SOME EXPERIENCES WITH INTRACARDIAC SURGERY USING THE HELIX-RESERVOIR BUBBLE OXYGENATOR WITH TOTAL CARDIO-PULMONARY BYPASS

A REVIEW OF THE FIRST 30 CASES TREATED AT THE GROOTE SCHUUR AND RED CROSS WAR MEMORIAL CHILDREN'S HOSPITALS, CAPE TOWN

C. N. BARNARD, M.D., M.MED. (CAPE TOWN), M.S., PH.D. (MINNESOTA); W. L. PHILLIPS, M.B., B.CH. (RAND), M.R.C.P. (LOND.), F.R.C.S. (ENG.); D. R. DE VILLIERS, M.SC.PHYS., M.B., CH.B. (CAPE TOWN); R. D. CASSERLEY, CH.M. (CAPE TOWN); R. P. HEWITSON, M.B., CH.B. (CAPE TOWN), F.R.C.S. (EDIN. and ENG.); R. L. VAN DER RIET, M.B., CH.B. (CAPE TOWN), F.R.C.S. (ENG.); and M. B. MCKENZIE, M.B., CH.B. (CAPE TOWN)

Department of Surgery, University of Cape Town

The rapid progress in cardiovascular surgery makes advisable periodic review of problems and achievements. The discussion of surgical procedures must be in part personal, and this review reflects to some extent the opinions and experiences of the authors. It must be remembered that, in a field where progress is so rapid, further experience may invalidate some concepts which at present appear proper.

It is not within the scope of this paper to give credit to the many pioneers in this field but it may be worth while noting the great role played by the experimental laboratory in the development of this branch of surgery. There is no doubt that the vast body of young surgeons from all over the world who have had access to such laboratories are the ones to whom one must give the most credit for the rapid advances in this field.

The object of this paper is to present the first 30 patients operated on under total cardio-pulmonary bypass with the helix-reservoir bubble oxygenator in Cape Town. After initial experimentation with total body perfusion in the Department of Surgery[1] clinical development followed[2] and open heart surgery is now performed every week both at Groote Schuur Hospital and the Red Cross War Memorial Children's Hospital.

Selection of Patients

During the early stages, patients with easily repairable lesions were selected but, as experience of the team increased, more complex lesions were corrected (Table I). These patients were all first investigated by members of the Cardiac Clinic, Groote Schuur Hospital, to determine the nature and severity of their lesions. Only those patients in whom objective studies indicated significant haemodynamic derangement were submitted for surgery.

The ages of the 30 patients ranged from 7 years to 44 years. There were 15 Europeans, 13 Coloured and 2 Bantu. Of these patients 3 had been operated upon before for their cardiac lesions. Case 7 had had a left thoracotomy and left atrial exploration for suspected mitral stenosis, when a myxoma was diagnosed. Case 20 had undergone left thoracotomy with pulmonary-systemic anastomosis in 1949, and in 1954 right thoracotomy and blind pulmonary valvotomy were performed. Case 30 had had a left thoracotomy and mitral valvotomy.

Pre-operative Preparation

The important aspects of the pre- and post-operative care of patients undergoing open heart surgery have been dealt with fully in a previous publication.[3] The careful preparation of the operative field with 'phisohex' and the pre-operative use of antibiotics and a good diet supplemented by vitamins will assist in obtaining smooth convalescence free from infection and complications.

Patients with a high pulmonary flow, or pulmonary congestion, are prone to infections of the tracheo-bronchial tree and lungs and should be adequately treated before surgery. Overt or incipient heart failure should be treated with salt restriction, diuretics and digitalization, but routine pre-operative digitalization is not recommended.

Tracheotomies are sometimes performed pre-operatively in patients with high pulmonary-artery pressures and resistances, to enable one to deal more adequately with secretions post-operatively and to assist respiration in this period if necessary.

Accurate pre-operative weight should be obtained to enable one to calculate the change in blood volume when compared with the immediate post-operative weight.

Anaesthesia

A simple technique with minimal disturbance to the patient from the anaesthesia has been used.

Premedication with secobarbital and atropine has been used; this produces a patient who is usually sleeping quietly when brought to the theatre. During the preliminary operative procedures of placement of the monitoring catheters, thoracotomy, and placement of the catheters linking the patient to the bypass machine, only light anaesthesia is required. This is achieved by controlled respiration *via* the endotracheal route, with nitrous oxide and oxygen and, if necessary, minimal doses of ether.

Requirements during the bypass are absence of respiratory movements, and hypnosis. These conditions are adequately obtained by means of curare and thiopentone, and it is found that minimal quantities only need be used.

Post-bypass, during closure of the thorax, nitrous oxide and oxygen anaesthesia is all that is necessary; as a result, the patients are fully conscious and cooperative within minutes of completion of the operation.

During these prolonged procedures—ranging from 5 to 10 hours—meticulous unceasing vigilance by the anaesthetist is necessary to make it possible to cope with any untoward occurrences.

SURGICAL TECHNIQUE

Surgical Approach

Three types of incision were used: bilateral thoracotomy with transverse sternotomy in 17 cases, median sternotomy in 11 cases, and right thoracotomy in 2 cases. The transverse thoracotomy gives good access to all the important structures but there is little doubt that with this approach the post-operative pulmonary complications are higher than with the median sternotomy because of the extensive and painful nature of the wound. The median sternotomy is an easier, faster and much less painful incision and we are making use of it whenever possible. This incision, however, has the disadvantage that dissection of the ductus area and control of a patent ductus is difficult, especially in cases where the heart and pulmonary artery are grossly enlarged. In all our cases it has been possible, where necessary, to expose the ductus by dissecting intrapericardially down the superior margin of the pulmonary artery. We prefer a right thoracotomy through the bed of the fifth rib in lesions to be repaired through a left atriotomy. A giant left atrium may interfere with the taping of the inferior vena cava, when an additional incision will be needed through a lower space.

With all three incisions it is possible to isolate an internal mammary artery, which is then cannulated with a thin polythene tube for arterial pressure recording.

