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Annular injuries following the insertion of heart valve prostheses[1]

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Bowes, V. F., Datta, B. N., Silver, M. D. and Minielly, J. A. (1974). *Thorax*, **29**, 530–533. **Annular injuries following the insertion of heart valve prostheses.** The clinical presentation and morphological findings in eight cases of annular injury associated with the insertion of prosthetic heart valves are discussed. The lesions presented as a separation of the left atrium and ventricle at the mitral annulus (one patient), a sinus or false aneurysm in the left posterior atrioventricular groove (two patients), or as a separation of the aortic root from the base of the heart (five patients). The pathogenesis of the lesions is similar. Annular tissue was weakened either by the removal of excess tissue or by an abnormal stress placed on normal tissue or by a slight stress placed on tissue altered by disease, and, as a result, it separated or tore. The resultant haemorrhage either caused the patient's death or, with time, produced the other morphological findings. Factors predisposing patients to the injuries are discussed and a differential diagnosis of annular pseudoaneurysms is provided.

The insertion of a prosthetic heart valve is an accepted method of treating valvular heart disease. During the surgical procedure injuries may occur at a valve annulus. Some cause a sudden fatal haemorrhage; others do not kill the patient but produce either a sinus tract that projects into the atrioventricular sulcus from the rim of the prosthesis or a false aneurysm that bulges the epicardial surface in this area, morphological findings that may puzzle a pathologist when encountered at necropsy for the first time. In this paper we discuss eight examples of annular injury encountered in the past 12 years, present the morphological findings, and discuss factors that may predispose patients to the injuries.

PATIENTS AND METHODS

PATIENT DATA (Table) Eight examples of annular injury were found at necropsy in patients who had one or more heart valves replaced by ball, disc, or homologous tissue prostheses at the Toronto General Hospital in the years 1962 to 1973. More than 1,500 patients were operated on in that period. The necropsied patients ranged in age from 22 to 60 years. Most were men and had had a single valve replaced; half had had rheumatic valvular disease.

HISTOLOGY Histological examinations were done on tissue specimens stained with haematoxylin and eosin, by a combined Verhoeff elastic–Masson trichrome stain and by the WHO stain (Masson trichrome–Alcian green).

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PATHOLOGICAL FINDINGS

Three morphologically distinct types of annular injuries were encountered; each will be discussed separately.

SEPARATION OF LEFT ATRIUM AND VENTRICLE AT MITRAL ANNULUS A 54-year-old woman with mitral insufficiency as a result of a prolapsed posterior mitral valve leaflet had the valve excised and a Starr–Edwards ball valve prosthesis inserted. Twenty minutes after bypass ended severe bleeding started from the anterolateral region of the mitral annulus. She died in the operating room.

The left atrium had separated from the ventricle anterolaterally. A tear with ragged edges extended 2·5 cm along the annulus and was up to 3 cm wide. A probe could be passed through it to the epicardial surface. Histologically, haemorrhage extended to the epicardial fat. The excised valve showed myxomatous degeneration, and an increased amount of mucopolysaccharide material was seen in the annular fibrous tissue. The latter change may have predisposed the patient to the injury.

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TABLE
PATIENT DATA

Age/Sex	Clinical Diagnosis	Valve Replaced	Type of Injury
54 F	Mitral incompetence due to prolapsed posterior mitral valve leaflet (myxomatous degeneration)	Mitral	Separation of left atrium and ventricle at mitral annulus
47 F	Mitral stenosis due to rheumatic valvular disease	Mitral	Sinus formation at mitral valve annulus
44 M	Mitral and aortic stenosis and incompetence due to rheumatic valvular disease	Mitral and aortic	False aneurysm formation at mitral valve annulus
52 M	Aortic stenosis and incompetence due to rheumatic valvular disease	Aortic	Separation of aortic root from base of heart at aortic annulus
55 F	Aortic stenosis and incompetence, mitral and tricuspid stenosis due to rheumatic valvular disease	Tricuspid, mitral, aortic	Separation of aortic root from base of heart at aortic annulus
44 M	Aortic stenosis (due to a congenitally bicuspid valve)	Aortic	Separation of aortic root from base of heart at aortic annulus
60 M	Calcific aortic stenosis (valve tricuspid – cause of change not established)	Aortic	Separation of aortic root from base of heart at aortic annulus
34 M	Aortic incompetence due to infective endocarditis on an aortic Starr–Edwards prosthesis	Aortic	Separation of aortic root from base of heart at aortic annulus

