Original articles

Echocardiographic examination of cardiac structure and function in elite cross trained male and female Alpine skiers

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Abstract

***Objective*—To assess cardiac structure and function in elite cross-trained male and female athletes (Alpine skiers).**

***Methods*—Sixteen athletes (10 male, six female) and 19 healthy sedentary control subjects (12 male, seven female) volunteered to take part in the study. Basic anthropometry determined height, body mass, body surface area, and fat free mass. Cardiac dimensions and function were determined by two dimensional, M mode, and Doppler echocardiography. Absolute data and data corrected for body size (allometrically determined) were compared by two way analysis of variance and post hoc Scheffé tests.**

***Results*—Absolute left ventricular internal dimension in diastole (LVIDd), septal and posterior wall thickness and left ventricular mass were larger in athletes than controls ($p<0.05$) and also increased in the men ($p<0.05$) compared with women (except for septal thickness in controls). An increased LVIDd, septal thickness, posterior wall thickness, and left ventricular mass in athletes persisted after correction for body size except when LVIDd was scaled by fat free mass. Cardiac dimensions did not differ between the sexes after correction for body size. All functional indices were similar between groups.**

***Conclusion*—There is evidence of both left ventricular chamber dilatation and wall enlargement in cross trained athletes compared with controls. Differences in absolute cardiac dimensions between the sexes were primarily due to greater body dimensions in the men.**

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Keywords: left ventricular mass; skiing; concentric enlargement; eccentric enlargement

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Cardiac structure and function are assessed in athletes for two primary reasons. Firstly, it may help underpin and explain increased athletic ability or physiological capacity. Secondly, it may help delineate physiological adaptation of the left ventricle due to exercise training, from various pathological processes that may predispose to sudden cardiac death.[1] Both these issues have been assessed in a number of elite athletic groups in cross sectional studies and longitudinally through periods of training. Many of these data have been extensively reviewed.[1–4] The consensus of opinion expressed at the 26th Bethesda conference (Recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities) was that aerobic training resulted in an increased left ventricular chamber size (eccentric enlargement), power training resulted in increased wall thickness (concentric enlargement), and athletes who combined these types of training (cross training or multiactivity training) would have a combination of eccentric and concentric enlargement.[5] References were supplied for the first two of these statements, but no data were supplied to support the final contention.

Although it would seem logical to assume that cross training induces periods of both haemodynamic pressure and volume overload that may result in a "mixed" pattern of left ventricular enlargement, few data exist to support this assertion empirically. Previous research has tended to investigate cardiac adaptation at one or both ends of the aerobic-anaerobic training spectrum.[6 7] Large cross sectional samples including data from many different sports have been published, most notably by Pelliccia and coworkers.[8–10] The aim of their studies was to determine general upper limits of cardiac size, rather than to investigate adaptations specific to training in single sport/activity groups. An extensive literature search produced only one article whose primary purpose was to investigate cardiac dimensions in cross trained subjects.[11] Elias *et al* provided some evidence for a mixed concentric/eccentric left ventricular enlargement in subjects who both ran and weight trained.[11] The data in Elias's study are, however, difficult to interpret because (*a*) there was no comparison with a control group; (*b*) body size normalisation of cardiac dimensions in Elias's study was achieved by a ratio-standard approach (this has been widely criticised[12–16] and may have led to

erroneous interpretation of the data); and (*c*) it is not clear whether the subjects represented elite athletic groups. As with most research into cardiac dimensions, Elias *et al*[11] only presented data from male subjects. It is unclear whether male and female subjects would respond similarly to the haemodynamic loading associated with cross training.

To address the methodological issues in the study by Elias *et al*, and to add to the available body of knowledge, our study investigated a group of male and female Alpine skiers and compared them with sedentary controls. Alpine skiers would seem to represent an ideal elite, cross trained group. Such athletes perform in races of roughly 40–120 seconds' duration and are thus characterised by high aerobic and anaerobic power,[17] increased isometric and low velocity concentric and eccentric muscle strength,[18] as well as improved skill, balance, and flexibility.[19] To deal with problems of body size normalisation, cardiac structural data were scaled for group differences in body dimensions by allometric procedures.