Once the pericardium is opened, the pre-operative diagnosis can be confirmed by noting the size of the various chambers of the heart and the roots of the great vessels, by digital exploration of the right atrium through the right auricular appendage, and by measurement of intracardiac pulmonary-artery and aortic pressures. A search should be made for anomalous venous return, especially for a left superior vena cava which, if present, should be occluded during the bypass. If a patent ductus has been diagnosed pre-operatively, it must be ligated but, in patients with ventricular septal defects, even if not diagnosed, a patent ductus should always be sought for.

Preparation for Cardio-pulmonary Bypass and Commencement of Bypass

In previous publications these have been fully discussed.[1,2] Since then we have made no major modifications in our technique.

INTRACARDIAC PROCEDURES

A wide variety of lesions have already been managed by open cardiotomy with total cardio-pulmonary bypass (Table I). There are still differences of opinion about the best technique for the correction of some defects encountered. Time and experience will solve whatever uncertainties may exist; in the meantime, each surgeon must decide from his own experience, and that of other surgeons, what technique gives the best results.

Ostium Secundum: Atrial Septal Defects

The only acceptable treatment for an atrial secundum septal defect is one that produces a complete correction with low mortality. We now employ extracorporeal circulation as a routine for the repair of these lesions and have successfully corrected 12 without fatality (Table II). The defects varied in size from 2 to 3·5 cm. in diameter. Multiple defects were found in 3 patients. One patient (case 21) had associated mitral stenosis and one (case 28), with a sinus-venosus type of secundum defect, had an anomalous return of the superior pulmonary vein.

Repair was effected by a running 3-0 silk suture doubly sewn and reinforced with interrupted mattress sutures. Except in the patient with the sinus-venosus defect, where an

TABLE I. ANALYSIS OF CASES

No.	*Age (yrs.)*	*Race Sex*	*Date of Operation*	*Type of Lesion*	*Result*
1	18	CF	28.7.58	Pulm. sten. (valv.)	Good
2	14	EF	18.8.58	ASD (ost. sec.)	Good
3	9	EF	8.9.58	ASD (ost. sec.)	Good
4	41	CM	29.9.58	ASD (ost. sec.)	Good
5	13	CF	13.10.58	VSD+PDA (*c* pulm. hyperten.)	Died 14.10.58
6	44	EM	20.10.58	ASD (ost. sec.)	Good
7	36	NM	26.10.58	L. atrial myxoma	Good
8	24	CF	10.11.58	ASD (ost. sec.)	Good
9	15	EM	17.11.58	VSD (*c* moderate pulm. hyperten.)	Good
10	21	CF	8.12.58	VSD (*c* pulm. hyper-ten.)	Good
11	10	EF	15.12.58	Ost. prim. (endocar-dial-cushion def.)	Good
12	13	CF	12.1.59	VSD (*c* pulm. hyper-ten.)	Good
13	7	NM	19.1.59	Tetral. Fallot	Good
14	11	EM	21.1.59	ASD (ost. sec.)	Good
15	13	EM	26.1.59	ASD (ost. sec.)	Good
16	15	CF	9.2.59	Ost. prim. (endocar-dial-cushion def.)	Died 10.2.59
17	12	EF	16.2.59	Aortic sten.+pulm. valv. sten.+ASD	Died 16.2.59
18	11	EM	2.3.59	Pulm. sten. (valv.)	Good
19	14	EF	9.3.59	ASD (ost. sec.)	Good
20	16	EM	16.3.59	Tetral. Fallot	Good
21	24	EF	23.3.59	ASD (ost. sec.)	Good
22	20	CF	8.4.59	VSD (*c* pulm. hyper-ten.)	Good
23	12	CM	13.4.59	Pulm. sten. (infund.) *c* tricuspid incomp.	Died 14.4.59
24	36	EF	20.4.59	ASD (ost. sec.)	Good
25	27	CM	27.4.59	Pulm. sten. (infund.) +VSD	Good
26	8	EM	18.5.59	VSD (*c* pulm. hyper-ten.)	Good
27	7	EM	25.5.59	VSD (+pulm. sten., valv.+infund.)	Good
28	12	CF	26.5.59	ASD (*c* anomalous pulm. venous return)	Good
29	8	CM	2.6.59	ASD (ost. sec.)	Good
30	31	CM	8.6.59	Mitr. sten.	Good

c = *cum* (with). ASD and VSD = auricular and ventricular septal defect.

TABLE II. ARTRIAL SEPTAL (OSTIUM SECUNDUM) DEFECTS

Case No.	*Type*	*Repair*	*Result*
2	Defect ± 3 cm. diam. Satellite defect (0·5 cm. diam.)	Suture	Good
3	Defect ± 3·5 cm. diam.	Suture	Good
4	Defect ± 2·5 cm. diam.	Suture	Good
6	Defect ± 2 cm. diam.	Suture	Good
8	Defect ± 2 cm. diam. Satellite defect 3 mm. diam.	Suture	Good
14	Defect ± 3·5 cm. diam. Satellite defect (0·5 cm. diam.)	Suture	Good
15	Defect 2·5 cm. diam.	Suture	Good
19	Defect 2·5 cm. diam.	Suture	Good
21	Defect ± 3 cm. diam. Assoc. mitral stenosis	Suture	Good
24	Defect ± 2·5 cm. diam.	Suture	Good
28	Defect ± 2·5 cm. diam. Assoc. anomalous pulmonary venous return	Ivalon patch	Good
29	Defect ± 2 cm. diam.	Suture	Good

Ivalon patch was sewn over the defect in order to partition off the entrance of the superior pulmonary vein and superior vena cava correctly, all lesions were completely and securely corrected by simple suture (Fig. 1) and there is no reason to believe that a residual shunt exists. The correctness of this statement will be tested later by re-catheterization.

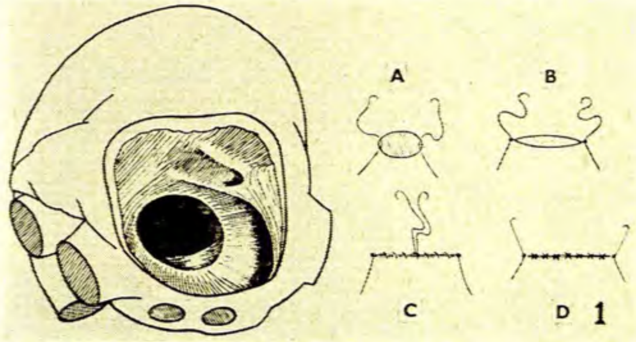


Fig. 1. Diagram of typical ostium-secundum defect. Insets A to D demonstrate simple method of direct repair used for most defects.

