Ventricular aneurysms after cardiac surgery

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Seven cases of ventricular aneurysm which developed after cardiac surgery are presented. Three were left ventricular aneurysms, all of which occurred after the operation of closed or 'blind' aortic valvotomy in adults, and four were right ventricular outflow tract aneurysms which developed in children after surgical procedures for the correction of severe forms of Fallot's tetralogy. Two of the left ventricular aneurysms arose at the site of the ventriculotomy used for introducing the Brock's dilator. The third aneurysm in an adult presented as a surprise finding at a second operation at which the calcified aortic valve was replaced. This aneurysm may have resulted from the creation of a 'false passage' through friable calcification at the base of the aortic leaflet of the mitral valve at the time of closed valvotomy. In the tetralogy cases a significant pressure gradient remained across the right ventricular outflow tract or pulmonary valve ring after operation. It is suggested that this is an important factor in the aetiology of this type of aneurysm.

Very few examples of ventricular aneurysm resulting from cardiac surgery have been reported since the first case of Smithy, Boone, and Stallworth (1950). The subject has been well reviewed by Kerr, Wilcken, and Steiner (1961) and Hudson (1965). In this paper, we report a further 7 cases.

Left ventricular aneurysms in adults: case reports

Case 1 A 35-year-old labourer had a transventricular valvotomy for aortic stenosis, using a Brock dilator, in April 1964. The systolic gradient over the aortic valve before and after operation was 50 mmHg. Convalescence was uneventful and the patient was subjectively better, though his electrocardiogram and chest radiograph remained unchanged.

The patient was referred again in November 1969, with a history of severe exertional retrosternal pain. His physical signs of aortic valve disease were essentially unchanged but the electrocardiogram and chest radiograph showed evidence of increased left ventricular size. The patient underwent aortic valve replacement on 27 January 1970, using conventional normothermic cardiac bypass technique. A thin pulsatile swelling approximately 2 cm in diameter was noticed between the roof of the left atrium and the left sinus of Valsalva (Fig. 1). The aorta was opened obliquely revealing a totally disorganized, heavily calcified valve with a rigid orifice 1.0×0.5 cm. This valve was excised revealing the mouth of the aneurysm

FIG. 1 Case 1. The thin pulsatile aneurysm noticed between the roof of the left atrium and the left sinus of Valsalva.

just below the base of the left coronary cusp of the aortic valve and penetrating the base of the anterior leaflet of the mitral valve. The mouth of the aneurysm measured 0.75 cm in diameter (Fig. 2). Three mattress sutures were used to close it before a No. 10 Starr Edwards prosthesis was secured in place. After completion of the operation, the region of the aneurysm was collapsed and non-pulsatile.

The postoperative recovery was uneventful.

Received 21 April 1971.

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FIG. 2 Case 1. The mouth of the aneurysm just below the base of the left coronary cusp of the valve.

Case 2 A 48-year-old spinster had a transventricular valvotomy for aortic stenosis in April 1964. The aortic gradient before and after surgery was 135 mmHg and 30 mmHg, respectively. The ventriculotomy, which was made anterior to the apex of the left ventricle, was repaired using two silk sutures. The early postoperative period was uneventful but later a large bulge, continuous with the left ventricular border, appeared on the chest radiograph (Fig. 3); this bulge was seen to pulsate paradoxically on screening the heart. A diagnosis of left ventricular aneurysm was made. The electrocardiogram was identical to the preoperative record.

The patient refused further investigation or surgery. She soon developed signs of congestive cardiac failure and was maintained on digoxin and diuretic therapy until her death in November 1967. Necropsy was not performed.

FIG. 3 Case 2. Postoperative radiograph showing a large bulge continuous with the left ventricular border.

Case 3 A 30-year-old man had a transventricular aortic valvotomy for aortic stenosis in July 1962. The systolic gradient was reduced from 95 mmHg to 20 mmHg and the ventriculotomy was repaired with two silk sutures.

The postoperative period was complicated by a collapsed left lower lobe but was otherwise uneventful. Postoperative chest radiographs showed no appreciable alteration in the cardiac silhouette and the electrocardiogram was unchanged. A grade 2/3 left parasternal diastolic murmur was clearly audible after operation.

