

A World Record Marathon Runner with Silent Ischemia without Coronary Atherosclerosis*

William J. Rowe, M.D.

A 62-year-old world record marathon runner was found to have silent ischemia manifested by a very abnormal stress test, whereas at autopsy nine months later, there was virtually no coronary atherosclerosis nor other disease of the coronary microvasculature. However, there was focal fibrosis of the papillary muscles consistent with remote ischemia secondary to possible CV. It is postulated that endurance-related high catecholamine levels might have been responsible. (*Chest* 1991; 99:1306-08)

CV = coronary vasospasm

In a study initiated nine months prior to death from lymphoma, a 62-year-old Chinese world record marathon runner was found to have by stress testing, ischemia with circadian variation suggesting possible CV¹ without angina. At autopsy, there was virtually no coronary atherosclerosis or other disease of the microvasculature, but there was focal fibrosis of the papillary muscles. To account for these findings, a mechanism related to high catecholamine levels was postulated.

CASE REPORT

The subject, an assistant professor of physical education at the University of Toledo, began participating in marathons at age 40, was first listed in the Guinness Sports Record Book in 1986² having at that time completed a record 374 marathon distances or greater, and two months prior to his death, had completed 524 marathons. Most were completed in less than 4 h. In addition, he competed in cross country ski and canoe races, triathlons, and ultramarathons

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Table 1—Endurance Races From 1981 Until May 1988*

	M	DNF	M (Groups of Consecutive Days)	UM Distances (Km)			Cross Country Ski Up to 50 Km	Triathlons	Canoe Races 13 Km	Injury
				50-100	120-225	490				
1981	36		3	3						
1982	38		5	3						
1983	43	2	5	10	1	1				
1984	42	2	6	5	3	1		3		
1985	43		6†	7				2	2	Struck by car‡
1986	44§		8	13				3		
1987	46		5	14				3		
1988	15		1	2			7	3		
Until May										
Total	524	(Since 1966 with last M 2 months before death)								

*M denotes marathon (42 km or greater), UM, ultramarathon (50 km or greater), DNF, did not finish.

†Includes 2 high altitude marathons on consecutive days. First day at Dillon, Colorado (2,700-3,000 m). Second day at Pike's Peak (4,300 m).

‡Injured knee. Ran M next day. Pain for 6 weeks.

§Guinness Sports Record Book, 1986—374 M.

||Includes M number 500 (Boston).

(Table 1).

Three symptom-limited maximum treadmill stress tests were performed. The first morning test was decidedly abnormal with downsloping ST segments developing during peak exercise consistent with profound myocardial ischemia³ (Fig 1,A), whereas the Holter recording (V₃) at peak exercise in the afternoon of the same day in conjunction with a high measured maximum oxygen uptake of 60.6ml/kg/min was not abnormal (Fig 1,B). However, with only a V₃ lead (Holter), there was a potential negative error of 15 to 20 percent.⁴ The 24-hour Holter recording otherwise (even during an hour training run) showed no ST segment displacement of > 1 mm in comparison with a normal resting ECG. A morning stress test six weeks later, in conjunction with normal thallium-201 imaging, was also not abnormal. M-mode and 2-D echocardiograms were normal with no evidence of left ventricular hypertrophy, significant mitral valve prolapse, or asymmetric hypertrophy. Results of chemistry profile, electrolytes, and complete blood count obtained on the day of the first stress test were also normal. Plasma catecholamines and ergonovine studies⁵ were not obtained. On the day of the first stress test, a physical examination was normal, and blood pressure was 110/75 mm Hg with no orthostatic hypotension.

At autopsy, there was a high grade malignant lymphoma with disseminated candidiasis. The heart weighed 360 g. There was ventricular dilatation but no evidence of septal or ventricular hypertrophy. A small patchy nontransmural scar was present in the posterior wall of the left ventricle. Sections of the left ventricular myocardium showed intramural fungal microabscesses with surrounding hemorrhage. There was focal fibrosis of the left papillary muscles consistent with remote ischemic insult to the myocardium (Fig 2). The coronary arteries sectioned at 5-mm intervals were widely patent with virtually no evidence of atherosclerotic coronary artery disease. There was no evidence of disease of the coronary microvasculature.

