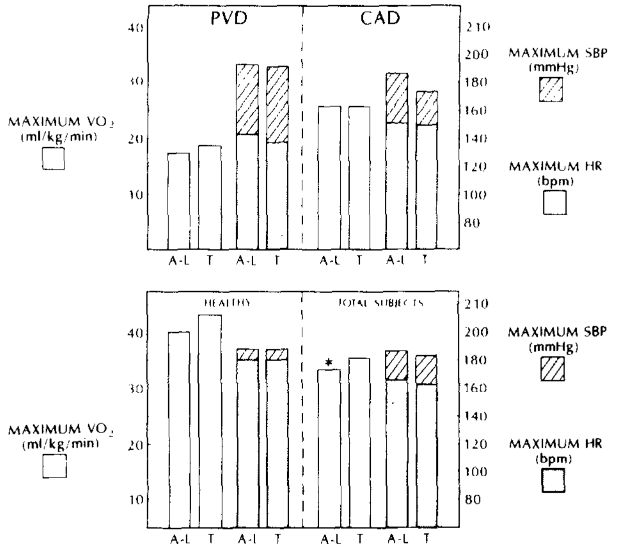

Fig. 1. Measured maximal oxygen consumption *(VO_2)*, heart rate, and systolic blood pressure. Data are for combined arm-leg ergometry *(A-L)* and treadmill *(T)* testing in the healthy, coronary artery *(CAD)*, and peripheral vascular disease *(PVD)* subjects, and for all subjects as a whole *(TOTAL)*. $*p < 0.05$.

Table III. Maximal rate-pressure products and respiratory exchange ratios for air-braked ergometry (A-L) and treadmill (T) testing

	Rate-pressure product (mm Hg × beats/min/100)		*Respiratory exchange ratio*	
	A-L	*T*	*A-L*	*T*
Total	306	292*	1.2	1.2
(*N* = 41)	7.0	8.5	0.02	0.02
PVD	282	263	1.1	1.1
(*n* = 9)	12.7	11.8	0.04	0.04
CAD	289	264*	1.3	1.3
(*n* = 10)	6.5	7.0	0.02	0.01
Healthy controls	327	324	1.3	1.2
(*n* = 22)	6.7	9.8	0.03	0.07

All data are expressed as means ± SEM.
$*p < 0.05$.

ing better tolerated in patients with PVD, and this finding has implications for both testing and training. A longer exercise time and greater work rate may confer an advantage for exercise testing if ST-segment evidence of ischemia can be elicited before exercise-limiting leg claudication. Also, if arm-leg ergometry is better tolerated than walking, the patient with PVD may be able to perform this exercise longer. Longer exercise bouts during exercise training would facilitate improvement in aerobic capacity (but not in claudication walking distance) in patients with PVD. However, both of these concepts are speculative and require further study. In conclusion, we report that maximal exercise efforts can be obtained with combined arm-leg ergometry testing. This device may have advantages for exercise testing and exercise training in patients with PVD.

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The role of gender in echocardiographically determined left ventricular mass in equally trained populations of runners

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Athletes participating in a wide variety of sports have been shown to have increased left ventricular mass.[1-3] Variables known to affect the degree of left ventricular hypertrophy seen in athletes include gender[4] and the length of exercise training.[5] Left ventricular mass has been reported to be significantly higher in men runners then in women runners,[4] but the effect of gender on left ventricular mass (LVM) in equally trained runners is not known. We studied competitive men and women runners with equal training status to determine the effect of gender on exercise-related hypertrophy.

We evaluated 8 men and 10 women (aged 19 to 50 years) competitive distance runners with similar training histories. All subjects were interviewed regarding their type of

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Table I. Baseline training and body composition data

	Men (n = 8)	Women (n = 10)	p Value
Age (yr)	32.8 ± 2.9	33.0 ± 2.3	N.S.
Height (cm)	172.8 ± 2.5	164.7 ± 2.5	<0.05
Weight (kg)	64.2 ± 1.7	53.9 ± 1.6	<0.01
BSA (m^2)	1.75 ± .03	1.52 ± 0.3	<0.01
LBM (kg)	57.6 ± 1.5	43.6 ± 1.4	<0.001
Body fat (%)	10.1 ± 0.7	18.9 ± 0.6	<0.001
Miles/week	46.4 ± 2.0	46.4 ± 2.0	NS
VO_{2max}	64.3 ± 3.3	56.5 ± 2.0	0.05
VO_{2max} (LBM)	71.9 ± 3.6	69.9 ± 2.2	NS

