratio is much more than 1 and significantly higher than the E/A ratio in the normal control group (Tab. 5).

Extremely striking were the very significant differences between the three groups concerning the strain values at the basal septum in the longitudinal axis at aortic valve closure and at mitral valve opening. We noted not only significant differences between the healthy subjects and the patients with severe ischaemic heart disease but also between the ETAs and the normal controls. The same statistically significant differences between the three groups were noted by measuring the strain values at the mid-septum in the longitudinal axis by aortic valve closure and by mitral valve opening (Tab. 6). Fascinating were the values of the end-diastolic strain at the end of the A-wave. In ETAs we noted enddiastolic and during the isovolumic contraction period a distinct and unmistakable negative strain value, while in normal controls the enddiastolic strain was near zero or slightly negative and in patients with ischaemic heart disease the enddiastolic

Table 2. Systolic left ventricular function

	Group I (triathletes)	Group II (normal controls)	Group III (ischaemic heart disease)
Ejection fraction	77.93	78.65	66.44
Posterior wall thickness diastole (cm)	6.76	6.15	4.28

Table 3. Diastolic left ventricular function

	Group I (triathletes)	Group II (normal controls)	Group III (ischaemic heart disease)	Mann-Whitney-U (p-value)		
				gr. 1 vs. gr. 2	gr. 1 vs. gr. 3	gr. 2 vs. gr. 3
Deceleration time (ms)	196	168	220	p < 0.05	nsd	p < 0.01
E/A ratio	1.79	1.21	1.13	p < 0.01	p < 0.01	p < 0.05
IVRT (ms)	80.28	84.80	97.41	nsd	p < 0.01	p < 0.01
ASEAC (cm)	0.55	0.37	0.18	p < 0.01	p < 0.01	p < 0.01

Table 4. Tissue Doppler imaging – left ventricular diastolic function

	Group I (triathletes)	Group II (normal controls)	Group III (ischaemic heart disease)	Mann-Whitney-U (p-value)		
				gr. 1 vs. gr. 2	gr. 1 vs. gr. 3	gr. 2 vs. gr. 3
Peak atrial (A) systolic motion velocity interventricular septum basal site longitudinal axis	-1.721	-2.77	-2.909	p < 0.01	p < 0.01	nsd
Peak early (E) diastolic motion velocity interventricular septum basal site longitudinal axis	-9.968	-7.78	-5.158	p < 0.01	p < 0.01	p < 0.05
Peak early (E) diastolic motion velocity posterior wall basal site longitudinal axis	-10.970	-8.30	-5.435	p < 0.01	p < 0.01	p < 0.01
Peak systolic velocity basal septum longitudinal apical axis (cm/s)	6.533	5.790	4.261	p < 0.01	p < 0.01	p < 0.01
Peak systolic velocity basal posterior longitudinal apical axis (cm/s)	7.217	5.470	3.412	p < 0.01	p < 0.01	p < 0.01
E/A ratio basal septum longitudinal apical axis	2.006	1.200	0.893	p < 0.01	p < 0.01	p < 0.01

Table 5. Strain imaging

	Group I (triathletes)	Group II (normal controls)	Group III (ischaemic heart disease)	Mann-Whitney-U (p-value)		
				gr. 1 vs. gr. 2	gr. 1 vs. gr. 3	gr. 2 vs. gr. 3
Peak systolic strain rate (s ⁻¹) basal posterior longitudinal apical axis	-1.922	-1.805	-1.359	nsd	p < 0.05	p < 0.05
E/A ratio strain rate (s ⁻¹) basal posterior longitudinal apical axis	2.206	1.320	1.377	p < 0.01	p < 0.01	nsd

strain presented a distinctly positive value. We came to the same observation for the integrated strain values in the different groups at the basal septum as well as at the mid septum in the longitudinal axis enddiastolic at the end of the A-wave (Tab. 7). The graphical diagram of these observations at the basal septum in the long axis shows us the significantly different courses of the strain curve between the three groups. At the mid-septum in the longitudinal axis we noted the same course of the curve in the different groups (Fig. 7).