Endocardial-cushion Defects

Failure of the embryologic endocardial cushions to develop normally and fuse in the mid-line of the common atrioventricular canal results in abnormalities of varying degree in atrial septation, ventricular septation, and atrioventricular valve-leaflet formation. The specific pathological abnormalities encountered vary from the simple ostium-primum atrial septal defect to a persistent atrioventricular canal. Atrioventricular valve malformation of varying extent is almost universally present.

Considerable progress has now been made in the surgical treatment of these lesions. Only open cardiotomy with extracorporeal circulation should be used for their repair. Closed techniques, hypothermia with inflow stasis, and the atrial well, are methods not satisfactory for the management of these repair procedures.

The extent of the operative repair depends on the type of defect. In patients with only an ostium-primum type of intra-atrial communication, it may be possible to approximate the edges with interrupted 3-0 mattress sutures. If the defect is large, direct repair will produce undue tension on the sutures, and an Ivalon patch sewn over the defect will provide a much securer closure (see under 'ventricular septal defects'). The sutures are accurately placed in the lower margin of the defect to avoid incorporating the mitral and tricuspid leaflets in the repair.

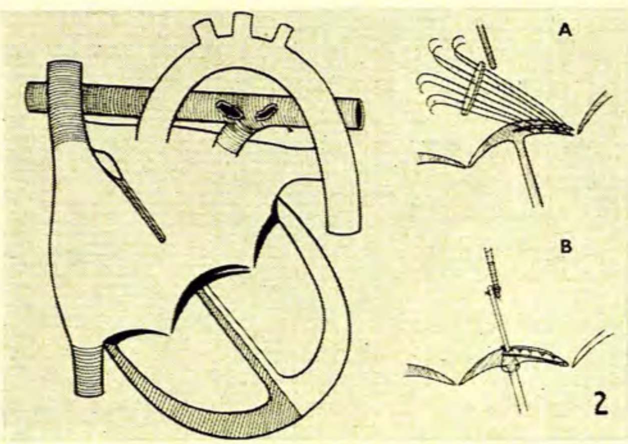


Fig. 2. Diagram of partial type of endocardial-cushion defect. Inset A shows closure of cleft leaflet of mitral valve with interrupted sutures reinforced with compressed Ivalon strip. Inset B demonstrates closure of ostium-primum defect with Ivalon patch after correction of valve defect.

When a partial type is encountered, the cleft mitral or tricuspid valve is first repaired with interrupted 4-0 silk sutures. We prefer to tie these sutures over a strip of compressed Ivalon (Fig. 2). After the valvular repair is accomplished the septal defect is repaired in the manner already described.

A complete atrioventricular-communis defect with common mitral and tricuspid leaflets and defects in atrial and ventricular septation is the most complex of all cardiac septal defects to repair. The first step is the repair of the cleft atrioventricular valves. A thin strip of compressed Ivalon is used to reinforce this repair, and the middle of this strip is used in the repair of the ventricular septal defect (Fig. 3). The atrial defect is then repaired as before. Cardiac asystole is not used, for in the beating heart one can better assess mitral and tricuspid regurgitation than in the arrested heart. Further, if the heart is beating during the placement

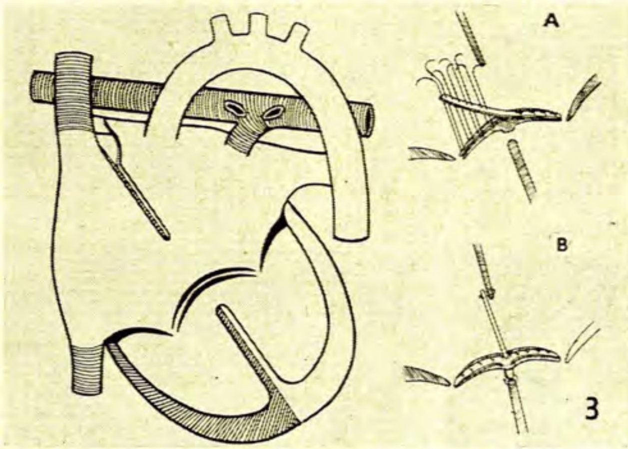


Fig. 3. Repair of complete endocardial-cushion defect showing extensive reconstruction of A-V valves and repair of defects in atrial and ventricular septation.

TABLE III. ENDOCARDIAL CUSHION (OSTIUM PRIMUM) DEFECTS

Case No.	*Type*	*Repair*	*Result*
11	Partial endocardial-cushion defect: Ost. prim. ASD (± 3 cm. diam.) and assoc. cleft of anteromedial leaflet of mitral valve producing mitral incompetence.	Suture of cleft mitral valve. Ivalon patch repair of septal defect.	Good
16	Complete endocardial-cushion defect: Ost. prim. ASD (± 3 cm. diam.). Complete splitting of aortic leaflet of mitral valve and mural leaflet of tricuspid valve VSD (± 1 cm. diam.).	Suture of mitral and tricuspid valve leaflets incorporating an Ivalon strip. Ivalon patch repair of ASD and VSD.	Died

and tying of stitches, the production of heart block can immediately be detected and the causative suture removed.

Two patients with endocardial-cushion defects were operated upon in this manner (Table III). One had a partial lesion with a cleft mitral leaflet (case 11), the other (case 16) suffered from the complete form. This last patient died 24 hours after the operation from pulmonary oedema. Autopsy revealed a complete correction of the lesion.

Ventricular Septal Defects

The majority of patients with ventricular septal defects selected for surgical treatment had a ventricular septal defect located immediately beneath the aortic-valve ring.

