Results of total correction of the tetralogy of Fallot

Long-term haemodynamic evaluation at rest and during exercise

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Postoperative haemodynamic evaluation was performed on 15 patients 7 to 14 years (mean, 11 years) after total intracardiac repair of Fallot's tetralogy. The average age of the patients at the time of postoperative catheterization was 20 years (range 12 to 29) and they were all asymptomatic. Before operation 11 patients were severely cyanosed and disabled and 4 had mild cyanosis with anoxic spells.

The cardiac output and right heart pressures were measured at rest and after 5 minutes of submaximal exercise on a bicycle ergometer. It was shown that the degree of residual obstruction of the pulmonary outflow tract was slight and that the right ventricular/pulmonary arterial systolic gradient (RV/PA gradient) exceeded 20 mmHg (2.6 kPa) at rest in only 3 patients. The mean cardiac index at rest was $5.4 \text{ l min}^{-1} \text{ m}^{-2}$ which increased to $8.3 \text{ l min}^{-1} \text{ m}^{-2}$ during exercise. The response of the cardiac output during exercise was either normal or supranormal in 14 of the 15 patients. In the remaining patient the response was marginally below the normal range.

Pulmonary valvular regurgitation was present in 8 patients but there was no detectable difference in the cardiac function of these patients compared with patients with competent pulmonary valves.

Six of the patients investigated had also had cardiac catheterizations performed 1 to 4 years after operation. The results of the present study show that remarkably little change has occurred in their haemodynamic status in the intervening years. Though these results are encouraging, further studies are required to determine the eventual outcome of the RV pressure and volume overload.

Total intracardiac correction of the tetralogy of Fallot is now well established as a highly successful surgical procedure (Kirklin and Karp, 1970). The symptomatic and clinical improvement which occurs after operation is usually dramatic. It is not surprising, however, that residual haemodynamic abnormalities have been shown by postoperative

catheterization studies (Hallidie-Smith *et al.*, 1967; Soulié *et al.*, 1971; Sunderland *et al.*, 1973; Kaplan *et al.*, 1973; Gersony *et al.*, 1973).

Two main abnormalities have been described:

- (1) Residual obstruction of the right ventricular outflow tract which is usually mild or moderate in degree.
- (2) Pulmonary valvular regurgitation, particularly when a prosthetic or pericardial patch is used to enlarge the right ventricular outflow tract and the pulmonary artery.

In a minority of patients the persistence of a shunt through a ventricular septal defect has been shown.

Many of the postoperative studies were done relatively soon after total correction and there is an

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obvious need to determine the long-term effects of the residual haemodynamic defects. In addition, the majority of follow-up catheterizations did not examine the effects of exercise on the right ventricular outflow tract gradient and the cardiac output. The haemodynamic response to submaximal exercise in the supine position has been reported (Malm *et al.*, 1966; Gotsman, 1966; Bristow *et al.*, 1970; Joransen and Moller, 1972; Shah and Kidd, 1966) and intense exercise in the upright position has been the subject of one report (Epstein *et al.*, 1973).

The present studies were undertaken to evaluate the long-term haemodynamic results at rest and during exercise in 15 patients approximately 11 years after total correction. It was also possible to make an assessment of the serial haemodynamic changes in 6 patients who had been studied 1 to 4 years after operation (Gotsman, 1966).

Patients and methods

Fifteen patients, 8 male and 7 female, were investigated 7 to 14 years after total correction of Fallot's tetralogy, and in 11 patients the interval was greater than 10 years. Their average age at the time of investigation was 20 years (range 12 to 29 years) and they were all asymptomatic and leading normal lives. They were selected because they were the earliest survivors of the total correction operation in our unit. The purpose and nature of the investigation was fully explained to each patient or patients as appropriate and informed consent for its performance obtained.

Before operation 11 patients were severely cyanosed and 4 had mild cyanosis with anoxic spells. The diagnosis was based on the clinical signs, haemodynamic and angiographic investigation, and the findings were confirmed at operation.

Operation details

Five patients had preliminary palliative operations, viz. a pulmonary valvotomy in 3 and a Blalock anastomosis in 2. At the time of total correction pulmonary infundibulectomy was done in all 15 patients and in addition 8 patients had a pulmonary valvotomy. The ventricular septal defect was repaired with a 'teflon' patch in 9 patients and in the other 6 patients the defect was closed by direct suture. The right ventricular outflow tract was widened with a 'teflon' prosthetic patch in 7 patients and in 2 of these the prosthetic patch was extended to widen the proximal pulmonary artery.