Discussion

Important structural heart adaptations are found in ETAs. We noted the same findings of a both concentric and eccentric left ventricular hypertrophy in ETAs as well as in the coronary patients. Notwithstanding a manifest left ventricular hypertrophy, it was observed that systolic cardiac function was normal in all groups. In contrast with the systolic left ventricular function, we registered significant differences between the groups in the diastolic left ventricular function. In cases of pathological myocardial hypertrophy, the diastolic left ventricular function is clearly disturbed. These particular findings contrast sharply with the diastolic behaviour of the left ventricle in ETAs. In spite of an underlying left ventricular hypertrophy, the left ventricle of the ETA behaves in the diastole perfectly normally and even better than normal and yet is clearly different from that in the control group [16, 17].

In this context we would put our findings about the ASEAC value. These findings indicate distinct differences between both groups in the late-diastolic passive filling period. The passive elasticity of the myocardium seems to be much higher in the ETAs than in the control group. Probably we can explain ASEAC both by the contribution of the atrial contraction and by the pressure rise during the isovolumic contraction period.

The results of the diastolic left ventricular function, obtained by bidimensional cardiac echodoppler examination, were indicative of a transitional form between an impaired myocardial relaxation and a restrictive filling pattern with a decreased compliance in patients with ischaemic heart disease, these results also pointed to a normal filling pattern in the control group and provided arguments for a supernormal diastolic left ventricular function in ETAs. But, there are limitations in the evaluation of left ventricular relaxation with the use of non-invasively recording transmitral flow velocities. During the past several years, techniques have been developed to evaluate left ventricular diastolic function without the influence of preload by using diastolic measurements obtained from the motion velocities of the left ventricular posterior wall and of the interventricular septum, recorded by pulsed

Table 6. Strain imaging

	Group I (triathletes)	Group II (normal controls)	Group III (ischaemic heart disease)	Mann-Whitney-U (p-value)		
				gr. 1 vs. gr. 2	gr. 1 vs. gr. 3	gr. 2 vs. gr. 3
Strain at AVC (aortic valve closure) basal septum longitudinal apical axis (%)	-16.249	-12.498	-7.541	p < 0.001	p < 0.001	p < 0.001
Strain at MVO (mitral valve opening) basal septum longitudinal apical axis (%)	-20.535	-14.708	-10.018	p < 0.001	p < 0.001	p < 0.001
Strain at AVC (aortic valve closure) mid septum longitudinal apical axis (%)	-19.159	-15.407	-8.864	p < 0.001	p < 0.001	p < 0.001
Strain at MVO (mitral valve opening) mid septum longitudinal apical axis (%)	-18.110	-14.358	-9.137	p < 0.001	p < 0.001	p < 0.001

Table 7. Strain imaging

	Group I (triathletes)	Group II (normal controls)	Group III (ischaemic heart disease)	Mann-Whitney-U (p-value)		
				gr. 1 vs. gr. 2	gr. 1 vs. gr. 3	gr. 2 vs. gr. 3
Enddiastolic strain at the end of the A-wave basal septum longitudinal apical axis (%)	-9.522	-1.901	+2.782	p < 0.001	p < 0.001	p < 0.001
Enddiastolic strain at the end of the A-wave mid septum longitudinal apical axis (%)	-10.672	-1.665	+3.739	p < 0.001	p < 0.001	p < 0.001

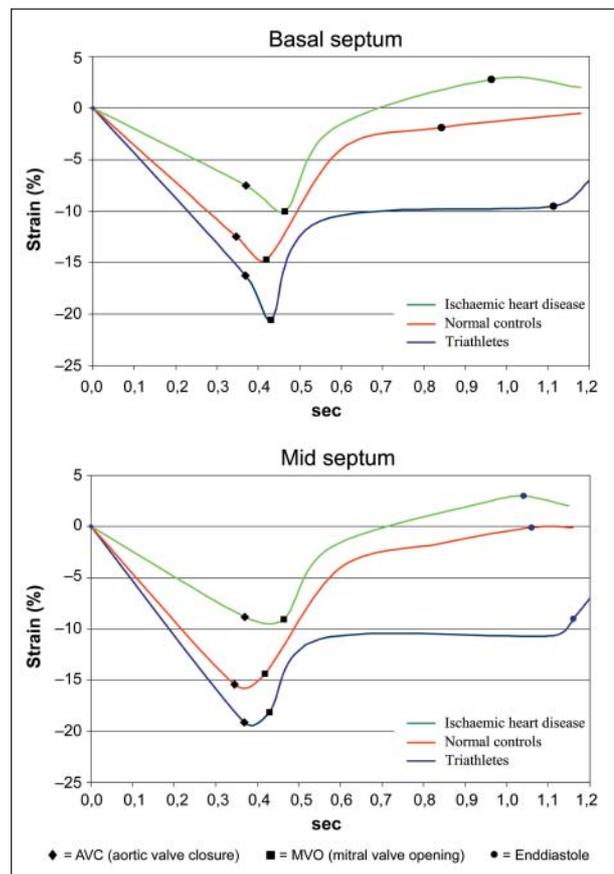


Figure 7. Course of the integrated strain curve at the basal and mid septum in the longitudinal axis in the different groups

