EDITORIAL

Pulmonary Atrial Banding in Babies with Large Ventricular Septal Defects

In 1952 Muller and Dammann showed that the production of pulmonary stenosis in an infant seriously ill from large ventricular septal defect resulted in palliation of many of the symptoms.1 Civin and Edwards2 had a few years earlier suggested that this might be the case, reasoning from their observations of the life history and pulmonary vasculature of individuals with large ventricular septal defect and moderate pulmonary stenosis. The important paper by Hunt and his colleagues in this issue of Circulation of the results of this procedure from the University of Minnesota documents in a valuable way the benefits from pulmonary artery banding. The large experience from Great Ormond Street Hospital in London has recently been published by Stark and associates.3

Some restrained enthusiasm for the procedure is in evidence in both papers, and I should like to comment on this specifically with regard to babies with large ventricular septal defect. In such individuals hospital mortality was less than 5% in both series. However, obviously intensive postoperative care is required, including often tracheostomy and respiratory assistance. This suggests that the procedure, although surgically simple, requires great expertise and is not suitable for use in institutions without this expertise. Occasionally ventricular septal defects close spontaneously even after banding of the pulmonary artery (see bibliography of Hunt’s paper) and still a second operation is required for relief of the surgically induced pulmonary stenosis. In a few patients in both series, inoperable multiple ventricular septal defects were encountered at the second definitive operation a few years after banding. It is true that the second stage has been admirably accomplished in most of the patients reported by Hunt and colleagues, with a hospital mortality rate of 12%. However, the previous banding occasionally made the ultimate result of definitive repair suboptimal.

Some years ago we showed that primary intracardiac repair of ventricular septal defects in babies less than 1 year of age could be done at a risk of less than 10%.4 In 1965 we reported primary repair of ventricular septal defects in babies aged 6–12 months, with a hospital mortality rate of 5%.5 Barratt-Boyes has recently successfully repaired large ventricular septal defects in the first few months of life in six babies seriously ill from their malformation (personal communication, 1970). Sloan has for some time successfully performed intracardiac repair in the first year of life.6

Because of these facts, we have banded the pulmonary artery infrequently in patients with large ventricular septal defect in recent years.
When the problem is interstitial pulmonary edema, recurrent pulmonary infections, and growth failure in the first year of life, intractable to medical treatment, primary intracardiac repair under proper circumstances is, we believe, preferable to banding and later repair. When there is evidence for increasing pulmonary vascular resistance at the age of 1 to 2 years, primary intracardiac repair without delay is clearly advisable, and the risk is low.

Hunt and colleagues refer also to the group of older children with severely elevated pulmonary vascular resistance and bidirectional shunting, and imply that banding of the pulmonary artery "may be beneficial" in this group. I would suggest that their data do not in fact indicate that banding has affected the life history of these patients. Theoretically, it should not, and probably the pulmonary artery should not be banded in that group of individuals.

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References
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