EDITORIAL

Value and Limitations of Systolic Time Intervals (Preejection Period and Ejection Time) In Patients with Acute Myocardial Infarction

THREE RECENT reports in this journal provide information on systolic time intervals in a total of 167 patients with acute myocardial infarction. It would seem, then, that a sufficient number of observations are available to permit critical assessment and meaningful conclusions. Are the data from these sources consistent and the results comparable? What are recordings of systolic time intervals in acute myocardial infarction designed to accomplish? What can currently be said of their value and limitations? Let us first examine the setting in which these studies were undertaken.

Coronary care units have provided optimum means for recognition and control of potentially fatal arrhythmias. Consequently, the prime cause of death in hospitalized patients with acute myocardial infarction is now inadequate performance of the damaged myocardium, i.e., inability of the heart as a pump to maintain circulatory integrity. The first step in an attempt to resolve this problem has been a more complete definition of the hemodynamic and pathologic spectra related to the functional state of the acutely infarcted heart. This step is now accomplished in large part through the efforts of investigators in Myocardial Infarction Research Units (MIRU). Their findings are important but were largely predictable. Left ventricular filling pressure is elevated while mean systemic arterial pressure, stroke volume, stroke work, ejection fraction, contractile element velocity, and maximum left ventricular dp/dt are all depressed to varying degrees. Stroke work emerges as a promising index of impaired left ventricular performance and its clinical consequences. The anatomic basis of cardiogenic shock has been found to reside in the extent rather than the location of the infarction. It appears that when 40% or more of left ventricular muscle mass is destroyed, the remaining myocardium cannot maintain cardiac function. Once cardiogenic shock is established, it tends to be self-perpetuating, with continuing necrosis in the form of marginal extension of the recent infarct in addition to areas of focal necrosis throughout both left and right ventricles.

The second step taken by MIRU investigators was a critical assessment of therapy for cardiogenic shock. It has been clear for some time that drug treatment is at best ineffectual.
Still more dismaying is the conclusion that mechanical circulatory assistance, even when coupled with aggressive surgical intervention, leads to only small salvage rates. The next step should therefore be therapeutic intervention in high-risk patients prior to the onset of overt cardiogenic shock. However, a sensitive and reliable prognostic index of deteriorating ventricular performance must first be developed. Moreover, if this index is to have a substantial impact on mortality, it must be relatively simple, widely available, and require little or no invasive instrumentation, since direct methods are likely to be associated with discomfort, risk, and technical difficulties in acutely ill patients. This, then, is the setting in which recent reports on systolic time intervals in acute myocardial infarction must be examined.

Initial reports of small series by Schoenfeld and colleagues and by Wayne indicate that in acute myocardial infarction—as in chronic heart failure—the preejection period (PEP) was prolonged. This observation seemed to be in accord with the view that PEP reflected the contractile state of the left ventricle. In addition, patients with acute myocardial infarction, like those with heart failure, exhibited a reduction in left ventricular ejection time (LVET). This conclusion was consonant with previous data that showed a close relationship between LVET and stroke volume. However, acute myocardial infarction was found to result in shortening of the total duration of electromechanical systole (QS2), whereas in chronic heart failure QS2 remains essentially unchanged.

Diamant and Killip serially recorded systolic time intervals for 5 consecutive days in 66 patients with acute myocardial infarction and found similar directional changes, i.e., prolongation of PEP with shortening of LVET and QS2. Their patients with transmural infarctions had more abnormal measurements than those with nontransmural damage, and patients dying with transmural infarcts had greater deviations from normal than survivors. Even though there was considerable overlap of individual values the authors concluded that the extent of infarction influences the magnitude of the changes in left ventricular systolic time intervals and that these measurements held promise as prognostic indicators.

Heikkilä et al. studied systolic time intervals in 50 patients with acute myocardial infarction and found that LVET and QS2 decreased from the first to the fifth day, then increased toward normal by the 20th day. A high prevalence of abnormal values indicated frequent and early impairment of left ventricular function. Furthermore, serial observations on days 1, 5, 20, or just preceding death were interpreted as showing that deterioration was reflected in progressive shortening of these intervals in contrast to gradual improvement in uncomplicated cases. On day 1, LVET was already significantly shorter in patients who later died than in survivors. However, the preejection period was not prolonged in relation to severity or ultimate course. The authors concluded that LVET was useful in the assessment of severity and prognosis in acute myocardial infarction.