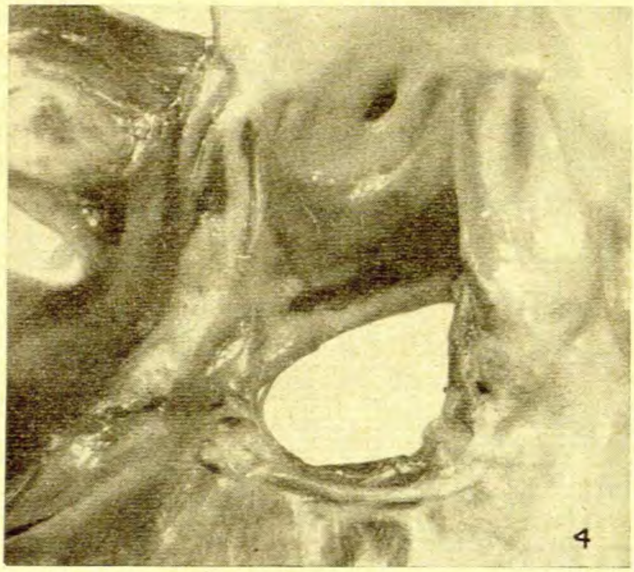


Fig. 4. High ventricular septal defect seen from the left ventricular aspect. Note the close proximity to the aortic valve.

When viewed from the left ventricular aspect, the opening is immediately inferior to the right aortic cusp, the adjacent portion of the posterior aortic cusp, and the intervening commissure. Thus there is no superior remnant of ventricular septum below the aortic ring itself (Fig. 4). From the right ventricular aspect the region of the defect is partially obscured by the crista supraventricularis, the tricuspid valve, and the papillary muscle of the conus (Fig. 5).

The remaining defects are situated either immediately beneath the pulmonary valve or in the region of the right ventricular inflow tract[4] and include defects in the muscular septum, which are often multiple and may be hidden between the muscle trabeculations. The position of the defect can easily be located when the right ventricle is opened by observing the site through which oxygenated blood enters the right ventricular cavity from the left ventricle with each heart beat.

Ventricular septal defects are best approached through a right ventriculotomy, which usually extends from a point just below the pulmonary-valve ring downwards nearly to the margin of the heart. This incision is placed to avoid major coronary branches. Induced cardiac arrest has clearly become a fundamental necessity to the accurate repair of

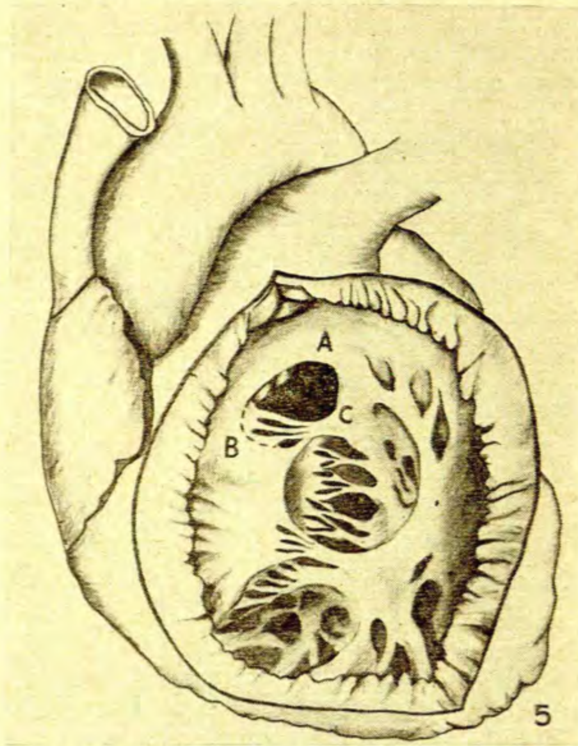


Fig. 5. Diagram showing the relations to a high ventricular septal defect viewed from the right ventricular aspect of (A) crista supraventricularis, (B) tricuspid valve, (C) papillary muscle of the conus.

TABLE IV. INDUCED CARDIAC ARREST WITH 2·5% SOLUTION OF POTASSIUM CITRATE

Case No.	*Amount Used*	*Duration of Arrest (minutes)*	*Recovery*
5	30 c.c.	20	Spontaneous return of sinus rhythm.
9	40 c.c.	32	Ventricular fibrillation *c* successful defibrillation
13	30 c.c.	48	Ventricular fibrillation *c* successful defibrillation.
17	25 c.c.	30	Initial return to sinus rhythm. Ventricular fibrillation followed cessation of by-pass—no response to electrical defibrillation.
20	50 c.c.	60	Spontaneous return of sinus rhythm.
22	50 c.c.	33	Ventricular fibrillation *c* successful defibrillation.
27	60 c.c.	20	Ventricular fibrillation *c* successful defibrillation.

c = *cum* (with)

these defects (Table IV). We use 2·5% solution of potassium citrate in oxygenated blood as suggested by Melrose[5] and induce arrest after the ventriculotomy, thus preventing distension of the relaxed muscle by blood still entering the heart from the lungs.

The edges of the incision are kept apart by suitably placed traction sutures. Two cardiotomy suckers are used, one placed through the tricuspid valve into the right atrium and one placed in the pulmonary artery. A dry field is obtained

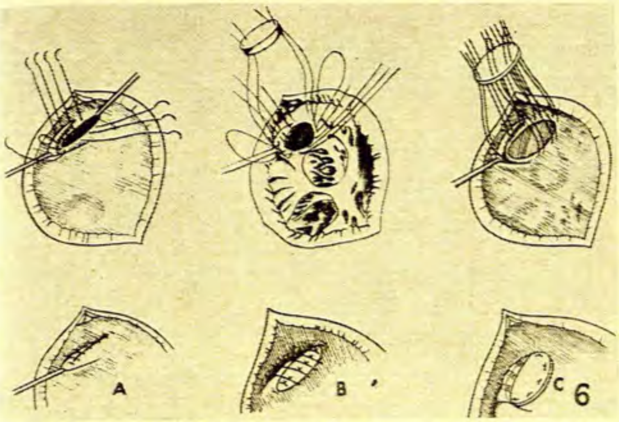


Fig. 6. Diagram illustrating the three methods of repair of ventricular septal defect. (A) Direct suture repair. (B) Sutures tied over Ivalon pledget. (C) Circumferentially sutured Ivalon patch.

in this way and the anatomy of the defect can be studied and a plan of the method of its closure formulated. Three methods of closure have been used (Fig. 6 and Table V), viz. direct sutures, sutures tied over an Ivalon pledget, and a circumferentially sutured Ivalon patch. As yet there is no unanimity

