We are aware that Gorlin’s formula underestimates mitral valve area in the presence of regurgitation; that is why we indicated the degree of regurgitation in our table. We consider severe mitral stenosis the dominant lesion in patients 5, 7, 9, 11, and 16 in the table. We did not consider cases with significant mitral regurgitation as having severe mitral stenosis except for patient #18 who had a flat diastolic slope on the echocardiogram. Had we excluded this last case, the message would not have been altered.

Since our paper has been published we have received personal communications from other institutions confirming our observations, albeit in a small number of cases.

We would like to reemphasize that posterior motion of the posterior mitral leaflet is seen in a small minority of cases with mitral stenosis, including moderate and severe stenosis. Echocardiographers should be aware of this in order to avoid misdiagnosis.

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Aortic Valvular Disease and Valve Replacement

To the Editor:

In their recent article discussing indices which may be helpful in predicting postoperative survival in patients with isolated aortic stenosis or insufficiency, Hirshfeld et al. have left several important questions unanswered.

1. What effect did the patient selection have on prognosis? Since patients were referred for surgery “based almost entirely on symptoms,” no data are available regarding prognostic criteria in patients with equally severe hemodynamic derangements but without symptomatology.

2. In patients with aortic regurgitation no correlation was found between survival and functional classification, whereas a significant relationship was shown between outcome and NYHA functional class in those with aortic stenosis. However, did the duration of symptoms have any effect on prognosis? Certainly those patients in whom symptoms were present for a longer time might be at higher risk of myocardial dysfunction and cardiac decompensation.

3. Though no association between hemodynamic “variables measured in the catheterization laboratory” and late survival was established, no mention was made of left ventricular systolic pressure or aortic valve area in patients with aortic stenosis. Lewis et al. have shown that patients with severe stenosis appear to be at significant risk of myocardial ischemia, particularly subendocardial ischemia. Brazier et al. have demonstrated a striking relationship in dogs with aortic stenosis between ischemia of the subendocardium and a left ventricular oxygen supply/demand ratio expressed as DPTI X C/SPTI. Where DPTI = diastolic pressure-time index, C = arterial oxygen content and SPTI = systolic pressure-time index (also known as the tension-time index). Ratios below 10 were associated with significant reductions in flow to the subendocardium. In valvar aortic stenosis the increase in left ventricular systolic pressure compromises subendocardial flow and lowers the supply/demand ratio by increasing SPTI. Severe aortic insufficiency may also cause subendocardial ischemia by reducing coronary arterial perfusion pressure during diastole and is thereby reflected by a fall in DPTI. The association between LVEDP and survival in patients with AI is compatible with this viewpoint since an increase in LVEDP produces a fall in DPTI and the supply/demand ratio. Thus, analysis of the oxygen supply/demand ratio in the group described by Hirshfeld et al. may be helpful in predicting those patients in whom myocardial ischemia may adversely affect their prognosis. Furthermore, the application of such an index to asymptomatic patients may identify individuals at risk of subendocardial ischemia from their aortic valve lesion prior to the onset of symptomatology. Early surgery in this group may avoid or lessen the incidence of late postoperative mortality.

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References


The authors reply:

Dr. Lewis’ first question is of great importance, and was carefully dealt with in our manuscript. At the National Heart and Lung Institute, the decision to proceed with operation in patients with aortic valvular disease is based on the presence of certain symptoms. Hence, the preoperative indices we found to be of value in predicting postoperative prognosis probably can be used prospectively to determine postoperative risk in those patients who satisfy our symptomatic criteria for operability, but very likely would not be applicable to those patients operated upon who have minimal or no symptoms.

We deliberately did not attempt to analyze the duration of symptoms in our patient population because of the retrospective nature of the study. The assessment of symptomatology is at best subjective. An attempt to assess the course of symptoms retrospectively would contain too many ambiguities to be meaningful.

In the analysis of the patients with aortic stenosis, both preoperative left ventricular systolic pressure and aortic valve area were examined as potential postoperative prognostic indicators. Neither proved to have prognostic value. We doubt that the left ventricular oxygen supply/demand ratio, as calculated by Dr. Lewis, has any relevance to postoperative prognosis in aortic valvular disease. First of all, the ratio which was developed from acute experiments in dogs, is an oversimplification of the complex variables operative in patients with chronic aortic valvular disease. It omits the influence which wall thickness and left ventricular cavity dimensions have on myocardial wall tension and coronary flow distribution. It also does not consider possible changes in the oxyhemoglobin dissociation curve (found to be present in some patients with cardiac decompensation).
Letter: Aortic valvular disease and valve replacement.

A Lewis

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