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

Acute 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 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 V4 was obtainable because of the marked agitation of the patient.

### 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 V4. (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.

#### Table 1

Heart Rate and Blood Pressure Before, During, and After Exercise

<table>
<thead>
<tr>
<th></th>
<th>Blood pressure (mm Hg)</th>
<th>Heart rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Best (examining room)</td>
<td>112/70</td>
<td>80</td>
</tr>
<tr>
<td>Best (on treadmill)</td>
<td>110/60</td>
<td>75</td>
</tr>
<tr>
<td>Exercise periods I</td>
<td>114</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>130</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>160</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>192</td>
<td></td>
</tr>
<tr>
<td>0 Recovery*</td>
<td>130/70</td>
<td>190</td>
</tr>
<tr>
<td>3-min recovery</td>
<td>160/70</td>
<td>144</td>
</tr>
<tr>
<td>6-min recovery</td>
<td>140/60</td>
<td>124</td>
</tr>
</tbody>
</table>

*Moment of cessation of exercise.
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

Figure 2

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.
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
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

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of coronary blood flow which would reduce
the lateral pressure on a partially stenosing
atherosclerotic plaque. This would theoreti-
cally 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 progres-
sive subintimal hemorrhage could have initi-
ated an intraluminal hematoma with the effect
of immediate coronary occlusion, myocardial
infarction, ventricular arrhythmia, and sudden
death. Paterson reported that 52 of 58 (89%)
autopsies on patients dying of coronary
thrombosis also showed intimal hemorrhages
adjacent to the thrombus. Comparable preva-
lences of such hemorrhage in the experience
of five other pathologists have ranged from
52% to 90%.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.”11 In this
instance, the time required to shower may
delay 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 avail-
ability of professional staff, emergency drugs,
oxygen, and a defibrillator in the room con-
verted a potential sudden death into a suc-
cessful resuscitation. Fortunately in this in-
stance, the opportunity for pathological
examination was lost.

It must be mentioned that Paterson’s hy-
thesis 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 Fulton12
showed that, although intimal hemorrhages
could be found in coronary arteries, the ma-
jority were unrelated to thrombosis. He con-
curred with Paterson that such hemorrhages
arise from rupture of thin-walled intimal ves-
sels 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 mon-
itoring 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 and in
over 2,000 men participating in epidemi-
ological 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 unchal-
lenged, although the specificity of an appar-
ently negative response to exclude the likeli-
hood of subsequent myocardial infarction was
fallible under the immediate circumstances
in this particular individual. From the cumula-
tive experience on thousands of individuals
over a period of 5 years in testing both
healthy subjects and properly screened ambu-
laratory 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 al-
lowed to stop the exertion on self-determined
symptoms. There is no requirement to com-
plete some arbitrarily prescribed number of
steps, workload, heart rate, oxygen intake,
or any other fixed criteria. In reality, when-
ever 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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