After discharge from hospital, the patient defaulted from the Follow-up Clinic. He was not seen again until seven years later when he was referred after a mass miniature chest radiograph, reported as showing a 'clear cut calcified aneurysm of the anterior and apical aspect of the left ventricle' (Fig. 4 and 5). Once again, he admitted to only slight exertional dyspnoea. He had the physical signs of aortic stenosis and regurgitation, and the electrocardiogram still showed grade 3 left ventricular hypertrophy and strain. The left ventricular bulge did not pulsate at fluoroscopy. Left heart catheterization was undertaken but the left ventricular cavity could not be entered by either the transseptal or retrograde aortic approach. A cineangiogram was performed using a left atrial injection through a transseptal catheter; the contrast medium in the left ventricle did not enter the sac, which was presumed to have thrombosed. This patient remains under review in the outpatient clinic.

Right ventricular aneurysms in children: case reports

Case 4 This 19-year-old girl was diagnosed as a case of tetralogy of Fallot by a right ventricular angiogram at the age of 2 years. A left subclavian artery to pulmonary artery anastomosis was performed at that time. Two years later she was readmitted because of severe cyanosis and syncopal attacks. A right ventricular infundibulotomy was performed using inflow occlusion technique. A probe was passed up from the body of the right ventricle, and with difficulty this was manipulated through a very narrow channel into a small subvalvar chamber; the stenosis was relieved with punch forceps. The cyanosis and exercise tolerance were much improved after this operation.

A chest radiograph taken 10 years later showed a rounded mass in the region of the pulmonary artery (Fig. 6). This was shown by angiography to be an aneurysm of the infundibulum of the right ventricle extending to the left and tending to be bilocular (Fig. 7). There was stenosis within the sac and the pulmonary valve was displaced. A gradient of 60 mmHg was shown between the infundibulum and the body of the right ventricle; the pressure in the former being 12 mmHg.

Three months later operation was performed using cardiopulmonary bypass. The aneurysm arising from the infundibular chamber was ex-

FIG. 4 Case 3. Chest radiograph taken 7 years after closed aortic valvotomy showing a calcified bulge on the anterior and apical aspect of the left ventricle.

posed and the right ventricle was opened. A sub-valvar diaphragm with an opening of 10 mm was removed. The pulmonary valve was narrowed to the same dimension and this was opened by valvotomy giving an orifice of 15 mm. The mouth of the aneurysm arising from the large subvalvar chamber was closed with sutures and patch closure of the ventricular septal defect (3×2 cm) was performed. A gradient of 50 mmHg existed between the body and infundibulum of the right ventricle at the conclusion of this procedure, and the electrocardiogram showed a complete right bundle-branch block pattern.

FIG. 5 Case 3. Lateral chest radiograph showing the calcification.

The patient made a satisfactory recovery until 12 months later when she collapsed and died suddenly at her home. No necropsy was performed.

Case 5 This 14-year-old girl was diagnosed as a case of tetralogy of Fallot at the age of 4 months and a left subclavian to pulmonary artery anastomosis was performed when she was 2 years old.

Cardiac catheterization was undertaken at the age of 12 years and showed a right ventricular pressure of 100 mmHg and also showed the presence of shunts at atrial and ventricular levels.

Operation was performed using cardiopulmonary bypass. The pulmonary artery was half the aortic diameter and was at low pressure. There was a small infundibular chamber with a thrill over it and over the pulmonary artery. The pulmonary valve had an orifice of 8 mm and this was opened to 16 mm with two incisions. Hypertrophied muscle was excised from the right ventricular outflow tract and the ventricular septal defect (2.0×2.0 cm) was closed with a Dacron patch. A patch of Dacron and pericardium was sutured into the ventriculotomy up to the level of the pulmonary valve ring. The Blalock-Taussig anastomosis was ligated.

The postoperative course was complicated by congestive cardiac failure and lower lobar collapse of the left lung. The electrocardiogram showed a right bundle-branch block. Cardiac failure persisted and the chest radiograph now showed a bulge in the region of the pulmonary bay.

The patient was recatheterized and the right ventricular pressure was found to be 100 mmHg with a gradient of 65 mmHg across the pulmonary valve. Angiography suggested a bilocular aneurysm of the outflow tract arising from the infundibulum.

Surgery was again performed in order to explore the heart and resuture the outflow tract bulge. There was a diffuse bulge with reasonable contraction and no paradoxical pulsation in the region of the right ventricular outflow tract. A large weavекnit patch was sutured over the outflow tract.

The postoperative course was uneventful. She was well when seen six months later, but a chest radiograph showed that the aneurysm had in fact increased in size (Fig. 8). No further operation is contemplated in the near future.