SINUS OR FALSE ANEURYSM FORMATION AT THE MITRAL ANNULUS Two patients had either a sinus or a false aneurysm at the mitral valve annulus. Neither lesion was detected during life. The first had mitral stenosis due to rheumatic valvular disease. The mitral valve was replaced by a Beall prosthesis six months before she died of a cerebral infarct. At operation a mitral commissurotomy was done first, but the posterior leaflet tore badly instead of the commissures separating. As a result the valve leaked badly and had to be replaced. A sinus was found in the posterior wall of the left ventricle at the atrioventricular junction. It extended 2 cm laterally and towards the apex of the heart, passing from the rim of the prosthesis on its ventricular aspect through the mitral annular tissue and into the epicardial fat between atrium and ventricle (Fig. 1). It did not cause the epicardial surface of the heart to bulge. Its diameter at the ostium was 2 mm and at its distal end 8 mm. The lumen was filled with thrombus. Histologically the walls were composed of dense

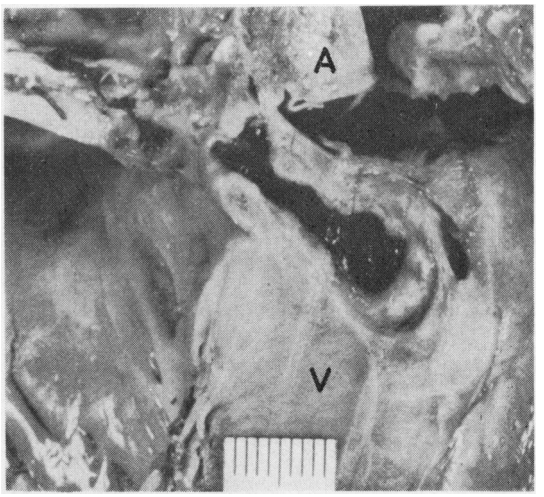

FIG. 1. *Sinus filled with black thrombus extending from ventricular lumen near rim of mitral prosthesis into epicardial fat between left atrium (A) and ventricle (V). Note the smooth mouth of the ostium and the walls of connective tissue. (Scale indicates 1 cm.)*

fibrous tissue and organizing thrombus and did not contain any myocardial fibres.

The second patient died of a myocardial infarction one year after both the mitral and aortic valves had been replaced by Starr–Edwards ball valve prostheses because of rheumatic disease. The surgeon found severe calcification in the mitral valve ring. He left a rim of calcium and placed pledgetted sutures when inserting the valve. Nevertheless at the end of the procedure he thought that the patient had developed a haematoma posteriorly in the area of the atrioventricular groove.

In this case a false aneurysm was found in the fat at the atrioventricular groove. It was 1·5 cm in diameter and caused the external surface of the heart to bulge outward (Fig. 2). Its ostium was 5 mm in diameter and was found on the posterolateral wall of the left ventricle at the rim of the mitral prosthesis. Its lumen was filled with thrombus and it had a wall of dense connective tissue 2 mm wide. No myocardium was found in the wall.

SEPARATION OF AORTIC ROOT FROM BASE OF HEART AT THE AORTIC ANNULUS The root of the aorta had partially separated from the aortic annulus in five patients. Each had had an aortic Starr–Edwards ball valve prosthesis inserted. All died in the operating room of a massive haemorrhage.

