Surgical treatment of persistent truncus arteriosus in the first year of life^{1, 2}

J. STARK, D. GANDHI, M. de LEVAL, F. MACARTNEY, AND J. F. N. TAYLOR

From the Thoracic Unit, The Hospital for Sick Children, Great Ormond Street, London

SUMMARY Between October 1974 and December 1977, 17 infants with persistent truncus arteriosus were admitted to this unit. Four had gross heart failure and severe metabolic acidosis. Two of these also had an interrupted aortic arch. All 4 died without operation. An urgent or semi-urgent correction was performed in 12 infants. Operations were done on cardiopulmonary bypass with deep hypothermia and circulatory arrest or periods of reduced flow. Continuity between the right ventricle and the pulmonary artery was established with a valved conduit (either fresh aortic homograft or porcine xenograft). Seven infants (aged 28 days to 8 months, weight 2.4 to 5.7 kg) survived the operation and are progressing satisfactorily. The causes of death in 5 infants are analysed. In addition, an 11-month-old infant with raised pulmonary arteriolar resistance died after operation. The lung histology showed grade III pulmonary vascular obstructive disease. The policies of medical treatment alone, palliation followed by correction, and early primary repair, are analysed on the basis of published data and our own results. It is concluded that the chances of a child surviving with a normal or near normal pressure in the pulmonary artery are best when the policy of early primary repair is followed.

Persistent truncus arteriosus is a rare congenital anomaly (Keith *et al.*, 1967): Collett and Edwards (1949) and Van Praagh and Van Praagh (1965) published detailed analyses and classifications of patients. Surgical correction of this condition was introduced in 1967 by McGoon and colleagues (1968). Provided pulmonary vascular disease is not severe, the results of correction in older children are excellent (Marcelletti *et al.*, 1977). However, most patients with persistent truncus arteriosus present in severe heart failure in the first days or weeks of life (Singh *et al.*, 1976). Many die despite intensive medical treatment. The median age at death was only 5 weeks in a series of 100 patients studied at necropsy by Calder and colleagues in 1976.

The published results of palliative surgery are also discouraging. A review by Poirier and colleagues (1975) showed that 43 out of 76 patients with persistent truncus died after pulmonary artery banding (56%). The mortality rate may be even higher as

single unsuccessful operations are not usually reported.

For these reasons, we decided in October 1974 to offer 'corrective' operations to all infants with heart failure unresponsive to medical treatment, irrespective of age and weight (Singh *et al.*, 1975). The results in the first 4 infants have already been reported (Singh *et al.*, 1976). The purpose of this paper is to review our overall experience during a 3-year period and to discuss some of the problems of treatment of persistent truncus arteriosus in the first year of life.

Subjects and results

Persistent truncus arteriosus is defined here as that condition in which a single arterial trunk leaves the heart via a single semilunar valve and which gives rise directly to the coronary, systemic, and one or both pulmonary arteries (Crupi *et al.*, 1977). Between October 1974 and December 1977, we have seen 17 infants with persistent truncus. These have been divided into three groups.

GROUP 1: DIED BEFORE REPAIR (TABLE 1)

Four patients were admitted in gross heart failure and with severe metabolic acidosis. Two

¹Presented in part at the Annual Meeting of the British Cardiac Society in London, November 1977.

²This work was supported in part by a British Heart Foundation grant.

Table 1 *Truncus arteriosus in infancy — group 1*

Age	Origin of pulmonary arteries	Additional cardiac anomalies	Comment
2 d	Pulmonary trunk	VSD + TVR + interrupted aortic arch + 'criss-cross heart'	Death at 6 days: low cardiac output
4 d	Widely separate origin from truncus at different levels	PDA + interrupted aortic arch	Death on operating table: banded PA, PDA infiltration with formaldehyde; subarachnoid haemorrhage
8 d	Pulmonary trunk	VSD + PFO	Death at cardiac catheterisation: subarachnoid haemorrhage, infarcted kidney and bowel
8 d	Pulmonary trunk	VSD + PFO	Death at cardiac catheterisation: severe acidosis

