Myocardial Infarction After Normal Responses to Maximal Exercise

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SUMMARY

The unexpected occurrence of acute myocardial infarction shortly after performance of a multistage test of maximal exercise by a normal 42-year-old subject is described. There was no evidence of infarction after this exhausting effort, but symptoms occurred immediately on exposure to hot water while he was taking a shower a few minutes later. Cardiac arrest from ventricular fibrillation occurred after ECG evidence of acute infarction. Following successful defibrillation and coronary care, there was satisfactory recovery from massive anterior wall infarction. Incidence, possible pathophysiological mechanisms, and precautions are cited.

Additional Indexing Words: Treadmill exercise Cardiac arrest Ventricular fibrillation

A CUTE MYOCARDIAL INFARCTION in an apparently normal individual with a normal electrocardiographic response to maximal exercise testing was heretofore an event unknown to the authors. This report describes the sudden occurrence of myocardial infarction during a shower following normal performance of a maximal exercise tolerance test. Ventricular fibrillation and cardiac arrest ensued. Both before and after successful defibrillation there was convincing evidence of major infarction. Despite this, recovery was satisfactory even though some evidence of transient pericarditis and mild heart failure appeared during the first week.

Report of Case

On November 22, 1967, E.H., a 42-year-old white male educator, reported to the exercise laboratory for performance of a routine treadmill test of maximal exercise tolerance. The subject was a member of a group of men whose electrocardiographic responses to exercise had been measured periodically. Evaluation 3 years previously had revealed a normal resting electrocardiogram, no cardiac pathology, and no segmental S-T response to maximal exercise on a treadmill. An interim history revealed no illness or symptoms. In terms of coronary risk factors, he had ingested a high fat diet and smoked two packs of cigarettes daily until 40 years of age. A few months before, he had experienced stressful interpersonal relations with his family, but these were relieved by a separation. At this examination, he was a well-developed, healthy white male, who was 188 cm tall and who weighed 70.8 kg. Blood pressure was 112/70 mm Hg. Heart rate was 80 beats/min and regular. Physical examination again failed to reveal evidence of cardiac pathology. An electrocardiogram (fig. 1A), identical to that of 3 years before, was considered to be within normal limits. The subject performed a standardized maximal exercise tolerance test which involved walking to exhaustion as both treadmill speed and grade increased at 3 min intervals as reported elsewhere.1

Continuous electrocardiographic monitoring was carried out employing a Frank X, Y, and Z orthogonal lead system, as well as a standardized CB-5 bipolar chest lead. All electrocardiograms were recorded on magnetic tape. All Frank leads
were also continuously displayed on a cathode ray oscilloscope and the bipolar CB-5 lead was intermittently recorded with direct writing apparatus. Blood pressure was recorded with a mercury sphygmomanometer before exercise, at 0 recovery (moment of cessation of exercise), and at 3 and 6 min after 0 recovery (table 1).

The subject performed well, and was able to walk to 97 sec of stage IV. Actually, this was 34 sec longer than during the same test performed 3 years previously. He had no chest pain, dizziness, or nausea, but stopped because of leg aching and dyspnea, which are common reasons for stopping in normal subjects. Immediate evaluation of the direct writing CB-5 lead revealed no segmental S-T displacement (fig. 2). At no time was there a horizontal or a “downward sloping” S-T segment in the bipolar or in any of the three orthogonal Frank leads.

Following exercise, the subject walked down two flights of stairs to the shower room where he was instructed to take a lukewarm shower, specifically “not too hot or too cold,” and then to report back to the examining physician. While in the shower, he was inadvertently scalded on two occasions when the water suddenly became hot as a toilet in an adjacent room was flushed. He immediately felt more tired than during the exercise test and suddenly became nauseated, felt faint, and found it difficult to dry and dress himself. He then reported to the examining physician on the same floor, as he had been instructed to do. At this time, he appeared hyperactive, irritable, anxious, and agitated, complaining of nausea which had developed while he was in the shower. Upon specific questioning he admitted to retrosternal chest pain extending to the arms which had developed concomitantly with the nausea.

An electrocardiogram taken immediately showed sinus rhythm and altered conduction in lead I. Of the precordial leads, only V5 was obtainable because of the marked agitation of the patient.

### Table 1

<table>
<thead>
<tr>
<th>Heart Rate and Blood Pressure Before, During, and After Exercise</th>
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<tbody>
<tr>
<td><strong>Blood pressure</strong> (mm Hg)</td>
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<tr>
<td>Rest (examining room)</td>
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<td>Rest (on treadmill)</td>
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<td>Exercise periods</td>
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<td>IV</td>
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<tr>
<td>0 Recovery*</td>
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<tr>
<td>3-min recovery</td>
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<td>6-min recovery</td>
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*Moment of cessation of exercise.