BSA, body surface area; *LBM*, lean body mass; *VO_{2max}*, maximum oxygen consumption.

training and average miles run per week (MPW). Anthropometric measurements included height, weight, and body surface area (BSA); lean body mass (LBM) was determined by averaging the results of bioelectric impedance,[6] five skinfold equations in men, and three skinfold equations in women.[7,8] A two-dimensional guided M-mode echocardiogram was performed according to the recommendations of the American Society of Echocardiography to evaluate LVM.[9] Measurements of posterior wall thickness (PWT), interventricular septal thickness (IVST), and left ventricular internal dimension in diastole (LVIDd) were determined and averaged over several cardiac cycles. To minimize bias and maintain a constant technique, the echocardiograms were read by an observer without knowledge of each subject's gender. Calculations for LVM were done using the formula $1.04[(IVSTd + LVIDd + PWTd)^3 - LVIDd^3]$, which has been validated in necropsy studies.[10] Maximal oxygen consumption was measured (2900, SensorMedics Corp., Yorba Linda, Calif.) on a treadmill to further assess training status. Testing was performed on the same day as the echocardiogram in all except two of the cases. (However, the training did not change in the interval between the echocardiogram and the treadmill test in those subjects.)

There was no significant difference in the mean age between the men and the women runners (Table I). Anthropometric measures were significantly different in all aspects including height, weight, BSA, LBM, and percentage body fat; women in our study had a higher mean percentage of body fat, were shorter, weighed less, had lower LBM, and had a lower BSA. Although there was a difference in the maximum oxygen consumption (VO_{2max}, in ml/kg/min), there was no difference between the two groups in the amount of miles run per week or in the maximal oxygen consumption per LBM (in ml/kg LBM/min). In the echocardiographic data (Table II), the calculated nonadjusted LVM showed a trend towards significance (p = 0.08), with the men having a larger LVM then the women. There were no significant differences between men and women in LVM/BSA, LVM/LBM, PWT, IVST, LVIDd, or LVIDs. Fig. 1 demonstrates that the LVM in trained women does not differ from that of the men, but rather follows the same relationship of LVM to LBM.

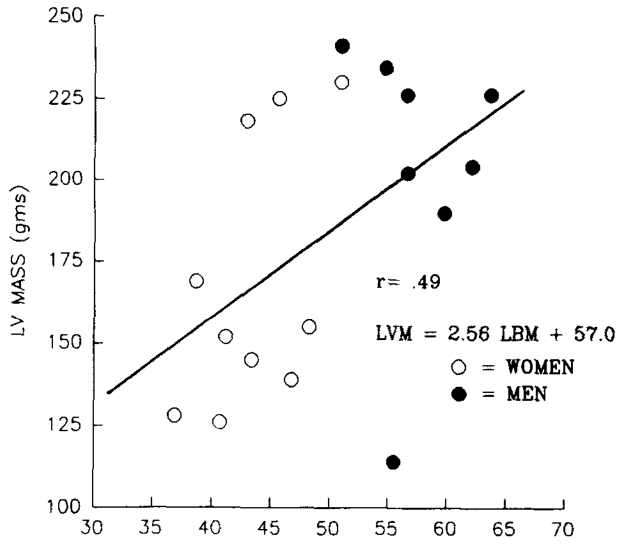

Fig. 1. Regression line for relation of left ventricular mass *(LVM)* to lean body mass *(LBM)* in men and women runners.

Table II. Echocardiographic data

	Men (n = 8)	Women (n = 10)	p Value
LVM	204.6 ± 14.3	168.8 ± 12.8	.08
LVM/BSA (gm/m^2)	117.4 ± 9.3	106.4 ± 7.1	NS
LVM/LBM (gm/kg)	3.57 ± 0.29	3.88 ± 0.25	NS
LVM/HGT (gm/m)	118 ± 9	102 ± 7	NS
PWT (mm)	8.4 ± 0.7	7.6 ± 0.5	NS
IVST (mm)	8.5 ± 0.6	8.0 ± 0.5	NS
LVIDd (mm)	52.8 ± 0.9	50.5 ± 0.8	NS
LVIDs (mm)	34.6 ± 0.7	32.0 ± 1.3	NS
IVSTi (mm/BSA)	4.9 ± 0.3	5.0 ± 0.3	NS
PWTi (mm/BSA)	4.8 ± 0.4	4.8 ± 0.3	NS

LVM, Left ventricular mass; *HGT*, height; *IVST*, interventricular septal thickness; *PWT*, posterior wall thickness; *LVIDd*, left ventricular internal dimension in diastole; *LVIDs*, left ventricular internal dimension in systole; *IVSTi*, interventricular septal thickness index; *PWTi*, posterior wall thickness index; other abbreviations as in Table I.

