

# Correction of persistent truncus arteriosus

M. A. ROGERS, W. S. WINSHIP, and A. J. COLEMAN

*Thoracic Surgical and Cardiac Units, Wentworth Hospital, and the Departments of Surgery, Medicine, and Anaesthetics, University of Natal*

Successful correction of a type 2 truncus arteriosus in an African boy of 10 years is reported. The surgical technique employed is described and preoperative and late postoperative haemodynamic data are documented.

Failure of the primitive truncus arteriosus to partition results in one of a group of congenital cardiac anomalies called persistent truncus arteriosus. A single vessel, guarded by a semi-lunar valve, leaves the heart, and from this arise the coronary arteries, the aorta, and the pulmonary arterial supply. Successful correction of this congenital anomaly was first recorded in 1968 (McGoon, Rastelli, and Ongley, 1968). Wallace and his associates have reported successful total correction in four patients (Wallace, Rastelli, Ongley, Titus, and McGoon, 1968). The purpose of this paper is to record the surgical technique employed in the correction of a type 2 truncus arteriosus and to outline the preoperative and late postoperative haemodynamic state.

## CASE REPORT

An African boy aged 10 years was referred from an outlying hospital where he had been treated for pulmonary tuberculosis and congestive cardiac failure. He had suffered recurrent respiratory infections since infancy and complained of palpitations and increasing dyspnoea on effort.

His weight was 51 lb (23 kg) and he was 4 ft 5½ in (1.37 m tall). Cyanosis was not recognizable at rest but he became cyanosed, if only slightly, with exercise; the peripheral pulses were collapsing in character; the liver was palpable 2 cm below the right costal margin. There was clinical evidence of both right and left ventricular enlargement and a systolic thrill was palpable at the left sternal border. A pansystolic murmur (grade 4/6), maximal at the 4th interspace, and an early diastolic murmur along the left sternal border were audible. At the apex a mid-diastolic murmur and a third heart sound were heard. The second heart sound was accentuated and single.

The electrocardiogram showed a mean frontal plane axis of +80° and evidence of bi-ventricular hypertrophy.

Radiographically (Fig. 1a) the cardiothoracic ratio was 0.58, the left atrium was enlarged, the ascending

aorta dilated, and the pulmonary segment concave. Both pulmonary arteries were large and arose high at approximately the same level.

Cardiac catheterization demonstrated equal systolic pressures in the aorta and right and left ventricles. The right and left pulmonary arteries were entered separately from the truncus, and there was not a systolic gradient between the pulmonary arteries and the truncus arteriosus. The oxygen saturation of arterial blood was 89 vol%. The ratio of pulmonary to systemic flow was 2.3 and pulmonary resistance was 38% of systemic resistance. The left-to-right shunt was calculated to be 68% of the pulmonary venous return and the right-to-left shunt to be 27% of the systemic venous return (Table I).