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**Figure 1**

(A) Twelve-lead ECG taken before exercise. (B) Seven-lead ECG taken after shower, approximately 10 to 15 min after exercise. Acute S-T elevation is present in lead V2. (C) Twelve-lead ECG taken 1 hour later. Acute S-T changes are present. (D) Twelve-lead ECG taken 5 days later shows evolution of acute anterior myocardial infarct and right bundle-branch block.
Electrocardiograms are recorded from a CB-5 bipolar lead during performance of multistage treadmill test of maximal exercise tolerance. At no time are abnormal S-T responses recorded. Heart rates are as follows: Resting, 76 beats/min; submaximal exercise, 126; maximal exercise, 194; initial recovery, immediately after cessation of exercise, 190; 3 min later, 144.

This lead revealed a markedly elevated S-T segment and a peaked T wave, indicative of acute anterior wall myocardial infarct (fig. 1B). Morphine sulfate, 10 mg, was given intramuscularly for pain and agitation. Within a 5-min period, the rhythm changed to a slightly irregular tachycardia (probably ventricular) at a rate of 240 to 260 beats/min, and progressed rapidly through a more irregular stage to ventricular fibrillation (fig. 3A and B). The patient had a convulsion and was defibrillated immediately with an AC defibrillator. Sinus tachycardia with right bundle-branch block developed at a rate of 120 beats/min (fig. 3C) and was followed by a tachycardia at a rate of 256 beats/min which was either ventricular or supraventricular with aberrant conduction. Quinidine (about 300 mg) was given intravenously. The ventricular rate became slower, dropping to 220 beats/min. Following an A-V nodal escape beat, conversion developed to sinus rhythm at a rate of 136 beats/min with right bundle-branch block (fig. 3D, E, and F).

The patient was moved to the coronary care unit where a 12-lead electrocardiogram taken within 1 hour after the onset showed extensive S-T elevation in the precordial leads consistent
with acute anterior myocardial infarction (fig. 1C).

During the next few days, he was alert, apprehensive, and in need of sedation. Heart size (by portable chest x-ray) was small, and blood pressure, normal. Heart sounds were distant with a transient ventricular gallop. An abnormal outward systolic impulse ("ischemic bulge") appeared in the precordial area. Heart rate ranged from 140 to 160/min until plasma volume was cautiously expanded with saline intravenously. Although heart size increased and basilar rales

All ECGs recorded from lead II. (A) ECG taken 5 min after tracing in figure 1B. Ventricular tachycardia at a rate of 240 to 260 beats/min progresses to a chaotic type of rhythm. (B) Ventricular fibrillation ensues. (C) Following AC defibrillation sinus rhythm with right bundle-branch block develops at a rate of 120 beats/min. (D) Within 5 min, tachycardia (either ventricular or supraventricular with aberrant conduction) supervenes again, at a rate of 256 beats/min. Intravenous administration of quinidine is begun. (E) Ventricular rate decreases to 220 beats/min. Following an A-V nodal beat, sinus rhythm with right bundle-branch block develops again, at a rate of 136 beats/min. (F) Sinus rhythm with right bundle-branch block continues.
were detected occasionally, the patient was treated with digoxin and no overt heart failure ensued. On the second day, the anterior S-T injury current was more marked and left axis deviation of the QRS forces appeared transiently. Occasional ventricular premature beats were controlled by intravenous administration of lidocaine. The serum creatine phosphokinase rose to 9.9 units, and the glutamic oxalacetic acid transaminase to 520 units. Lactic dehydrogenase reached a maximum of 1,040 units by the third day with a marked increase in the alpha fraction. On the fourth day, a prominent pericardial friction rub was noted, and the following day a high-pitched systolic murmur of mitral incompetence was heard during expiration.

Arteriographic examination of the coronary arteries by Dr. Werner Samson 6 months later revealed a normal right artery, minimal plugging of the left anterior descending artery and 90% obstruction of the junction of the proximal and middle one-third of the circumflex artery. A left ventricular angiocardiogram showed paradoxical pulsations of a minor aneurysm near the apex.

The patient’s subsequent course was that of steady improvement, with reduction in heart size, improvement of pericarditis, and disappearance of the apical systolic murmur as he gradually became ambulatory. He was discharged to go home on the twenty-second day. When seen in clinic 4 and 11 days later, he was doing well, although the blood pressure was only 96/70 mm Hg.

**Discussion**

Isolated instances of myocardial infarction or sudden death, or both in patients known to have heart disease have occurred in close relation to exercise stress testing. However, the occurrence of acute infarction following exercise testing in an apparently healthy person is virtually unknown. In the present instance, the interim history, physical examination, and resting 12-lead electrocardiogram were all unremarkable. Indeed, contemporary risk factors for coronary heart disease were seemingly negative also: The patient had discontinued his high fat diet and excessive smoking of cigarettes 2 years before. His weight was less than average for his height, and his blood pressure was not elevated. Although he had formerly been under emotional stress, he had resolved his problem by separation from his family a few months earlier. Finally, there was no electrocardiographic evidence of segmental S-T depression indicative of postexertional myocardial ischemia after maximal exercise less than one-half hour before the overwhelming event. Actually, his exercise capacity was slightly greater than that observed 3 years earlier by the same test.