TABLE V. VENTRICULAR SEPTAL DEFECTS

Case No.	*Type*	*Repair*	*Result*
5	PDA (1 cm. diam.) VSD (1·5 cm. diam.) in membranous portion of septum. Aortic insuff. due to prolapse of one aortic leaflet into the VSD	Ligation of PDA. Suture repair of defect.	Died
9	Defect in membranous area (± 2 cm. diam.)	Suture	Good
10	Defect in membranous area (± 2 cm. diam.)	Suture	Good
12	2 defects in membranous septum (0·5 cm. diam.). 1 defect in muscular septum (0·5 cm. diam.)	Suture repair of 2 defects. Ivalon pledget tied in sutures in repair of other defect	Good
22	Defect in membranous area (± 1·5 cm. diam.)	Ivalon patch repair	Good
26	Defect (± 2·5 cm. diam.) in posterior portion of muscular septum. 2 satellite defects in muscular septum (± 3 mm. diam.)	Suture repair of all defects	Good
27	Defect (± 2·5 cm. diam.) in membranous portion of septum. Associated valv. + infund. pulm. sten.	Suture repair of defect. Pulmonary valve commissurotomy. Infundibular resection	Good

of opinion about the best method, and it is for the surgeon to decide which method gives the best results in his hands. Whichever method is employed the following points are most important for the successful closure of these defects:

(*a*) A clear three-dimensional concept of the compact, complex and somewhat obscure anatomy of this region.

(*b*) Closure must be accomplished without undue tension. These defects are usually ovoid apertures and if direct suture is chosen as the method of repair the line of repair must be parallel to the long axis of the oval. In the commonly encountered high defects the line of repair runs parallel to the aortic annulus (Fig. 6).

(*c*) Sutures should be tied with just sufficient tension to approximate and this is accomplished best in the arrested heart.

(*d*) The repair should always start at the most inaccessible angle of the defect and end at the most accessible angle. Thus, in the high membranous defect, whether repair is effected by direct suture or by insertion of a patch, the repair should start at the important postero-inferior angle and end at the antero-superior angle just below the crista supraventricularis.

(*e*) Sutures should be placed in such a manner that they have a firm hold preventing them from tearing loose but, on the other hand, they should be placed so as not to damage vital structures. Damage to the conduction system causing a complete heart block is usually caused by sutures placed at the postero-inferior angle of the high membranous defect.[6,7] This danger can be minimized by using the edge of the insertion of the septal leaflet of the tricuspid valve for holding the sutures in this area (Fig. 6).

Damage to the aortic valve in the placing of stitches in the aortic annulus, which will form the superior border in the repair of high membranous defects, can always be prevented if the aortic valve leaflets are clearly visualized. During cardiac arrest the cusps do not bulge and their margins may be difficult to identify. This difficulty can be overcome by momentarily releasing the clamp on the ascending aorta and allowing the blood to fill the cusps and bulge them into view. The heart is restarted before the last two or three stitches are tied. This allows an escape for air and blood from the left side of the heart until the beat is effective. After the last stitches are tied the closure is carefully inspected for residual leaks, which must be corrected, and the remainder of the ventricular septum is then examined for other defects as described above.

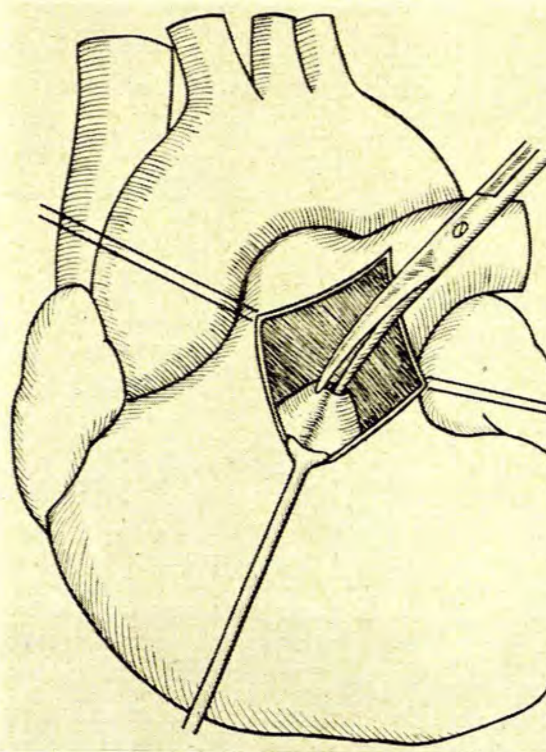
The ventriculotomy is closed with running 3-0 silk sutures doubly sewn. Before complete closure the tape around the catheter in the superior vena cava is released and the right ventricular cavity allowed to fill with blood.

In 7 patients (Table V) in this series a ventricular septal defect was the major disability. An Ivalon patch was necessary for adequate closure of the defect in 2 patients (cases 9 and 22). In one (case 12) sutures were tied over an Ivalon pledget, and in the remaining cases the direct suture technique was used. Valvular pulmonary stenosis was present in one patient (case 27) and this was corrected. One patient (case 5) had a patent ductus arteriosus, which was ligated before going on bypass. This patient also suffered from aortic incompetence, which was not corrected. Two patients (cases 12 and 26) had multiple ventricular septal defects, all of which were closed by direct suture. In 2 cases the pulmonary-artery pressure was greater than 75% of the systemic pressure.

One patient (case 5) died 24 hours after the operation. This was due to the residual aortic incompetence and excessive post-operative bleeding. The remaining 6 patients are all well and have left hospital. No heart blocks were encountered.