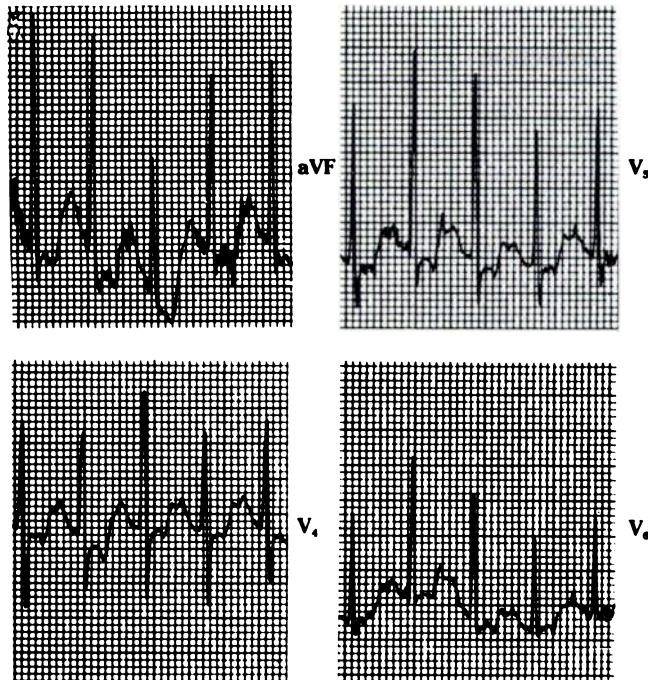
DISCUSSION

The circadian variation in the first two stress tests performed on the same day is consistent with CV¹. This is more frequently associated with exercise-induced ST segment elevation¹, but, as in this case, may be associated with ST segment depression.⁵ Local coronary vessel supersensitivity⁵ arising fortuitously could have been responsible. It should be stressed, however, that CV has been postulated to occur in patients with catecholamine cardiomyopathy secondary to pheochromocytoma,⁶ and was pos-

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Marathon Runner with Silent Ischemia (William J. Rowe)

A—MORNING



B—AFTERNOON

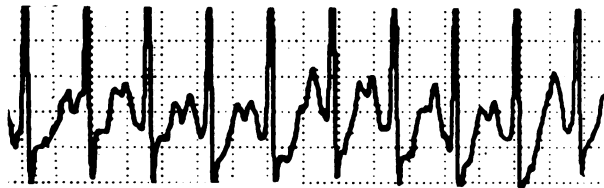


FIGURE 1 A. First morning stress test showing raw tracings because of potential motion artifact, at maximum at end of stage 5 (Bruce). Heart rate was 175 beats per minute. There is > 2 mm of J point depression with ST segments downsloping in lead aVF and 1 mm horizontal ST depression in leads V_4 and V_5 . B. modified V_5 from Holter at maximum oxygen uptake. Heart rate was 173 (afternoon of same day.) There is J point depression with slowly upsloping ST segments.

tulated to have been responsible for an epinephrine-induced myocardial infarction.⁷ Similarly, the subject's plasma catecholamine levels would be expected to have been elevated significantly, but intermittently, during his last 20 years related to his extraordinary endurance activities. Blomqvist and Saltin⁸ have emphasized that training does not reduce exercise plasma catecholamine levels when comparisons are based on relative work loads. Plasma catecholamines may increase 170 percent following a marathon and 200 percent after a 10 km race,⁹ and probably increased to an even greater percentage with the energy expenditure required for a marathon at Pike's Peak (Table 1). Although there was nothing seen that would suggest the presence of catecholamine cardiomyopathy, there was focal fibrosis of the papillary muscles (Fig 2). Steer et al¹⁰ emphasized the distinction between diffuse fibrosis of the papillary muscles associated with aging and focal fibrosis which they termed healed infarcts. Since the fungal myocarditis did not occur until several days to a few weeks prior to death, it would not have been responsible for the focal fibrosis consistent with remote ischemia. In the absence of left ventricular hypertrophy,

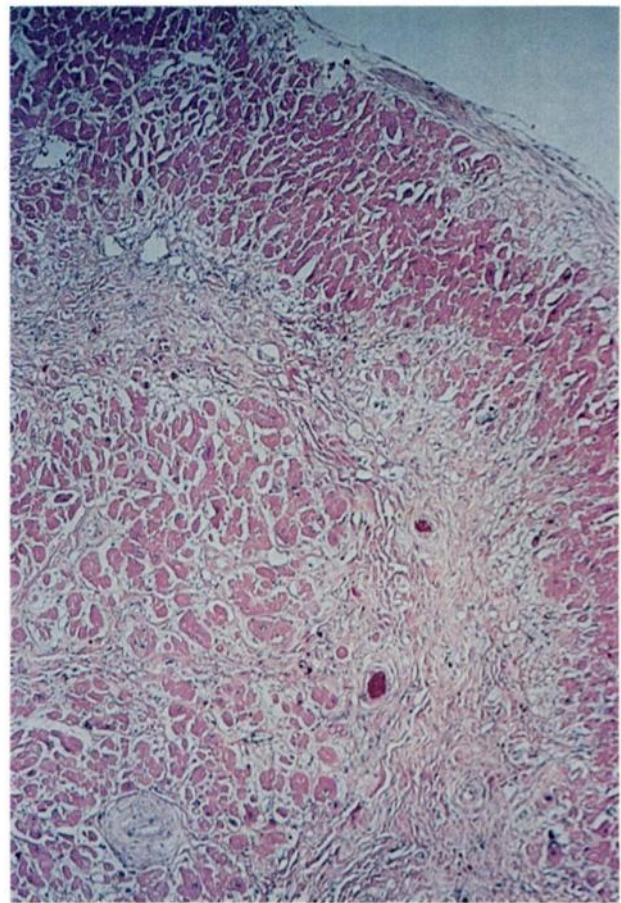


FIGURE 2. Left ventricular papillary muscle showing focal fibrosis (hematoxylin-eosin, original magnification $\times 16$.)

coronary atherosclerosis, or disease of the microvasculature, the cause of the subject's focal fibrosis is speculative. CV, as previously noted, has been postulated,^{6,7} and myocardial infarction without demonstrable coronary artery disease may occur in patients with elevated catecholamines.^{6,7} In patients with the latter condition and in this subject, a similar mechanism for a myocardial infarction or focal fibrosis may be responsible, *ie*, an imbalance between myocardial oxygen supply, reduced secondary to possible CV, and increased demand related to high catecholamine levels.

