Athletic left ventricular hypertrophy: long-term studies are required

Since the description by Henschen at the turn of the century[1] of cardiac enlargement in cross-country skiers, debate has continued as to whether this adaptation to regular high intensity exercise training is a purely physiological condition or has pathological consequences. As athletic left ventricular hypertrophy or an athlete’s heart has been better characterized in the modern era, mainly by echocardiographic studies, most authors believe that this syndrome is a purely physiological adaptation without harmful consequences[2–5]. The recognized features of athletic left ventricular hypertrophy compared to matched controls are: a resting and relative bradycardia at equivalent workloads, mild concentric left ventricular hypertrophy (15–20% increase in wall thickness), a mildly dilated left ventricular end-diastolic volume (10% increase), normal diastolic filling and normal systolic function[2]. Endurance sports such as running, cycling, rowing and cross-country skiing are said to be particularly associated with these features[2,5].

The prevalence of left ventricular hypertrophy in athletes is difficult to quantify as most studies present mean results and rarely give the number of subjects with a wall thickness >12 mm or a left ventricular mass index >134 g.m⁻². In the largest single echocardiographic study, at least 10% of the athletes had a wall thickness (≥12 mm) outside the traditionally accepted normal range[6]. A similar percentage of female athletes demonstrate hypertrophy if a sex-specific cut-off of left ventricular mass index ≤110 g.m⁻² is used[6]. Moderate left ventricular hypertrophy to a level which may be confused with pathological disorders such as hypertrophic cardiomyopathy, i.e. interventricular septum ≤13 mm, occurs in less than 2% of male athletes and usually only in sports involving a degree of isometric upper limb exercise such as rowing, canoeing and cycling[5,6].

Several arguments have been advanced to support the concept that athletic left ventricular hypertrophy has no pathological significance, largely based on cross-sectional studies comparing groups of athletes with sedentary controls. The main tenets are: athletes have better cardiorespiratory fitness as measured by maximal oxygen consumption (VO₂max)[7] associated with increased left ventricular mass; the left ventricular hypertrophy regresses if athletes decondition[5,8]; left ventricular systolic function and Doppler measured mitral diastolic filling patterns are usually normal[5]; and, lastly, follow-up studies of elite international standard competitors suggest that they live longer than expected compared to the population mean[9].

However, athletic left ventricular hypertrophy does not only occur in Olympic standard athletes, as has been shown by Montgomery et al.[10]. They studied 460 army recruits prior to and after an initial 10 week strength and endurance training programme, and demonstrated that mean left ventricular mass increased by 18% from 167 g to 197 g at the end of the study. Moreover, increase in left ventricular mass appears to have a strong genetic component which may explain why only some athletes develop left ventricular hypertrophy. Subjects were subdivided into three groups depending on angiotensin-converting enzyme (ACE) genotype polymorphism: homozygous insertion (II); heterozygous insertion/deletion (I/D) or homozygous deletion (D/D). The groups with the deletional allele showed a much greater rise in left ventricular mass, suggesting that increased ACE levels play a major role in the development of myocardial hypertrophy in response to exercise training. The increase in left ventricular mass was mirrored by an increase in brain natriuretic peptide, particularly in those with the D/D genotype.

We are uncomfortable with the currently accepted view that athletic left ventricular hypertrophy is a purely physiological adaptation with no pathological consequences, for several reasons. Firstly, although sudden cardiac death in competitive athletes under the age of 35 years is rare, up to 18% of post-mortems of athletes dying during sport suggest a condition which has been termed idiopathic left ventricular hypertrophy, where no clear reason has been found for the increase in left ventricular mass[11]. We feel that some of these cases may be attributed to the athlete’s heart syndrome. Secondly, the life expectancy results of Sarna et al. were not controlled for cardiovascular risk factors including smoking status, which one would expect to have a major bearing on cardiovascular mortality[9]. Also, the fact that left ventricular hypertrophy caused by endurance training regresses on cessation of regular exercise, merely confirms that athletic left ventricular hypertrophy acts the same way as other pathological conditions.

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such as treated hypertension and left ventricular hypertrophy in aortic valve disease after aortic valve replacement. Once the abnormal stimulus to hypertrophy is removed (pressure overload in hypertension/aortic stenosis, volume overload in valve regurgitation or endurance training) the left ventricular hypertrophy regresses. Similar to hypertension, diastolic filling may become abnormal, compared to age-matched controls, after many years of high intensity exercise as has been demonstrated in professional veteran cyclists and this finding may not be reversible up to 2 years after cessation of regular training. An increase in brain natriuretic peptide with the athlete’s heart syndrome is also seen in other accepted pathological causes of left ventricular hypertrophy and may indicate an adverse prognosis, as has been shown in acute myocardial infarction.

As far as we are aware, no studies in homogenous groups of athletes have shown that those with an increase in left ventricular mass have a performance advantage over those with normal cardiac dimensions. In our unpublished series of 20 elite soccer players, there was a weak non-significant negative correlation between left ventricular mass index and VO_{2\max}. Finally, we have recently shown in a larger group of professional athletes (n=120) that those with left ventricular hypertrophy, mass index >134 g, m^{-2} (n=30), have a greater QT dispersion than both athletes without left ventricular hypertrophy and sedentary matched controls. Increased QT dispersion has been shown to be a risk factor for sudden cardiac death in left ventricular hypertrophy secondary to hypertension and predicts ventricular arrhythmias and sudden death in hypertrophic cardiomyopathy.

We conclude on the basis of the current evidence that athletic left ventricular hypertrophy behaves exactly the same way as every other pathological cause of an increase in cardiac mass. We therefore believe that long-term studies are required to assess the impact of athletic left ventricular hypertrophy on morbidity and mortality before we can reassure these subjects that they are not at increased risk of future cardiac events.

Such studies will not be easily performed. Athletic left ventricular hypertrophy is uncommon in the general population, with perhaps only 10% of adolescents and young adults actively involved in competitive sport with approximately 10% of these exhibiting left ventricular hypertrophy. The incidence of sudden cardiac death is estimated to be 1-2/100 000 per athlete years and therefore is a rare phenomenon which is why large long-term studies would be required to demonstrate an increased risk of mortality. Further information regarding left ventricular hypertrophy as a possible independent risk factor for ischaemic heart disease could also be obtained. As pre-participation athletic screening is already in place in many countries, it should be possible to coordinate a multicentre, multinational study, collating and analysing the data at one centre. The following, which may be associated with an adverse prognosis, should be measured: (a) left ventricular mass/mass index, (b) brain natriuretic peptide, (c) QT dispersion and (d) ACE genotype. The Framingham Heart Study has demonstrated that echocardiographically determined left ventricular hypertrophy is associated with an approximate two-fold increased relative risk (corrected for traditional cardiovascular risk factors) of cardiovascular mortality. Although there is no direct evidence in the current literature to suggest that athletic left ventricular hypertrophy is an additional risk factor, we cannot say that it is a purely physiological adaptation until long-term studies with large subject numbers demonstrate no adverse outcomes compared to similarly matched athletic subjects without left ventricular hypertrophy.

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References


