JACC Vol. 41, No. 10, 2003
May 21, 2003:1848–53

REFERENCE

1. Malouf JF, Enriquez-Savano M, Pellikka PA, et al. Severe pulmonary hypertension in patients with severe aortic valve stenosis: clinical profile and prognostic implications. J Am Coll Cardiol 2002;40:789–95.

REPLY

We appreciate the interest of Dr. Rahimtoola in our study of pulmonary hypertension in patients with aortic stenosis (1), and we appreciate his comments underscoring the importance of the data. Unfortunately, it was not possible to present all the data we collected as space is limited by the *Journal*. Below are the responses to the specific queries made:

1. Left ventricular ejection fraction was derived by two-dimensional echocardiography using visual estimate in all patients and combining it to M-mode measurements in 23 patients (49%). This approach has been proven in previous studies to be highly predictive of outcome, at least as well as angiography (2).
2. The mean ± SD (range) Doppler-derived calculations of the pulmonary artery systolic pressure (PASP) assuming a mean right atrial pressure of 10 mm Hg were:

   79.5 ± 8.0 mm Hg (74–106) for the entire group
   82.0 ± 11.6 mm Hg (74–106) for the No-AVR (aortic value replacement) group
   78.8 ± 6.8 mm Hg (74–102) for the AVR group

   These data confirm, irrespective of the estimated right atrial pressure, that the pulmonary hypertension in our study population was indeed severe.

3. With regard to coronary disease, 34 patients (72%) underwent selective coronary angiography—AVR group, 32 patients (86%); No-AVR group, 2 patients (20%). These rates are well expected, as the small but definite risk of coronary angiography is not warranted in patients who either refuse surgery or are not considered candidates for surgery.

   A total of 19 patients had initially unprotected obstructive coronary artery disease—17 among the AVR-group patients, all of whom underwent coronary artery bypass graft surgery (CABG). The remaining 15 patients (all in the AVR group) had either normal or minimally diseased coronary arteries (12 patients) or patent grafts from prior CABG (3 patients). The five-year survival rate of AVR patients who underwent CABG (58%, SE 0.16) was not significantly different ($p = 0.87$) from that of AVR patients without obstructive coronary disease on angiography, including those with previous bypass and patent grafts (48%, SE 0.18). Moreover, there was no significant difference in the severity of pulmonary hypertension between these two AVR subgroups (PASP 78.4 ± 4.8 mm Hg [74–87] for isolated AVR patients vs. 79.6 ± 8.7 mm Hg (74–102) for AVR patients who needed CABG; $p = 0.9$). This suggests that the poor outcome in this subset of patients is indeed the result of pulmonary hypertension rather than that of coronary disease.

4. The five-year survival (mean ± SE) for AVR patients, including operative mortality, was 48 ± 12%; the p value in comparison to expected survival was <0.0001. Indeed, despite treatment, this group is at high risk, although less than historical controls with similar levels of pulmonary hypertension but no aortic stenosis (AS) (3). Hence, an important conclusion of our study is that it is essential that patients be operated on before they reach such a considerable level of pulmonary hypertension. Nevertheless, as operative results continuously improve, the postoperative survival excluding operative mortality, in our mind, is also important and supports hope of a better outcome for patients with AS and severe pulmonary hypertension who otherwise are at very high risk.

We appreciate the interest of Dr. Rahimtoola, which allows underscoring further the importance of detecting pulmonary hypertension in patients with aortic stenosis.

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doi:10.1016/S0735-1097(03)00324-3

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Coronary Atherosclerosis and Body Iron Stores

We read with great interest the study by Gaenzer et al. (1) in a recent issue of the *Journal*. The investigators studied associations between iron status and early functional and structural vascular abnormalities in patients with hereditary hemochromatosis and found that impaired endothelial function and increased intima-media thickness (IMT) may be associated with iron overload, with subsequent induction of oxidative stress. Gaenzer and colleagues suggested that iron-depletion therapy, which normalizes endothelial function, may reduce the increased risk of cardiovascular events.

A possible association between body iron status and the risk of coronary heart disease was first supported by findings from a Finnish study relating increased levels of both serum ferritin and dietary iron to an increased risk of myocardial infarction in men (2). It is believed that inflammation and oxidation are important mechanisms involved in the complex pathological process of atherogenesis (3). Free radical production is catalyzed and accel-