Procedure for cardiac catheterization

Right heart catheterization was performed in the supine position and in the fasting state without earlier sedation. A No. 9 double-lumen Cournand catheter was used in most studies and the intracardiac pressures were measured by Sanborn transducers (Model 267 BC) and recorded on an ultraviolet photographic recorder. The pulmonary arterial and right ventricular pressures were determined by planimetry over several respiratory cycles. The right ventricular end-diastolic pressure (RVEDP) was measured after the 'a' wave. The zero reference level for pressure was 10 cm above the plane of the catheterization table. The oxygen saturation of blood samples from the superior vena cava, inferior vena cava, right atrium, right ventricle, pulmonary artery, and brachial artery was determined by a spectrophotometric method. The cardiac output was measured by the direct Fick method. The expired air was collected in a Tissot spirometer while samples of blood were withdrawn from the pulmonary artery and brachial artery.

TABLE 1 Postoperative data in 15 patients with tetralogy of Fallot

	Rest Mean	Range	Exercise Mean	Range
Mean RAP (mmHg)	5	-1-10	7	1-10
RVSP (mmHg)	39	22-61	54	23-83
RVEDP (mmHg)	6	0-13	7	-2-12
PASP (mmHg)	24	14-36	33	18-49
PADP (mmHg)	8	-1-18	13	5-22
Mean PAP (mmHg)	15	6-26	22	8-33
RV/PA systolic pressure gradient (mmHg)	15	1-46	23	3-59
PAWP (mmHg)	8	0-19	13	4-30
BA systolic (mmHg)	128	99-150	160	120-181
BA diastolic (mmHg)	84	67-102	101	72-119
PVR (dynes·s·cm ⁻⁵)	87	36-175	65	11-141
Systemic resistance (dynes·s·cm ⁻⁵)	1080	508-1724	956	487-899
Systemic arterial oxygen saturation (%)	95	88-99	95	86-98
AV oxygen difference (vol %)	3.3	2.1-4.8	8.0	5.4-11.3
Cardiac index (l min ⁻¹ m ⁻²)	5.4	3.2-8.5	8.3	5.1-11.8
Heart rate (beats/min)	88	72-108	135	108-166
Stroke volume (ml/beat)	96	62-185	100	61-173
Oxygen consumption (ml min ⁻¹ m ⁻²)	153	113-192	597	444-755

Abbreviations: RAP, right atrial pressure; RVSP, right ventricular systolic pressure; RVEDP, right ventricular end-diastolic pressure; PASP, pulmonary artery systolic pressure; PADP, pulmonary artery diastolic pressure; PAP, pulmonary artery pressure; PAWP, pulmonary artery wedge pressure; BA, brachial artery; PVR, pulmonary vascular resistance; AV, arteriovenous.

Conversion factors from Traditional Units to SI Units; 1 mmHg ≈ 0.133 kPa; 10 dynes·s·cm⁻⁵ = 1 MPa·s·m⁻³.

simultaneously. The gas samples were analysed for oxygen content by the Scholander micro-method. The cardiac output was measured at rest and during the fifth minute of exercise on a bicycle ergometer. The degree of workload selected for each patient was approximately two-thirds of the predetermined maximum load and it was maintained for 5 minutes. Simultaneous pulmonary arterial (PA) and right ventricular (RV) pressures were recorded at the beginning and at the end of the gas collection.

Electrocardiography

All the patients had complete right bundle-branch block on their electrocardiograms but there was no evidence of left anterior hemiblock or complete atrioventricular dissociation.

Results

The physical characteristics of the patients and haemodynamic results at rest and during exercise are shown in Tables 1 and 2.

Right heart pressures

The mean values for the RV systolic pressure and the RV-PA systolic pressure gradient at rest and during exercise are given in Table 1 and the individual values are shown in Fig. 1 and 2. The RV-PA systolic pressure gradient at rest was 20 mmHg (2.7 kPa) or less in 12 of the 15 patients. An increase in the gradient during exercise was recorded in 14 patients but the increase was <10 mmHg (1.3 kPa) in 7 patients. The RV-LV peak pressure ratio

FIG. 1 Right ventricular systolic pressures at rest and during exercise.

averaged 0.31 (range 0.16 to 0.42) and there was no significant difference between the patients who had a patch repair of the RV outflow tract and those who did not.