Methods

Written informed consent was obtained from all subjects after the study gained ethical approval from the local ethics committee. Sixteen athletes, from a national Alpine ski squad (10 male, six female; mean (SD) age 22 (3) years), and 19 healthy sedentary control subjects, from a similar aged student population (12 male, seven female; mean (SD) age 21 (3) years), volunteered to take part in the study. All athletes provided information about their training and competitive activity during a brief informal interview. All the athletes had been training for national/international competition for a minimum of six years and were competing in Europa Cup races, World Club races, or on the World Cup circuit. Training regimens for all athletes were relatively similar as they took part in squad training programmes.

The profiles of elite Alpine skiers have shown that success demands a high degree of muscular power, strength, endurance, flexibility, agility, balance, and aerobic endurance.[19] In this study such theoretical physiological attributes were supported by training information provided by the athletes themselves. All athletes were coming to the end of a period of pre-season fitness training (at low altitude) that included weight training, plyometrics, sprint exercise, aerobic running and cycling, and dry land ski simulation exercises, such as roller blading. Resistance training comprised low volume high intensity exercise, whereas aerobic training sessions were designed to improve aerobic capacity by steady state, moderate intensity, prolonged exercise. The athletes reported that they trained for a minimum of six days a week, and each day typically involved a combination of both aerobic and anaerobic activities. Haemodynamic studies specific to Alpine skiing are not available, but it seems realistic to assume that a combination of both transient pressure and volume overload would occur during training and competition.

The control group were all apparently healthy. No control subjects were formerly or currently engaged in any structured exercise training programme associated with taking part in competitive sport. They reported that their physical activity was limited to one to two hours a week of recreational activities such as jogging, cycling, and step aerobics. No subjects reported any personal history of hypertension or any other manifestation of cardiovascular disease.

All subjects attended the physiology laboratory on one day and a weight training facility on a separate occasion within one to two days of the initial test. Data were collected in the same order for all subjects. A health questionnaire was given to subjects and controls followed by a brief interview about training history. Subjects were then assessed anthropometrically for height (m) and body mass (kg), which allowed the estimation of body surface area (m^2).[20] Skinfold thicknesses at the biceps, triceps, subscapular and suprailiac sites were averaged from triplicate measures and used to predict body fat percentage and fat free mass (kg).[21] Subjects then rested in a supine position for a minimum of 30 minutes. After this, resting heart rate (electrocardiogram, (ECG)) and blood pressure (manual sphygmomanometry) were recorded, immediately followed by echocardiographic analysis (HP Sonos 100 ultrasound imaging system; Hewlett Packard, Andover Massachusetts). A 2.5 MHz transducer was used to obtain a two dimensional long axis view of the left ventricle from the parasternal window. M mode recordings were taken at the tip of the mitral valve leaflets. From a concomitant ECG, septal thickness (ST), posterior wall thickness (PWT), and left ventricular end diastolic dimension in diastole (LVIDd) were digitised at the peak of the R wave according to guidelines set down in the Penn convention.[22] Left ventricular end systolic dimension (LVIDs) was measured as the minimum separation of the septal and posterior walls. Left ventricular mass was estimated by a previously validated "cube formula" corrected for regression.[22] The ratio of wall thickness (h=ST+PWT) to internal dimension (R=LVIDd) was calculated as an index of concentric enlargement (h:R). Fractional shortening percentage (FS (%) = LVIDd−LVIDs/ LVIDd $\times 10^{-2}$) was calculated as an index of left ventricular contractility. Diastolic function was assessed by pulsed wave Doppler echocardiography. A two dimensional sector scan of a four chamber view from the apex position allowed the placement of the sample volume at the level of the mitral valve, parallel to mitral inflow. This facilitated the measurement of peak flow velocities (cm/s) for early passive (E) and late atrial contraction (A) filling of the left ventricle.[23] The ratio E:A was then calculated. Ultrasound images of the left ventricle were obtained by a single experienced echocardiographer. Measurements were made over five consecutive heart cycles by a separate, experienced individual who was unaware of group allocation.

