nique is mentioned, only palliative procedures.

Some years ago, after the appearance of the first papers on cava-pulmonary anastomosis (Glenn and Patiño, 1954; Glenn, 1958), we conceived the theoretical basis of the operation we report. Experimental research on dogs enabled us to check the technical feasibility of this procedure, but there were no survivals for more than a few hours, perhaps because the haemodynamic status of a normal dog heart does not allow a circulation which involves circulatory bypass of the right side of the heart. We were of the opinion that the right atrium of a normal heart could not provide the required work, whereas a hypertrophied right atrium, as in tricuspid atresia, could supply the additional work represented by a pulmonary arterial pressure higher than the left atrial pressure. However, it seemed to us indispensable to provide the right atrium with valve homografts, one inserted into the inferior vena cava at the level of the right atrium, and the other at the exit from the right atrium to the left lung, to prevent free flow between the inferior vena cava, the right atrium, and the pulmonary artery and, in this way, stasis in the lower half of the body and inadequate cardiac filling.

The pliability and plasticity of the homograft facilitates suturing; but the long-term fate of valve homografts is unknown. Rastelli, Wallace, and Ongley (1969) have reported that calcification of the aortic wall of homografts used to repair truncus arteriosus defects has occurred in each of five patients operated on, without preventing continued function of the leaflets.

Reports from other surgeons (Bigelow *et al.*, 1967; Bigelow, 1968) on the fate of aortic homografts, in place for as long as 13 years in the thoracic aorta, have indicated that the segment of aorta and aortic valves have remained functionally satisfactory with time. Indeed, secondary calcifications seem to occur only in aortic valve homografts; pulmonary valve homografts are rarely affected by these changes and they can be used not only for 'valvation' of the inferior vena cava, but also for an anastomosis between the right atrium and the proximal end of the right pulmonary artery. Barratt-Boyes *et al.* (1969) and Ross (1971) have noted that fresh aortic valve homografts are less likely to become calcified than those that are sterilized and preserved.

A homograft between the right atrium and the proximal end of the right pulmonary artery is not indispensable, because in our first patient we did not use one for want of having one small enough. The result was satisfactory two and a half years postoperatively. But we are of the opinion that the homograft is probably useful, for the postoperative course was more difficult in this patient and an inferior vena caval syndrome was observed (hepatomegaly, melaena, oligoanuria), though this last syndrome could be explained by the child's congenital bilateral hydronephrosis.

A striking fact in the postoperative course was the need to provide a large amount of fluid infusion (blood and physiological solution) and maintain a tachycardia to ensure a correct haemodynamic balance. During the first three days postoperatively we had to ensure an overcompensation and a tachycardia (about 100 to 120/min) as if the right atrium, 'ventricle-like', could supply a satisfactory left pulmonary blood flow only by ensuring a venous hyperpressure which would help filling and a tachycardia permitting a suitable flow, until a spontaneous balance was obtained. The need for a large volume of fluid infusion is well known in cava-pulmonary anastomosis and is explained by a liquid storage in the upper half of the body. This new technique, which is a double cava-pulmonary anastomosis, could only aggravate this syndrome.

Respiratory assistance should be stopped early because positive pressure prevents central venous return.

Another less explicable feature of the postoperative course was, in both cases, a right or bilateral pleural effusion which required a few pleurocenteses.

The operation is not technically difficult. We have waited for as long as 30 months after operation before reporting this technique. The immediate result is remarkable and remains satisfactory. One element remains unpredictable—the haemodynamic consequences of an eventual atrial rhythm disturbance such as an atrial fibrillation or flutter.

## INDICATIONS

The indications for this corrective procedure, though remaining limited, apply to many patients. Our first two patients were anatomically and haemodynamically privileged; they had pulmonary arteries of normal size and low pressure.

The anatomical classification of tricuspid atresia, from Edwards and Burchell (1949) (Fig. 12) and Keith, Rowe, and Vlad (1958), distinguishes two principal types—type I, with normally related great arteries, and type II, with transposition of the great arteries; and three groups in each type—group A, with pulmonary atresia, group B, with

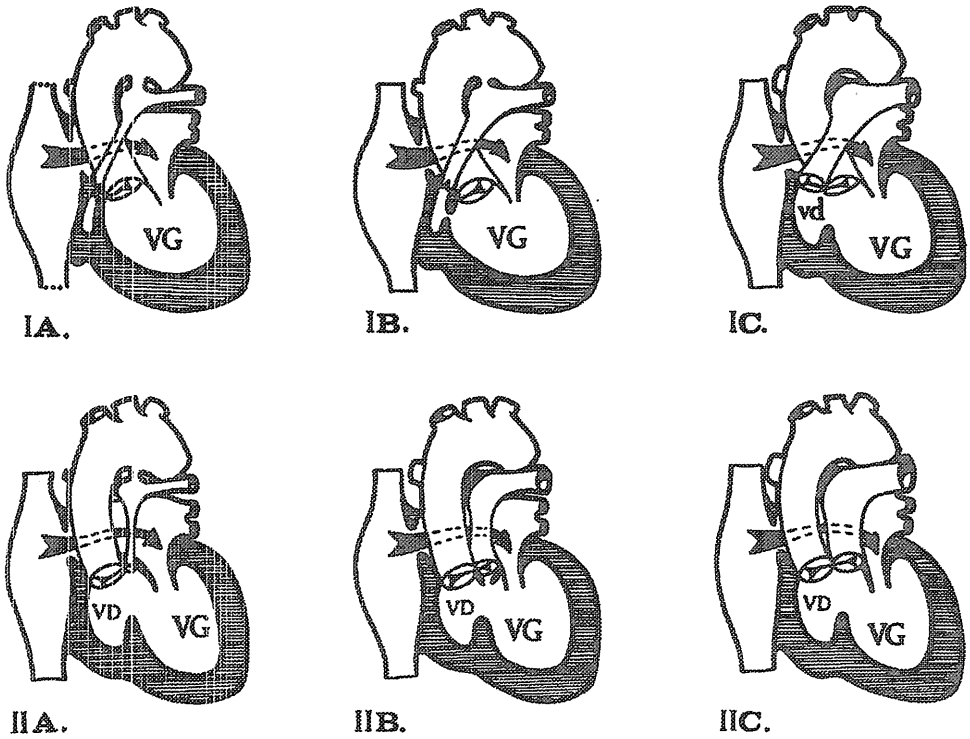


FIG. 12. *Drawing illustrates the different types of tricuspid atresia (from Edwards and Burchell, 1949).*

pulmonary valvular or subvalvular stenosis, and group C, with a normal pulmonary artery and increased pulmonary blood flow.

Most of the children with tricuspid atresia have a poor prognosis and die rather early. In these circumstances, only palliative surgical procedures can be considered (anastomosis in groups A and B and banding of the main pulmonary artery in group C), but we are of the opinion that they could profit from this corrective procedure as soon as they are older and have a bodily development compatible with the anatomical, haemodynamic, and technical necessities of this operation.

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# Ⅲ．研究成果の刊行に関する一覧

別紙４

研究成果の刊行に関する一覧表レイアウト（参考）

雑誌

| 発表者氏名 | 論文タイトル名 | 発表誌名 | 巻号 | ページ | 出版年 |
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