Recovery of Left Ventricular Function Following Acute Myocardial Infarction

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The evaluation of patients sustaining an acute myocardial infarction (MI) has traditionally included a description of symptoms, observations on physical examination, radiographic assessment of the heart and lungs, and a thorough review of the electrocardiogram. Immediate and effective treatment of electrical instability in acute MI is essential and has greatly reduced the incidence of mortality from ventricular arrhythmias. However, hemodynamic abnormalities secondary to acute left ventricular (LV) dysfunction are invariably present and account for significant mortality. Because acute MI is primarily a disease of the left ventricle, the estimation of LV function is vitally important in assessing the severity of circulatory impairment produced by the infarction.

Recognition of the importance of LV function in the setting of acute MI led to extensive investigations aiming to document the severity of this functional impairment (1-6). As a result, the degree of LV dysfunction was shown to be a major determinant of morbidity and mortality after acute MI (7). A direct relationship between the magnitude of this functional impairment and enzymatic estimates of infarct size was also found (8). Furthermore, evaluation of LV filling pressures and cardiac output in acute MI were shown to be of value in defining subsets of patients with different prognoses and responses to therapy (7,9).

The assessment of LV function in acute MI also provided an important insight into the pathophysiology of this event, i.e., that LV function can improve during early convalescence. In dogs, complete occlusion of an epicardial coronary artery causes acute impairment of global LV function which gradually improves during the first week following the acute injury (10,11). Global LV function also has been shown to occur in humans during the healing phase of acute MI (6,12-14).

This review examines the nature of the recovery of LV function following acute MI in terms of its magnitude, the rate at which it can take place, and the mechanisms responsible for its manifestation. These observations, previously published in detail (15-17), are based primarily on data collected at our institution using noninvasive continuous-wave Doppler velocimetry.

Quantitation of LV Function in Acute MI

Global LV function has been evaluated in patients during both the early and late stages of an acute MI. However, data on serial measurements of LV performance during the course of recovery from an acute MI are sparse (14,18,19). The paucity of such data is most likely due to the technical difficulties of obtaining an accurate assessment of LV function on a daily basis. Invasive techniques of right-heart catheterization have been used to evaluate cardiac function during the acute and convalescence phases of acute MI (18). These studies provide a reliable assessment of cardiac output and pulmonary artery wedge pressure, but neither of these indices are direct measures of global LV performance. Direct catheterization of the left ventricle also has been used to determine end-diastolic pressure in the early and late stages of an acute MI (14). While this approach provides a better assessment of global LV function, it is associated with discomfort and risk to the patient and cannot be performed on a daily basis. Radionuclide studies using 99mTc-technetium have been performed to evaluate serially LV ejection fraction during the course of recovery from acute MI (19). This approach is useful but requires repeated injections of radioactive material and its availability is limited. Other noninvasive approaches such as M-mode echocardiography and systolic time intervals have been used to evaluate LV performance in patients with acute MI but have inherent problems which limit their use for this application. Two-dimensional echocardiography may be ideally suited for the serial evaluation of global LV performance and regional wall motion abnormalities in the setting of acute MI. Studies of this nature, however, have not yet been performed.

An alternative approach to the noninvasive bedside evaluation of LV performance is the use of peak acceleration (PACC) of blood in the ascending aorta measured with continuous-wave Doppler ultrasound (15). Continuous-wave Doppler ultrasound allows for accurate noninvasive measurement of phasic aortic velocity from the suprasternal notch. PACC can easily be derived from the phasic velocity waveform using electronic differentiation (15,20). This method is both simple and reproducible and is based on a solid theoretical foundation. PACC has long been recognized as a sensitive index of global LV performance.