Case 6 A 12-year-old boy in whom a diagnosis of tetralogy of Fallot was made when aged 6 months. At the age of 3 years total correction using cardiopulmonary bypass was performed. The aorta was 19 mm and pulmonary artery 8 mm in diameter, the latter having little flow in it. There was both valvar and subvalvar stenosis. An incision was made into the infundibular region and the valve was opened and dilated. The outflow tract was cored out as much as possible. A ventricular septal defect of 2.0 cm diameter was sutured. The final pressures were pulmonary

FIG. 6 Case 4. Chest radiograph taken 10 years after operation shows the rounded expansion in the region of the pulmonary artery.

arterial 45 mmHg; right ventricular 80 mmHg, equal to the systemic pressure.

Postoperatively there was congestive cardiac failure requiring digoxin and diuretics. The electrocardiogram showed complete right bundle-branch block with ST segment depression in leads V₃R and V₁ to V₄. His effort tolerance improved gradually and when seen 7 years later he was noted to have conspicuous pulsation over the right ventricle. A chest radiograph revealed rotation of the upper border of the heart on the left side (Fig. 9) which pulsated on screening. This was thought to be an aneurysm of the outflow

FIG. 7 Case 4. Right ventricular angiogram showing a large true aneurysm of the infundibulum.

tract. It has been decided to keep the patient under review and not intervene surgically for the time being.

Case 7 This 11-year-old girl was diagnosed as having tetralogy of Fallot at the age of 18 months. Cardiac catheterization revealed a gradient across the right ventricular outflow tract and pulmonary valve of 108 mmHg. The aorta was entered across the ventricular septal defect from the right ventricle.

Four months later total correction was performed using cardiopulmonary bypass. The pulmonary artery was 'very small', being one-third the aortic diameter. A vertical right ventriculotomy was performed. The pulmonary valve ring was very small and the valve cusps were thickened and rudimentary. There was conspicuous hypertrophy of the crista supraventricularis but 'not much tissue could be removed without danger of damaging the aortic ring'. The ventricular septal defect was closed with a patch of Dacron, and a large pear-shaped patch was sutured into the ventriculotomy as a gusset, up to but not across the pulmonary valve ring. This patch was further covered with a pericardial onlay patch.

The postoperative period was uneventful; the electrocardiogram showed right bundle-branch block. A year later a routine chest radiograph showed a large bulge in the region of the right ventricular outflow tract which appeared to contain calcium in its wall. She was admitted for cardiac catheterization. The right ventricular pressure was 110/0 mmHg and a left-to-right shunt of 1.2 l./min was shown. The right ventricular angiogram was reported as showing trabeculation of the right ventricle with a large aneurysm of the outflow tract distorting the anatomy. There is calcification in its wall and possibly clot is present. The pulmonary valve is displaced (Fig. 10).

This patient is under consideration for a 'reconstruction procedure' of the right ventricular outflow tract.

Discussion

Ventricular aneurysms after cardiac operations have not been commonly reported; but of those recorded incisional aneurysms are the commonest (Table 1). This type of aneurysm usually follows the introduction of a dilating instrument through the ventricular wall. Though this type of operation has become much less common with the use of open-heart procedures, cases may still occur and some present with this complication at open-heart surgery. Kerr *et al.* (1961) gave an excellent review of these aneurysms which, they pointed out, could be 'false' or 'true'. Infection and faults in suturing technique leading to disruption or ischaemic necrosis were considered the most important causes of a false aneurysm, while infarction, 'unsound' muscle, and high intraventricular pressure

FIG. 8 Case 5. Chest radiograph taken 6 months after reoperation shows that the aneurysm has increased in size.

resulting from incompletely relieved valve stenosis were the principal antecedents of the true variety.

There are only three reported examples of false ventricular aneurysms after operation. The two cases described by Smith, Goldberg, and Bailey (1957) were attributed to faults in the technique of suturing the ventricle. In the first case the surgeon had inadvertently perforated the left ventricle with his finger during mitral commissurotomy and had repaired this

FIG. 9 Case 6. Chest radiograph taken 7 years postoperatively showing an enormous bulge continuous with the left border of the heart.

rather quickly. The second case was only noticed two years after closed aortic valvotomy, when the patient had been involved in a road accident. It seems unlikely that the trauma had any bearing on the development of this aneurysm since retrospective appraisal of the chest radiographs showed that it was present, though much smaller, soon after the valvotomy. A failure to place the sutures through the full thickness of the muscle was thought to have led to disruption and subsequent leakage of blood into the pericardium to form the false aneurysm.