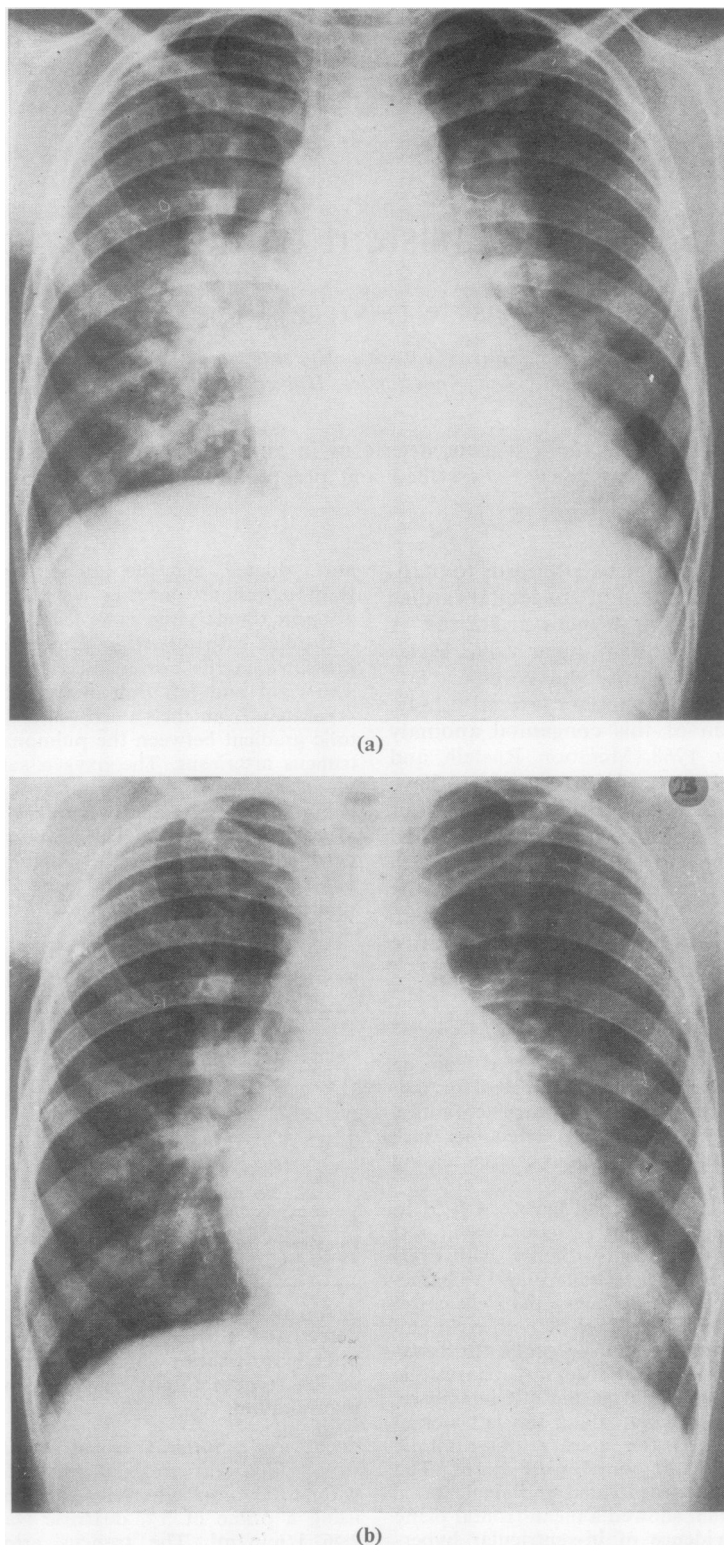
TABLE I

RESULTS OF PREOPERATIVE CARDIAC CATHETERIZATION

Site	Pressures (mmHg) SD Mean	Blood Oxygen Saturation
SVC		71
RA	5/1.5	73
IVC		74
RV	87/0-5	76
RPA	87/42	88
LA	12/6	95
Aorta	87/50	89
Systemic blood flow (l/min/m <sup>2</sup> )		5
Pulmonary blood flow (l/min/m <sup>2</sup> )		11.5
Pulmonary/systemic flow ratio		2.3
PVR/SVR %		38

Angiocardiography showed a ventricular septal defect and a truncus arteriosus with right and left pulmonary arteries which arose from the dorsal aspect of the truncus arteriosus. There was minimal aortic regurgitation.

**OPERATION** Surgical repair, through a median sternotomy, was undertaken on 25 November 1969 with total normothermic cardiopulmonary bypass, using a prime of 5% dextrose water and a flow of 2.46 l/min/m<sup>2</sup>. The truncus arteriosus was cross-clamped distal to the origin of the pulmonary arteries and the left ventricle vented through its apex. The pulmonary arteries arose separately but close together



**FIG. 1.** (a) *Preoperative postero-anterior chest radiograph. There is increased pulmonary vascularity and flattening in the region normally occupied by the pulmonary trunk. The cardiothoracic ratio is 0.58.* (b) *Post-operative chest radiograph in the postero-anterior view showing the prominent pulmonary artery segment and a marked decrease in pulmonary vascularity.*

from the dorsal aspect of the truncus arteriosus. The truncus arteriosus was transected at the level of the pulmonary arteries, the incision being so arranged that it encircled the orifices of the pulmonary arteries posteriorly. The valve which guarded the entrance to the truncus had four cusps, one being divided by a median raphe. The right coronary artery arose anteriorly and the left coronary artery posteriorly and to the right. Both coronary arteries were perfused.