Table 1 Anthropometric, performance and clinical characteristics of athlete and control subjects (data are means (SD))

	Skiers		*Controls*	
	Men	*Women*	*Men*	*Women*
Age (years)	22 (4)	21 (4)	23 (2)	21 (3)
Height (m)	1.80 (0.03) †	1.62 (0.03)	1.78 (0.06)†	1.67 (0.05)
Body mass (kg)	78.4 (3.6)†	62.3 (2.5)	74.8 (3.6)†	64.0 (7.1)
Body surface area (m^2)	1.98 (0.06)†	1.66 (0.05)	1.92 (0.07)†	1.71 (0.09)
Body fat (%)	11.5 (2.1)*†	20.6 (1.8)*	14.8 (2.3)†	24.7 (2.4)
Fat free mass (kg)	69.3 (3.5)*†	49.5 (2.4)	63.7 (3.7)†	48.1 (5.5)
Maximum oxygen uptake (ml/min/$kg^{0.67}$)	241 (13)*†	200 (18)*	189 (24)†	133 (18)
1RM bench press (kg)	82.2 (7.3)*†	45.5 (6.)1*	57.1 (12.1)†	32.7 (6.4)
Resting heart rate (beats/min)	58 (8)*	57 (10)*	69 (6)	68 (9)
Resting systolic BP (mm Hg)	120 (9)	116 (6)	122 (10)	118 (7)
Resting diastolic BP (mm Hg)	74 (7)	70 (12)	74 (10)	74 (6)

*Significantly different from the control group ($p<0.05$).
†Significantly different between men and women ($p<0.05$).
BP= blood pressure.

To determine indices of aerobic and anaerobic fitness, maximum oxygen uptake ($\dot{V}O_2MAX$) and maximal bench press performance (1RM) were determined in all subjects. Subjects familiarised themselves with procedures before a graded bicycle exercise test to volitional exhaustion, which was conducted in the physiology laboratory after the echocardiographic analysis. Online gas analysis (Sensormedics metabolic measurement Cart 2900, Bilthoven, Netherlands) and heart rate data (ECG) were collected. On a subsequent day, after at least 12 hours of recovery, all subjects performed graded bench press exercises to failure (1RM). Subjects familiarised themselves with the technique before exercise, and progression from first lift to failure normally involved five to eight single lifts with adequate recovery in between. Aerobic warm up, stretching, and activity exercises preceded both performance tests.

Echocardiographic data were expressed in absolute units and then scaled allometrically for individual differences in anthropometric data. Exponents for allometric scaling were generated according to dimensionality theory.[24 25] This theory states that one dimensional/linear cardiac dimensions should be scaled by height (HT) raised to the power 1.00 ($HT^{1.00}$), by $BSA^{0.50}$, by $BM^{0.33}$, and $FFM^{0.33}$. Left ventricular mass should be scaled by $HT^{3.00}$, $BSA^{1.5}$, $BM^{1.00}$, and $FFM^{1.00}$. The use of these dimensionally consistent allometric exponents is supported by cardiological studies,[12 13] exercise science research,[14 15] and, specifically, in an elite athlete group.[16] Maximum oxygen uptake data were also scaled allometrically adopting a body mass exponent of 0.67.[26] Bench press 1RM was not scaled owing to current controversy about the scaling of body size in very short term maximal human muscular performance.[27] All data were compared by two way analysis of variance. A Scheffé post hoc analysis was used owing to the unequal sample sizes. Pearson product-moment correlations were performed between absolute cardiac dimensions and maximum oxygen uptake for the entire sample. A critical α of 0.05 was adopted. Where appropriate, effect sizes were calculated to aid interpretation of the data.[28] Statistical analyses were performed on Statistica software (Statsoft Ltd, Tulsa, Oklahoma).

Results

Athletes and controls were of a similar age, but their anthropometric variables were distinctly different (table 1). Both male groups were significantly taller, heavier, and possessed greater body surface area and fat free mass than their respective female counterparts ($p<0.05$). The male athletes were slightly, but non-significantly ($p>0.05$), taller, heavier, and had a greater body surface area than the male controls. The male athletes, owing to a significantly reduced percentage of body fat, had a greater fat free mass ($p<0.05$) than the male controls. The female athletes were slightly, but non-significantly ($p>0.05$), shorter, lighter and had a reduced body surface area than the female controls. However, owing to a significant reduction in the percentage of body fat in the female athletes, they possessed slightly more ($p>0.05$) fat free mass than the female controls.