Pulmonic Stenosis

Pulmonic stenosis with presumably intact ventricular septum is frequently a complex abnormality, for the stenosis may be valvular, infundibular or both, and may be associated with an atrial septal defect or even with a small ventricular septal defect. The surgical treatment which provides optimal conditions for restoration of normal anatomy utilizes extracorporeal circulation and any or all of ventriculotomy, atriotomy and pulmonary arteriotomy. Valvular stenosis is corrected (through an anterior incision in the main pulmonary artery) by radial incisions made with scissors to produce a tricuspid or bicuspid valve (Fig. 7). The infundibular region

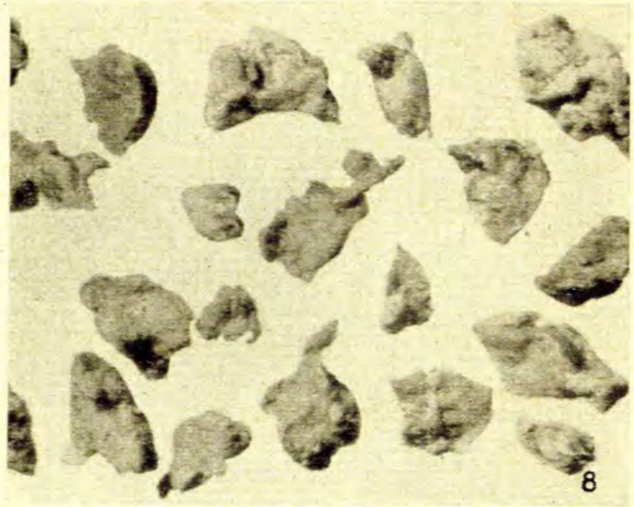


7

Fig. 7. Stenotic pulmonic valve incised along vestigial commissure through pulmonary arteriotomy.

is then inspected and palpated. If an infundibular stenosis is expected the ventricle must be opened. Infundibular stenosis may be relieved by excision of redundant infundibular muscle and myocardium. Great care must be taken on the postero-medial aspect of the floor of the infundibulum not to injure the aortic annulus, which is in close proximity to this area. In some patients it may be necessary to excise the hypertrophied moderator band to provide an adequate outflow to the right ventricle (Fig. 8). In some cases, especially where the infundibular stenosis is high, resection of redundant muscle may not completely relieve the stenosis and the insertion of a pear-shaped compressed Ivalon patch (base towards the pulmonary artery) in the right ventricular outflow tract may be required.[8] If the valve ring is narrow the surgeon must not hesitate to cut across this and extend the patch well onto the main pulmonary artery. Residual pulmonary stenosis is much more dangerous than a slight pulmonary incompetence.

A total of 4 patients with this condition have been operated on in this series (Table VI). In 2 of these only valvular stenosis was present, in one pure infundibular stenosis, and in one infundibular stenosis plus a small ventricular septal defect. One patient had extensive peripheral oedema with gross tricuspid incompetence and died 48 hours after the operation in pulmonary oedema. It will be seen from Table VI that pre-operative right ventricular pressures ranged from 219/5 mm. Hg to 130/15 and the pulmonary-artery pressures from 31/17 to 20/2. The gradient across the stenosis ranged from 178 mm. Hg to 88. Immediately after surgery pressures were again measured and the gradient now varied from 20 mm. Hg to 10, indicating an adequate relief of the obstruction.



8

Fig. 8. Photograph of some of the muscle resected from outflow tract of right ventricle in patient with infundibular pulmonary stenosis. × 3.

TABLE VI. PULMONARY STENOSIS

Case No.	*Type of Lesion*	*Repair*		*Pressures* *Pre-op.*	*Post-op.*	*Result*
1	Valv. sten. (5 mm. diam. aperture)	Pulm. valve commissurotomy	IMA RV PA	110/80 130/15 27/17	80/60 40/7 25/12	Good
18	Valv. sten. (4 mm. diam. aperture)	Pulm. valve commissurotomy	IMA RV PA	125/80 145/0 20/0	110/80 40/5 30/20	Good
23	Infund. sten. (*c.* assoc. tricuspid incomp.)	Infund. resection	IMA RV PA	80/50 140/40 31/17	84/40 52/10 12/2	Died
25	Infund. sten. VSD in membranous part of septum	Infund. resection. Suture repair of VSD	IMA RV PA	122/75 219/5 20/2	— 40/5 20/15	Good

Tetralogy of Fallot

The significant abnormalities in this syndrome are the ventricular septal defect, pulmonic stenosis, and stenosis or atresia of the pulmonary artery. The septal defect is generally large and located in the membranous area of the septum. The pulmonic stenosis is usually infundibular or infundibular and valvular, rarely only valvular. Although the pulmonary artery is always smaller than the aorta, it may vary in actual size from an atretic cord of hypoplastic vessel to an artery scarcely smaller than normal.

As little can be done surgically to increase the size of the pulmonary artery the correction of this anomaly consists in closure of a ventricular septal defect and correction of the pulmonic stenosis. The techniques of dealing with both of these have been discussed above.

Two patients were operated upon for correction of tetralogy of Fallot, with no deaths (Table VII). As stated above, one patient (case 20) had undergone 2 previous operations for relief of his disability, with only temporary success. With total cardio-pulmonary bypass we were able to close the ventricular septal defect with an Ivalon patch and correct the pulmonic stenosis by an adequate infundibular resection. The other patient (case 13) also needed an Ivalon patch for closure of the septal defect but, in addition, for complete relief of the infundibular stenosis an Ivalon patch was sutured nto the roof of the outflow tract of the right ventricle.

TABLE VII. TETRALOGY OF FALLOT

Case No.	*Lesions*	*Repair*	*Pressures (mm. Hg)*			*Result*
				Pre-op.	*Post-op.*	
13	VSD in membranous part of septum (2·5 cm.). Overriding of aorta. Pulm. infund. sten.	Ivalon patch repair of VSD. Infundibular resection and Ivalon patch incorporated in roof repair of RV outflow tract	IMA	95/70	110/95	Good
			IVC	20	14	
			RV	81/4	52/5	
			MPA	22/15	47/12	
20	VSD in membranous part of septum (3·5 ×2 cm.). Extensive overriding of aorta. Infund. sten. *c* partial correction of valv. sten.	Ivalon patch repair of VSD. Infundibular resection	IMA	120/90	100/75	Good
			IVC	18	15	
			RV	125/5	60/15	
			MPA	20	40/18	
			Infund. region	30/10	40/12	

Other Lesions

One patient (case 17) with aortic stenosis, pulmonic stenosis, and an ostium-secundum defect, was operated upon. The aortic stenosis was relieved through an aortic incision after potassium citrate arrest, the pulmonic stenosis through a pulmonary arteriotomy, and the ostium-secundum defect through a right atriotomy. The heart failed soon after going off bypass and although the patient was placed again on total cardio-pulmonary bypass all attempts to re-establish an effective heart beat failed. Autopsy revealed extensive endocardial fibro-elastosis.

A left atrial myxoma was successfully removed through a left atriotomy (case 7); this case is the subject of a separate report.[9]

The last case in this series was the correction of mitral stenosis in a patient (case 30) in whom a closed valvotomy was performed 3 years before. This was successfully done with the use of total cardio-pulmonary bypass. In all we have operated upon 30 patients with 4 deaths (13·3%).