VSD, ventricular septal defect; PFO, patent foramen ovale; PA, pulmonary artery; TVR, truncal valve regurgitation; PDA, persistent ductus arteriosus.

arrested and died during cardiac catheterisation. A third patient was admitted at the age of 2 days and was found to have severe truncal valve regurgitation, with type I truncus arteriosus; in addition there was interruption of the aortic arch and 'criss-cross heart' (Anderson *et al.*, 1974). The infant was in a low output state, which probably contributed to the severe burns suffered during attempts to rewarm after cardiac catheterisation. The fourth patient had persistent truncus arteriosus with widely separated pulmonary arteries, a persistent ductus arteriosus, and interrupted aortic arch.

GROUP 2: CORRECTION FOR INTRACTABLE HEART FAILURE (TABLE 2)

An urgent or semi-urgent operation was performed in 12 infants in intractable heart failure. Seven survived the operation and are progressing satisfactorily.

A 5-day-old infant was admitted moribund in severe heart failure with gross metabolic acidosis. The arterial pH remained below 7.0 despite massive doses of sodium bicarbonate and trometamol. Intensive treatment with catecholamines, diuretics, and assisted ventilation did not improve the child's condition. She started to convulse and became

anuric. An urgent operation was uneventful despite the presence of left superior vena cava and some truncal valve regurgitation. Peritoneal dialysis was instituted immediately after operation, but despite this intensive treatment the infant deteriorated. Acidosis persisted, she developed disseminated intravascular coagulation, and died 12 hours after operation.

An 8-day-old infant had a massive intramyocardial injection during angiocardiology. Tamponade was relieved by an emergency operation, but at the completion of the repair the left ventricle did not contract and the infant died on the operating table. It was presumed that the damage to the left ventricular myocardium was responsible for this unfortunate outcome.

A 17-day-old infant died one day after operation. Excessive bleeding and partial compression of the conduit were probably major factors.

Another death occurred in a 5-month-old child with a very unusual anatomy. The right pulmonary artery, which was mistaken for a main pulmonary trunk, originated from the left side of the truncus and divided into the upper and lower lobe arteries. The operation was uneventful and the child did well. Ventricular fibrillation occurred on the fourth

Table 2 *Truncus arteriosus in infancy — group 2*

Age	Weight at operation (kg)	Origin of pulmonary arteries	Comment
5 d	3.8	Pulmonary trunk	Death: moribund on admission
8 d	3.4	Pulmonary trunk	Death on table: intramyocardial injection of contrast material
17 d	3.1	Separate	Death after 1 d
28 d	2.4	Widely separate at different level	Well at 15 m
2 m	3.5	Pulmonary trunk	Well at 2 y
2 m	3.5	Pulmonary trunk	Recovering at 1 m
3 m	3.7	Pulmonary trunk	Well
5 m	3.9	Pulmonary trunk	Well at 16 m
5 m	3.9	Interrupted LPA originated from persistent ductus arteriosus	Death after 4 d: persistent ductus arteriosus not recognised
6 m	4.7	Pulmonary trunk	Well at 3 y
8 m	5.7	Separate	Well at 3 m
9 m	6.0	Separate	Death on table: grade III pulmonary vascular obstructive disease

d, day; m, month; y, year; LPA, left pulmonary artery.

postoperative day and was resistant to treatment. At necropsy, the true situation became apparent. The interrupted left pulmonary artery arose from a persistent ductus arteriosus and not from the right pulmonary artery.

The last child, the oldest in our series, had an uneventful repair. Ventricular fibrillation occurred without warning about 30 minutes after discontinuation of cardiopulmonary bypass and all resuscitative measures were unsuccessful. Histological examination of the lungs showed grade III pulmonary vascular obstructive disease (Heath and Edwards, 1958).