The third example (Case 1 of Kerr *et al.*, 1961) was thought to be the result of staphylococcal infection of the suture line leading to wound disruption. This patient had developed a staphylococcal pulmonary infection with an associated pleural effusion on the third postoperative day. At reoperation the original cardiac sutures were removed and subsequently *Staph. albus* was cultured from them.

In all three cases the false aneurysm was successfully repaired.

Our third case was probably a false aneurysm; the diagnosis lies between a thrombosed aneurysm and an intrapericardial haematoma. The former was considered more likely since we might have expected evidence of a haematoma much earlier; there was no evidence of a ventricular bulge six weeks after operation when the patient was discharged.

Cineangiograms showed that contrast material in the left ventricle did not enter the sac, indicating thrombosis.

True aneurysms are commoner in both this series and also in the published reports. Kerr *et al.* (1961) were of the opinion that interstitial haemorrhage and oedema after instrumentation could devitalize surrounding muscle fibres and produce a small area of infarction in the heart wall. Their third case, a man aged 26, developed the electrocardiographic changes of an anterior myocardial infarct on the day after a transventricular aortic dilatation. A true left ventricular aneurysm developed 11 weeks later and was presumed to be the result of the infarct. Their second case with mitral stenosis had angina pectoris with a positive electrocardiographic effort test before operation; thus 'unsound' muscle may have contributed to the subsequent development of a true ventricular aneurysm.

None of our patients had any electrocardiographic evidence of myocardial infarction. The classical persistent ST segment rise seen in cases of ventricular aneurysm, due to coronary artery disease, is not usually a feature of

FIG. 10 Case 7. Right ventricular angiogram showing an infundibular aneurysm.

ably played an important part in the subsequent development of an aneurysm at that site.

In the case described by McCord and Blount (1955) a large thin aneurysm arose from the infundibular chamber while the previous cardiectomy incision was well healed. They attributed the aneurysm to the sudden increase in pressure and blood flow through the thin-walled chamber which had previously been protected by the infundibular stenosis. This abrupt change did not allow sufficient time for compensatory hypertrophy to occur. Our Case 5 is similar in the respect that a true aneurysm of the infundibular wall developed, in the absence of a previous incision at this site. The use of punch forceps to enlarge the small channel through the narrowed outflow tract may have perhaps damaged the myocardium.

These four cases illustrate that postoperative ventricular aneurysms may arise from 'normal' ventricular musculature, through ventriculotomy incisions or through a bulging patch of synthetic material. In all cases the causative factor appears to have been distal obstruction, either within the outflow tract of the right ventricle or at the pulmonary valve or valve ring. The satisfactory correction of such severe cases of Fallot's tetralogy poses difficult problems. Opinions differ as to the long-term effects of leaving a child with free pulmonary regurgitation, and the surgeon's view on this question will determine his practice in carrying the ventriculotomy across a narrow valve ring and into the pulmonary artery beyond. If this practice is adopted, a gusset is inserted to enlarge the whole outflow tract, but this should abolish any pressure gradient. In recent years reconstructive procedures have been advocated employing homograft valve leaflets or grafts of the pulmonary artery or aorta including valve leaflets. More recently still, fascia lata reconstructions have been employed with promising early results. The longer-term follow-up of these techniques is awaited with interest, as indeed is the clinical follow-up of cases left with free pulmonary regurgitation.

The intraventricular pressure is also important in adult cases. In the two cases of Segel *et al.* (1957) there was a considerable degree of aortic incompetence after closed aortic valvotomy which probably increased the strain of the left ventricle, while in both cases of aortic stenosis reported by Kerr *et al.* the final left ventricular pressures were higher than normal.

Case 1 is unique in that this particular type of aneurysm has not previously been

this type of 'surgical' aneurysm (Kerr *et al.*, 1961). Some authors recorded T-wave changes, which they thought could have been due to myocardial damage (McCord and Blount, 1955; Smith *et al.*, 1957). The electrocardiographic record did not change appreciably in our 3 adult cases, but all 4 young children developed right bundle-branch block after operation. The occurrence of right bundle-branch block after surgical repair of tetralogy of Fallot and ventricular septal defect is well known (Rosenbaum *et al.*, 1970) and is unlikely to have been important in the subsequent development of the aneurysms.