The RVEDP was slightly increased (>5 mmHg; 0.7 kPa) in 10 patients at rest (Fig. 3). During exercise the RVEDP increased in 10 patients but the increase exceeded 5 mmHg (0.7 kPa) in only 1 patient.

The mean pulmonary artery pressure (PAP) and pulmonary wedge pressures (PAWP) were within

TABLE 2 Physical characteristics and values for cardiac output, cardiac index, AV O₂ difference, and oxygen consumption at rest and during exercise

Case No.	Age	Sex	BSA (m ²)	AV O ₂ Differences (vol %)		Cardiac output (l/min)		Cardiac index (l min ⁻¹ m ⁻²)		Oxygen consumption index (ml min ⁻¹ m ⁻²)	
				Rest	Exercise	Rest	Exercise	Rest	Exercise	Rest	Exercise
1	21	M	1.69	3.8	8.9	6.4	13.6	3.8	8.0	143	714
2	24	F	1.49	2.3	6.7	10.6	9.9	7.1	6.6	166	444
3	23	M	1.94	4.0	10.7	7.3	13.3	3.7	6.9	148	731
4	23	F	1.59	2.7	5.0	6.7	15.0	4.2	9.3	113	465
5	12	F	1.44	2.3	5.7	9.7	12.7	6.8	8.8	155	500
6	17	M	1.63	2.3	6.2	13.4	15.3	8.5	9.4	192	585
7	17	M	1.72	3.9	6.8	6.9	14.0	4.0	8.2	158	552
8	19	M	1.81	3.8	10.9	6.5	11.7	3.6	6.5	138	703
9	14	M	1.30	3.8	6.4	5.8	15.4	4.5	11.8	172	755
10	29	F	1.64	2.4	6.5	10.6	14.2	6.4	8.6	153	561
11	18	F	1.54	3.2	8.5	6.5	10.0	4.3	6.5	137	557
12	17	F	1.71	2.6	7.1	10.6	15.1	6.2	8.8	163	621
13	24	M	1.60	4.8	11.3	5.5	8.2	3.4	5.1	164	579
14	17	M	1.78	2.1	5.4	15.0	18.7	8.4	10.5	180	566
15	25	F	1.63	3.6	10.0	5.2	10.2	3.2	6.3	116	625

Normal values at rest: (1) AV oxygen difference, 2.4 to 5.7 vol. %; (2) cardiac index, 2.6 to 5.8 l min⁻¹ m⁻²; (3) oxygen consumption index, 110 to 192 ml min⁻¹ m⁻². Fuller details are given in a paper by Segal *et al.* (1964).

FIG. 2 Right ventricular-pulmonary arterial systolic pressure gradient at rest and during exercise.

FIG. 3 Right ventricular end-diastolic pressures at rest and during exercise.

normal limits at rest in all except one patient in whom the PAWP was 19 mmHg (2.5 kPa). The mean pulmonary vascular resistance was also normal at rest and it decreased during exercise.

Cardiac index (Table 2)

The mean cardiac index at rest was 5.4 ± 1.9 l min⁻¹ m⁻² and was above the normal range for this laboratory (2.6 to 5.8 l min⁻¹ m⁻²) in 6 patients. The relation between cardiac index and oxygen uptake during exercise was below normal in only 1 patient (Fig. 4).

At rest the mean AV oxygen difference and oxygen uptake were within the normal range. During exercise the mean oxygen uptake increased almost fourfold and the mean AV oxygen difference more than doubled. The values for 6 subjects were less than those for normal subjects doing a comparable level of exercise (Donald *et al.*, 1955). The relation between the AV oxygen difference and the oxygen uptake for individuals is shown in Fig. 5.

Stroke volume

The stroke volume increased in 8 patients during exercise and decreased in 7; the changes were generally small (Fig. 6).