The distribution of the right coronary artery was such that a right ventriculotomy for exposure of the ventricular septal defect would have resulted in division of major coronary arterial branches. The ventricular septal defect, which was typically situated immediately below the truncal valve, was therefore repaired from within the truncus arteriosus. A Dacron patch was anchored to the rim of the defect using interrupted figure-of-eight sutures. The right ventricular cavity, at the site calculated to be that from which, normally, the pulmonary artery should have arisen, was a thin-walled vestibule. The anterior wall of the right ventricle, at the site devoid of coronary vessels, was excised so as to leave an elliptical defect, approximately 3 cm in length.

A homograft which comprised ascending aorta, aortic valve, and the anterior leaflet of the mitral valve was used to fashion a pulmonary artery. The homograft had been sterilized in ethylene oxide and frozen to  $-70^{\circ}\text{C}$  for storage. The diameter of the graft at the level of the aortic annulus was 26 mm.

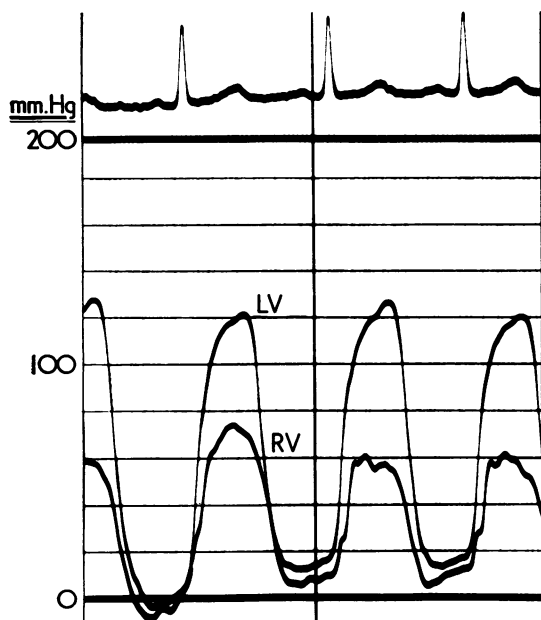


FIG. 2. A simultaneous recording of the pressures (mmHg) in the left ventricle (LV) and right ventricle (RV) three months after surgical correction.



FIG. 3. Postoperative right ventriculogram in the left lateral view showing unimpeded flow through the homograft pulmonary valve and main pulmonary artery. Note the unusually anterior position of the surgically created outlet of the right ventricle.

The distal anastomosis to the cuff of tissue which had been left surrounding the orifice of the pulmonary arteries was completed first, the homograft being orientated to allow the anterior leaflet of the mitral valve to lie anteriorly. Incorporation of the aortic leaflet of the mitral valve lent obliquity to the anastomosis between the right ventricular chamber and the homograft valve, so that the valve lay in a transverse plane. Finally, continuity of the truncus arteriosus, now ascending aorta, was achieved by end-to-end anastomosis.

Perfusion was discontinued after 120 minutes and the haemodynamic status of the patient was immediately good. However, continued bleeding from the posterior aspect of the aortic suture line necessitated the re-institution of cardiopulmonary bypass. The suture line was reinforced and finally supported by a band of Teflon felt which served also to narrow the 'truncus' and allow the aortic homograft to lie undisturbed. Thereafter the patient made an uneventful recovery.

The patient was readmitted three months after operation for restudy. He was asymptomatic and active. A grade 3/6 systolic murmur was heard, best in the pulmonary area; a soft pansystolic murmur and a short diastolic murmur were heard at the left sternal



border. Two components of the second heart sound could be clearly distinguished.

Electrocardiography showed sinus rhythm and incomplete right bundle-branch block and the mean frontal plane axis was  $+80^\circ$ .

Radiographically the cardiothoracic ratio was unchanged (0.58), there was a prominent pulmonary segment, and the lung fields appeared less plethoric than before operation (Fig. 1b).

