STRUCTURE—FUNCTION CORRELATIONS IN CARDIOVASCULAR AND PULMONARY DISEASES (CPC)

Running to Death*

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Herein we will discuss findings in a conditioned runner who died suddenly, shortly after completing a 5-km run.

A 51-year-old man had been healthy until his sudden death in March 1980. In 1975 (aged 46 years), he started jogging and quickly began averaging about 19 km (12 mi) weekly (4.8 km four times a week). At that time, his blood pressure was 140/95 mm Hg, and he began taking a diuretic daily. The total serum cholesterol was 273 mg/dl. In 1976, he began lifting weights and doing other types of calisthenics regularly. The total serum cholesterol level in 1978 was 250 mg/dl, and his systemic arterial pressure was 130/80 mm Hg. In November 1979 (four months before death), he noted “tiredness in one arm” while running, and went to a hospital emergency room, where the ECG shown in Figure 1 was recorded. The heart rate was 55 beats/min, and the blood pressure 120/80 mm Hg. A chest roentgenogram (Fig 2) was normal. He continued to run 19 km weekly thereafter without chest pain or “arm tiredness.” He collapsed and died in his bathroom while preparing to shower shortly after completing a 5 AM, 5-km run. Clinical manifestations of cardiac disease were absent in other family members.

At necropsy the heart weighed 390 g. Transverse ventricular sections showed a healed transmural myocardial infarct extending from base to apex and involving primarily the ventricular septum (Fig 3). The left anterior descending and left circumflex coronary arteries were narrowed 76 to 100 percent in cross-sectional area by atherosclerotic plaques (Fig 4). Of 53 five-mm coronary segments of the left main, left anterior descending, left circumflex, and right coronary arteries, 9 (17 percent) on histologic examination were narrowed 76 to 100 percent in cross-sectional area by atherosclerotic plaques, including one segment narrowed

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**Figure 1.** ECG recorded four months before death. Absence of R wave in V2 and nonspecific ST-T segment and T waves changes.

**Figure 2.** Posteroanterior chest roentgenogram obtained four months before death.
95 to 100 percent (Fig 5); 15 (28 percent) were narrowed 51 to 75 percent; 25 (47 percent), 26 to 50 percent; and 4 (8 percent), 0 to 25 percent. Thus, 34 (64 percent) of the 53 five-mm segments were narrowed >50 percent in cross-sectional area by plaques. By assigning a number for the category of narrowing for each 5-mm coronary segment (4 = 76 to 100 percent narrowing; 3 = 51 to 75 percent; 2 = 26 to 50 percent, and 1 = 0 to 25 percent), a total score of 135 was found for the 53 five-mm segments, giving each segment a mean score of 2.5 (135 ÷ 53). Thus, the lumen of each 5-mm coronary segment was narrowed on the average about 63 percent in cross-sectional area by atherosclerotic plaques. None of the coronary arteries contained fibrin or platelet thrombus or extravasated erythrocytes into atherosclerotic plaques.

Running appears to be the most efficient regular exercise, and it is estimated to be done regularly by about 25 million Americans. Few conditioned runners smoke cigarettes, are overweight, or go to psychiatrists. Nearly all “feel better” and perform their tasks better than they did before they began running regularly. Although unproved, running is believed to dilate previously normal or relatively normal coronary arteries (one of the means to increase the amount of blood perfusing myocardium is to dilate the coronary arteries), but it is uncertain whether dilation is possible in runners with
previous coronary events, ie, patients with known coronary atherosclerotic narrowing. Although reason dictates that it must, regular running has not been shown to increase longevity compared with not running.

Recently we reported cardiac findings in five men, aged 40 to 53 years (average, 46 years) who ran 22 to 176 km weekly (mean, 53 km) for one to ten years (mean, five years).1 None had had clinical evidence of cardiac disease before they became habitual runners, and all died while running. After running regularly for several years, one showed an abnormal resting ECG and a positive exercise stress test; he also had angina pectoris while running. At necropsy all five had 76 to 100 percent cross-sectional area narrowing by atherosclerotic plaques of the right, left anterior descending, and left circumflex coronary arteries. In three, who had the entire lengths of these three arteries plus the left main coronary artery available for examination, 73 (48 percent) of the 153 five-mm segments were narrowed 76 to 100 percent in cross-sectional area by atherosclerotic plaques.

In comparison with the previously described five runners,1 the present patient had less severe coronary narrowing; only 17 percent of the 5-mm segments of his four major arteries were narrowed 76 to 100 percent, compared with 34, 50, and 56 percent, respectively, in the three previously described runners studied quantitatively. Although severe, the amount of narrowing in our patient is not as great (range, 30 to 48 percent) as that usually observed in patients with fatal coronary heart disease.2-4 The present patient also had hypercholesterolemia and systemic hypertension. He had not started to run regularly until aged 46 years, and died at the age of 51. He probably had considerable coronary atherosclerosis by the time he first began to run regularly. Bassler5 stated that marathon running provides "immunity to atherosclerosis." Although the present patient was not a marathon runner, three of our five previously described runners were marathoners; none began running regularly, however, until aged 39 years, and all died by the age of 50.1 It is likely, therefore, that they also had considerable coronary atherosclerosis before they became conditioned runners.

Our patient had a relatively large transmural left ventricular scar. Four of the five runners previously described by Waller and Roberts1 also had transmural left ventricular scars, all of which were clinically silent events, as in our patient. Whether acute myocardial infarcts are more commonly clinically inapparent in conditioned runners compared with nonrunners is uncertain.

Several reports have described sudden death in runners, but few have provided the distance run per week (or per day) or the length of time that the person ran regularly.6-14 Furthermore, these reports often lack information regarding the serum cholesterol and systemic blood pressure levels and whether other family members had coronary heart disease. Despite the scarcity of reported information in reported deaths among conditioned runners,6-14 coronary atherosclerosis nevertheless appears to have been the villain in most of the previously described patients.

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