References


Alterations in Ventricular Mass and Performance Induced by Exercise Training in Man Evaluated by Echocardiography

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SUMMARY Few data are available regarding the effects of exercise training upon cardiac structure and performance in man. We evaluated the echograms of 24 normals before (PRE) and after (POST) 11 weeks of endurance exercise training. Conditioning consisted of a walk-jog-run protocol at 70% maximal heart rate for one hour four days per week. Training reduced heart rate and increased maximal duration and estimated oxygen consumption of treadmill exercise. Compared to PRE, the echogram in the POST training period revealed an increased left ventricular (LV) end-diastolic dimension (EdD), a decreased end-systolic dimension (EsD) and thus an increased stroke volume (EdD - EsD) and shortening fraction (EdD - EsD)/EdD). Cardiac output (CO) and peripheral vascular resistance (BP/CO x 80) were identical PRE and POST conditioning. Importantly, an increase in mean fiber shortening velocity was observed POST training as were increases in LV wall thickness, ECG voltage of S in V1 + R in V5, and LV mass. Thus endurance training was accompanied by increases in both LV dimension and mass as well as LV shortening fraction and contraction velocity as observed by echocardiogram.

THE POSSIBILITY THAT PHYSICAL CONDITIONING might favorably affect the appearance and course of coronary artery disease has stimulated a resurgence of interest in exercise training programs in recent years.1–8 However, the effects of exercise training upon intrinsic ventricular structure and performance in man remain uncertain. Thus, although nearly all investigations have demonstrated the development of ventricular hypertrophy in animals following a program of strenuous physical conditioning,9–11 no data exist concerning the influence of similar exertional activities upon cardiac muscle mass in humans. Moreover, despite the constant observation of a reduced heart rate at rest and submaximal work loads and an increased maximal oxygen consumption in normal subjects who have completed an exercise conditioning program,12–14 it remains unresolved whether these beneficial effects are the result of enhanced cardiac performance, or of improved oxygen delivery and enhanced oxygen utilization by the peripheral circulation and musculoskeletal system.15, 16

The paucity of precise data regarding the effects of physical conditioning upon intrinsic cardiac function has been largely related to the necessity of utilizing invasive procedures to obtain reliable measurements of the anatomy and performance of the heart. The recent advent of echocardiography has provided an atraumatic technique which has been demonstrated to be capable of reliably evaluating several aspects of cardiac anatomy and performance. Echocardiography provides accurate measurements of left ventricular cavity size, wall thickness and wall motion.17–20 The present study was undertaken to assess by means of echocardiography the influence of exercise training on cardiac structure and function in normal human subjects.

Methods and Materials

Twenty-six normal subjects participating in a program of endurance physical conditioning as part of the curriculum of the Sacramento Policy Academy volunteered for the present study. The group was comprised of 15 males and 11 females
ranging in age from 20 to 34 years (mean age 26 years) without clinical evidence of cardiac disease.

The protocol for this study entailed performance of a physical examination, an electrocardiogram, an echocardiogram and a maximal treadmill exercise test prior to and following an eleven week program of endurance physical conditioning. The training sessions consisted of a walk-jog-run program designed to maintain the heart rate at 70% of exercise-determined maximum for a one hour period four days per week. In addition, the subjects performed flexibility calisthenics for one hour on another day, and participated in daily self-defense maneuvers. The self-defense maneuvers consisted primarily of exercise in the martial arts involving coordination and body control, although some drills requiring isometric exertion of one participant against another were utilized. All training sessions were supervised by faculty of the Police Academy.

Resting electrocardiograms (ECG) were performed with careful attention to ascertain consistency of precordial electrode positioning. ECGs were analyzed in the standard fashion and, in addition, the total voltage of the S wave in lead V1 and the R wave in lead V6 (SV1 + RV6) was measured in both the pre and post conditioning state. Maximal treadmill exercise testing was performed using a commercially available treadmill (Quinton Instruments, Model 18-49C) according to the protocol proposed by Bruce and associates. Maximal exertion was assured in all patients by terminating exercise only when the subject expressed an inability to continue due to fatigue or shortness of breath. Measurements obtained during exercise testing consisted of maximal heart rate, heart rate after six minutes of exercise, blood pressure by cuff sphygmomanometer, and duration of treadmill exertion. An estimate of maximal oxygen consumption was calculated based upon the relation of duration of exercise to oxygen consumption previously demonstrated by Bruce and co-workers as: 

\[ VO_2 \text{ max} = \left( 3.88 + (0.056 \times \text{duration in seconds}) \right) \times \text{VO}_2 \text{ max} \text{ for males} = 1.06 + (0.056 \times \text{duration in seconds}) \]

