Thorax, 1980, **35**, 318–320

Correspondence

Pulmonary hypertension accompanying ventricular septal defect and patent ductus arteriosus: management in infancy and early childhood

Sir,—We read with great interest the article by Reid and colleagues.[1] We would like to congratulate the authors for once more bringing this important topic to the attention of your readers. We would like to make two observations.

The authors are to be congratulated on using pulmonary vascular resistance as an index of pulmonary vascular disease, but it is a pity they used a fall in the ratio between the pulmonary and systemic resistance as evidence for regression in pulmonary vascular disease. There is now good evidence[2 3] in patients with pulmonary vascular disease secondary to cardiac shunts, that systemic vascular resistance rises *pari passu* with pulmonary vascular resistance. This phenomenon makes the resistance ratio a poor measure of pulmonary vascular resistance, at least when this is markedly elevated.[3]

The second point relates to conclusions. The authors suggest that the final decision will have to await the publication of larger series of primary closure of ventricular septal defect in infancy. We would like to point out that several such large series have been published.[4–8] All these reports show that the mortality associated with early primary repair of ventricular septal defect is lower than the mortality rate resulting from two-stage procedures.

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Sir,—We are well aware that most indices of pulmonary vascular disease in infants and small children are to some extent inaccurate, as in many of the calculations several assumptions, particularly oxygen consumption, have to be made. The evidence quoted (reference 3 in preceding letter) demonstrating a concomitant rise in both systemic, and pulmonary vascular resistance in patients with pulmonary vascular disease relates largely to older children than those in our series. Although the ratio between pulmonary and systemic resistance is by no means an accurate measurement we contend that serial estimations at repeat catheter studies are helpful in the overall assessment of either regression or progression of pulmonary vascular disease in young children.

We would agree that nowadays early primary repair of ventricular septal defect in infancy is preferable to the two-stage procedure. Several of the articles referred to appeared in print after submission of our article. We would still maintain, however, that in many young patients with the combined lesions of ventricular septal defect and patent ductus arteriosus accompanying pulmonary hypertension, ligation of the ductus alone is all that is necessary; we have shown quite clearly that spontaneous closure or reduction in size of the ventricular septal defect occurs frequently (22 out of 41) and that, therefore, closure of the VSD is only required in a small number of patients with the combined defects.

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