GROUP 3: EARLY ELECTIVE REPAIR FOR INCREASING PULMONARY ARTERIOLAR RESISTANCE

One patient with a raised pulmonary arteriolar resistance was referred for correction at the age of 11 months. His calculated pulmonary arteriolar resistance was 10 units m^2 BSA and arterial oxygen saturation was only 80 per cent breathing air. In an older child this would suggest an inoperable condition. Because he was only 11 months old, and after much discussion, we decided to offer him a corrective procedure. The operation was uneventful but the child died 28 hours later. Histology showed grade 3 pulmonary vascular obstructive disease.

Surgical considerations

(a) TECHNIQUE OF PERFUSION

We do not believe that it is important whether cardiopulmonary bypass or a technique of deep hypothermia and circulatory arrest is used, and indeed we often combine the two techniques. Older

and larger children (over about 6 months of age and 5 kg) usually undergo operation using cardiopulmonary bypass with core cooling and one or two periods of low flow, sometimes including a short period of circulatory arrest at a low temperature. Two venous cannulae are inserted and the standard technique of cardiopulmonary bypass is employed.

In small sick infants we prefer to use surface cooling after the arterial monitoring line is inserted. The chest is opened when the nasopharyngeal temperature is lowered to 26 to 28°C. The infant is connected to the heart/lung machine using a thin walled metal cannula placed in the aorta above the origin of the pulmonary arteries and one venous cannula in the right atrial appendage. The pulmonary arteries are snared or clamped to avoid overperfusion of the lungs. Using cardiopulmonary bypass, the nasopharyngeal temperature is lowered to 19°C and the circulation stopped.

(b) OPERATIVE TECHNIQUE

The operative technique follows the principles outlined by McGoon and colleagues (1968). The aorta is cross-clamped, the root of the aorta perfused with cardioplegic solution, and then the pulmonary artery is detached from the aorta. Care should be taken when detaching the lower part of the pulmonary artery in infants as the origin of the pulmonary trunk may be close to a coronary artery orifice (Bharati *et al.*, 1974) (Fig. 1). An unusual problem was presented by an infant whose pulmonary arteries originated separately from the truncus at different levels. The origin of the right pulmonary artery was so high that it would have been difficult to cannulate the aorta above its origin (Fig. 2). The cannula was therefore placed

Fig. 1 Ostium of the left coronary artery (LCA) is situated very close to the origin of the pulmonary arteries (PA). Ventricular septal defect (VSD) and right coronary artery ostium (RCA) are seen.

Fig. 2 (a) The arterial cannula is placed between the truncal valve and the origin of the right pulmonary artery. Continuous lines indicate an oblique excision of a segment of the truncus including the origin of both pulmonary arteries. Dotted line shows the opening in the anterior wall which will be used for the anastomosis with the conduit. (b) Anastomosis between the excluded segment of the truncus and the valved conduit is completed. Division of the aorta facilitates access to the anastomosis. (c) Aortic continuity is re-established by end-to-end anastomosis. Air is evacuated from the aorta and aortic clamp released.

between the truncal valve and the origin of this pulmonary artery. Under circulatory arrest, the truncus was transected above and below the origins of the pulmonary arteries and a cuff of the wall opened for the anastomosis with the conduit. Aortic continuity was re-established by an end-to-end anastomosis (Fig. 2).

The ventriculotomy is vertical pointing towards the pulmonary artery. The ventricular septal defect is closed with a generous patch of Dacron velour. The patch is brought up to the anterior right ventricular wall to provide a large ventricular outflow for the aorta (Fig. 3). It also serves to reinforce the attachment of the conduit to the right ventricle later. An extracardiac valved conduit is then used to establish continuity between the right ventricle and pulmonary artery. The anastomosis of the conduit to the pulmonary artery is performed using a continuous stitch of 5-0 Prolene. When this anastomosis has been completed, the foramen ovale is closed through the right atrium. If the operation is being performed using circulatory arrest with one venous cannula, it is possible to place two venous cannulae once the ventricular septal defect has been closed. The aortic clamp can then be removed and the patient completely rewarmed while the conduit is being anastomosed to the right ventricle. Time is thus saved and when the right atrium is closed and the anastomosis of the conduit to the right ven-

tricle completed, perfusion can be terminated.