Echocardiography was performed in the supine position with a commercially available echograph (Ekoline 20A, Smith-Kline Instruments), and the ultrasound tracing was displayed and recorded on a multichannel oscilloscope recorder (Model DR-8 Electronics for Medicine). Considerable care was taken to obtain pre and post echographic records from the same interspace with identical transducer angulation. Simultaneous electrocardiography provided a measurement of heart rate. Mean blood pressure was calculated as the sum of one-third of the pulse pressure and diastolic pressure. The following measurements were made from the echographic record as demonstrated in figure 1. Left ventricular (LV) end-diastolic (EdD) and end-systolic (EsD) dimensions, measured from the endocardial surfaces of the interventricular septum (IVS) and posterobasal left ventricular wall (LVPW) 40 msec after the onset of the QRS complex, and at the point of maximal approximation of these structures, respectively; left ventricular wall thickness (WT) as half of the sum of IVS thickness (measured 40 msec after the onset of the QRS complex as the distance from the first echo signal obtained from the right ventricular surface of the septum to the initial echo of the left septal surface), and LVPW thickness (measured 40 msec after the onset of the QRS complex as the distance from the first echo signal obtained from the endocardial surface of the posterior wall to the origin of the epicardial echo obtained during electrical damping of the ultrasound signal during diastole), 

\[ WTd = (WT_{PW} + WT_{IVS})/2; \text{amplitude of IVS and LVPW systolic contraction as the distance from the position of the left ventricular endocardial surface of the structure at end diastole to its location at peak posterior or anterior movement, respectively; mean IVS and LVPW velocity of contraction as the amplitude of motion of the endocardial surface of these walls divided by systolic ejection time (ET) measured as the interval from end diastole to peak posterior wall motion less 50 msec for isovolumic systole.} \]

All echocardiographic measurements were obtained in the following manner: the pre and post training echograms of each individual were examined and comparable areas of the left ventricle were marked from the available apex to base ultrasound scans; the echograms were measured at a later date in a random order without knowing either the identity of the patient or his conditioning status at the time of ultrasound examination. All ultrasound measurements were obtained during three cardiac cycles by two observers, were corrected for echo scale according to 1 cm calibration markers, and were expressed to the nearest millimeter. In addition, in order to ascertain the variability in measurements between the observers and from a single individual at different times, an echogram was measured for LV size and wall thickness for each patient by both observers on two separate days. The results of these measurements were then analyzed in terms of linear regression and standard error of the estimate for both inter and inr observer comparison.

Previous studies have shown that in the normal heart the cube function of the left ventricular EdD and EsD closely correlates with the respective ventricular volumes.
Therefore the following values were derived from echocardiographic measurement: end-diastolic and end-systolic volumes as the cube of the respective dimensions; stroke volume as the difference between end-diastolic and end-systolic LV volumes; and cardiac output as the product of stroke volume and heart rate. In addition, echographic shortening fraction (SF) was calculated as the difference between end-diastolic and end-systolic left ventricular dimensions normalized for end-diastolic dimension, 

\[
SF = \frac{(EdD - EsD)}{EdD} \times 100
\]

and an index of left ventricular mass (LVM) was obtained as a modification of the method of Troy and associates as the product of 1.05 and the difference of EdD and diastolic wall thickness cubed and EdD cubed, 

\[
LVM = [(EdD + WTd)^3 - (EdD)^3] \times 100
\]

with the caveat that the submaximal test was not performed. 

Statistical differences between data obtained prior to and following exercise training were evaluated by means of Student's t-test for paired data. 

