Two months of endurance training does not alter diastolic function evaluated by TDI in 9–11-year-old boys and girls

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ABSTRACT

Objective: Superior global cardiac performance (ie stroke volume) is classically reported after training in children. Current knowledge of the impact of exercise training on myocardial relaxation, a major component of left ventricular (LV) filling and subsequently stroke volume, is, however, limited in the paediatric population. This study aimed to investigate the effect of aerobic training on LV wall motion velocities by tissue Doppler imaging (TDI) in healthy children.

Methods: 25 children (11 girls, 14 boys) were enrolled in a 2 month high-intensity aerobic training programme and 25 (12 girls and 13 boys) served as controls. The children (9–11 years old) performed a graded maximal exercise test on a treadmill to evaluate maximal oxygen uptake. Standard Doppler echocardiography and TDI measurements were performed at baseline and end of the study. Tissue Doppler systolic, early and late myocardial velocities were obtained at the mitral annulus in the septal, lateral, inferior and posterior walls.

Results: Maximal oxygen uptake increased by 6.5% (before: 51.6 (SD 4.2), after: 55.0 (4.5) ml/min/kg $p<0.001$) after training. A modest but significant increase in left ventricular end-diastolic diameter was also noticed (before: 46.1 (3.4), after: 48.3 (4.3) $mm.BSA^{-1/2}$, $p<0.001$), whereas left ventricular wall thickness and mass were unchanged. Neither transmitral inflow velocities nor early and late wall motion (Em: before = 18.4 (2.7), after = 18.0 (2.3) cm/s, Am: before = 6.8 (1.2), after = 6.7 (1.3) cm/s) were affected by training. Shortening fraction and regional systolic function (Sm: before = 10.1 (1.6), after = 10.2 (1.4) cm/s) by TDI were also unchanged.

Conclusion: High-intensity aerobic sessions repeated over a 2 month period failed to improve regional diastolic function assessed by TDI in healthy young children.

A comprehensive understanding of cardiac adaptive changes to aerobic training is still needed in the paediatric population. Superior global cardiac functional capacities (ie stroke volume) as a result of aerobic training programmes are classically reported in children.[1–5] Diastolic function, by setting left ventricular filling, is a major determinant of left ventricular (LV) performance. LV filling depends on a complex interplay between cardiac dimensions, loading conditions and myocardial relaxation and compliance. Surprisingly, the effect of exercise training on LV diastolic function in the young child remains to date extremely poorly documented. In healthy children, most of the studies used conventional echocardiography parameters and compared transmitral flow velocity profile between trained athletes and inactive counterparts or examined changes after endurance training programmes. Controversial results have been obtained, early diastolic velocities being reported enhanced[4 6] or unchanged[7–11] in the trained state. Transmitral blood flow reflects global cardiac diastolic function. It is an indirect technique for the evaluation of myocardial properties with a distinct influence from loading conditions.[12] Tissue Doppler imaging (TDI) provides quantitative assessment of wall motion, and myocardium longitudinal velocities by TDI behave as relatively good indexes of LV relaxation.[13] Although this is controversial, TDI has been suggested to be less load-dependent than Doppler flow measures.[13 14] To our knowledge, only two studies[6 15] have examined TDI indexes in trained children. When compared with age-matched inactive children, highly trained cyclists demonstrated higher early wall motion velocities,[6] whereas similar values were obtained in well-trained swimmers.[15] Whether conventional exercise training is able to improve myocardial function and especially regional diastolic function in healthy children remains currently unknown.

Accordingly, the present study investigated the effect of a 2 month endurance training programme on regional diastolic function estimated by TDI in healthy boys and girls.

METHODS

This study was conducted in 50 9–11-year old boys and girls randomly recruited from two primary schools. Children from one school (11 girls, 14 boys) participated in a high-intensity aerobic training programme. To avoid bias selection, the training programme was included in their curriculum. It consisted of high-intensity aerobic running sessions (including exercise/recovery sequences of 25–30 min with exercise intensity varying between 100 to 130% of each child's maximal aerobic velocity) repeated three times per week over a 2 month period. Further details are available elsewhere.[16] Such a training programme has been shown to induce significant improvement in aerobic fitness in children over a short-term period, but also similar improvements to more conventional endurance programmes.[17] Attendance was quite high (>96%). Children from the other school (12 girls and 13 boys) were instructed to simply carry on as normal during the same period and were recruited to control for potential growth effect on cardiac measurements. The protocol was approved by the local ethics committee and carefully explained to the children and their parents to obtain their written informed consent.