There are a few other technical points which should perhaps be emphasised. The conduit is not trimmed too short, so as to allow for growth of the patient. It is cut obliquely at the ventricular end to achieve a wide opening without a gradient (Fig. 4). We routinely resect the thymus to make more room for the conduit and also open the pericardium and pleura widely behind the heart. The heart then rotates to the left and allows the conduit to move away from the midline. Compression of the conduit by the sternum is thus avoided.

Perioperative care

Intensive care is required throughout all stages of treatment, that is before, during, and after operation. It does not differ from the care used for other ill infants with congenital heart defects. Intermittent positive pressure ventilation is used for at least 24 hours after operation and weaning from the ventilator is aided by using constant positive airway pressure with spontaneous breathing.

Discussion

The results of early 'correction' of persistent truncus arteriosus are encouraging. After initial successful case reports (Girinath, 1973; Kirklin,

Fig. 3 Closure of the ventricular septal defect. The patch is brought up to the anterior right ventricular wall. This creates a large outflow from the left ventricle to the aorta and the patch can be used to anchor the posterior part of the conduit to the right ventricle. Anastomosis of the conduit with the pulmonary artery is started.

Fig. 4 The conduit is trimmed obliquely at the ventricular end (see insert) to achieve a wide anastomosis. The anastomosis between the conduit and the right ventricle is done using 4-0 Prolene.

1973; Singh *et al.*, 1975; Sullivan *et al.*, 1976), Stanger and colleagues (1977) reported a series of 21 infants with a remarkable result. Eighteen out of 23 infants (79%) survived the operation. Persistent truncus arteriosus is sufficiently uncommon for it to be impossible with any accuracy to compare the alternative management policies of (a) medical treatment in infancy followed by corrective surgery at the age of 5 years, (b) banding in infancy followed by debanding and corrective surgery later, and (c) primary correction in infancy. It is nevertheless instructive to ask, on the basis of such information as is available, how many patients out of 100 can be expected to be alive and well at 9 to 10 years of age with each of these management policies. The rough comparison in Table 3 is based on the following assumptions:

(i) 20 per cent of all infants with persistent truncus arteriosus survive the first year of life on medical

treatment. The actual figures quoted are 0 (Marcelletti *et al.*, 1976), 15 per cent (Keith *et al.*, 1967), and 30 per cent (Nadas and Fyler, 1972).

(ii) The immediate mortality of banding of the pulmonary artery is 60 per cent. This is based on the figures of 56 per cent reported by Poirier *et al.* (1975) and 73 per cent by Singh *et al.* (1975).

(iii) 10 per cent of all patients who have had pulmonary artery banding in infancy are inoperable at 5 years. The figures reported by McFaul *et al.* (1976) were 1/22 (4.5%) for patients with two pulmonary arteries and 3/5 (60%) for patients with one pulmonary artery.

(iv) The immediate mortality for correction of truncus arteriosus after pulmonary artery banding is 11 per cent. Parker *et al.* (1975) reported an overall mortality of 20 per cent, but a 6 per cent mortality in a group of 17 children operated more recently.

(v) Of those patients who survive infancy, only half

Table 3 Comparison of three management policies for patients with persistent truncus arteriosus

Primary correction at 5 years		Pulmonary artery banding in infancy; debanding and correction at 5 years		Primary correction in infancy and conduit replacement at 5 to 8 years*	
Born	100	Born	100	Born	100
Die < 1 year	80	Die at banding	60	Die at correction	40
Inoperable at 5 years	10	Inoperable at 5 years	4	Die at conduit replacement	2
Die at correction	1	Die at correction	6		
Alive and well at 9 years	9		30		58

*Deaths in infancy without operation are not included.

are suitable candidates for elective surgery at the age of 5 years. Of 70 patients investigated since 1967 at the Mayo Clinic, 40 were regarded as operable (Mair *et al.*, 1974), though several of these would not be deemed operable by current criteria.

