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 x 0.5 cm. This valve was excised revealing the mouth of the aneurysm 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.
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 angiocardiogram 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 angiocardiography 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-
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 val-
votomy giving an orifice of 15 mm. The mouth of
the aneurysm arising from the large subvalvar
 chamber was closed with sutures and patch clo-
sure of the ventricular septal defect (3 x 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.

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

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 anasto-
mosis 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 cardiopulmo-
 nary 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 pul-
monary valve had an orifice of 8 mm and this was
opened to 16 mm with two incisions. Hypertro-
fied muscle was excised from the right ven-
tricular outflow tract and the ventricular septal
defect (2.0 x 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 col-
lapse 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 aneu-
rysms of the outflow tract arising from the
infundibulum.

Surgery was again performed in order to ex-
 plore 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 weaveknit patch was sutured over the out-
flow 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 out-
flow tract was cored out as much as possible. A
ventricular septal defect of 2.0 cm diameter was
sutured. The final pressures were pulmonary
ventricle. A chest radiograph revealed rotation of the upper border of the heart on the left side (Fig. 9) which pulsed on screening. This was thought to be an aneurysm of the outflow 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
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 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
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, Deuchars, and Brock (1954), the myocardium of the right ventricle was lacerated by the infundibular punch during its withdrawal. This probably 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 cardiotomy 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 hypertension 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
TABLE I  Review of published cases of ventricular aneurysms after cardiac operation

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

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 on 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.

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.

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

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Site of obstruction</th>
<th>Preop. gradient (mmHg)</th>
<th>Postop. gradient (mmHg)</th>
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<tbody>
<tr>
<td>1</td>
<td>Aortic valve</td>
<td>50</td>
<td>50</td>
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<td>2</td>
<td>Aortic valve</td>
<td>135</td>
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<td>3</td>
<td>Aortic valve</td>
<td>95</td>
<td>20</td>
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<tr>
<td>4</td>
<td>Right ventricular outflow tract</td>
<td>?</td>
<td>50</td>
</tr>
<tr>
<td>5</td>
<td>Right ventricular outflow tract</td>
<td>?</td>
<td>65</td>
</tr>
<tr>
<td>6</td>
<td>Pulmonary valve</td>
<td>RV 100</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Right ventricular outflow tract</td>
<td>108</td>
<td>RV 110</td>
</tr>
</tbody>
</table>
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References


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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 outflow 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.

Reference