Relation between stroke index and RVEDP during exercise (Fig. 7)

The stroke index increased during exercise in 7 patients and this increase was accompanied by a rise in RVEDP in 5 patients. However, the increase in RVEDP did not exceed 5 mmHg (0.7 kPa). A

FIG. 4 Relation between cardiac index and oxygen uptake during exercise. The normal regression line and 95 per cent confidence limits are shown (Donald *et al.*, 1955).

substantial fall in stroke index was observed in 2 patients and in 1 of these the RVEDP remained unchanged while in the other the RVEDP increased by 4 mmHg (0.5 kPa). In one further patient the stroke index fell by $8 \text{ ml min}^{-1} \text{ m}^{-2}$ while the RVEDP increased from 1 mmHg (0.1 kPa) to 9 mmHg (1.2 kPa).

Oxygen saturations

The mean arterial oxygen saturation at rest and during exercise was identical (95 ± 3 per cent). Two patients showed a significant reduction of the arterial saturation at rest (90 and 88%). In one of these patients an increase occurred during exercise from 90 to 93 per cent but a further reduction to 86 per cent was observed in the other patient.

Analysis of the right heart blood samples by spectrophotometer revealed no evidence of a left-to-right shunt.

Effects of pulmonary regurgitation

Eight patients had early diastolic murmurs in the pulmonary area and these showed haemodynamic evidence of pulmonary regurgitation (pulmonary artery diastolic pressure within 0 to 2 mmHg (0 to 0.3 kPa) of the RVEDP).

In patients without pulmonary regurgitation the mean RVEDP at rest was 6 mmHg (0.8 kPa) and it was unchanged during exercise. The mean RVEDP at rest was also 6 mmHg (0.8 kPa) in the patients with pulmonary regurgitation and exercise caused a slight increase to 9 mmHg (1.2 kPa). The

highest individual value recorded at rest was 13 mmHg (1.7 kPa) in a patient with pulmonary regurgitation (Fig. 3).

The mean pulmonary artery pulse pressure was 19 ± 6 mmHg (2.5 ± 0.8 kPa) in the patients with pulmonary regurgitation compared to 13 ± 5 mmHg (1.7 ± 0.7 kPa) in those without pulmonary regurgi-

FIG. 6 The stroke volume for individual patients at rest and the changes occurring during exercise.

FIG. 5 Relation between AV oxygen difference and oxygen uptake during exercise. The normal regression line and 95 per cent confidence limits are shown (Donald et al., 1955).

FIG. 7 Changes in stroke index and right ventricular end-diastolic pressure during exercise.

TABLE 3 Comparison of early and late haemodynamic results at rest in 6 patients

Case No.	RVSP (mmHg)		RV/PA systolic gradient (mmHg)		RVEDP (mmHg)		RAP (mmHg)		PASP (mmHg)		PADP (mmHg)		PAWP (mmHg)		Cardiac index ($l\ min^{-1}\ m^{-2}$)	
	Early	Late	Early	Late	Early	Late	Early	Late	Early	Late	Early	Late	Early	Late	Early	Late
2	31	25	1	5	7	3	5	-1	30	20	6	-1	8	2	3.9	7.1
7	60	50	35	19	5	7	5	4	25	31	6	8	4	10	3.4	4.0
8	30	36	14	12	7	6	8	8	16	24	8	10	7	2	2.4	3.6
9	25	35	7	10	6	13	6	10	18	25	8	13	10	15	4.7	4.5
10	43	39	16	18	6	1	5	5	27	21	9	9	10	9	3.9	6.4
15	31	37	5	7	7	7	7	8	30	30	8	6	7	19	4.5	3.2
Mean	37	37	13	12	6	6	6	6	24	25	8	8	8	10	3.8	4.8
SD	±13	±8	±12	±5	±1	±4	±1	±4	±6	±5	±1	±5	±2	±7	±0.8	±1.6

Abbreviations: as for Table 1.

tation; this difference is not statistically significant ($P>0.05$). Minor differences in the cardiac output were observed in the two groups at rest and on exercise. The mean cardiac index at rest in patients with pulmonary regurgitation was $6.1\ l\ min^{-1}\ m^{-2}$ increasing to $9.0\ l\ min^{-1}\ m^{-2}$ on exercise compared to $4.6\ l\ min^{-1}\ m^{-2}$ increasing to $7.6\ l\ min^{-1}\ m^{-2}$. These differences were not statistically significant ($P>0.1$ rest; $P>0.05$ exercise).