Submitted for publication: July 31, 1991.
Accepted for publication: September 11, 1991.
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It is closely related to the maximum force exerted by the left ventricle in early systole (21) and the maximum initial velocity of shortening of LV muscle (21) and is a primary determinant of the rate of change of power developed by the left ventricle during ejection (22). In dogs, PACC was shown to be very sensitive to alterations of the inotropic state and minimally influenced by changes of preload and afterload (21). In dogs, PACC is a sensitive indicator of regional myocardial ischemia produced by ligation of the coronary arteries (22) and has been shown to be linearly related to the extent of LV ischemic mass at risk (23). In patients, PACC has been shown to be highly sensitive in distinguishing those with normal LV function from those with abnormal LV function (24). In patients undergoing cardiac catheterization, PACC has been shown to be linearly related to LV ejection fraction measured angiographically (15).

**Studies of PACC During Recovery From Acute MI**

We have examined the course of recovery of LV function in 26 consecutive patients with acute MI (16). In each patient, acute MI was confirmed by characteristic electrocardiographic changes and by a typical rise and fall of serum creatine kinase and elevation of creatine kinase isoenzymes. In this cohort of patients, 20 had a Q wave infarction and six had a non-Q wave infarction. Ten patients had an anterior wall infarction and 16 had an inferior wall infarction. None of the patients had aortic valve disease, none developed heart block, and six developed clinical signs of congestive heart failure.

In all patients, phasic aortic blood velocity and PACC were measured transcutaneously from the suprasternal notch with a conventional continuous-wave Doppler transmitter and receiver operating at 3.0 MHz (ExerDop, Quinton Instruments, Seattle, WA). The Doppler system and its use in patients has been described (15). An initial measurement of PACC was obtained within 20 ± 2 hours of the acute onset of chest pain. Measurements were repeated daily for six consecutive days. All measurements were made with the patient supine. Serial Doppler measurements were also made in an identical manner in 11 normal volunteers for comparison with acute MI patients. The measurements in normal subjects were performed to insure that daily changes of the Doppler measurements of PACC were not due to intrinsic variability of the measurements themselves. None of the 11 normal subjects had any symptoms or history of cardiovascular disease at the time of the study. Only minimal changes of PACC were observed in normal adult volunteers over the course of six consecutive days. PACC was 23 ± 2 m/sec/sec on day 1 and 24 ± 2 m/sec/sec on day 6. The daily variability was ± 2 m/sec/sec based upon repeated measures analysis of variance used to calculate the mean squared error for the time factor.

**Serial changes of PACC in patients with acute MI**

In patients with acute MI, PACC increased gradually over the course of six days. The rate and magnitude of this functional improvement is depicted in the Figure. As expected, PACC was markedly depressed on day 1 in MI patients in comparison to normal subjects (13 ± 1 versus 23 ± 2 m/sec/sec) but increased to 18 ± 1 m/sec/sec on day 6 of the study. To gain additional insight into the rate and extent of recovery of LV function in patients with acute MI, the 26 patients were separated into two groups. Group I (n = 15) included patients who had an acute MI for the first time and who did not develop congestive heart failure during the course of recovery. Group II (n = 11) included patients who had a history of acute MI or who developed congestive heart failure as a consequence of their current MI. A comparison of the clinical characteristics among the two groups is shown in the Table. A comparison of the rate and magnitude of the recovery of LV function in the two patient groups over the course of the study is shown in the Figure. The recovery of LV function in Group I patients was marked and rapid. PACC increased from 13 ± 1 m/sec/sec on day 1 to 20 ± 2 m/sec/sec on day 6 (P < 0.001). In contrast, LV function remained depressed in Group II patients with evidence of only slight but insignificant improvement of function over the course of six days. PACC in this group of patients was 12 ± 1 m/sec/sec on day 1 and increased to only 15 ± 1 m/sec/sec on day 6.

These data indicate that a gradual recovery of global LV performance generally occurs during the first week after acute MI. The magnitude and pace of this recovery was far more pronounced in patients who had an acute MI for the first time uncomplicated by congestive heart failure in comparison to patients with a history of acute MI or those whose course of recovery was complicated by congestive heart failure. The lack of re-
covery of LV function in this latter group is not surprising. Multiple infarctions lead to cumulative loss of viable contractile myocardium beyond the point of intrinsic compensation. Similarly, patients whose course of recovery from acute MI was complicated by congestive heart failure may have experienced a larger infarction and consequently a greater loss of viable contractile myocardium. The preponderance of anterior acute MI in Group I and inferior acute MI in Group II adds yet another dimension which further explains the apparent lack of recovery of LV function in this cohort of patients.