Maximum oxygen uptake (table 1) was significantly greater ($p<0.05$) in both athlete groups than in controls (27% in men; 50% in women) and also in both male groups than in female groups (21% in athletes; 42% in controls). Differences were noted in the 1RM bench press data (44% in men; 39% in women; 81% in athletes; 75% in controls). Resting heart rates were similar in male and female athletes and significantly lower than in the control groups ($p<0.05$). Blood pressures tended to be lower in women, though no difference between groups was significant ($p>0.05$).

Mean values for cardiac dimensions (table 2) in the athlete and control groups were within "normal ranges".[29] Absolute LVIDd was greater in men than women ($p<0.05$) and also in the athletes than in the corresponding control group ($p<0.05$). The difference between athletes and controls persisted after allometric scaling of LVIDd by height, body mass, and body surface area ($p<0.05$). When LVIDd was scaled for fat free mass a significant main effect for "group" was still evident in the two way analysis of variance, although a post hoc Scheffé test reported a p value of 0.11 (for male skiers *v* male controls) and a p value of 0.12 (for female skiers *v* female controls). Effect size data (1.33 for male skiers *v* male controls; 0.83 for female skiers *v* female controls) supported

Table 2 Absolute and allometrically scaled left ventricular structural data (data are means (SD) with ranges for absolute data in parentheses)

	Skiers		*Controls*	
	Men	*Women*	*Men*	*Women*
LVIDd (mm)	55.5 (1.9)*† (53–59)	50.5 (1.8)* (48–53)	50.9 (2.6)† (46–54)	46.6 (2.9) (42–48)
LVIDd/HT$^{1.00}$	30.7 (0.7)*	31.3 (1.4)*	28.7 (1.4)	27.9 (2.1)
LVIDd/BM$^{0.33}$	13.1 (0.4)*	12.9 (0.5)*	12.3 (0.6)	11.8 (1.1)
LVIDd/BSA$^{0.50}$	39.4 (1.0)*	39.2 (1.6)*	36.8 (1.7)	35.5 (2.9)
LVIDd/FFM$^{0.33}$	13.7 (0.4)	13.9 (0.5)	12.9 (0.6)	12.9 (1.2)
LVIDs (mm)	33.4 (1.4)† (32–35)	30.2 (1.6) (27–31)	31.3 (1.8)† (30–34)	28.1 (2.0) (26–30)
LVIDs/HT$^{1.00}$	18.6 (0.8)	18.8 (1.1)*	17.6 (0.8)	16.8 (1.3)
LVIDs/BM$^{0.33}$	7.94 (0.32)	7.74 (0.41)	7.54 (0.39)	7.13 (0.54)
LVIDs/BSA$^{0.50}$	23.8 (1.0)	23.5 (1.3)*	22.6 (1.1)	21.4 (1.6)
LVIDs/FFM$^{0.33}$	8.26 (0.34)	8.35 (0.43)	7.94 (0.43)	7.83 (0.60)
PWT (mm)	10.6 (1.0)*† (9–12)	8.7 (0.7)* (8–10)	8.3 (0.9)† (7–10)	7.1 (0.5) (6–8)
PWT/HT$^{1.00}$	5.89 (0.51)*	5.38 (0.31)*	4.67 (0.53)	4.29 (0.39)
PWT/BM$^{0.33}$	2.52 (0.23)*	2.22 (0.15)*	1.99 (0.20)	1.81 (0.16)
PWT/BSA$^{0.50}$	7.55 (0.67)*	6.75 (0.43)*	5.97 (0.56)	5.46 (0.47)
PWT/FFM$^{0.33}$	2.62 (0.24)*	2.40 (0.16)*	2.10 (0.23)	1.99 (0.16)
ST (mm)	10.1 (0.9)*† (9–11)	8.5 (0.5)* (8–10)	7.6 (0.8) (6–9)	7.0 (0.6) (6–8)
ST/HT$^{1.00}$	5.59 (0.51)*	5.24 (0.22)*	4.26 (0.48)	4.21 (0.40)
ST/BM$^{0.33}$	2.39 (0.23)*	2.17 (0.09)*	1.82 (0.18)	1.78 (0.16)
ST/BSA$^{0.50}$	7.16 (0.67)*	6.57 (0.28)*	5.46 (0.56)	5.37 (0.54)
ST/FFM$^{0.33}$	2.49 (0.24)*	2.34 (0.09)*	1.92 (0.19)	1.96 (0.21)
LVM (g)	272 (47)*† (241–292)	194 (21)* (171–222)	180 (32)† (115–223)	133 (22) (100–155)
LVM/HT$^{3.00}$	46.5 (6.4)*	46.1 (4.5)*	32.3 (6.2)	28.9 (6.4)
LVM/BM$^{1.00}$	3.47 (0.52)*	3.12 (0.31)*	2.40 (0.38)	2.11 (0.51)
LVM/BSA$^{1.50}$	97.7 (13.9)*	90.6 (8.7)*	67.6 (11.1)	59.8 (13.7)
LVM/FFM$^{1.00}$	3.93 (0.58)*	3.92 (0.38)*	2.83 (0.48)	2.81 (0.70)