Comparison of serial haemodynamic results

The results of the early and late postoperative catheterization studies are compared in Table 3 and they show remarkably little change in the intervening years. The mean RV systolic pressure, RVEDP, RAP, and PA diastolic pressures are identical and there are only minor changes in the RV-PA gradient, PA systolic, PA mean, and PAWP. However, one patient showed a significant increase in the PAWP from 7 to 19 mmHg (0.9 to 2.5 kPa) at rest and from 14 to 30 mmHg (1.9 to 4.0 kPa) during exercise. The effects of exercise were measured in only 3 patients who showed no significant haemodynamic change between the two studies.

The mean cardiac index in the early study was $3.8\pm0.8\ l\ min^{-1}\ m^{-2}$ compared to $4.8\pm1.6\ l\ min^{-1}\ m^{-2}$ in the later study. The difference is not statistically significant ($P>0.1$). The mean AV oxygen difference at rest in both studies was within the normal range and only 1 patient had a value significantly greater than normal during exercise.

Discussion

The majority of haemodynamic studies after total correction for Fallot's tetralogy have shown a decrease in the RV/PA systolic pressure gradient in later studies compared with measurements made immediately after operation (Kirklin and Karp, 1970; Muraoka *et al.*, 1973). The results of catheterizations performed 5 to 6 years postoperatively compared with 1 year after operation have shown little subsequent haemodynamic change (Gotsman *et al.*, 1969; Bristow *et al.*, 1970). The RV/LV ratio was selected by Hawe *et al.* (1972) as the most valid indicator of the degree of pulmonary stenosis. They studied 37 patients and showed in 25 patients that the RV/LV ratio decreased between the completion of repair and restudy by 10 to 53 per cent. The ratio was unchanged in 4 patients and in 8 it was increased by 10 to 73 per cent.

In our study the RV/LV peak pressure ratio was 0.31 (range 0.16 to 0.42) and this indicates satisfactory relief of the pulmonary stenosis. It was also noted that the ratio did not differ significantly between patients who had a prosthetic patch in the right ventricular outflow tract and those who did not. Exercise caused an increase in the RV/PA systolic pressure gradient but the increase was slight or trivial in the majority of patients though more intense exercise would probably have shown a further increase (Epstein *et al.*, 1973). Nevertheless, the increase in pulmonary blood flow during exercise was well tolerated. Another important observation was the unchanged RV/PA systolic pressure gradient in the patients catheterized previously. The relatively small increase in the RV/PA gradient during exercise and the constancy of the haemodynamic measurements with the passage of time strongly suggests that the restoration of a normal pulmonary blood flow provides an adequate stimulus for the growth of the right ventricular outflow tract and the pulmonary arteries.

Right ventricular myocardial function

The RVEDP was greater than 5 mmHg (0.7 kPa) in 10 patients and instead of the normal fall during exercise the RVEDP showed a further slight increase. Pulmonary regurgitation is one possible reason for the increased RVEDP but diminished

right ventricular compliance caused by persisting muscular hypertrophy or myocardial fibrosis may also be important. A fall in the stroke volume index and a concomitant increase in the RVEDP during exercise has been interpreted as indicating abnormal right ventricular myocardial function in patients with Fallot's tetralogy after total correction (Joransen and Moller, 1972) and also after pulmonary valvotomy for pulmonary stenosis (Finnegan *et al.*, 1974; Stone *et al.*, 1974). A similar occurrence was observed in 5 patients in the present study. Because of the presence of pulmonary regurgitation and the brevity of the isovolumic period in the right ventricle, it was not possible to derive indices of myocardial contractility (V_{max} , V_{pm}) and therefore a more direct assessment of myocardial contractile function was not made.

Pulmonary hypertension

An increase in the pulmonary vascular resistance has been observed in the early postoperative period in the majority of patients (Albertal, Swan, and Kirklin, 1964) and in some patients thereafter (Lillehei *et al.*, 1964; Hallidie-Smith *et al.*, 1967; Gersony *et al.*, 1973; Kinsley *et al.*, 1974). However, other studies have shown a normal pulmonary vascular resistance and mean pulmonary artery pressures (Gotsman, 1966; Bristow *et al.*, 1970; Epstein *et al.*, 1973; Shah and Kidd, 1966). In our study there was no evidence of pulmonary arterial hypertension at rest or during exercise. The fall in the pulmonary vascular resistance during exercise shows the ability of the pulmonary vascular system to accommodate the increased pulmonary blood flow. This finding lends support to the view that the thrombotic lesions, seen in the pulmonary arteries of cyanotic patients, usually resolve when the pulmonary blood flow increases (Ferencz, 1960).