Results

With the exception of a single individual who incurred an orthopedic injury unrelated to the conditioning protocol and was excluded from the study, all subjects successfully completed the 11 week training program. One additional subject was omitted from analysis due to extreme apprehension with the occurrence of hypertension and tachycardia observed on the physical examination following the conditioning period. No evidence of cardiac disease was elicited by history and physical examination, resting electrocardiogram, or maximal treadmill exercise test in any of the remaining 24 subjects included in this study. Echocardiograms of adequate technical quality to obtain measurement of all aspects of cardiac structure and function were obtained in each individual. Results in female participants were not significantly different from those observed in males; therefore, data for both groups have been analyzed together. Linear regression analysis carried out upon measurements from two observers and from an individual observer on separate days revealed an excellent correlation with coefficient of greater than .90 for both variables. Thereby, utilizing the methodology employed in the present study, excellent reproducibility of echographic measurements was observed.

Quantification of Training Effect

Unequivocal evidence of a training effect was observed following the physical conditioning program. Thus, as compared to the control period, resting heart rate decreased for the group of subjects from 69.3 ± 2.7 to 62.9 ± 2.9 beats/min (mean and standard error of the mean), \( P < 0.005 \); maximal duration of treadmill exercise increased from 9.78 ± 0.45 to 11.28 ± 0.35 min, \( P < 0.001 \); estimated maximal oxygen consumption increased from 35.5 ± 1.7 to 40.6 ± 1.3 cc/kg/min, \( P < 0.001 \); and heart rate at the submaximal work level achieved after six minutes of exercise decreased from 164 ± 4.1 to 147 ± 4.1 beats/min \((P < 0.001)\) post exercise training (fig. 2). Physical conditioning did not produce an alteration in blood pressure: 120.2 ± 2.8 to 119.9 ± 1.9 systolic, 73.8 ± 2.1 to 70.8 ± 1.9 diastolic, and 89.2 ± 2.0 to 87.7 ± 1.6 mm Hg mean (fig. 3), although a decrease in maximal heart rate-blood pressure product occurred, 36859 ± 920 to 33802 ± 755 \((P < 0.005)\). In addition, no change in body surface area occurred during the training period, 1.89 ± 0.18 m² before and 1.88 ± 0.17 m² after completion of the program.

Cardiac Structure

The results of echocardiographic studies indicated that significant alterations in cardiac anatomy were induced by
physical conditioning. Thus ultrasound left ventricular end-diastolic dimension increased from 4.8 ± 1 to 5.0 ± 1 cm, \( P < 0.005 \); while end-systolic dimension decreased from 3.0 ± 1 to 2.9 ± 1 cm, \( P < 0.05 \) following exercise training (fig. 4). A greater left ventricular wall thickness was observed following exercise training (9.1 ± 3 before to 10.1 ± 2 mm after the program, \( P < 0.001 \)). Thus, calculated left ventricular mass was found to be augmented from 80.6 ± 4.5 to 97.2 ± 5.8 g following conditioning, \( P < 0.001 \) (fig. 5). Although certain subjects exhibited post exercise training values for cardiac structure which were disparate from those of the group as a whole, no distinctive features could be identified in these individuals. Therefore, we interpreted these findings as representing the effects of additional physiological variables present in the pre and post conditioning periods as well as minor variations in measurements related to limitations in the technique and methodology applied. Consistent with the echographic evidence of an increase in LVWTd was the observation of an increase in total voltage in SV\(_1\) + RV\(_1\) on ECG from 24.9 ± 1.4 mV in the control period to 30.7 ± 1.8 mV post training (\( P < 0.001 \)).

**Cardiac Function**

The changes observed in LV diastolic dimensions accounted for an increase of derived left ventricular end-diastolic volume and a decrease in end-systolic volume in the post conditioning period: 113.9 ± 7.7 to 125.2 ± 7.4 cc, \( P < 0.01 \) and 28.5 ± 2.5 to 25.6 ± 2.1 cc, \( P < 0.05 \), respectively.

**Discussion**

The respective roles of enhanced intrinsic cardiac function and increased peripheral oxygen utilization in the generation of the greater exertional capacity which invariably accompanies the completion of a physical conditioning...
program remain unresolved. Thus, despite the evidence of augmented ventricular performance after training provided by some investigators, including a greater velocity of aortic pressure rise and ejection rate, as well as elevation of the rate of right ventricular pressure development, other workers have found that the increase in maximal oxygen consumption following exercise training could best be related either to alterations in the regulation of the peripheral circulation or to a greater arteriovenous oxygen difference in exercising skeletal muscle. Adding to the controversy is that no consistent findings have been observed regarding the influence of physical conditioning upon systolic time intervals. It is likely that the variable results reported in previous studies are largely related to differences in the age and physiologic status of the subjects evaluated and the design of the protocol. The present investigation, utilizing echocardiography, demonstrates the ability of a controlled program of exercise training in a homogeneous group of normal individuals to produce favorable effects upon left ventricular structure and performance at rest.

