be amplified and that sympathetic block would cause a marked decrease in pulmonary artery pressure unless sympathetic innervation is minimal, as Dr. Mallampati pointed out. More likely, however, is that the patient had developed pulmonary vascular changes sufficient to preclude significant effect from sympathetic blockade. The cardiac catheterization data reveal pulmonary artery pressure greater than one-half systemic with a markedly elevated pulmonary vascular resistance. The pulmonary artery pressure was seen to be minimally responsive to the inhalation of 100 per cent oxygen or the administration of sodium nitroprusside and without calculation of the pulmonary vascular resistance it is difficult to state whether or not the pulmonary vascular resistance was affected at all. It is not surprising therefore that no effect was seen from sympathetic denervation.

The data Dr. Mallampati presents does not substantiate any of the comments regarding lack of effect of sympathetic blockade. Such data should be best obtained from an individual with normal pulmonary vasculature.

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REPLY

I would like to thank Dr. Burrows for his comments. Please allow me to make it clear that the observations published pertain to the perioperative care of an unusual case. As has been pointed out in the discussion, there were no precedents in the anaesthesia literature for comparative purposes.

The diagnosis was established by an experienced team including a clinician, an invasive cardiologist and a diagnostic radiologist. The patient had undergone both right and left heart catheterization, left ventriculogram, and echocardiography. Manual injections of contrast medium into vessels encountered during right heart catheterization revealed one anomalous pulmonary vein from the midportion of the right lung draining into the right atrium and another one from the left lung draining into the left innominate vein. An atrial septal defect or ventricular septal defect was not demonstrable by echocardiography or catheter search. Injection of large doses of contrast medium for pulmonary angiography, which Dr. Burrows refers to, is not without complications in patients with elevated pulmonary vascular resistance. Acute hypotension and bradyarrhythmias are common. This was therefore deemed an undesirable risk to the patient considering that the information obtained would not have altered the course of management. Most of Dr. Burrows' remaining comments refer to the pulmonary vascular disease in infants and children secondary to congenital heart defects. His comments on the pathologic changes in pulmonary vessels are informative.

As he points out, there is more smooth muscle in pulmonary arteries in individuals with congenital heart disease and increased pulmonary blood flow. But that pathologic finding does not tell us anything about the response of the smooth muscle to sympathetic stimulation or denervation, as compared with this response in individuals with normal pulmonary vasculature. Dr. Burrows suggests that such data should be best obtained from patients with normal pulmonary vasculature. To the best of my knowledge, no one has conducted a study to ascertain the effect of pulmonary sympathetic blockade on pulmonary haemodynamics in individuals with normal pulmonary vasculature.

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