LETTERS TO THE EDITOR


The authors reply:
To the Editor:

As stated by Dr. Bete, studies of Purkinje fiber bundles perfused with physiologic salt solution have shown that procaine amide prolongs the action potential duration (APD) and the effective refractory period (ERP), the latter to a greater extent than the former.1 We did not discuss the differences Dr. Bete has noted in calculating the ΔAPD/ΔERP ratio for our studies because our method for determining the ERP (as outlined in the text) differs somewhat from that used in other investigations. To measure the ERP we delivered a premature stimulus (S2) after each basic drive (S1), not intermittently as is often done. The value we reported for the APD was that at the basic stimulus rate; that for the ERP (with an S2 being delivered after each S1) was obtained at a rate twice as fast as the basic drive.

Hence, although the method was sensitive to changes in APD and ERP it did not lend itself to the direct determination of the ΔAPD/ΔERP which is calculated when both parameters are being measured at the same stimulus rate. Our studies did indicate that at therapeutic plasma procaine amide concentrations there is no significant change in membrane responsiveness and the ERP is only slightly prolonged; while when high concentrations (equivalent to those attained in other studies) are attained membrane responsiveness is depressed and the ERP is more markedly prolonged. We stated that the rather small prolongation of ERP and the lack of effect on membrane responsiveness which occur during maintenance of therapeutic plasma procaine amide concentrations "suggest that altered response to premature impulses may not be as consistent a mechanism for abolition of arrhythmias as are changes in conduction and automaticity." (The latter changes were invariably noted during maintenance of therapeutic concentrations.)

I am not certain of the intent of Dr. Bete's comment regarding automaticity and conduction time. We demonstrated that following a single procaine amide injection depression of automaticity occurred well before changes in other parameters including conduction time in the Purkinje fiber bundles. The implication was that tissue concentrations of procaine amide required for suppression of automaticity might be lower than those required for depression of conduction. As a result of these observations we speculated that ventricular arrhythmias which respond rapidly to intravenously administered procaine amide might be due to enhanced automaticity and those which respond more slowly, or require higher concentrations of procaine amide, might be due to abnormal conduction.

Michael R. Rosen, M.D.
Department of Pharmacology
College of Physicians and Surgeons of Columbia University
New York, New York

Reference

Abnormalities in Ventricular Function following MI
To the Editor:

The paper entitled "Evolution of Abnormalities in Left Ventricular Function after Acute Myocardial Infarction" (Circulation 46: 731, 1972) uses a left ventricular dimension to reflect changes in ventricular volume. This dimension is obtained by an echographic technic now in use in several laboratories. However, I am distressed to note that figure 4 of Drs. Broder and Cohn's paper does not show measurement of the left ventricle. Inspection of this illustration shows motion of the "PWLV" away from the transducer between the QRS and T waves, and motion toward the transducer between the P and QRS waves of the electrocardiogram. The PWLV is closest to the transducer at the time of QRS inscription. This is the pattern of the posterior left atrial wall. The posterior left ventricular wall should demonstrate anterior motion (toward the transducer) in systole and posterior motion in diastole. Clearly the "left ventricular diameters" as measured in this figure would not be valid. LVIDd is measured at a mid-to-late diastolic point judging from the ECG and mitral valve pattern while LVIDs is measured at the onset rather than the end of systole by these same criteria. It is hoped that the authors used echographic left ventricular studies in the remaining four patients, but this represents a considerable defect in study and would suggest that the authors may have to modify their conclusions regarding the state of left ventricular function and compliance in the postinfarction period.

Richard L. Popp, M.D.
Assistant Professor of Medicine
Director, Noninvasive Laboratory
Cardiology Division
Stanford University Medical Center
Stanford, California

References
LETTERS TO THE EDITOR

Figure 1
Echocardiogram of left ventricle.


The authors reply:
To the Editor:
Dr. Popp's sharp eye has detected an error in our labelling of the echocardiogram reproduced in our paper. The timing of the posterior wall movement does indeed suggest that the echo is from the posterior wall of the left atrium. Unfortunately, the wrong picture was inadvertently sent for reproduction. The accompanying picture (fig. 1) was taken immediately after the one that was reproduced in figure 4 with the transducer moved slightly inferiorly. The posterior wall now clearly represents left ventricle, since its movement is in proper phase with the QRS wave of the electrocardiogram. The measured internal diameter is identical in the two pictures. Another possibility is that the "paradoxical" motion on the published figure represents a localized area of dyskinesia in this patient with an acute posterolateral infarction.

The small number of left ventricular measurements reported in our paper is a reflection of the stringent criteria we used in accepting the echocardiograms for analysis. All those analyzed showed an identifiable septum and a posterior wall which fulfilled Dr. Popp's prerequisites for left ventricular echoes. Therefore, we must stand by our observations that the high LVEDP in this small group of patients probably was not accompanied by a large left ventricular cavity. As pointed out in our paper, however, this apparent "noncompliance" of the left ventricle could represent a normal ventricular response to a very small acute increase in volume rather than a state of abnormal distensibility unique to the ischemic or infarcted myocardium. Too little is known about ventricular diastolic pressure-volume changes in normal man to allow for a more definite conclusion at this time.

JAY N. COHN, M.D.
Veterans Administration Hospital and Georgetown University
Washington, D.C.

MARTIN I. BRODER, M.D.
Metropolitan Hospital
Cleveland, Ohio
Abnormalities in Ventricular Function following MI
RICHARD L. POPP

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