It was not possible in this study to perform direct analysis of respired gases in order to obtain precise measurements of maximal oxygen consumption. Therefore, an estimate of maximal oxygen uptake was obtained from treadmill duration by the method of Bruce and associates. Previous studies have demonstrated that maximal oxygen consumption can be only grossly estimated by means of the length of the exercise period, and that a greater treadmill duration may actually accompany a decrease in oxygen uptake in some subjects. However, the uniformity and magnitude of the increase in treadmill duration observed in the present investigation renders it almost certain that the augmented exercise period represented a true increase in maximal oxygen consumption.

Although inclusion in this project was voluntary and did not influence the successful completion of the Police Academy curriculum, it is likely that the individuals who participated were highly motivated. Substantial decreases in resting and submaximal heart rate and increases in maximal oxygen consumption were achieved during this training period, despite the fact that the group was comprised of active young adults who could not be considered completely sedentary. Since the female cohort in this study engaged in less physical activity and exhibited a constellation of findings compatible with a lower level of physical fitness prior to conditioning, and also manifested a greater improvement in maximal exertional capacity following reconditioning (fig. 2), it is probable that training would have induced greater effects upon exertional capacity if a more sedentary group of subjects had been selected for participation.

Using echocardiography, we were able to evaluate directly the influence of exercise training upon left ventricular intracavitary size, wall thickness, and contractile pattern. Although the ultrasound beam may traverse a limited area of myocardium which may not correlate closely with actual...
ventricular volumes in the presence of advanced cardiac disease, this limitation was minimized in our protocol by virtue of the fact that the study group was comprised only of normal young subjects in whom technically high quality echograms could be recorded. In addition, in an inpatient study such as the present investigation, evaluation of directional changes remain valid since such subjects serve as their own control.

Precise measurements of cardiac structures are limited by current ultrasonic techniques. Thus, in comparing measurements of an echogram by two investigators, variations of greater than 1 mm were occasionally encountered; however, differences between the two observers were constant. When data were analyzed for interobserver variability and for differences in repeated measurements by the same individual at different times, the correlation between values was excellent. This close correlation was aided by the restriction upon the number of echographic complexes which could be measured, imposed by the necessity to compare identical ultrasonic sectors before and after conditioning. Although the changes in echographic measurements in the present investigation are small, we believe the alterations detected represent a true reflection of the cardiovascular changes induced by exercise training.

All estimates of systolic ejection time were obtained as the interval from end diastole to peak posterior wall motion less 50 msec for isovolumic systole. We recognize that systolic ejection time measured in this manner may not be entirely accurate in defining the actual ejection period. However, previous studies have demonstrated the validity of systolic ejection time obtained by echocardiography in this manner compared to carotid pulse tracings and angiography.

We considered the possibility that the decrease in heart rate which occurred post conditioning contributed substantially to the increase in left ventricular internal size in this period (fig. 4). However, in a previous study, Hirshleifer and co-workers were able to demonstrate only a 2 mm decrease in LV dimension accompanying a change in heart rate of 34 beats per minute induced by atropine, an identical change in endocardial dimension to that observed in the present study in which only a 6 beat per minute alteration in heart rate occurred. Thus, it is extremely unlikely that pulse rate alone accounted for the increase in LV dimension observed in this study. The increase in ventricular size is consistent with reported increases in total heart volume measured by chest roentgenogram following conditioning in young normal subjects. However, a similar increase in heart size was not detected in patients with coronary artery disease, in middle-aged and older normal men, or in dogs following endurance physical conditioning. Thus, it is likely that the influence of exercise training upon ventricular dimensions is largely dependent upon the age and physiological status of the subjects involved. Further, it is understandable that the relatively small changes in LV cavity size induced by training could easily go undetected by techniques other than ultrasound.

