to be expected since the leaflets are attached to the ring. On the other hand, I can’t understand Dr. Dock’s feeling that the usual mitral valve echo is only the pattern of ring motion. I am not aware of any ring motion which has the classical ‘M’-shaped appearance during diastole. Even in the figure which he gives, the valve is clearly moving anteriorly or toward the chest following atrial contraction. We have done extensive studies of mitral ring motion, and I am not aware of any ring which moves away from the left atrium following atrial contraction.

The use of a prosthetic valve to analyze normal mitral leaflet motion is totally misleading. The ball motion is only vaguely similar to the motion exhibited by the normal mitral leaflets. The axis of motion is totally dissimilar. Furthermore, the ball has far more inertia than the leaflets and does not exhibit all of the fine motion seen in the true valve. The dog is also an unfortunate example for trying to demonstrate mitral leaflet motion with echocardiography. Because of the shape of the dog’s chest it is not always easy to get technically good echograms. Furthermore, since most dogs are anesthetized with pentobarbital, their heart rates are very rapid. As a result, diastole is so short that one cannot see the usual "M"-shaped appearance of the mitral valve. Lastly, I disagree with Dr. Dock regarding the recording of the posterior leaflet. With experience, the posterior leaflet can be recorded in almost every patient. The problem is recognizing the posterior leaflet and having the proper equipment for recording this structure.

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References

Myocardial Function in CAD Determined by Atrial Pacing

To the Editor:

Dr. Linhart reports on the use of atrial pacing in the assessment of myocardial function in patients with coronary artery disease (Circulation 44: 203, 1971). This technique of determining ventricular function curves, as previously described,1,2 relates alterations in left ventricular end-diastolic pressure to alterations in left ventricular stroke work as the heart rate is increased by atrial pacing. The author has demonstrated impairment of left ventricular function in nine of 11 patients during pacing-induced angina pectoris. In five of these patients abnormal pacing ventricular function curves were demonstrated prior to the onset of angina. Abnormal ventricular function curves were also demonstrated in five of 10 patients who did not develop angina during myocardial ischemia.

Our studies during pacing-induced ischemia show a variable hemodynamic response.3,4 While on the average left ventricular end-diastolic pressure is unchanged in contrast to the marked decline seen in normal subjects, in some patients the left ventricular filling pressure does decline, while in others it will rise to grossly abnormal levels. Our studies with atrial pacing in patients who had proven coronary disease but who did not develop clinical or metabolic evidence of myocardial ischemia during atrial pacing have shown a regular fall in left ventricular end-diastolic pressure and a normal pacing ventricular function curve. The patients whom we have studied in these nonangina groups all have had normal left ventricular end-diastolic pressures during the control period and had not significant abnormalities on left ventriculography. Of 10 patients reported by Dr. Linhart with abnormal ventricular function curves in the absence of chest pain, eight had significant abnormalities on left ventriculography and seven had left ventricular end-diastolic pressures exceeding 13 mm Hg.

In a recent publication5 we have assessed left ventricular function by atrial pacing in 21 normal subjects and in a group of patients with elevated left ventricular end-diastolic pressure. This latter group included 11 with coronary disease and two with primary myocardial disease. None of the patients with coronary disease developed angina

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or evidence of myocardial ischemia during rapid atrial pacing. Ventricular function curves were drawn relating stroke volume index to left ventricular end-diastolic pressure, and for any change in stroke volume index, there was a greater decrease in left ventricular end-diastolic pressure in the normal subjects than in the patients with elevated left ventricular end-diastolic pressure. There was however considerable overlap between the normal and abnormal patients. This type of analysis, however, was extremely useful in demonstrating alterations in ventricular function as induced by myocardial ischemia, digitalis glycosides, and isoproterenol.

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The authors reply:

To the Editor:

As Dr. Parker mentions, an impairment of left ventricular function, as determined by atrial pacing, is usually readily apparent during angina pectoris. However, there is some question regarding the value of atrial pacing in assessing myocardial function in the absence of induced angina. In our experience, abnormal pacing ventricular function curves without induced angina have been disclosed in 18 of 34 patients, and we consider these quite helpful in patient evaluation.

Both Parker and Bahler believe that exercise, probably because the left ventricle is subjected to greater stroke work, more graphically illustrates ventricular dysfunction. It appears from the studies performed so far that exercise is more sensitive than pacing in detecting these abnormalities. This is probably best illustrated by comparing Dr. Parker’s and our coronary patients who do not develop clinical evidence of ischemia during pacing. All of his patients had normal control LVEDP and left ventriculograms. Most of our patients, who apparently come to us later in the course of their disease, had control pressure and angiographic abnormalities. One might expect the latter patients to have abnormalities of function while the former might be normal, as in some of our patients, until angina is induced by sufficient stress.

Sufficient stress in patients whose myocardial function is only mildly depressed may necessitate the use of exercise studies. In Dr. Parker’s study of coronary disease patients with elevated control LVEDP, although some overlap existed, the pacing ventricular function curve slopes for these patients were significantly different from those in his normal patients (P < 0.001). He therefore has had some results similar to our own in patients with significant coronary artery disease, elevated LVEDP, and left ventricular angiographic abnormalities in the absence of angina pectoris.

Since a good correlation frequently does not exist between the extent of coronary artery disease demonstrated angiographically and its functional significance, some form of stress is required during hemodynamic evaluation. The relative ease of atrial pacing makes this method ideal, if we bear in mind some of its limitations, as previously noted, when compared to exercise. In our institution, we have found that by combining precatheterization treadmill exercise with atrial pacing during catheterization, we have a satisfactory means of assessing myocardial performance (J. W. Linhart, B. M. Beller, R. C. Talley: Unpublished observations).

Atrial pacing may also be used in clinical situations where one may be loath or unable to exercise patients, such as those with arthritis, neurologic disorders, claudication, after a recent myocardial infarction, or in association with “preinfarction” angina, since one need not always induce pain to obtain ischemic ECG changes or to disclose abnormal myocardial performance. The ability to precisely control the heart rate, and, other things being equal, the stress applied, is certainly important in studies utilizing drug intervention where pacing may be repeated frequently or in postoperative comparisons.

In addition, since the patient need not be aware of the initiation of pacing, the psychic components associated with exercise are lacking, and a more objective evaluation can be made in patients with atypical chest pain, those with normal coronary arteries, and in patients who might be malingering. The lack of generalized

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