LETTERS TO THE EDITOR

Reduced LV Myocardial Blood Flow

To the Editor:

The interesting article on myocardial blood flow in aortic stenosis by Johnson et al.\(^1\) makes some statements that need further discussion. The authors commented that in their patients the ratio DPTI/SPTI was not related to myocardial blood flow per beat. However, that ratio was never proposed as a predictor of total myocardial blood flow by its originators: In animals, the DPTI/SPTI ratio predicts relative subendocardial blood flow (the inner:outer ratio), not total flow.\(^2\)\(^-\)\(^3\) Secondly, the authors state that there is no evidence that long standing, compensated aortic stenosis is associated with ischemia at rest, and that myocardial lactate production at rest is not encountered in these patients. It is true that there are no measurements of subendocardial blood flow in man, so there is no evidence that long standing, compensated aortic stenosis is associated with ischemia at rest, and that myocardial lactate production at rest is not encountered in these patients. It is true that there are no measurements of subendocardial blood flow in man, so that evidence of reduced subendocardial flows cannot be provided. Nevertheless, in children with aortic stenosis there is a fairly good correlation between the DPTI/SPTI ratio (or its equivalent) and changes in the resting electrocardiogram that suggest myocardial ischemia.\(^4\) Furthermore, patients with severe aortic stenosis usually have well-marked subendocardial fibrosis, and references to this appear in a recent publication.\(^6\) It is likely that if there is severe ischemia at rest for more than a short time, muscle cells will die and be replaced by fibrous tissue. Subendocardial blood flow will then be below normal but will be adequate to oxygenate the surviving muscle cells at rest. As a result there is no net myocardial lactate production at rest, but with exercise there may be angina, lactate production, and electrocardiographic signs of subendocardial ischemia due to ischemia of the remaining subendocardial muscle.

Finally, replacement of some subendocardial muscle by fibrous tissue will provide mass without a corresponding increment of myocardial oxygen consumption or blood flow. Thus, although Johnson et al. found a reduced myocardial blood flow per unit mass of left ventricle, it is possible that flow per unit mass of muscle might have been within normal limits at rest.

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References


The authors reply:
To the Editor:

We appreciate the comments by Dr. Hoffman about our discussion of the ratio DPTI/SPTI from our article on myocardial blood flow in aortic stenosis.\(^1\) Our study indicated that in a group of patients with severe aortic stenosis with normal coronary arteriograms and normal ejection fractions, mean left ventricular (LV) myocardial blood flow (MBF) per unit mass of tissue was reduced significantly below that of normal control subjects. In both groups mean LVMBF per beat was significantly related to peak left ventricular wall stress (\(r = 0.97\)). The slopes of the two regression lines were not different, but the \(y\) intercept for aortic stenosis was lower.

The ratio DPTI/SPTI was reduced in the aortic stenosis patients. However, when DPTI/SPTI in the aortic stenosis group was plotted against LVMBF per beat there was no significant relationship (\(r = 0.24\)). In reply to his four major points:

1. Although the ratio DPTI/SPTI has been shown to be a predictor of the endocardial-to-epicardial blood flow ratio,\(^7\) the technique we use to estimate regional myocardial blood flow using xenon-133 and a multi-crystal scintillation camera is unable to measure subendocardial or subepicardial blood flow.\(^8\) The reason for testing whether transmural blood flow was related with DPTI/SPTI was based on a hypothesis that since the DPTI/SPTI was reduced in the patients, low subendocardial perfusion might lower average transmural blood flow and the latter might relate to the ratio DPTI/SPTI. Even though average LVMBF per beat was significantly reduced in the aortic stenosis group, there was no significant correlation between mean LVMBF/beat and DPTI/SPTI in the individual patients. The failure to find a significant correlation between the ratio and our measurements of blood flow does not exclude the possibility that there was relative under-perfusion of subendocardium in this group of aortic stenosis patients.

2. We agree with Dr. Hoffman that at this time there are no measurements that provide direct evidence for reduced subendocardial blood flow at rest in patients with aortic stenosis.

3. For several reasons we do not think that subendocardial fibrosis can explain our finding of reduced transmural blood flow per unit mass in patients with aortic stenosis. The primary data for measuring myocardial blood flow using \(^133\)Xe consists of initial slope rate constants of \(^133\)Xe washout. A bolus injection preferentially distributes tracer to tissue with higher blood flow rates, i.e., to muscle. In addition, the washout of \(^133\)Xe from fibrous tissue is very slow and thus contributes little to the first 40 seconds of the washout curves which were measured. The measurements therefore primarily reflect blood flow per unit mass of cardiac muscle.

4. Dr. Hoffman mentions that replacement of subendocardial muscle by fibrous tissue would provide mass without a corresponding increment of myocardial oxygen consumption or blood flow. It seems unlikely to us that the amount of subendocardial fibrosis known to exist in the hearts of patients with aortic stenosis is sufficient to account for the 23% reduction in mean LVMBF per unit mass found in the aortic stenosis patients. However, our data suggested that contractility may be reduced in the patients with aortic stenosis. It is conceivable that subendocardial fibrosis might explain reduced LV performance in aortic stenosis and, indirectly lower MBF because of lowered myocardial oxygen consumption. However, recent reports of marked improvement in LV function after aortic valve replacement due to reduction in afterload and/or regression of hypertrophy make this possibility less tenable.\(^9\)\(^-\)\(^10\)

In sum, we believe that the DPTI/SPTI ratio is a valuable index relating myocardial oxygen supply and demand. Our data showing reduced mean LVMBF per unit mass in a selected group of patients with aortic stenosis provides no information concerning the possibility that the reductions in MBF were selectively greater in the subendocardium of these patients.

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References

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