A major finding in the present study was that of a small but significant increase in left ventricular muscle mass following exercise training (fig. 4). Thus, a slight increase in left ventricular wall thickness was documented by direct measurements obtained with echocardiography, and by indirect assessment performed by electrocardiography. In addition, left ventricular mass derived from echographic measurements was significantly increased in the post-conditioning period. These data are in agreement with the post-exercise training observations of an increased ratio of heart weight to total body weight in rats, and an augmentation of left ventricular wall thickness and mass demonstrated radiographically by the bead and clip technique as well as by the spatial magnitude of the QRS complex of the ECG in dogs, and an increase in maximum spatial QRS magnitude in man. Further, the results of previous studies demonstrating that myocardial hypertrophy induced by exercise is associated with an increased actomyosin ATPase activity in contrast to the decrease in the activity of this enzyme recorded with hypertrophy due to hemodynamic overload, suggest that the increment in ventricular mass observed after the training program may enhance cardiac function.

Two previous echographic studies have reported substantially greater left ventricular dimensions in endurance athletes compared to those of normal controls. However, it could not be ascertained from these studies whether the LV enlargement in athletes was due to physical training or, conversely, if the propensity to athletic activity was secondary to the genetic predisposition to an enlarged heart. In previous studies the echograms of endurance athletes manifested a greater left ventricular internal dimension while competitors involved in isometric exercise such as wrestlers exhibited an increased LV wall thickness compared to normals. However, posterobasal and interventricular septal wall thickness, which were evaluated independently in previous work, actually revealed a combined mean increase of 1 mm in endurance runners as compared to controls. Further, all athletes demonstrated a significant elevation of left ventricular mass compared to their sedentary counterparts. In the present study, small increases of left ventricular dimension, wall thickness, and mass were observed following conditioning. Thus, the data from this investigation support the concept that endurance, or moderate intensity-long duration isometric exercise is capable of producing increases in both left ventricular internal size and wall thickness. Since the changes in LV dimension before and after exercise training in this study were considerably smaller than the difference observed between athletes and controls in previous reports, an important subject for further investigation is to determine if endurance training alone is capable of inducing the marked ventricular enlargement seen in champion athletes.

Exercise training also resulted in an increase in LV shortening fraction and calculated stroke volume indicative of slightly enhanced cardiac performance. However, other parameters of hemodynamic function including cardiac output, blood pressure, and peripheral vascular resistance were not altered following conditioning (fig. 3). Thus, compared to the control state, circulatory dynamics in the post training period were characterized by a maintenance of identical peripheral blood flow at a reduced heart rate due to an increment in stroke volume accomplished by an augmented LV diastolic volume and shortening fraction. The absence of changes in peripheral vascular resistance observed in the present study is in agreement with those of prior investiga-
tions, 12, 16, 28, 31 These data indicate that any benefit conferred on resting hemodynamic function by alterations in peripheral circulation would have to be related to changes in the distribution of flow to the various vascular beds as has been proposed by Clausen and co-workers. 1, 28

Although previous studies using isolated cat papillary muscle have indicated that preload has an effect upon the velocity of myocardial contraction, measurements of velocity of circumferential fiber shortening obtained from the intact ventricle in man by echocardiography have been demonstrated to provide an index of myocardial contractility which is dependent upon ventricular afterload but only minimally influenced by acute changes in preload. 44, 45

Therefore, although the alteration in preload described herein appears to be of a chronic nature, the fact that no decrease in afterload was recorded in the present study suggests that the modest improvement in VCF observed following training may represent an increase in left ventricular contractile state; a finding consistent with an increase in peak LV dp/dt under constant loading conditioning observed in conditioned rat hearts. 46

The data from the present investigation must be viewed in proper perspective in regard to the biologic significance of the alterations following exercise training. Our data were obtained in healthy young adults at rest and may not apply to middle age or older sedentary adults or patients with coronary heart disease. Although statistically significant changes in cardiac structure and function were induced by conditioning, the magnitude of these alterations was relatively modest and individual values of wall thickness and left ventricular dimension remained within the normal range. Therefore, although these data clearly demonstrate that exercise training is capable of inducing definite alterations in cardiac structure and function, the precise biologic significance of these changes cannot be determined at present.