*Significantly different from the control group ($p<0.05$).
†Significantly different between men and women ($p<0.05$).
Abbreviations: LVIDd = left ventricular internal dimension in diastole; LVIDs = left ventricular internal dimension in systole; PWT = posterior wall thickness; ST = septal wall thickness; LVM = left ventricular mass; HT = height; BM = body mass; BSA = body surface area; FFM = fat free mass.

the suggestion that the group difference in LVIDd/FFM$^{0.33}$ was statistically "meaningful". When LVIDd was scaled by any of the anthropometric variables the previous sex difference (in absolute data) was removed in both athletes and controls. The absolute LVIDs was greater in men than women ($p<0.05$) but not different between athletes and controls of the same sex. When LVIDs was scaled by any of the anthropometric variables the sex difference (in absolute data) was removed in both athletes and controls. LVIDs in male skiers and male controls was not significantly different after scaling for any body size variable. However, female skiers had a significantly larger LVIDs when scaled for height and body surface area ($p<0.05$), but a similar LVIDs when scaled for body mass or fat free mass ($p>0.05$), than the female controls.

Both posterior wall thickness and septal thickness were significantly different between athletes and controls ($p<0.05$). The posterior wall thickness and septal thickness of male athletes were on average 2.3 and 2.5 mm greater, respectively, than those of male controls, and of female athletes on average 1.6 and 1.5 mm greater, respectively, than those of female controls. These differences are beyond the resolution limits of the system employed. The absolute posterior wall thickness was greater in men than women in both the athlete and the control groups ($p<0.05$). For absolute septal thickness the difference between the sexes was only significant for the athlete groups ($p<0.05$). After scaling for differences in body size variables, there were consistent differences between the athletes and their sex matched control groups ($p<0.05$). Any initial difference between the sexes (within the athlete or control group) was removed by all of the scaling procedures. The h:R ratio (0.37 (0.03) male skiers, 0.34 (0.02) female skiers, 0.31 (0.03) male controls, and 0.30 (0.02) female controls) was significantly greater in both athlete groups than in controls ($p<0.05$).

The percentage difference in absolute left ventricular mass between athletes and controls was 51% and 46% in men and women, respectively ($p<0.05$). The percentage difference between the sexes was 40% and 35% in athletes and controls, respectively ($p<0.05$). Although the significant difference between athletes and controls persisted after scaling for all body size variables ($p<0.05$), the difference between the sexes was reduced and became non-significant ($p>0.05$).

Fractional shortening was similar across all groups (table 3). There were no significant group differences in the peak filling velocities of either the early or atrial diastolic inflow. A ten-

Table 3 Resting systolic and diastolic cardiac functional indices (data are means (SD))

	Skiers		*Controls*	
	Men	*Women*	*Men*	*Women*
Fractional shortening (%)	40 (3)	40 (2)	38 (2)	40 (3)
Early diastolic peak filling velocity (cm/s)	90 (12)	87 (7)	83 (16)	81 (14)
Late-atrial diastolic peak filling velocity (cm/s)	45 (7)	42 (5)	43 (9)	41 (8)
E:A ratio	2.04 (0.39)	2.12 (0.26)	1.95 (0.42)	2.04 (0.51)

E:A ratio = ratio of early passive (E) to late atrial contraction (A) filling of the left ventricle.

dency for higher early inflow velocity data in athletes was accompanied by a large degree of individual variability within each group. The E:A data were also non-significant between the groups. Pearson product-moment correlations between maximum oxygen uptake and LVIDd, LVIDs, posterior wall thickness, septal thickness, and left ventricular mass were 0.68, 0.57, 0.68, 0.66, and 0.70, respectively. All correlation coefficients were significant ($p<0.05$).