Doppler tissue imaging [18, 19]. The values of the pulsed Doppler tissue imaging peak atrial systolic (A) velocities, peak early diastolic (E) velocities and E/A ratios clearly indicated that the ETAs had a supernormal left ventricular function compared with the nonathletic men. Both the early active diastolic relaxation period and the late passive diastolic filling period showed supernormal characteristics in the ETAs, notwithstanding undeniable signs of eccentric and concentric left ventricular hypertrophy.

Myocardial velocities measured by tissue Doppler echocardiography vary throughout the left ventricle because of tethering effects from adjacent tissue. Strain Doppler echocardiography is a new tool for measuring myocardial deformation excluding the effect of adjacent myocardial tissue [20–23]. Myocardial fibre strain is directly related to left ventricular contractility [24, 25]. In competitive ETAs, left ventricle recoil is vigorous and myocardial relaxation is swift; therefore, most filling is completed during early diastole, with only a small contribution at atrial contraction, and, therefore, an E/A ratio much more than 1. In healthy control subjects, left ventricular recoil and myocardial relaxation are normal but less vigorous than in ETAs, therefore, the E/A ratio will be lower but still more than 1. In patients with ischaemic heart disease there is a gradual decrease in the rate of myocardial relaxation as well as in elastic recoil, resulting in slow left ventricle pressure decline. Filling becomes slower with a prolonged isovolumic relaxation with a decreased E-velocity, an increased A-velocity, and a varying E/A ratio, dependent on the nature of the left ventricular diastolic dysfunction: E/A ratios will be less than 1 with problems of delayed relaxation and will be more than 1 with restrictive filling pattern.

The explication and interpretation of the markedly different enddiastolic strain values between the three groups is not so evident and not so obvious. In patients with ischaemic heart disease the positive enddiastolic strain value probably could be caused by an important active contribution of the atrial contraction which can produce a supplementary elongation of the myocardium that still is characterised by a decreased force of contractility and by a reduced capacity to maintain a certain muscular tone. The marked negative enddiastolic strain in ETAs could be connected with a possible recoil phenomenon of the left ventricle: after an almost complete early diastolic filling of the left ventricle, made possible by a swift and increased myocardial relaxation capacity, we later on in the diastole suppose a vigorous left ventricle recoil with an increased muscular tone of the myocardium and with shape adaptation during the isovolumic contraction period.

It is a fact that at the end of the diastole the zero line must be reached. Possibly in ETAs a different enddiastolic strain value could be obtained if we examined another myocardial segment or if the strain measurements were performed in another plane. We only performed strain measurements at the basal and mid septum in the longitudinal apical axis and we determined in that manner the longitudinal shortening. We also could try to determine strain measurements in a radial plane to define radial thickening, and in a circumferential plane to define circumferential shortening. Probably in this setting we could record that there is compensation between the strain values in the different planes, the result being that the global enddiastolic strain value of the entire left ventricle reaches the zero line. If this supposition is correct, we believe there are arguments for the athletic heart to give evidence of an underlying cardiomyopathy with a non-uniform contraction pattern of the left ventricle. In these circumstances we are far away from the classical opinion that an athletic heart is a physiological adaptation of the heart to increased physical activity.

Conclusions

- In spite of signs of combined eccentric and concentric left ventricle hypertrophy, the left ventricle systolic and diastolic functions remain normal and even supernormal in ETAs.
- Two-dimensional echocardiographic Doppler examination enables us to show that in particular the late diastolic filling period in the ETAs has specific characteristics.
- Pulsed Doppler tissue imaging demonstrated in ETAs specific characteristics of both the late passive diastolic filling period and the early active diastolic relaxation period.
- Strain imaging not only proved specific characteristics of the diastolic left ventricular function in ETAs but also gave evidence for an increased systolic function with a forced up systolic myocardial contraction; these findings conceived an intrinsic characteristic of the myocardial wall.
- In the structural and functional heart adaptations in ETAs other factors than way of life, nutrition, training and physical activity play an important role: we suspect in particular genetic factors.

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