(vi) The immediate mortality of surgical correction in infancy is 40 per cent. This is based upon 5/23 (Stanger *et al.*, 1977), 5/7 (Appelbaum *et al.*, 1976), and 6/13 in this series.

(vii) The immediate mortality of conduit replacement in childhood after primary 'correction' in infancy is 3 per cent. This is derived from the mortality for replacement of obstructed conduits of 0/17 in older children (0%) (Moodie *et al.*, 1976).

This analysis does not include late mortality after primary correction in later childhood, which has been reported and which is particularly common in patients with pre-existing pulmonary vascular disease (Marcelletti *et al.*, 1977). However, the late mortality after correction in infancy is not yet known. As is clear from this report, some infants are so sick that they do not even reach the operating table alive. These infants appear in Table 3 under the heading of 'Primary correction at 5 years' as deaths under 1 year, but not under the headings of 'Banding in infancy' and 'Correction in infancy.' Even so, the difference in overall survival between medical and surgical treatment in infancy is so great as to rule out medical treatment alone in all but the few cases who respond rapidly to a moderate anti-failure regimen.

Accepting that surgical treatment is inevitable in patients in intractable heart failure, the difference between the policy of the two-stage correction and early primary correction (Table 3) is remarkable. Almost twice as many children can be expected to be alive and well using the latter approach. With increased experience, further reduction of operative mortality of early repair can be expected.

The type of valved conduit to be used is still open to discussion. Both homografts and heterografts have advantages and disadvantages (Table 4). Late complications, namely severe calcification and stenosis of the homograft conduit (Moodie *et al.*, 1976), led to the development of alternative materials. At the present time, a Dacron conduit containing a porcine valve is favoured by most surgeons. It is, however, probable that the complications of aortic homografts result from the technique of sterilisation and preservation rather than the properties of the homograft itself. Those which were irradiated and deep-frozen showed a high incidence of obstruction, but this was considerably greater for patients with complex transpositions (22%) than for patients with truncus arteriosus (3%) (Moodie *et al.*, 1976). Homografts preserved fresh in an antibiotic solution functioned well up to 9 years in patients operated upon by Ross (Moore *et al.*, 1976). We believe that the aortic homograft extended towards the right ventricle with a woven Dacron tube is the most suitable conduit particularly in infancy. We find it easier to suture the homograft to the small pulmonary artery in an infant, and also we can fit in a larger homograft than the more rigid 'Hancock' heterograft. Using an 18 to 22 mm homograft in infants, it is conceivable that replacement may not be necessary in the future.

The optimal timing of early elective repair is another unsolved problem. There are no difficulties in patients from groups 1 and 2 who were either desperately ill or in heart failure that was completely refractory to medical treatment. An urgent or semi-urgent operation is indicated, as any longer delay could be detrimental (group 1). The question remains as to when one should operate on patients

Table 4 Valved conduit

	For	Against
Homograft:	Cheap	Irradiated and frozen calcify
	Larger size fits Easier distal anastomosis	Availability
Heterograft:	Availability Quality control	Expensive Bulky in infants Gradients in small sizes

Table 5

PERSISTENT TRUNCUS ARTERIOSUS	
↓	
EARLY INVESTIGATION	
↓	↓
INTRACTABLE CHF	MEDICALLY CONTROLLED CHF
↓	↓
'CORRECTION'	ELECTIVE SURGERY
Irrespective of age and weight	At 12 to 24 months* (as for VSD)

*This age may need revision subsequent to further experience.
CHF, congestive heart failure; VSD, ventricular septal defect.