The investigations of Hodges et al. serially examined systolic time intervals in 51 patients with acute myocardial infarction. QS2 and LVET were significantly shorter than normal on each day during the first 3 weeks, reaching their lowest values on day 6 and gradually returning toward normal during the fourth hospital week. Despite these trends, the authors found themselves unable to conclude that LVET correlated with clinical severity or prognosis. In addition, PEP exhibited no meaningful patterns, with measurements ranging from short to normal to long.

In these studies, the greatest area of agreement is in the directional change in group means for LVET. Both Diamant and Heikkilä extracted diagnostic and prognostic significance from measurements of LVET, whereas Hodges et al. did not. LVET is dependent upon a number of variables in addition to stroke volume. Many of these variables, such as heart rate, afterload, and inotropic state vary systemically in acute myocardial infarction but affect LVET in opposite ways. Thus it is no surprise to find on
EDITORIAL

931

close scrutiny, that, even when group means for LVET differ significantly, the absolute magnitude of the differences is relatively small and individual variations wide. Nevertheless, if it could be shown that LVET were decreased on day 1, lowest on days 5 or 6, and rises toward normal by the end of the third week, then it would be difficult to avoid the assumption that failure of LVET to increase significantly after its expected nadir should be viewed as unfavorable. Hodges' conclusion to the contrary may have been influenced by the comparatively small number of measurements made late in the course of acute myocardial infarction.

The explanation for the discrepant observations on PEP in acute myocardial infarction is by no means clear. In part these discrepancies may reflect differing interpretations of similar data or may reflect significant methodologic differences which are incompletely evident in published information. It is apparent, however, that an ample basis exists for broad variations in PEP among patients with acute myocardial infarction of comparable severity. Determinants of PEP include QRS duration, left ventricular dp/dt, left ventricular end-diastolic pressure, and aortic diastolic pressure. In cardiogenic shock, a high LVEDP and a low aortic diastolic pressure may result in a short PEP despite a low LV dp/dt. On the other hand, a normal or mildly elevated LVEDP and a normal or elevated aortic diastolic pressure may be accompanied by appreciable prolongation of PEP if LV dp/dt is depressed. If one examines individual rather than group data, wide variations in PEP among similar patients and considerable overlap among various groups are indeed present in most reported series. Agress et al. have recently shown that correction of PEP for aortic diastolic and left ventricular filling pressures enhances its sensitivity as a prognostic index in acute myocardial infarction, but this requires modifying a "noninvasive" with an invasive technic.

Some comment must be made on problems of methodology and experimental design. Recordings of low-frequency waveforms are exquisitely dependent on the time constant of the transducer used, but data on this variable are not available in the reports under consideration. Further, one would anticipate that regression equations for systolic time intervals against heart rate in normal controls would be similar if not identical; yet these vary widely. Most of the reports, except that of Diamant and Killip, correct abnormal values for heart rate using regression lines derived from normal controls, a procedure of doubtful validity in the absence of direct evidence that the regressions are identical. Finally, the inclusion of groups designated as "chest pain but no myocardial infarction," in which the presence and extent of heart disease is unclear, serves only to complicate the interpretation of data.

In summary, we are persuaded that, while systolic time intervals undoubtedly reflect alterations in left ventricular performance in acute myocardial infarction, they do not yet consistently distinguish among individual patients with acute infarcts of varying severity in a practical way. To do so may require more data on serial measurements in the second, third, and fourth weeks as well as correction of systolic time intervals for dependency on multiple variables having opposing effects. Such corrections would have to be made using noninvasive technics alone. Appropriate methods to achieve this end are not presently available.

JOSEPH K. PERLOFF
NATHANIEL REICHEK

References

4. SCHEIDT S, ALONSO DR, POST MR, KILLIP T: Quantification of myocardial damage in cardio-

Circulation, Volume XLV, May 1972


Value and Limitations of Systolic Time Intervals (Preejection Period and Ejection Time) In Patients with Acute Myocardial Infarction
JOSEPH K. PERLOFF and NATHANIEL REICHEK

Circulation. 1972;45:929-932
doi: 10.1161/01.CIR.45.5.929

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1972 American Heart Association, Inc. All rights reserved.
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:
http://circ.ahajournals.org/content/45/5/929.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/