rected sensing problem with CPI pacemakers is concerned, those units already had high impedance circuitry as Dr. Furman points out, rather deflecting his argument that increasing sensing circuit impedance is a cure-all. In fact, the sensing problem with the CPI units was solved by widening the band width of the sensing circuit amplifier. The accompanying increase in sensing circuit impedance was based on the same assumption, unsupported by data, that Dr. Furman is now making.

The fact that "the industry" is now straying into the area of high impedance sensing circuitry in no way means that it is right, especially considering the recall record of the last three years. As pointed out in the discussion of our paper, there are many detrimental effects of high impedance circuitry which are well known to those skilled in signal processing. Based on extensive experimental data at all impedance levels and over a wide range of electrode surface areas, we stand firmly behind our statements that optimal sensing circuit impedance is in the 5,000 to 10,000 ohm range. It is the duty of those engaged in basic pacemaker research to lead the industry, not to follow it.

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Echo and Cardiomyopathy

To the Editor:

In his recent excellent review article on echocardiography,1 Doctor Popp, in contrasting constrictive pericarditis and restrictive cardiomyopathy, (p. 548), states that patients with restrictive myopathy "generally show large ventricular diameters." This conflicts with our series2-4 of seven cases of amyloid cardiomyopathy, each presenting as a restrictive cardiomyopathy, and each uniformly had small or normal left ventricular dimensions. This has been true in one case from Massachusetts General Hospital.5 In addition Doctor Chew and associates recently reported their findings in three patients with amyloid heart disease6 and all had normal left ventricular end-diastolic volumes by cineangiography.

I understand Doctor Popp's notation at the end of his article which essentially states that in such a review article, he could not refer to everyone working in the field. However, with our information on restrictive cardiomyopathy conflicting with his, perhaps he could supply a reference in his support. There may ultimately be a spectrum of findings described in this entity as further patients are evaluated, perhaps with some showing left ventricular dilatation, but at this point in time I can only quote the data available to me, albeit on a small number of patients.

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The authors reply:

Dr. Child raises an interesting point. Certainly we agree that amyloid infiltration of the heart usually is associated with a normal or small sized left ventricular chamber.1 However, the patients we refer to with "restrictive myopathy" include a much broader spectrum and amyloid heart disease is only one facet of this category. The hemodynamic picture of restricted ventricular filling may occur in idiopathic myopathies as well, and simulate some of the hemodynamic features of constrictive pericarditis. I'm sure Dr. Child is aware that "...the clinical picture of restrictive cardiomyopathy is a non-specific hemodynamic pattern of diverse causes."2 The gradual development of characteristic hemodynamics of restriction has been noted in the course of cardiomyopathy patients.3 The statement in my review that patients with restrictive myopathy "generally have large left ventricular diameters and uniformly have reduced percent change in ventricular dimension during ejection" is true in our experience.4 In our series we have two patients with amyloid infiltration and both have rather small echocardiographic ventricular dimensions. But the others have ventricular dimensions greater than the constrictive group.

We favor use of the anatomic classification of myopathies proposed by Roberts and Ferrans5 as an addition to the hemodynamic classification used traditionally.6-7 Dilated myopathies, hypertrophic myopathies, infiltrative myopathies, and nonhypertrophic, nondilated myopathies may be sorted out largely on the basis of the echocardiographic and clinical picture. The possibility that there is a rather distinctive echocardiographic pattern of infiltrative disease such as amyloidosis is a recent addition to this differentiation of myopathic forms.1

I appreciate Dr. Child calling attention to this point regarding differentiation of constrictive from restrictive heart disease. This remains a clinical problem because of the therapeutic implications of these diagnoses. The echocardiographic picture of a thickened pericardium, with normal myocardial thickness and percent change in ventricular dimension during ejection, will be seen very rarely in any form of myopathy I believe. Conversely, the opposite profile strongly will suggest cardiomyopathy. Moreover, the echocardiographic features will help the hemodynamicist and clinician come to the proper diagnosis in most cases, and this combination of methods may further our understanding of these conditions.

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Echo and cardiomyopathy.

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