whose heart failure is controlled medically, but who have systemic pressure in the pulmonary arteries and are, therefore, at risk of early development of pulmonary vascular obstructive disease. It is certain that the age of 5 years suggested earlier (Marcelletti *et al.*, 1977) is far too late. Though the risk of correction for patients with low pulmonary vascular resistance will be very low at this age, too many patients will be inoperable by that time. One possible approach is to follow the criteria outlined for infants with ventricular septal defect by Blackstone *et al.* (1976). The initial operative mortality, late risk, and survival with normal or near normal pulmonary artery pressure should be taken into consideration. Stanger and colleagues (1977) recommended elective correction before the age of 6 months because of their observation of severe pulmonary vascular obstructive disease in patients aged 6 to 12 months. In their series, the mortality rate was 13 per cent (2 out of 15) under 6 months of age, and 37 per cent (3 out of 8) in the 6 to 12 month age group. In the latter group, pulmonary vascular obstructive disease was a significant factor. Our present policy (Table 5) is to correct asymptomatic patients between the age of 1 and 2 years, but we are constantly reviewing our experience and accept that we might have to alter this policy and operate earlier.

In conclusion, we think that infants with persistent truncus arteriosus and additional complex intracardiac anomalies and/or truncal valve regurgitation are likely to be at a very high risk irrespective of the type of treatment offered. Those who present with 'uncomplicated' persistent truncus arteriosus are most likely to benefit from the policy of early repair. It is important in determining a policy for the treatment of infants with persistent truncus arteriosus that one considers *all* patients born with this anomaly and not only those admitted for

operation. Furthermore, the object of treatment is far more than merely to achieve survival. Our aim should be normally active children with a normal pulmonary artery pressure and no residual defect.

References

- Anderson, R. H., Shinebourne, E. A., and Gerlis, L. M. (1974). Criss-cross atrioventricular relationships producing paradoxical atrioventricular concordance or discordance. Their significance to nomenclature of congenital heart disease. *Circulation*, **50**, 176-180.
- Appelbaum, A., Barger, L. M., Jr., Pacifico, A. D., and Kirklin, J. W. (1976). Surgical treatment of truncus arteriosus with emphasis on infants and small children. *Journal of Thoracic and Cardiovascular Surgery*, **71**, 436-440.
- Bharati, S., McAllister, J. A., Jr., Rosenquist, G. C., Miller, R. A., Tatroles, C. J., and Lev, M. (1974). The surgical anatomy of truncus arteriosus communis. *Journal of Thoracic and Cardiovascular Surgery*, **67**, 501-510.
- Blackstone, E. H., Kirklin, J. W., Bradley, E. L., DuShane, J. W., and Appelbaum, A. (1976). Optimal age and results in repair of large ventricular septal defects. *Journal of Thoracic and Cardiovascular Surgery*, **72**, 661-679.
- Calder, L., Van Praagh, R., Van Praagh, S., Sears, W. P., Corwin, R., Levy, A., Keith, J. D., and Paul, M. H. (1976). Truncus arteriosus communis: clinical, angiographic, and pathologic findings in 100 patients. *American Heart Journal*, **92**, 23-38.
- Collett, R. W., and Edwards, J. E. (1949). Persistent truncus arteriosus: a classification according to anatomic types. *Surgical Clinics of North America*, **29**, 1245-1270.
- Crupi, G., Macartney, F. J., and Anderson, R. H. (1977). Persistent truncus arteriosus, a study of 66 autopsy cases with special reference to definitions and morphogenesis. *American Journal of Cardiology*, **40**, 569-578.
- Girinal, M. R. (1973). Case presentation: truncus arteriosus: repair with homocraft reconstruction in infancy. In *Heart Disease in Infancy: Diagnosis and Surgical Treatment*, p. 234. Ed. by B. G. Barratt-Boyes, J. M. Neutze, and E. A. Harris. Churchill Livingstone, Edinburgh.
- Heath, D., and Edwards, J. E. (1958). A pathology of hypertensive pulmonary vascular disease. A description of six grades of structural changes in the pulmonary artery with special reference to congenital cardiac septal defects. *Circulation*, **18**, 533-547.
- Keith, J. D., Rowe, R. D., and Vlad, P. (1967). *Heart Disease*