THE PERFUSION

The perfusion (Table VIII) presented no difficulty in any of the cases. In the last 12, stainless steel cannister 'debubblers' were employed (Lillehei—personal communication).

Flow rates were calculated in c.c. per kg. per minute, varying according to the size of the patient, a larger flow per kg. being used in the smaller-sized patients. The minimum flow rate in this series was 45 c.c. per kg. per minute, and the maximum 100.

The perfusion time varied between 14 and 135 minutes (average 51·7). Venous drainage was obtained by gravity, a difference of approximately 25 cm. being established between the height of the operating table and that of the venous well. The venous pressures varied between 5 and 25 mm. Hg with a mean of 13·3.

Return of oxygenated blood was always into the right common femoral artery. The mean perfusion pressures varied from 55 mm. Hg to 100, with an average of 66·3.

Oxygenation and carbon dioxide removal were always adequate and blood destruction minimal.

Removal of blood from the open heart was never a real problem. It was accomplished by gentle suction and the blood was returned to the oxygenator immediately. The

TABLE VIII. ANALYSIS OF BLOOD FLOW, PRESSURES, BLOOD DESTRUCTION AND GAS ANALYSIS DURING PERFUSION

Case No.	*Name*	*Wt. (kg.)*	*Duration* Anaes. (hrs. and mins.)	*Duration* Op. (hrs. and mins.)	*Duration* Per-fusn. (mins.)	*Flow Rate per min. (c.c.)* Total	*Flow Rate per min. (c.c.)* Per kg.	*Catheters (French gauge)* SVC	IVC	FA	*Venous Perfusion Pressure (mean) (mm. Hg)*	*Arterial Perfusion Pressure (mean) (mm. Hg)*	*EEG*	*Gas Analysis of Blood from Oxygenator at end of Bypass* Oxygen % Art.	Oxygen % Ven.	CO_2 vols. % Art.	CO_2 vols. % Ven.	*Haemolysis (mg. %)*
1	J.P.	40	6/12	5/50	15	2800	70	36	40	18	10	55	Satis.	100	93	28	32·9	Not done
2	R.J.	36	5/22	5/10	25½	2700	75	36	40	18	15	55	(*a*)	96	89	26	31	23·2
3	H.vW.	33	5/36	5/10	29	2300	70	34	36	16	19	50	Satis.	98	76	34	34	28·0
4	N.V.	70	7/44	7/2	26½	3260-3500	45-50	40	40	22	20	50	Satis.	Not analysed				Not done
5	M.J.	37	8/30	7/28	55	2200-2550	60-65	36	40	18	8	70	Satis.	100	78	30	38·1	Not done
6	N.vR.	—	6/28	5/35	31	3000-3350	50-55	40	40	24	9	60	Satis.	100	79	51	52·5	Not done
7	S.W.	57	8/56	8/12	45	2850-3705	50-65	40	40	20	10	65	Satis.	97	63	46	46	55·1
8	R.B.	53	8/50	7/36	22	2920	55	40	40	20	20	65	(*a*)	100	70	38	39	Not done
9	S.W.	54	8/30	8/0	55½	2720-3180	50-60	40	40	20	11	50	Satis.	100	67	35·4	40	52·0
10	A.S.	52	9/30	8/36	50	2860	55	40	40	20	15	55	Satis.	100	73	44	48	43·2
11	M.M.	24	7/31	7/2	73	1440	60	36	40	16	23	55	Satis.	98	70	33·2	40	68·9
12	S.H.	50	8/5	7/3	64	2750	55	36	40	18	18	70	Satis.	98	70	25·1	42·5	61·0
13	T.K.	20	9/7	8/30	88	1200-1400	60-70	36	36	14	14	100	Satis.	100	75	36·4	43	81·0
14	C.B.	26	7/30	6/39	24	1560-2080	60-80	30	40	14	20	65	Satis.	100	85	36·7	36·7	19·6
15	P.R.	46	6/31	5/35	31	2760	60	36	40	18	21	75	Satis.	95	73	35·7	40·8	31·2
16	D.H.	30	8/42	8/10	93	2100	70	40	40	20	17	95	Satis.	94	42	29·5	34	83·1
17	J.C.	26	7/2	6/42	135	1820-2080	70-80	36	36	16	—	75	(*b*)	Not taken				Not done
18	W.vD.	35	6/20	5/19	14	2450	70	40	40	16	15	70	Satis.	99	78	35	45·5	Not done
19	E.G.	40	5/40	5/13	27	2800-3000	70-75	40	40	18	10	80	Satis.	100	75	39·5	41·8	Not done
20	P.K.	57	10/22	9/32	99	3135-2850	50-55	40	40	20	15	80	Satis.	95	75	30·9	45·0	101·0
21	Y.H.	42	6/49	6/0	29½	2520-2730	60-65	40	40	18	16	55	Satis.	Not analysed				Not done
22	E.A.	48	7/32	6/50	80	2880-3120	60-65	40	40	18	16	55	Satis.	Not analysed				78·0
23	J.A.	32	7/14	6/8	69	2240-3200	70-100	40	40	16	25	100	Satis.	100	66	37·2	46	80
24	S.R.	52	8/29	7/52	32	3120-3380	60-65	36	40	22	10	65	Satis.	100	76	36·0	36·4	21·9
25	J.G.	62	7/56	7/21	45	3410-3720	55-60	40	40	22	20	70	Satis.	100	38	39·7	44·9	75
26	A.G.	22	6/55	5/54	75	1650-1870	75-85	34	34	18	13	85	Satis.	96	53	38·9	43·9	64·6
27	J.P.	20	6/47	6/1	77	1600-1900	80-95	32	36	16	20	90	Satis.	94	65	34	50	68·3
28	R.S.	32	5/17	4/21	62	2400-2720	75-85	32	40	18	5	75	(*a*)	93	52	37·8	38·2	40·3
29	A.R.	—	4/33	4/33	28	1600-1400	70-80	32	40	18	8	—	(*a*)	98	60	42·6	48·6	32·2
30	E.A.	69	6/17	5/39	54	3450-3795	50-55	40	40	22	9	65	Satis.	97	39	44·4	47·6	121·5

(*a*) Not recorded. (*b*) Gradual deterioration from cessation of cardiac arrest period.

electro-encephalogram was satisfactory throughout perfusion in all cases.