Discussion

Scaling for the group differences in anthropometric characteristics was achieved by allometric power function procedures. These are gaining wider acceptance within sports medicine publications as the most statistically appropriate way to account for the influence of body size variables upon cardiac dimension data.[12–16] This facilitates a theoretically and practically more valid process of comparing the cardiac dimensions in groups of disparate body size and composition. This is especially important in athlete-control studies where training may significantly alter body composition, independent of body size. Although this study adopted power function exponents calculated by dimensionality theory as predicted by Gutgesell and Rembold,[25] and supported by Batterham *et al*,[14] Batterham and George,[15] and George *et al*,[16] it has been suggested that specific power function exponents should be determined for each individual group to be studied.[14] This approach was not adopted in the current study owing to the small sample size and the limited range of both anthropometric and cardiac dimension data. Both these aspects of the present subject group would probably have produced very weakly specified power function exponents (wide 95% confidence limits). Thus it was deemed appropriate to adopt generic values that have been previously supported.

The physiological performance tests provided evidence that skiers were both aerobically and anaerobically superior to the control groups. Given the haemodynamic loading that would occur with the type of training required to develop an increase in maximum oxygen uptake and 1RM, it seemed that the skiers represented an ideal group in which to determine the effects of prolonged elite level, cross training on cardiac dimensions. The cardiac data presented (table 2) seem to support the contention that both walls, and the chamber cavity of the left ventricle of the skiers had adapted to haemodynamic pressure and volume overloads, respectively. This pattern would be predicted based on the work of Grossman *et al*.[30] The only variable not significantly different between athletes and controls was $LVIDd/FFM^{0.33}$. This parameter is still greater in both male and female athletes than in their respective control groups. However, whether this represents true ventricular dilatation (eccentric enlargement) requires verification. In general, LVIDs was not significantly different between groups, except between female skiers and controls when it was scaled for height and body surface area. This is likely to be a consequence of general enlargement of the left ventricle, as noted with the LVIDd value.

Wall thicknesses were consistently larger in athletes than controls after all body dimension corrections. These data, together with the differences noted for the h:R ratio, may provide evidence of a slight bias towards high intensity activity (and thus a haemodynamic pressure overload) in the athlete's training and competition. This might have been predicted based on the suggested 60%:40% split in anaerobic:-aerobic energy sources being used during ski races.[31] Grossman *et al* also stated that any chamber dilatation due to volume overload would result in a small compensatory wall thickening to normalise wall stress.[30] Therefore the h:R data might be explained by a combination of a pressure and volume overload acting as a stimulus for wall thickening.

A key finding from this subject sample was the lack of statistically significant differences for any cardiac dimension between men and women after correction for body size. This would suggest that most sex related differences in left ventricular size are due to differences in male-female body size and composition, and not to any other sex related factor. These data contradict previous research that has directly compared groups of elite male and female athletes. Pelliccia *et al*[10] compared 600 elite female with 738 elite male Italian athletes and reported significant differences in absolute cardiac dimensions, findings similar to those we report in this study. Significantly greater wall thickness and left ventricular mass values for men were maintained after ratio-standard scaling (for height and body surface area). Cavity dimension normalised to height (ratio-standard) was still greater in men, but when normalised to body surface area was significantly larger in the women. The lack of a difference in cardiac dimensions between the sexes in this study is supported partially by the findings of Morales *et al*,[32] who showed that cardiac dimensions were similar after scaling (ratio-standard) for body dimensions in equally trained male and female runners. This issue is still confused by the varied choice of scaling process and variable. Future research may wish to clarify the nature and extent of any sex differences in cardiac dimensions in elite athletes. Specific attention may be paid to the importance of differences in body composition between the sexes.

In the current study cardiac function was normal in all subjects. The lack of statistically significant differences in any resting systolic or diastolic functional index is consistent with most previous research in men and women.[3 33] It should be noted that there is no functional decrement in the athletes despite significant increases in left ventricular mass, and a small but significant relative concentric adaptation as shown by the h:R data.