- in *Infancy and Childhood*, 2nd ed., p. 770. Macmillan, New York.
- Kirklin, J. W. (1973). Surgical treatment of patients with absence of direct anatomic continuity between pulmonary arterial system and the heart and ventricular septal defect. In *Heart Disease in Infancy. Diagnosis and Surgical Treatment*, p. 219. Ed. by B. G. Barratt-Boyes, J. M. Neutze and E. A. Harris. Churchill Livingstone, Edinburgh.
- McFaul, R. C., Mair, D. D., Feldt, R. H., Ritter, D. G., and McGoon, D. C. (1976). Truncus arteriosus and previous pulmonary arterial banding: clinical and hemodynamic assessment. *American Journal of Cardiology*, **38**, 626-632.
- McGoon, D. C., Rastelli, G. C., and Ongley, P. A. (1968). An operation for the correction of truncus arteriosus. *Journal of the American Medical Association*, **205**, 69-73.
- Mair, D. D., Ritter, D. G., Davis, G. D., Wallace, R. B., Danielson, G. K., and McGoon, D. C. (1974). Selection of patients with truncus arteriosus for surgical correction. Anatomic and hemodynamic considerations. *Circulation*, **49**, 144-151.
- Marcelletti, C., McGoon, D. C., Danielson, G. K., Wallace, R. B., and Mair, D. D. (1977). Early and late results of surgical repair of truncus arteriosus. *Circulation*, **55**, 636-641.
- Marcelletti, C., McGoon, D. C., and Mair, D. D. (1976). The natural history of truncus arteriosus. *Circulation*, **54**, 108-111.
- Moodie, D. S., Mair, D. D., Fulton, R. E., Wallace, R. B., Danielson, G. K., and McGoon, D. C. (1976). Aortic homograft obstruction. *Journal of Thoracic and Cardiovascular Surgery*, **72**, 553-561.
- Moore, C. H., Martelli, V., and Ross, D. N. (1976). Reconstruction of right ventricular outflow tract with a valved conduit in 75 cases of congenital heart disease. *Journal of Thoracic and Cardiovascular Surgery*, **71**, 11-19.
- Nadas, A. S., and Fyler, D. C. (1972). *Paediatric Cardiology*, 3rd ed., p. 665. Saunders, Philadelphia.
- Parker, R. K., McGoon, D. C., Danielson, G. K., Wallace, R. B., and Mair, D. D. (1975). Repair of truncus arteriosus in patients with prior banding of the pulmonary artery. *Surgery*, **78**, 761-767.
- Poirier, R. A., Berman, M. A., and Stansel, H. C., Jr. (1975). Current status of the surgical treatment of truncus arteriosus. *Journal of Thoracic and Cardiovascular Surgery*, **69**, 169-182.
- Singh, A. L., de Leval, M. R., Pincott, J. R., and Stark, J. (1976). Pulmonary artery banding for truncus arteriosus in the first year of life. *Circulation*, **53** and **54**, Suppl. III, 17-19.
- Singh, A. K., de Leval, M. R., and Stark, J. (1975). Total correction of Type I truncus arteriosus in a 6-month old infant. *British Heart Journal*, **37**, 1314-1316.
- Stanger, P., Robinson, S. J., Engle, M. A., and Ebert, P. A. (1977). 'Corrective' surgery for truncus arteriosus in the first year of life (abstract). *American Journal of Cardiology*, **39**, 293.
- Sullivan, H., Sulayman, R., Replogle, R., and Arcilla, R. A. (1976). Surgical correction of truncus arteriosus in infancy. *American Journal of Cardiology*, **38**, 113-116.
- Van Praagh, R., and Van Praagh, S. (1965). The anatomy of common aortopulmonary trunk (truncus arteriosus communis) and its embryologic implications. *American Journal of Cardiology*, **16**, 406-425.

Requests for reprints to Dr J. Stark, Thoracic Unit, The Hospital for Sick Children, Great Ormond Street, London WC1N 3JH.