Exercise-induced CV,^{1,5} with silent ischemia, might have been the mechanism¹¹ preceding acute pulmonary edema (possibly recurrent) in two ultramarathoners with angiographically normal coronary arteries;¹² this provides an additional applications of the hypothesis described.

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Right-Sided Aortic Arch Presenting as Refractory Intraoperative and Postoperative Wheezing*

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A 29-year-old woman presented with severe refractory intraoperative wheezing and airflow limitation that resolved spontaneously. Contrast-enhanced computed tomographic (CT) scan of the thorax confirmed a right-sided aortic arch. Variable intrathoracic large airway obstruction that worsened markedly when the patient was in a supine position and slightly more following intravascular volume expansion was shown on flow-volume studies. We postulate the right-sided aortic arch caused airflow obstruction that worsened intraoperatively because of position and intravenous fluids.

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A minority of patients with right-sided aortic arch have symptoms from tracheal compression. Most reports are of children; case reports of right-sided arch causing symptoms in adults have been scarce.¹ We present herein

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the case of a 29-year-old woman in whom severe intraoperative airflow obstruction developed because of a right aortic arch; moderate symptoms, in retrospect, had been present since childhood.

CASE REPORT

A 29-year-old woman was referred for evaluation of severe wheezing and airflow obstruction that developed during administration of a general anesthetic for an orthopedic procedure. Wheezing, dyspnea, and airflow obstruction were refractory to nebulized albuterol, nebulized ipratropium, and intravenous aminophylline. These symptoms resolved gradually over several postoperative days with the patient receiving albuterol, ipratropium, aminophylline, and intravenous methylprednisolone sodium succinate (40 mg every 6 hours). At induction of anesthesia, the anesthetist reported the subjective feeling of a "square wave" inspiratory pressure curve.

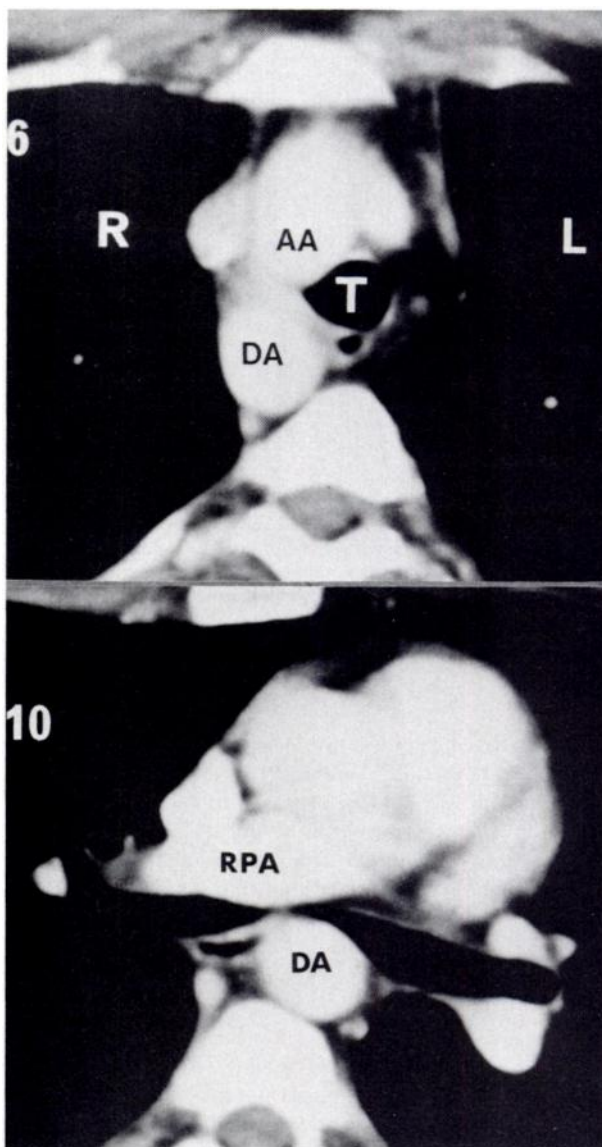


FIGURE 1. Mediastinal cuts of contrast-enhanced computed tomographic (CT) scan of chest 6 cm (1A, upper) and 10 cm (1B, lower) below sternal angle. AA = ascending aorta; DA = descending aorta; RPA = right pulmonary artery; R = right; and L = left. The trachea is compressed between the descending aorta (as it crosses the spine from right to left) and the right pulmonary artery at the level of the carina.