At cardiac catheterization right ventricular systolic pressure was less than half that in the left ventricle (Fig. 2). The main pulmonary artery was entered easily and there was not a systolic gradient between the distal pulmonary arteries and the right ventricle (Fig. 3). Oxygen saturation of arterial blood was 95 vol%, of pulmonary arterial blood 79 vol%, and of mixed venous blood 76 vol%. Systemic blood flow was calculated to be 6 l/min/m<sup>2</sup> and pulmonary blood flow 7 l/min/m<sup>2</sup>. There was a left-to-right shunt of 16% at high right ventricular level. The pulmonary vascular resistance was 19% of systemic (Table II).

TABLE II

RESULTS OF POSTOPERATIVE CARDIAC CATHETERIZATION

Site	Pressures (mmHg)		Blood Oxygen Saturation
	SD	Mean	
SVC			74
RA			75
IVC	10/4	6	77
RV	58/5-12		79
MPA	58/24	36	79
RPA	58/20		79
LA	17/12	15	95
LV	130/8-14		
Aorta	130/75	96	95
Systemic blood flow (l/min/m <sup>2</sup> )			6
Pulmonary blood flow (l/min/m <sup>2</sup> )			7
Pulmonary/systemic flow ratio			1.2
PVR/SVR %			19.4

An indicator dye-dilution curve confirmed the presence of a small left-to-right shunt. There was trivial aortic regurgitation.

## DISCUSSION

Before the use of composite homografts of the ascending aorta, aortic valve, and anterior leaflet of the mitral valve for construction of the pulmonary artery, attempts at total correction of truncus arteriosus failed. Failures were from either persistent low cardiac output or continuing haemorrhage (Cooley, and Hallman, 1966; McGoon *et al.*, 1968). With the use of homografts, haemostatic suture lines are more easily

achieved and the intact aortic homograft valve spares the right ventricle the additional burden of pulmonary regurgitation in the presence of increased pulmonary vascular resistance. The surgical technique employed in this patient is essentially the same as that described by McGoon and his colleagues; the ventricular septal defect was, however, closed from within the truncus arteriosus. Haemorrhage from the posterior aspect of the aortic suture line would probably have been avoided by the insertion of a gusset of material such as Dacron, as the wall of the truncus arteriosus is normally thin and does not withstand the tension accepted by the normal aorta.

Collective studies have shown that the majority of infants with truncus arteriosus die within the first six months of life (Edwards, Carey, Neufeld, and Lester, 1965). Pulmonary flooding is the cause of death. Patients most likely to survive infancy are those with small pulmonary arteries or increased pulmonary vascular resistance. While increased pulmonary vascular resistance promotes survival beyond infancy it will also jeopardize the success of total correction.

The surgical management of patients with this anomaly therefore appears to be the same as for ventricular septal defects — banding of the pulmonary artery or pulmonary arteries in the severely ill infant, who is not a candidate for open heart surgery, in preparation for later total correction. Total correction is undertaken when the patient is large enough to allow the insertion of an aortic homograft of adequate size for adult life, provided the pulmonary vascular resistance is less than some 0.75 times the systemic vascular resistance.

## REFERENCES

- Cooley, D. A., and Hallman, G. L. (1966). *Surgical Treatment of Congenital Heart Disease*, pp. 185-189. Lea and Febiger, Philadelphia.
- Edwards, J. E., Carey, L. S., Neufeld, H. N., and Lester, R. G. (1965). *Congenital Heart Disease*, vol. 1, p. 296. Saunders, Philadelphia.
- McGoon, D. C., Rastelli, G. C., and Ongley, P. A. (1968). An operation for the correction of truncus arteriosus. *J. Amer. med. Ass.*, **205**, 69.
- Wallace, R. B., Rastelli, G. C., Ongley, P. A., Titus, J. K., and McGoon, D. C. (1968). 'Complete Repair of Persistent Truncus Arteriosus Defects.' Read at the 48th Annual Meeting of the American Association for Thoracic Surgery, Pittsburg, Pennsylvania, April 24, 1968.