Table 1 Clinical and echocardiographic data at baseline and after 2 months of aerobic training

	Experimental group		Control group	
Variables	**Baseline**	**2 months**	**Baseline**	**2 months**
Body height (cm)	138 (10)	141 (10)*	140 (7)	141 (7)*
Body mass (kg)	35 (9)	36 (9)*	34 (67)	35 (8)*
VO_{2max} (ml/min/kg)	51.6 (4.2)	55.0 (4.5)*	50.3 (7.4)	50.5 (7.6)
Heart rate (beats/min)	74 (12)	75 (13)	75 (12)	74 (11)
Stroke volume (ml/m^2 BSA)	41.3 (6.4)	47.1 (13)*	43.2 (10.2)	45.3 (6.8)
Systolic blood pressure (mm Hg)	105 (7)	102 (8)*	103 (12)	100 (11)*
Diastolic blood pressure (mm Hg)	35 (9)	36 (8)*	34 (7)	35 (8)*
LV end-diastolic diameter (mm.BSA$^{-1/2}$)	46.1 (3.4)	48.3 (4.3)*	46.0 (6.1)	46.8 (5.7)
LV end-systolic diameter (mm.BSA$^{-1/2}$)	26.3 (3.4)	27.1 (3.3)	26.4 (3.9)	26.5 (4.0)
Septal wall thickness (mm.BSA$^{-1/2}$)	6.19 (0.88)	6.20 (0.77)	6.32 (0.73)	6.48 (0.99)
LV posterior wall thickness (mm.BSA$^{-1/2}$)	5.95 (0.94)	6.06 (0.74)	6.21 (1.11)	6.31 (1.00)
LV mass (g.BSA$^{-3/2}$)	57.8 (11.6)	58.6 (12.5)	55.9 (7.2)	57.8 (8.0)
LV shortening fraction (%)	42.4 (4.6)	43.5 (4.8)	43.0 (4.5)	43.6 (5.1)

Results expressed as mean (SD).
*$p<0.05$.
BSA, body surface area; LV, left ventricular.

Subjects performed at the beginning and end of the study period a graded maximal exercise test on a treadmill (Pulsar, HP-Cosmos, Germany) in the same laboratory conditions. The velocity at the first stage was set at 6 km/h and increased by 0.5 km/h every 1 min until exhaustion. Respiratory gas exchanges were measured by using a metabolic measurement system (Cosmed K4b^2, Rome, Italy). VO_{2max} corresponded to the highest VO_2 determined over a 15 s period.

Standard Doppler echocardiography and TDI measurements were performed with the subjects in a partial left decubitus position using ESAOTE ultrasound equipment (MyLab30, Esaote SpA, Firenze, Italy) with a 3.5 MHz transducer. All echocardiographic measurements and calculations were obtained according to standard procedures recommended by the American Society of Echocardiography.[18] M-mode recordings were obtained from the standard parasternal two-dimensional long axis and short axis planes. The echocardiographic parameters measured or derived included: LV end-diastolic and end-systolic dimensions (LVEDd and LVEDs, respectively), posterior wall thickness (PWT) and interventricular septal thickness (ST) at end-diastole as well as LV shortening fraction (SF). LV mass (LVM) was calculated according to the modified Devereux's formula.[19]

Pulsed Doppler LV inflow recordings were performed in the apical four-chamber view, with the sample volume at the tips level of mitral valves. E and A peak velocities (m/s) were measured as previously reported[6] and served as indexes of global diastolic function. Pulsed TDI was performed by transducer

Table 2 Standard Doppler inflow and tissue Doppler measurements at baseline and after 2 months of endurance training

	Experimental group		Control group	
Variable	**Baseline**	**2 months**	**Baseline**	**2 months**
Standard Doppler				
E (cm/s)	86.8 (8.7)	86.0 (13.9)	89.7 (8.9)	90.3 (9.4)
A (cm/s)	41.2 (9.2)	39.6 (9.4)	45.3 (12.6)	44.8 (14.0)
E/A ratio	2.21 (0.48)	2.23 (0.50)	2.10 (0.45)	2.21 (0.80)
Tissue Doppler Imaging : regional diastolic function				
Em septal (cm/s)	15.7 (1.6)	15.3 (1.5)	14.8 (1.3)	15.2 (1.2)
Am septal (cm/s)	6.6 (0.9)	6.5 (1.0)	6.4 (1.1)	6.5 (0.8)
Em/Am septal ratio	2.4 (0.4)	2.4 (0.5)	2.3 (0.4)	2.4 (0.4)
Em lateral (cm/s)	22.8 (3.0)	20.9 (3.0)	21.1 (3.2)	21.0 (3.3)
Am lateral (cm/s)	7.0 (1.6)	6.9 (1.8)	7.0 (1.3)	7.1 (1.2)
Em/Am lateral ratio	3.3 (0.9)	3.1 (0.6)	3.0 (0.7)	3.0 (0.4)
Em inferior (cm/s)	16.3 (3.3)	17.0 (2.8)	15.3 (2.8)	16.9 (3.5)
Am inferior (cm/s)	7.1 (1.4)	7.0 (1.3)	6.7 (2.1)	7.2 (1.1)
Em/Am inferior ratio	2.4 (0.7)	2.3 (0.3)	2.3 (0.6)	2.4 (0.6)
Em anterior (cm/s)	18.8 (3.1)	18.9 (2.4)	19.1 (1.7)	19.2 (2.4)
Am anterior (cm/s)	6.8 (1.2)	7.0 (1.4)	6.9 (1.8)	7.4 (1.5)
Em/Am anterior ratio	2.8 (0.6)	2.7 (0.8)	2.9 (0.7)	2.7 (0.8)
E/Em septal	5.6 (0.9)	5.5 (0.8)	6.0 (0.6)	5.9 (0.8)
Tissue Doppler Imaging : regional systolic function				
Sm septal (cm/s)	8.6 (0.9)	8.1 (0.9)	8.9 (0.9)	8.2 (0.5)
Sm lateral (cm/s)	11.8 (1.9)	11.9 (1.7)	11.9 (2.0)	11.9 (2.3)
Sm inferior (cm/s)	10.6 (2.7)	11.4 (1.8)	10.8 (1.9)	11.1 (1.7)
Sm anterior (cm/s)	9.6 (1.2)	9.7 (1.5)	9.6 (1.1)	9.5 (1.3)

