Hemodynamic Response to Isometric Handgrip in Acute Myocardial Infarction*

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Thirteen patients underwent right cardiac catheterization during the first 24 hours after the onset of symptoms of acute myocardial infarction. All had normal (less than 12 mm Hg) pulmonary arterial diastolic pressure or normal mean pulmonary wedge pressure (7.6 ± 0.6 mm Hg). The patients did isometric forearm contraction to a measured level of 100 mm Hg. All patients had elevation of systemic systolic blood pressure, with a mean rise of 14.2 ± 2.9 mm Hg; heart rate rose by 12 ± 2 beats per minute. When compared to a control group, patients with acute myocardial infarction had a significant (P < 0.005) elevation of 5.4 ± 1.3 mm Hg in the pulmonary arterial diastolic or pulmonary wedge pressure. This may be a result of either increased left ventricular stiffness or decreased myocardial functional reserve. In the setting of acute myocardial infarction, patients with normal left ventricular filling pressures have abnormal ventricular performance. Isometric effort is poorly tolerated and should be avoided.

Evaluation of left ventricular performance in patients suffering from an acute myocardial infarction has provided a great advance in the care of this particular group of patients. Bedside hemodynamic monitoring has been used not only to evaluate different types of therapy during the acute stage, but also for the assessment of provocative interventions directed to define the myocardial reserve during this critical period. Static effort has not been used previously for this purpose. Isometric exercise is known to impose a disproportionate pressure load on the left ventricle. In normal individuals, isometric exercise has been proven to result in increased systolic blood pressure, cardiac index, stroke work index, and heart rate with significant change in the left ventricular filling pressure. Systemic vascular resistance remains unchanged or rises minimally during isometric exercise in the normal state. This suggests a shift in inotropic state from the basal to a higher level. On the other hand, patients with heart disease exhibit a rise in blood pressure and heart rate, plus a significant elevation of the left ventricular end-diastolic pressure and a reduction in cardiac index and stroke work index.

When analyzed by angiographic methods, normal hearts exhibit reduction in both end-systolic and end-diastolic volumes while ejection fraction remains unchanged. Patients with heart disease have shown an increase of the end-systolic volume with reduction in ejection fraction.

This study was undertaken to evaluate the response of patients with acute myocardial infarction and normal resting left ventricular filling pressure to a measured amount of isometric stress. This approach was taken in an attempt to unmask an underlying abnormal hemodynamic state.

Materials and Methods

Patients were selected from those admitted to the coronary care unit of the San Juan (Puerto Rico) Veterans Administration Hospital. The patients underwent right cardiac catheterization within 24 hours of the onset of symptoms of acute myocardial infarction. Myocardial infarction was documented by the typical electrocardiographic changes, plus a rise in the serum levels of creatine phosphokinase, lactic dehydrogenase, and glutamic-oxaloacetic transaminase. Right cardiac catheterization was performed using the percutaneous technique through a basilic or femoral approach. Balloon-tipped Swan-Ganz catheters were used, and pressures were monitored via a transducer (Statham P231A) and a pressure amplifier (American Optical). Pressures were recorded in a two-channel direct recorder. The electrocardiogram was recorded continuously. Criteria for inclusion in the study were as follows: (1) right cardiac catheterization within 24 hours of uncomplicated acute myocardial infarction; (2) resting pulmonary arterial diastolic pressure or a pulmonary capillary wedge pressure less than 12 mm Hg; and (3) resting systemic blood pressure not lower than 90/60 mm Hg and not higher than 160/110 mm Hg. Thirteen patients fulfilled these criteria and constitute the group under study.

Isometric handgrip exercise was performed for a period of 30 seconds against a partially filled blood pressure cuff with a resting tension of 20 mm Hg, which was maintained at a minimal level of 100 mm Hg. Valsalva's maneuver was
prevented by having the patient breathe slowly through his mouth. Brachial blood pressure was measured by cuff before, during, and after isometric exercise. The heart rate, ECG, and pulmonary arterial diastolic or wedge pressure were monitored during a control period of one minute preceding exercise, during isometric exercise, and for one minute thereafter. In the four cases where the basilic approach was used for right cardiac catheterization, blood pressure was measured from the opposite arm. Isometric handgrip exercise was done by using the catheterized arm. To avoid possible distortion on the recordings of pressure during isometric handgrip, the exercise was limited to the muscular group distal to the insertion of the catheter, and the pressure tracings were examined for pressure artifacts.

Four patients without acute myocardial infarction or heart failure underwent similar evaluation as part of a diagnostic cardiac catheterization at the cardiac catheterization laboratory. Two of these patients were normal, and two had coronary arterial disease with no prior myocardial infarction and normal left ventricular function. These four patients represent our control group. Informed consent was obtained in all subjects studied. Results were analyzed using Student's t-test for paired groups and are given as the mean ± SE. Values for P of less than 0.05 were considered significant.

RESULTS

The mean age of the 13 patients in the group under study was 53 years, with a range of 44 to 58 years (Table 1). Eleven of the patients had transmural myocardial infarction, and two had nontransmural myocardial infarction. Eight of the 13 patients sustained an inferior wall myocardial infarction, and five suffered an anterior myocardial infarction. Four of the patients had rales or an S₃ gallop rhythm. The mean time from the onset of symptoms of acute myocardial infarction to isometric exercise was 8.5 hours.