The results of our study demonstrate no significant difference in LVM in the men and women runners. There was no difference in the amount of miles run per week or in the maximal oxygen consumption per LBM between the men and women runners, suggesting that we had two groups that were equally trained. Echocardiographic data revealed only a trend toward the men having a larger LVM than the women. When indexed by BSA or LBM, this trend disappeared. This is consistent with data from Deveraux et al.,[10] where in a study of normal subjects (without specific reference to exercise status) they demonstrated that indexing for LBM helped to equalize the difference in LVM between men and women.

Our results contrast with those of Mumford et al.,[4] who reported that LVM index was significantly higher in male runners compared with female runners. However, in the study performed by Mumford et al. there was no attempt to control for training status between the two groups. We were able to compare two groups with equal training status, as determined by average miles run per week and by VO_{2max} testing. In conclusion, gender does not play a significant role in the exercise-related echocardiographically determined left ventricular hypertrophy seen in habitual long distance runners. Further study is needed to determine the effect of gender on other sports, particularly sports involving different types of exercise (i.e., isometric).

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Right coronary artery-to-left ventricle fistula identified by transesophageal echocardiography

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Right coronary artery-to-left ventricle fistula is a very rare clinical manifestation.[1] The presence of the fistula is usually identified by angiography. Noninvasive diagnosis of coronary fistula with conventional two-dimensional echocardiography, pulsed Doppler ultrasound, and color flow imaging has been previously described.[2,3] Herein we report a case with the fistula coming from the right coronary artery to the left ventricle; before cardiac catheterization, anatomic data regarding the origin, course, and drainage site of the fistula could be correctly obtained only by transesophageal echocardiography. This case demonstrates the usefulness of transesophageal echocardiography for the precise evaluation of coronary artery fistula.

A 31-year-old man was referred to our hospital because of prolonged fever of unknown etiology. He had been healthy until 6 weeks before admission when he underwent a dental procedure for toothache, and intermittent fever ensued thereafter. The fever was occasionally associated with a sensation of chilliness and a temporal headache, but it did not disturb his daily activities. On admission, the body temperature was 38.4° C, the blood pressure was 126/70 mm Hg, and the pulse rate was 100/min and regular. Physical examination revealed a well-developed young man with essentially negative findings except an elevated body temperature and a rapid heart rate. The first and second heart sounds were normal and there was no cardiac murmur. There was no skin sign of embolic phenomena and no clubbing of the digits. Blood examination showed mild anemia and an elevated erythrocyte sedimentation rate, as well as C-reactive protein; it was otherwise within normal limits. Electrocardiography revealed left ventricular hypertrophy by voltage criteria. The chest x-ray film demonstrated a mildly enlarged cardiac silhouette and clear lung fields. A blood culture was positive for *Streptococcus viridans* in five of six sets of blood samples. The patient was then placed on a regimen of intravenous penicillin therapy with the clinical impression being infective endocarditis. Two-dimensional echocardiography showed a mildly enlarged left ventricle with normal function and no evidence of valvular pathology. Color flow imaging showed an abnormal mosaic jet arising from the posterior wall of the left ventricle at the junction of the posterior mitral leaflet (Fig. 1, *left panel*). The turbulent flow pattern was found only during diastole by pulsed Doppler echocardiography (Fig. 1, *right panel*). However, despite approaches from multiple views, transthoracic examination failed to define the origin and the precise course of the turbulent flow. Transesophageal echocardiography was subsequently performed with a 5 MHz transesophageal imaging transducer (Hewlett-Packard Co., Andover, Mass.). It clearly demonstrated a markedly dilated and tortuous right coronary artery that coursed along the right atrioventricular groove (Fig. 2, *upper panels*). Color flow imaging from multiple modified views further showed that the dilated coronary artery was filled with diastolic flow, ran through the posterior atrioventricular groove (Fig. 2, *left lower panel*), and drained into the posterior aspect of the left ventricle (Fig. 2, *right lower panel*). Thus a right coronary artery-to-left ventricle fistula was diagnosed after the transesophageal examination. After a course of antibiotic therapy for 4