The four aneurysms developed in children suffering from severe forms of Fallot's tetralogy; in each case a significant pressure gradient remained across the right ventricular outflow tract or pulmonary valve after operation. The high intraventricular pressure presumably favours the development of an aneurysm, particularly if the ventricular wall has been weakened by ventriculotomy. The insertion of a Dacron patch as in Cases 6 and 7 would further weaken the outflow tract. In the case reported by Campbell, Deuchar, and Brock (1954), the myocardium of the right ventricle was lacerated by the infundibular punch during its withdrawal. This prob-

TABLE 1 *Review of published cases of ventricular aneurysms after cardiac operation*

Author	No. of cases	Operative procedure	Site of aneurysm	Type of aneurysm
Smith <i>et al.</i> (1957)	2	(a) Transatrial digital mitral valvotomy	(a) Left ventricle	False
		(b) Aortic valvotomy	(b) Left ventricle	False
Smithy <i>et al.</i> (1950)	1	Mitral valvulotomy	Left ventricle	True
Campbell <i>et al.</i> (1954)	1	Closed pulmonary valvotomy (Fallot's tetralogy)	Right ventricle	True
McCord and Blount (1955)	1	Infundibular resection (Fallot's tetralogy)	Right ventricle (infundibular chamber)	True
Segel <i>et al.</i> (1957)	2	(a) Aortic valvotomy	(a) Left ventricle	?
		(b) Aortic valvotomy	(b) Left ventricle	?
Derra and Loogen (1959)	1	Closed pulmonary valvotomy	Right ventricle	True
Fatti (1959)	1	Closed pulmonary valvotomy (Fallot's tetralogy)	Right ventricle	True
Kerr <i>et al.</i> (1961)	4	(a) Aortic valvotomy	(a) Left ventricle	(a) False
		(b) Mitral valvotomy	(b) Left ventricle	(b) True
		(c) Aortic valvotomy	(c) Left ventricle	(c) True
		(d) Closed pulmonary valvotomy (Fallot)	(d) Right ventricle	(d) True

described. It seems likely that the dilator failed to enlarge the valve orifice in view of the failure to lower the systolic gradient. It is probable that the false passage was produced directly by the dilator. The aneurysm could not be detected in the straight chest radiographs and was a 'surprise' finding at open-heart surgery. It is conceivable that with increasing left intraventricular pressure, rupture of the aneurysm could have occurred.

The operation of closed aortic valvotomy has now generally given way to open-heart procedures with valve replacement, and fewer of these aneurysms should be encountered in the future. The principle, however, still holds that the surgeon's aim must be to abolish any existing valve gradient and this may influence his choice of valve substitute. Unfortunately, some of the available prostheses still leave a

significant gradient across the valve (Wright, 1970). Cardiac failure is a feature of nearly all traumatic aneurysms unless they are small or have thrombosed. Case 2 rapidly developed cardiac failure after the appearance of the aneurysm, while in Case 3 the thrombosed aneurysm had no adverse haemodynamic effects. Case 4 required reoperation because of her symptoms and Case 7 has considerable symptoms.

Only two of these aneurysms have been successfully treated by operation (Cases 1 and 4, respectively). The increasing size of the aneurysm in Case 5, despite further surgery, is not surprising since the right ventricular pressure was still at systemic level.

There was no indication to intervene in Case 3 since the aneurysm was already thrombosed and had not given rise to systemic emboli. Cases 6 and 7 will almost certainly come to surgery in the future. Kerr *et al.* dealt successfully with both of their true aneurysms while Segel *et al.* did not attempt to repair their two and the patients were alive two years later. The patient of McCord and Blount died after operation after his infundibular aneurysm was repaired.

Patients may survive for long periods with this type of aneurysm (Kerr *et al.*, 1961), but the presence of cardiac failure or systemic embolism justifies surgical treatment.

TABLE 2 *The pre- and postoperative gradients in our 7 cases*

Case No.	Site of obstruction	Preop. gradient (mmHg)	Postop. gradient (mmHg)
1	Aortic valve	50	50
2	Aortic valve	135	30
3	Aortic valve	95	20
4	Right ventricular outflow tract	?	50
5	Right ventricular outflow tract	?	65
	RV 100		
6	Pulmonary valve	?	35
7	Right ventricular outflow tract	108	?
	RV 110		

We are grateful to Mr. F. Ronald Edwards, Mr. B. J. Bickford, Mr. L. J. Temple, and Mr. M. Hickey for permission to report these cases.

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Addendum

After this paper was written Wychulis *et al.* (1971) reported 3 patients with idiopathic hypertrophic subaortic stenosis who developed true ventricular aneurysms after surgical resection of hypertrophic muscle from the left ventricular out-flow tract. Two of these aneurysms were thought to be due to interruption of the coronary blood supply, resulting in myocardial infarction, at the time of operation.

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