The mean resting arterial blood pressures in the group with myocardial infarction were 117 ± 5 mm Hg (systolic) and 85 ± 4 mm Hg (diastolic), with a mean heart rate of 73 ± 4 beats per minute. The mean resting pulmonary arterial diastolic pressure was 8 ± 1 mm Hg, with a range of 4 to 12 mm Hg. The mean maximal arterial blood pressures at isometric exercise were 131 ± 6 mm Hg (systolic) and 93 ± 4 mm Hg (diastolic), with a mean heart rate of 85 ± 5 beats per minute. The pulmonary arterial diastolic pressure in the group under study rose to a mean of 13 ± 1 mm Hg. Of the 13 patients with acute myocardial infarction, seven (54 percent) had elevations of the pulmonary arterial diastolic pressure to 12 mm Hg or more during isometric exercise. In three (23 percent) of the 13 patients, the pulmonary arterial diastolic pressure rose to 20 mm Hg or more during isometric exercise.

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<th>Patient Age (yr)*</th>
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*Mean age, 53 years.
**AL, anterolateral; I, inferior; A, anterior; IL, inferolateral; and PI, posteroinferior.
†T, transmural; and NT, nontransmural.
‡S₃, third heart sound.
§PAEDP, pulmonary arterial end-diastolic pressure; and PCWP, pulmonary capillary wedge pressure.
||P < 0.05 compared to control.
Isometric handgrip in the group with myocardial infarction resulted in a rise of 14 ± 3 mm Hg in the mean systemic systolic blood pressure and a rise of 8 ± 2 mm Hg in the mean systemic diastolic pressure. The heart rate accelerated an average of 12 ± 2 beats per minute. The pulmonary arterial diastolic pressure rose a mean of 5 ± 1 mm Hg. All of these values were significant (P < 0.005).

In the control group of patients, the mean resting systemic blood pressures were 128 ± 6 mm Hg (systolic) and 65 ± 5 mm Hg (diastolic), with a mean resting heart rate of 68 ± 5 beats per minute. The mean resting pulmonary arterial diastolic pressure was 7 ± 1 mm Hg. After isometric exercise, there was a rise in mean systolic blood pressure of 26 ± 7 mm Hg, with a mean rise in heart rate of 20 ± 2 beats per minute. Both values were significant (P < 0.05). The mean pulmonary arterial diastolic pressure in the control group rose 0.5 ± 0.3 mm Hg, which was not significant (P < 0.25). The rise of 18 ± 6 mm Hg in the mean systemic diastolic blood pressure was also not significant (P < 0.1).

During isometric handgrip exercise, none of the subjects developed hypotension, pain in the chest, or arrhythmias. In all patients the elevation of heart rate, blood pressure, and pulmonary arterial diastolic pressure returned to a resting level within one minute after the end of isometric exercise.

**DISCUSSION**

In the normal individual who performs handgrip exercise to less than 60 percent of maximal voluntary contraction, the flow to the forearm is increased, and the heart rate and blood pressure also rise. Elevation of blood pressure is due to increased cardiac output, since systemic vascular resistance is maintained unchanged. The pressure load imposed upon the left ventricle results in a rise in stroke work index, with minimal or no change in the left ventricular filling pressures. These are the mechanisms by which the normal left ventricle reacts to the pressure load by increasing its contractile state.\(^6\) Patients with known heart disease respond to the isometric challenge not only with augmentation of both heart rate and blood pressure, but also with an increase in systemic vascular resistance and left ventricular filling pressure. These hearts must resort to the Frank-Starling mechanism in order to withstand the burden of pressure resulting from the isometric exercise, since their contractile state cannot be improved.\(^8,11,12\)

We have used this test in the early phase of acute myocardial infarction in order to observe the hemodynamic response of patients with normal resting left ventricular filling pressure. Filling pressures rose above the normal limit of 12 mm Hg in most of our patients; and in 23 percent (3/13) of them, it reached or exceeded the level of 20 mm Hg (Fig 1). This response is abnormal and may be explained by two different mechanisms. These hearts may be working on a flattened curve of left ventricular function. This is similar to those curves seen in patients with diminished contractility who operate on the plateau of the curve of the Frank-Starling relation. Similar results can also be obtained in hearts with diminished left ventricular compliance, where higher filling pressures are needed but the slope of the curve of function is preserved.

The response that we have demonstrated suggests a diminished cardiac reserve in the very early stage of acute myocardial infarction, even in patients with normal left ventricular filling pressure. This abnormal response may be due to a loss in the contractile efficiency, loss of left ventricular compliance, or a combination of both.\(^10,13\)

This study suggests that in the setting of acute myocardial infarction, those patients with normal left ventricular filling pressures (in whom one would usually predict a benign course) may exhibit abnormal left ventricular performance when challenged with a minimal amount of afterload stress. Activity involving isometric effort should be avoided in all patients with acute myocardial infarction in the early convalescent period. Reversibility of this abnormal response probably depends on the total amount of functional myocardial loss.

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