Results presented as mean (SD).
E and A, peak early and late transmitral flow velocities; Em and Am, peak early and late myocardial diastolic velocities; Sm, peak myocardial systolic velocity.

frequency of 3.5 to 4 MHz, adjusting the spectral pulsed Doppler signal filters to a Nyquist limit of 15 to 20 cm/s, and using the minimal optimal gain. Wall motion velocities were assessed at the mitral annulus level on the septal, lateral, inferior and anterior walls, from two and four-chamber views. Myocardial systolic (S_m) and diastolic (early, E_m and atrial, A_m) velocities were measured, allowing assessment of regional systolic and diastolic function. The ratio of peak E to Peak E_m was used as an index of LV filling pressures.[13] Stroke volume (SV) was obtained by LV Doppler outflow method. Measurements were performed from an apical five-chamber view, with the sample volume placed between the aortic leaflet tips. The time-velocity integral of aortic flow (Ao-TVI) was calculated. The aortic diameter was measured at the level of the aortic valve annulus from a long-axis parasternal view. The cross-sectional area of the annulus (Ao-CSA) was then calculated. Finally, SV was calculated as Ao-TVI x Ao-CSA. The heart rate (Hr) was measured simultaneously with all echocardiographic and Doppler examinations. Qc was calculated by multiplying the SV and Hr evaluated during the Ao-TVI measurement. Systolic and diastolic blood pressures (mm Hg) were measured at rest using an automated system (Dynamap, GE Medical Systems, Milwaukee, USA).

All values presented here are the average of a minimum of three technically adequate cardiac cycles. LV internal diameter and wall thickness, as well as stroke volume, were indexed to body surface area (BSA).[20] Linear cardiac dimensions were scaled by $BSA^{0.5}$ and LV mass scaled by $BSA^{1.5}$. Interobserver or intra-observer variabilities of M-mode and Doppler measurements have been reported elsewhere.[3 4 6] The children were examined each time by the same experienced echocardiographer and in the same conditions.

Statistical analysis

The results are presented as mean (SD). Effect of training on biometric and cardiovascular data was examined by means of two-way ANOVA with repeated measures using dedicated software (Statview, SAS Institute, Cary, USA). Statistical significance was defined as $p<0.05$.

RESULTS

Maximal oxygen uptake increased significantly after the 2 month period in the trained children only (table 1). Both groups exhibited a slight decrease in blood pressure. Standard Doppler echocardiography and TDI measurements did not change in the control group. As shown in table 1, our training programme increased left ventricular internal cavity dimensions, although differences were significant for end-diastolic diameter only ($p = 0.10$ for end-systolic diameter). Left ventricular wall thickness and mass, as well as shortening fraction, were not affected by endurance training (table 1). For both groups, resting heart rate did not change at the end of the study period, whereas stroke volume increased in the trained children only (table 1).

Neither standard Doppler inflow velocities nor tissue Doppler measurements were changed after 2 months of high-intensity aerobic training (table 2).

DISCUSSION

To our knowledge, this study is the first to document the longitudinal effect of endurance training on myocardial function by DTI in children. We showed that vigorous aerobic training running sessions repeated over a 2 month period failed to improve longitudinal peak systolic and diastolic wall motion velocities.