Acknowledgment

We wish to thank Dr. Juan Angel and Ms. Lynn Weinert for their invaluable technical assistance, Mr. Donald Farnsworth and the Cadets and Staff of the Sacramento Police Academy without whose cooperation this study could not have been performed, Mrs. Leigh Segal for her helpful review of the manuscript, and Betty Paro for her secretarial assistance.

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Two-dimensional Echocardiographic Recognition of Ruptured Chordae Tendineae

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SUMMARY Real-time, phased-array, two-dimensional echocardiographic studies identified ruptured chordae tendineae in five patients: four patients had a flail mitral valve and one had flail mitral and tricuspid valves. The characteristic abnormality was a rapid systolic motion of the involved leaflet beyond the line of valve closure into the atrium. The maximal abnormal systolic motion was greatest at the tip of the leaflet with a loss of the normal coaptation point. By contrast, the two-dimensional echocardiographic feature of mitral valve prolapse is an abnormal systolic motion that is maximal in the body of the leaflet with intact leaflet coaptation. Thus, two-dimensional echocardiography can identify flail mitral and tricuspid valves and is useful in distinguishing ruptured chordae from valvular prolapse.

M-MODE ECHOCARDIOGRAPHY may be helpful in the diagnosis of mitral regurgitation secondary to ruptured chordae tendineae. Several investigators have described echocardiographic findings for the diagnosis of ruptured chordae tendineae. These include systolic intracavitary left atrial echoes, holosystolic mitral valve prolapse, systolic mitral leaflet fluttering, chaotic diastolic anterior motion of a flail posterior mitral valve leaflet, or coarse diastolic fluttering of a flail anterior mitral valve leaflet. Because of the limited number of patients reported in the literature, the sensitivity and specificity of these findings have not been conclusively determined. Thus, the preoperative diagnosis of a flail mitral valve is often based on hemodynamic and angiographic findings. Furthermore, criteria for the M-mode diagnosis of flail tricuspid leaflets have not been described.

Real-time, two-dimensional echocardiography can provide spatial information concerning intracardiac structures that cannot be obtained by M-mode echocardiography. The purpose of this report is twofold: 1) to demonstrate the ability of real-time, two-dimensional echocardiography to visualize flail mitral and tricuspid valves secondary to ruptured chordae tendineae and 2) to demonstrate that two-dimensional echocardiography may be more sensitive than M-mode echocardiography in diagnosing ruptured chordae tendineae.

Material and Methods

M-mode and two-dimensional echocardiograms were recorded in five patients in whom the surgical findings later confirmed the diagnosis of flail mitral or tricuspid valves. Four patients had flail mitral valves and one patient had flail mitral and tricuspid valves. Clinical data based on symptoms, physical findings, electrocardiograms, and chest X-rays were analyzed. The clinical, echocardiographic, angiographic, and anatomic data on these five patients form the basis of this report.

M-mode echocardiographic studies were performed in the supine or left lateral decubitus position utilizing a Smith Kline Ekie 20A ultrasonoscope with a 2.25 MHz medium (7.5 cm) internally focused transducer. Permanent records were obtained with an Irex 101 Continutrace Recorder. Slow and rapid full M-mode echocardiographic sweeps from the aortic valve leaflets to below the level of the mitral valve leaflets were performed in each patient. Careful attention was paid to transducer position and gain setting to avoid missing small valvular or intra-atrial cavitary abnormalities. Large scale mitral valve echograms were recorded through several cardiac cycles to magnify the details of mitral valve motion. Similar attention was directed to assessing tricuspid valve motion.

Two-dimensional echocardiographic studies were performed using a Grumman Health Systems RT-400 phased-array sector scanner. Serial excitation of the 32 transducer elements produces an acoustic wave which is directed toward the target. The hand-held focusable linear array generates an 80° field in a circular sector format to produce tomographic images of the heart in real time. A digital computer controls the scan format. The images are displayed on a television screen and, via an optical link, permanent records of images are made on one-half inch video tape for later playback and analysis. A simultaneously recorded electrocardiogram provided a time reference within the cardiac cycle specifically to identify the mid and late diastolic and systolic frames shown in the illustrations. Illustrations presented in this manuscript were photographed from single
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Circulation. 1978;57:237-244
doi: 10.1161/01.CIR.57.2.237

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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