Other Indices of LV Function During Recovery From Acute MI

The recovery of LV function following acute MI based upon serial improvement of PACC is supported by a host of studies utilizing other indices of global LV performance. Cardiac index and LV stroke work were shown to increase three to four weeks after acute MI in patients whose LV end-diastolic pressure decreased but not in those whose LV end-diastolic pressure remained elevated (14). The persistent elevation of LV end-diastolic pressure in patients who show a lack of improved LV function may be a manifestation of a larger infarct size or possibly the development of pump failure. Others demonstrated improved LV function at day 3 after an acute MI based upon the observation of an upward shift of LV function curves relative to day 1 (25). A limited number of studies used sequential radionuclide angiography to evaluate LV ejection fraction during the course of recovery from acute MI (26,27). In these studies, considerable improvement of LV ejection fraction was shown to occur as early as one week after acute MI (26,27). This improvement, however, appeared to be limited to patients who showed signs of clinical improvement (26). These studies clearly indicate that one can expect improvement of LV function following acute MI. This functional improvement, however, does not occur at the same rate or to the same extent in all patients. Instead, the degree of recovery of LV function may be dependent upon multiple factors including infarct size, cumulative loss of viable myocardium, anatomical site of the acute MI, and complications associated with the acute MI, particularly the development of heart failure.

Mechanisms of Recovery of LV Function After Acute MI

Although there is general agreement that recovery of LV function can take place in patients following acute MI, the precise mechanism of this recovery remains elusive. Several hypotheses have been proposed which merit discussion. One possible explanation implicates stiffening of the infarcted myocardium as a factor responsible for the recovery of LV function following acute MI (28). Investigators ascribing to this notion argue that the aneurysmal bulging or regional myocardial dyskinesia that is often present at the onset of infarction tends to diminish during the recovery period due to stiffening of the infarcted tissue. The diminution or disappearance of this regional systolic bulging is thought to account for the increase in the effective stroke volume during recovery (28). Studies in patients and in experimental animal models have provided direct evidence supporting the concept of increased stiffness of the infarcted myocardium during the period of recovery from acute MI (10,29). Improvement of global LV function during the recovery from acute MI has also been suggested to result from increased sympathoadrenergic activity (30). This hypothesis is supported by studies in dogs which showed increased plasma catecholamine concentration following coronary artery ligation (30). Increased adrenergic discharge represents an independent pathway for increasing the inotropic state of the noninfarcted viable myocardium. Studies by Ross and Franklin (31) and by Theroux et al (32) in dogs showed increased systolic shortening and end-diastolic fiber length of the normal LV myocardial wall segments at one and three weeks after experimental acute MI. Another possibility is the existence of ischemic but viable myocardium within or at the periphery of the infarct zone compatible with the presence of "stunned" or "hibernating" myocardium (33,34). This condition is likely to exist in patients with a non-Q wave MI or so-called "incomplete infarction." Functional recovery of this ischemic but viable myocardium in the days following acute MI may also partly explain the gradual overall recovery of global LV function. At present, and without any direct evidence to implicate a single mechanism in this recovery process, it is reasonable to suggest that all of the above mechanisms act in concert to produce the type of recovery of global LV function seen in patients following acute MI.

Conclusion

Based upon our work and that of others, it is safe to suggest that significant recovery of global LV function can occur as early as one week following acute MI. The rate at which this recovery takes place and the magnitude of the recovery vary considerably among patients and are dependent on multiple factors including infarct size, anatomical location of the infarction, history of MI, and the development of congestive heart failure.
From a pathophysiological point of view, little doubt remains that the size of the infarction is the major determinant of the rate and extent of recovery of global LV function.

References