In accordance with previous studies in children,[4 16 21] our training programme increased maximal oxygen uptake by about 6.5%, confirming that training-induced improvement in aerobic potential is lower in children than in adults (15–30%). The cardiac remodelling in our trained boys and girls corresponded to that previously reported in the literature after short-term[4 9] or long-term[8] endurance training programmes or in highly trained children.[3 6 10] LV end-diastolic diameter increased modestly but significantly whereas no changes were noticed for LV wall thickness. Improvement in preload distinctly alters LV filling and thereby LV dimensions. Training-induced plasma volume expansion has been previously reported in trained prepubertal children.[22] Whether similar changes in blood volume occurred after training in our children is unknown, and, if they did occur, to what extent they affected LV dimensions. Structural adaptations could also be advanced[23] in order to explain our training-induced cardiac remodelling. The increase in LV filling was associated with an increase in resting stroke volume (table 2), as previously described in trained children.[4–6]

The effect of training on diastolic function in children has been essentially studied by using conventional Doppler examination of mitral flow patterns.[4 6–11] Most of these studies,[7–11] but not all,[4 6] did not report enhancement in mitral inflow indexes in the trained state, which agrees with the present experiment. However, since transmitral inflow velocities are largely influenced by preload conditions,[12] results are difficult to interpret in terms of training effect on myocardial function. Longitudinal velocities of the myocardium recorded by TDI have been suggested to be less affected by loading conditions than Doppler flow measures.[13 14] The major, yet never established, result from the present study was that longitudinal LV velocities estimated by DTI were not improved by endurance training. We could postulate that this lack of training effect was due to the duration and intensity of our programme, and assume therefore that intensive and prolonged stimuli are required to induce significant improvement in diastolic function and especially relaxation properties in the young healthy heart. Indeed, Nottin *et al*[6] established higher wall motion at the LV inferior, posterior and septal walls in 12–13-year old child cyclists who had been

What is already known on this topic

There is still a debate in the literature regarding the influence of endurance training on left ventricular relaxation properties evaluated by TDI in adults, even if the majority of studies reported favourable effects of exercise training. Numerous studies from our group and others have shown that cardiac adaptive changes to endurance training in adults do not strictly mimic those obtained in children. Only two cross-sectional studies evaluated left ventricular longitudinal velocities by TDI in children and controversial results were reported.

What this study adds

Our study is the first to investigate regional left ventricular function by TDI using a longitudinal approach. Our results demonstrated that 2 months of endurance training failed to improve regional systolic and diastolic function in children.

training 5–9 h per week for at least 5 years than in untrained counterparts. However, Ayabakan *et al*[15] did not report differences in tissue Doppler measurements at the LV lateral, posterior and septal walls between prepubertal boy swimmers who had been training 8 h per week for at least 3 years and age and sex-matched inactive controls. Maturation could be involved to explain this apparent blunted response to training. Only prepubertal children were enrolled in Ayabakan's study and the present work, whereas both prepubertal and pubertal boys were investigated by Nottin *et al*.[6] Furthermore, it is important to note in this context that very recently Rodrigues *et al*[24] reported significant improvements in TDI measurements in healthy young adults after 6 months of endurance training. It has, however, to be acknowledged that controversial results have also been obtained regarding TDI indexes in highly trained adult athletes,[25 26] with values being reported enhanced or normal when compared with age-matched sedentary counterparts. We cannot, therefore, rule out the potential effect of genetic factors, as they have been shown to contribute to cardiac dimensions as well as cardiac structure adaptive changes after endurance training programmes.[27] Whether this applies also to cardiac functionalities is unknown.

To conclude, despite an increase in maximal oxygen uptake, LV chamber size and stroke volume, none of the conventional Doppler or tissue Doppler indexes of global and regional left ventricular diastolic function were modified with endurance training in healthy children.

Competing interests: None declared.

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Notice

On 6 June 2007, Springer-Verlag London Limited published a book entitled *The achilles tendon* (ISBN-10: 184628628X; ISBN-13: 978-1846286285). It now transpires that Chapter 27 of the book (entitled "Genes and the Achilles Tendon"), which was authored by Dr Adam Ajis and co-authored by Professor Nicola Maffulli, included unpublished material substantially written by Professor Malcolm Collins of the UCT/MRC Research Unit for Exercise Science and Sports Medicine at the University of Cape Town, South Africa. The material was included in the book by Dr Adam Ajis without the prior knowledge of Professor Nicola Maffulli and without the permission or any acknowledgement of Professor Collins, and is in breach of Professor Collins' copyright subsisting in the material. Professor Maffulli appreciates that full credit should have been, but was not, given to Professor Collins, and wishes to express his regret at this matter and apologise to Professor Collins. Professor Maffulli would also like to point out that both the publishers of the book—Springer-Verlag London Limited—and the co-editor of the book—Professor Louis C Almekinders—were entirely unaware that the inclusion of the material in question infringed copyright and that no responsibility whatsoever attaches to them.