The exact pathophysiological mechanisms responsible for myocardial infarction and cardiac arrest with the potentiality for sudden death of this patient are not known. Three possible mechanisms might be considered:

1. The possibility of a transient ventricular arrhythmia cannot be absolutely excluded. Palpitations were not noticed, and an electrocardiogram within 5 min of the onset of weakness, fatigue, nausea, and faintness showed normal sinus rhythm with no premature beats. Acute anterior infarction was already manifest by the appearance of a substantial S-T force. Symptomatically, he progressed rapidly and ventricular tachycardia and fibrillation ensued with coma and convulsions.

2. Acute coronary insufficiency, with subsequent myocardial necrosis, might have resulted from the cumulative effects of sudden reduction in cardiac output and coronary perfusion secondary to reduced venous return from orthostatic pooling in acutely dilated venous capacitance vessels. In addition to any residual postexertional vasodilatation in skeletal muscle, sudden exogenous thermal stress may have initiated substantial cutaneous vasodilatation. Since he was standing, gravitational displacement of additional blood in the dependent vessels could have occurred. If so, the supine posture shortly afterward when the electrocardiogram was recorded again should have been restorative. Furthermore, many individuals have taken hot showers after maximal exertion without such untoward complications.

3. The occurrence of severe symptoms immediately after exposure to hot water may have been fortuitous. This would necessitate the coincidental development at that time of a major vascular lesion following performance of exercise. Paterson has suggested that strenuous physical exercise may initiate high rates
of coronary blood flow which would reduce the lateral pressure on a partially stenosing atherosclerotic plaque. This would theoretically result in rupture into the plaque of acutely dilated “acquired capillaries” which have direct connection to the high pressure arterial lumen adjacent to the lesion. This progressive subintimal hemorrhage could have initiated an intraluminal hematoma with the effect of immediate coronary occlusion, myocardial infarction, ventricular arrhythmia, and sudden death. Paterson\textsuperscript{9} reported that 52 of 58 (89\%) autopsies on patients dying of coronary thrombosis also showed intimal hemorrhages adjacent to the thrombus. Comparable prevalences of such hemorrhage in the experience of five other pathologists have ranged from 52\% to 90\%.\textsuperscript{10}

Paterson noted that “hours or even days elapse between time of the inception of the thrombus and moment when occlusion with its resulting cardiac pain occurs.”\textsuperscript{7,11} In this instance, the time required to shower may have delayed the patient’s leaving the hospital laboratory long enough so that the cardiac arrest occurred within the building rather than out on the street. The immediate availability of professional staff, emergency drugs, oxygen, and a defibrillator in the room converted a potential sudden death into a successful resuscitation. Fortunately in this instance, the opportunity for pathological examination was lost.

It must be mentioned that Paterson’s hypothesis of capillary rupture with subsequent thrombus formation following exercise is totally theoretical and unproven. There is no documentation of the definite occurrence of this hypothesized train of events. Even pathological findings may not support the hypothesis. The careful studies of Fulton\textsuperscript{12} showed that, although intimal hemorrhages could be found in coronary arteries, the majority were unrelated to thrombosis. He concurred with Paterson that such hemorrhages arise from rupture of thin-walled intimal vessels originating from the arterial lumen, but he found no causal relationship of intimal hemorrhages and thrombosis.

It is noteworthy that myocardial infarction is an extremely rare complication of maximal exercise testing when appropriate precautions are employed. These include selection to avoid patients with recent active myocardial or pulmonary lesions, and professional monitoring during the testing. With such selection and supervision, safety has been assured in over 2,000 individuals (the majority of whom were cardiac patients) in Seattle\textsuperscript{13} and in over 2,000 men participating in epidemiological studies in Taiwan. The supervised monitoring as demonstrated in this patient should not end abruptly when the test is completed.

Finally, the concept of predictive value of exercise electrocardiography remains unchallenged, although the specificity of an apparently negative response to exclude the likelihood of subsequent myocardial infarction was fallible under the immediate circumstances in this particular individual. From the cumulative experience on thousands of individuals over a period of 5 years in testing both healthy subjects and properly screened ambulatory cardiac patients, as well as additional experience over the ensuing 4 months, this instance remains the only known example of myocardial infarction occurring shortly after maximally tolerated exercise utilizing the multistage treadmill test. Indeed, it should be noted that the initial work load of stage I for 3 min is substantially less stressful in energy expenditure than the well-established and clinically familiar Master “two-step” test. Perhaps the safety of this multistage test, in addition to the principles of prior medical examination and professional supervision of the test, is the fact that the patient is also monitoring his own performance and is allowed to stop the exertion on self-determined symptoms. There is no requirement to complete some arbitrarily prescribed number of steps, workload, heart rate, oxygen intake, or any other fixed criteria. In reality, whenever patients are unable to complete such fixed load tests, the stress may well be in excess of their capacity to perform that much exertion. In view of these considerations,
the multistage treadmill test is still used, both for physiological research and clinical testing purposes, with appropriate selection and supervision to assure the safety of each individual.

References
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Circulation. 1968;38:552-558
doi: 10.1161/01.CIR.38.3.552

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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