Pulmonary valvular regurgitation

The problem of pulmonary valvular regurgitation after total correction continues to cause concern as to its ultimate effects on right ventricular function. Though some authors consider pulmonary regurgitation to be the major postoperative problem (Soulié *et al.*, 1971), others have concluded that it appears to be well tolerated (Malm *et al.*, 1963; Gotsman, 1966; Jones *et al.*, 1973; Sunderland *et al.*, 1973).

In our study there was no significant difference in the resting cardiac output or the response to exercise in patients with and without pulmonary regurgitation. The slight increase in RVEDP

during exercise was the main difference between the two groups. It appears, therefore, that pulmonary regurgitation is well tolerated but further observation is necessary to determine its ultimate significance because a long-term experimental study of pulmonary regurgitation has shown the development of right ventricular dilatation and hypertrophy and a reduced resting cardiac output (Ellison *et al.*, 1970).

Recurrence of ventricular septal defect

Several authors report that the ventricular septal defect reopens in a small number of patients after total correction (Bristow *et al.*, 1970; Sunderland *et al.*, 1973; Joransen and Moller, 1972). Analysis of the right heart blood samples by spectrophotometer showed no evidence of left-to-right shunting in any of our patients. This does not exclude the presence of a small left-to-right shunt but more sensitive methods of shunt detection were not used by us.

Systemic oxygen saturations

The arterial oxygen saturations were normal in all except 2 patients at rest. One patient had an oxygen saturation of 90 per cent which increased to 93 per cent during exercise. The other patient had an oxygen saturation of 88 per cent and a further decrease to 86 per cent occurred during exercise. This patient was asymptomatic and had a high resting cardiac output, a normal response of the cardiac output during exercise, and a trivial degree of right ventricular outflow tract obstruction (RV/PA systolic gradient at rest = 5 mmHg (0.7 kPa)). The reason for the arterial unsaturation is, therefore, unexplained but it may be attributable to a persistent abnormality of pulmonary blood flow distribution.

Left ventricular function

Abnormal left ventricular function has been reported before and after total correction of Fallot's tetralogy (Jarmakani *et al.*, 1972). The main abnormality described was a decreased left ventricular ejection fraction. Left ventricular function was not studied directly by us but the pulmonary wedge pressure was within normal limits in 14 patients. One patient had a PAWP at rest of 19 mmHg (2.5 kPa) which increased to 30 mmHg (4.0 kPa) during exercise. In the majority of patients, however, the failure to develop pulmonary hypertension and the response of the stroke volume during exercise probably indicates normality of LV function.

Cardiac output

The mean resting cardiac index for the group was $5.4 \text{ l min}^{-1} \text{ m}^{-2}$ which is at the upper limit of normal and 6 patients had cardiac outputs above the normal range. Six subjects had a normal response of the cardiac output during exercise and 1 patient was slightly below the normal range. The remaining 8 patients had high cardiac outputs during exercise and this was associated with low AV oxygen differences. Though the patients were generally anxious there was no evidence of hyperventilation during the catheterization procedure or any other reason for the high output state such as anaemia, thyrotoxicosis, or arteriovenous shunting. However, the presence of small left-to-right shunts undetected by analysis of oxygen saturations is a possible explanation.

In normal subjects the response of the stroke volume during exercise is variable; it usually increases or is unaltered but it may fall (Donald *et al.*, 1955). In patients with pulmonary stenosis studied postoperatively, a similar variability was observed (Finnegan *et al.*, 1974; Stone *et al.*, 1974). Another study of patients with pulmonary stenosis showed that in the presence of mild stenosis the stroke volume increased on exercise but when the stenosis was more severe the stroke volume remained constant (Moller, Rao, and Lucas, 1972). In our study the changes in stroke volume during exercise were also variable. The stroke volume increased in 8 subjects and decreased in 7, though a substantial decrease was observed in only 2 patients (38 ml and 27 ml).

The response of the cardiac output during submaximal exercise testing is clearly very satisfactory, but testing during more intensive exercise has shown a consistently reduced response (Epstein *et al.*, 1973). Nevertheless, considering the presence of residual defects such as right ventricular outflow tract obstruction and pulmonary regurgitation, the performance of these patients during exercise is remarkably good. The ultimate effect of the residual abnormalities can only be determined by further clinical and haemodynamic studies.

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