Peripartal Heart Failure and Untreated Hypertension

To the Editor:

In a recent article, Fillmore and Parry presented interesting data on the natural history of peripartal heart failure. The data on the degree of hypertension in groups 3–5 was omitted, however, and this diluted the thrust of the article. It is possible that several normotensive patients with peripartal heart failure may have intrinsic myocardial disease. The excess morbidity and mortality seen in the hypertensive group, however, leads one to think that hypertension is the major contributing factor and accelerates the progression of myocardial dysfunction as demonstrated by cardiothoracic ratios in table 4 of the article.

It has been shown that untreated blood pressure elevation in excess of 140/90 carries a significant risk of excess mortality over normotensive subjects under 40 years of age. The danger of untreated hypertension as compared to the benefit derived from treatment has also been clearly defined in the VA Cooperative Study Group. Dorph et al. have also shown that treatment of hypertension favorably influences visceral organ changes, and these findings have been documented by echocardiographic studies. In the latter study and others, the authors have further shown that echocardiographic left ventricular structural and functional changes may precede electrocardiographic and roentgenographic changes. It is inconceivable, therefore, that the authors would continue to study the natural history of peripartal heart failure without controlling the factor (hypertension) that carries excess morbidity and mortality on its own right.

Chronic pressure overload appears to produce left ventricular changes in three stages: 1) a stage of contractile functional impairment (preceding hypertrophy), 2) a stage of increased myocardial mass to maintain normal myocardial function and 3) a stage of progressive deterioration of myocardial function and eventual cardiac failure. This progression has been substantiated in the animal model with spontaneous hypertension that could be equivalent to essential hypertension. Thus, available data indicate that if hypertension is untreated, the untreated patients will develop increasing cardiothoracic ratios and eventually, cardiac failure, especially in the face of subsequent pregnancies. Digitalis and diuretic therapy alone are not sufficient, unless the hypertension can be controlled with diuretics alone. Randomization of these hypertensive subjects into treated and untreated groups is no longer feasible, for this study has already shown a definite trend favoring nortensives. Possibly, if the risk of hypertension were controlled early in the course of peripartal heart failure, the associated excess morbidity and mortality could be reduced to nearly that of group 2, although it is not clear whether the treatment of hypertension could reduce the risk of sudden death seen in these patients.

I should hope that the authors would treat these hypertensive patients in order to reverse the unfavorable trend they have demonstrated so well.

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References


The authors reply:

To the Editor:

The authors wish to thank Doctors Fowler and Spodick for their comments concerning our manuscript. In response to Dr. Fowler's comments, we did not obtain hemodynamic studies during exercise either before or after operation in any of the patients with occult constrictive pericardial disease. We are in agreement that such information would aid immensely in providing additional objective evidence for hemodynamic compromise before surgery and improvement after surgery. We suspect that exercise hemodynamics would be abnormal in this group of patients, and we anticipate performing exercise studies on future patients whom we find with occult constrictive disease.

The clinical significance of the physiologic demonstrations, stated simply, is that the pericardium is abnormal. We are reluctant to say that the group of patients with occult constrictive pericardial disease represent only a group of patients where pericardial constriction has been identified early in the course of a progressive disease. We are intrigued by the possibility that some diseases of the pericardium may not be progressive to classic overt constriction, but rather, may be occult and fairly stable in their symptomatology. We are unable to say that these patients will eventually develop overt constriction and at least in some patients (those who are not operated on) there appears to have been no progression of their illness by either symptoms or hemodynamic findings. Thus, the major clinical significance of the physiologic demonstrations is to define an abnormal pericardium and to be able to differentiate patients with such an abnormal pericardium from patients with abnormal diastolic ventricular function. We are in total agreement with Dr. Fowler and wish to reemphasize the need to individualize and carefully consider surgical intervention in these patients. We have taken such patients to surgery only in situations of marked disability and the absence of other findings to account for such disability.

The borderline cardiomegaly noted in the patient population was borderline at most. Careful measurement of the cardiac silhouettes in these patients demonstrated cardiothoracic ratios of under 50% in all patients. The two patients with globular enlargement each had extremely thick pericardiums. One of these did in fact have a small pericardial effusion.

The authors agree with Dr. Spodick's comments regarding the difference in the hemodynamics of tamponade and constriction. The reference to the expansion of intravascular volume in pericardial constriction was in relation to the recognized or unrecognized case of chronic pericardial constriction that is volume depleted or over diuresed. In such a situation, as in almost any other form of chronic cardiac disease that is over diuresed, rapid expansion of intravascular volume may be lifesaving.
Peripartal heart failure and untreated hypertension.
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