POST-OPERATIVE

Post-operative care has been previously discussed.[3] In the immediate post-operative phase careful measurement and replacement of blood loss from the chest catheters and care of the tracheobronchial tree are amongst the most important aspects leading to a low mortality. Clearing the secretion from the mouth, nasopharynx and hypopharynx by careful suctioning is recommended. Oxygen and humidity often supplemented with bronchial detergents were used by routine.

Unless there are unusual losses, electrolyte imbalance is not a problem in the post-operative phase. Intravenous fluids were limited to $1\cdot5-2$ c.c. per kg. for the first 24 hours, including intravenous fluids given during the operation. The prevention of acidosis in the post-operative period is of the greatest importance. An adequate circulation is the most important single factor in the prevention of this grave complication. However, control of restlessness and high temperatures and the use of intravenous sodium bicarbonate will be of some help.

Post-operative heart failure was treated in the usual manner. Temporary cardiac decompensation that occurs 10-14 days after surgery as the result of the traumatic myocarditis was seen occasionally, especially in patients with low pre-operative cardiac reserve. It is detected early by following daily weights; and it responds well to digitalization and salt restriction and in no way jeopardizes an ultimately good prognosis if properly managed. Antibiotics were given for 14-21 days post-operatively.

Post-operative Complications

Chest haemorrhage was excessive in 3 patients and as soon as this complication was detected the patients were taken back to the operating room and the bleeding dealt with. Prompt action with this form of complication is the only form of treatment. The usual blood loss was less than 750 ml.

As coughing and deep breathing are difficult immediately after operation, retention of bronchial secretion became a complication in several cases. In 2 patients direct suction of the bronchi had to be resorted to, but in none of the patients was the performance of a tracheotomy necessary. One patient developed broncho-pneumonia, which responded to the appropriate antibiotics.

Superficial wound sepsis occurred in 2 patients and deeper sepsis in one, but in no instance was there any suggestion of infection of the blood stream, mediastinum, sternum or pleural cavities.

We were fortunate in not encountering heart block of any form in this series.

SUMMARY AND CONCLUSION

Our results agree with those of workers in other countries in showing that many congenital cardiac anomalies and some acquired heart lesions can be corrected as a routine surgical procedure with an acceptable low mortality by a well trained team of doctors, nurses and technicians. There is little doubt today that for complex cardiac lesions that were incurable before the advent of extracorporeal circulation the treatment of choice is open cardiotomy with total body perfusion. On the other hand, some difference of opinion may still exist concerning intracardiac conditions in which an improvement in the patients' status was possible by the use of indirect or blind methods of surgical treatment. In this issue the only objective worthy of consideration is the patients' welfare; that is to say, the treatment should be selected which is known to give the best results at the least risk. Considerations such as economic factors, the special equipment or assistance needed, the work thrown upon the blood transfusion service and other personnel, are of secondary importance when human life and happiness are at stake.

In this brief review of our first 30 cases treated by open cardiotomy with extracorporeal circulation, the method of correction of certain common congenital defects is discussed in some detail. We should like to emphasize again that in this rapidly expanding field views necessarily change as the result of new experiences. Firm ideas should thus not be held until opinion is crystalized in the light of further knowledge of the problems involved.

ADDENDUM

Since the completion of this article another 22 cases were treated by open cardiotomy with extracorporeal circulation. Of these cases 2 died. Thus of the 52 (30 + 22) cases treated, 6 died. The 22 cases included complicated clinical conditions such as tetralogy of Fallot, endocardial-cushion defect, rheumatic mitral incompetence, and rheumatic aortic stenosis.

We are indebted to Prof. J. H. Louw, Professor of Surgery at the University of Cape Town, Dr. J. G. Burger, Medical Superintendent of Groote Schuur Hospital, and Dr. J. F. W. Mostert, Medical Superintendent of the Red Cross War Memorial Children's Hospital, for their continued encouragement and support; Dr. V. Schrire and other members of the Cardiac Clinic, Groote Schuur Hospital, not only for referring all these patients to us, but also for their great help in the operating theatre during surgery; Dr. E. R. Rudman, Medical Superintendent of the Western Province Blood Transfusion Service and his staff for their unfailing cooperation; Dr. S. Berman, Head of the Department of Neuropsychiatry and his staff for monitoring and interpreting EEG recordings during operation; Dr. C. S. Jones, Head of the Department of Anaesthesia, Dr. J. Ozinsky and Dr. A. B. Bull; the various surgical registrars and housemen who have 'specialed' these patients; and the Matrons and nursing staff of Groote Schuur Hospital and the Red Cross War Memorial Children's Hospital. To Mr. C. Goosen, Mrs. V. Connell and Miss D. Shepstone, the technical staff of the Department of Surgery, we owe a special debt of gratitude.

To the United States Public Health Department and the Cape Provincial Hospitals Administration, we are greatly indebted, for their financial assistance and their support of the project as a whole, not only ensured the development of the apparatus but also its clinical success.

REFERENCES

1. McKenzie, M. B. and Barnard, C. N. (1958): S. Afr. Med. J., 32, 1145.
2. Phillips, W. L. and Barnard, C. N. (1958): Med. Proc., 4, 722.
3. Barnard, C. N., DeWall, R. A., Varco, R. L. and Lillehei, C. W. (1959): Dis. Chest, 35, 194.
4. Kirklin, J. W. and McGoon, D. G. (1958): J. Thorac. Surg., 35, 584.
5. Melrose, D. G., Dreyer, B., Bentall, H. H. and Baker, J. B. E. (1955): Lancet, 2, 21.
6. Truex, R. C. and Bishof, J. K. (1958): J. Thorac. Surg., 35, 421.
7. Kirklin, J. W., Harshburger, H. G. Donald, D. E. and Edwards, J. E. (1957): *Ibid.*, 33, 45.
8. Warden, H. E. DeWall, R. A., Cohen, M., Varco, R. L. and Lillehei, C. W. (1957): *Ibid.*, 33, 21.
9. Barnard, C. N., Swanepoel, A., McKenzie, M. B. and Phillips, W. L. (1959): In press.