Training and competitive activity are likely to impose a combination of haemodynamic pressure and volume overload. It is difficult to be certain whether this loading alone is sufficient to produce the cardiac adaptation

noted in this study. Alternatively, as is the potential in any cross sectional study, genetic predisposition and self selection to the sport may have been an influence, and may have accounted for a significant percentage of the variance in cardiac dimensions between the groups. Similarly, whether the patterns of cardiac adaptation are essential in supporting successful competitive performance or, as Tesch[34] proposed for maximum oxygen uptake, are simply a consequence of the training regimens adopted, is difficult to ascertain. Interestingly, in our study, significant and positive correlations were noted between maximum oxygen uptake and cardiac dimensions. These correlations were similar to those published by Osborne *et al*.[35]

Clinically, the data demonstrated a healthy left ventricular adaptation in both male and female athletes that is different from the pattern generally observed in athletes who are purely aerobically or anaerobically trained. No data, structural or functional, were of a magnitude to suggest that these adaptations resembled pathological heart disease. Only one male athlete reported a wall thickness of 12 mm, and only one female athlete had a wall thickness of 10 mm. Thus the athletes were not structurally within the "grey zone" of differentiation from hypertrophic cardiomyopathy.[2] However, clinicians should be aware that elite athletes who cross train, such as Alpine skiers, may be found to have moderate wall and cavity enlargement of the left ventricle upon echocardiographic investigation.

In conclusion, both male and female Alpine skiers showed evidence of left ventricular wall and internal cavity adaptation compared with sedentary controls. The clinical significance of this was negligible as cardiac function remained normal in both athlete groups. Significant differences in absolute cardiac dimensions between the sexes were removed after allometric scaling for body size and composition differences.

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Take home message

Athletes who combine both endurance and resistance training may have greater left ventricular wall and cavity dimensions than controls. This information may be of relevance in any cardiovascular screening process and could be of importance in underpinning increased performance capacity.

Commentary

The authors report their assessment of cardiac structure and function in a small number (10 male, six female) of elite cross trained male and female athletes (Alpine skiers) and use a group of healthy sedentary subjects as controls. They have assessed the relation between echocardiographic findings and absolute and body size corrected data (allometrically determined). They conclude that the athletes studied have larger left ventricular diastolic dimensions (LVIDd), wall thickness, and left ventricular mass than controls. Their results confirm that these athletes have a combination of eccentric and concentric enlargement, as suggested by Spirito *et al* in their review of a large group of Italian athletes which included 32 Alpine skiers (24 male, eight female).[1] However, in this study the authors have also performed allometric scaling of the anthropometric data and have shown that the differences between athletes and controls persist even after body size correction (except when LVIDd was scaled by fat free mass). The most interesting finding of the study is the fact that no difference exists between the sexes for any of the cardiac dimensions after correction for body size.

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1 Spirito P, Pelliccia A, Proschan MA, *et al.* Morphology of the "athlete's heart" assessed by echocardiography in 947 elite athletes representing 27 sports. *Am J Cardiol* 1994;**74**:802–6.

Commentary

One of the principal physiological adaptations to intensive exercise is a modest increase in cardiac dimensions. In a small proportion of athletes, absolute cardiac dimensions may exceed the upper limits of normal for the general population and resemble dimensions seen in cardiomyopathy. This is highlighted in a study of almost 1000 elite Italian Olympic athletes which showed that 2% athletes had a left ventricular wall thickness (LVWT) exceeding 12 mm (13–15 mm), which could be compatible with hypertrophic cardiomyopathy, the commonest cause of sudden cardiac death in young elite athletes. Interestingly, all but one athlete with an LVWT >12 mm had a body surface area exceeding 2 m^2. Correction for body surface area in each of these subjects would have shown that the LVWT was within normal limits for size. In this study the authors have set out to assess cardiac dimensions in highly trained, cross trained athletes after correction for body size and shown that cardiac dimensions are modestly increased but do not exceed the upper limits of normal. Scaling cardiac dimensions for body size is important in the cardiovascular evaluation of an athlete being assessed for possible underlying cardiomyopathy.

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