This lesion occurs at the posterior aspect of the annulus and presents as a jagged tear 2–4 cm long. Two of the patients had histological changes in the

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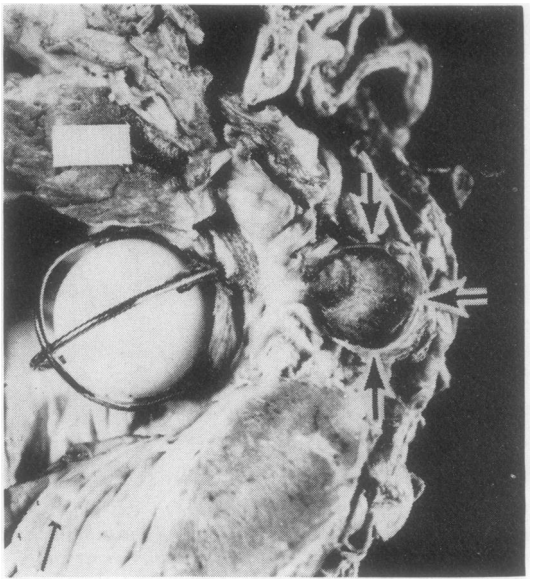

FIG. 2. *Pseudoaneurysm (arrows) bulging into the posterior left antrioventricular groove. Its small ostium in the left ventricle is in intimate association with the rim of the mitral prosthesis. (Scale indicates 1 cm.)*

annular region that may have predisposed them to the injury. The tear in one, with a dehisced Starr–Edwards ball valve prosthesis as a result of infective endocarditis, developed in the friable bed of the old prosthesis. The second patient had had isolated aortic stenosis. Increased amounts of mucopolysaccharides were found in both the aortic annulus and the dilated ascending aorta. This change may have weakened the tissue. The patient had no manifestations of Marfan's syndrome nor, on histological findings, was a diagnosis of either cystic medial necrosis or myxomatous degeneration warranted.

DISCUSSION

Roberts and Morrow (1967) described the necropsy findings in a patient who died of a massive haemorrhage during replacement of a mitral valve. The left atrium and ventricle had separated at the mitral annulus because of the removal of a large calcium deposit that extended from the mitral leaflets through the annulus and into the left ventricular myocardium. MacVaugh, Joyner, and Johnson (1971) thought that this had happened in four of their patients who bled. This mechanism was responsible for a lesion in one of our patients who survived, but the morphological result differed in that a pseudoaneurysm developed. Roberts, Bulkley, and Morrow (1973) illustrated a case in which a haematoma formed in the epicardial fat because a portion of the annulus was excised during mitral valve replacement. The haematoma compressed the coronary artery and eventually ruptured. A ruptured epicardial haematoma also caused the death of a patient described by MacVaugh *et al.* (1971). We did not encounter such a case. A patient in our series and one described by MacVaugh, Joyner, Pierce, and Johnson (1969) developed a pseudoaneurysm in the posterior atrioventricular groove. A pseudoaneurysm, found in the second of two patients described by Spellberg and O'Reilly (1972), may have been of similar type. Most of these mitral annular injuries affected the posterior or posterolateral aspect of the annulus and occurred in patients whose posterior mitral valve annulus was heavily calcified. Surgeons are aware of the risk of this complication if they remove too much tissue from this area.

In our patients, separation of the aortic root tended to occur towards the end of the operative procedure when the heart was lifted forward and up in order to inspect and clear the posterior pericardial cavity. We believe that the vessel was stretched to tearing point by the rotation and leverage of the heart on a fixed fulcrum provided by the prosthetic rim.

These annular injuries are rarely encountered at necropsy but may be slightly more common in clinical practice. All are iatrogenic and so preventable. Although we describe three morphologically distinct types the pathogenesis of each must be similar, a weakening of the annular tissue that allows it to separate or tear. It may be caused by the surgical removal of too much tissue; the application of an excessive physical stress to normal tissue, as we suppose happened in some cases of aortic root separation; or the application of a slight stress to tissue previously altered by underlying disease, for example infective endocarditis, myxomatous degeneration, etc. In our view, the variation in morphological findings reflects a variation in the lesion size, and/or the interval between its occurrence and the patient's death. Thus, an annular tear may cause a catastrophic haemorrhage, an epicardial haematoma that ruptures subsequently or a haematoma that with organization produces a pseudoaneurysm in the atrioventricular groove or presents as a sinus leading through the annular tissue. Each of these lesions occurs in the mitral area, but we observed only one type in the aortic area, probably because it causes a severe haemorrhage. Presumably, in other circumstances, either of the other two lesions found in the mitral area could evolve in the aortic annular region,

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while all three might develop at the tricuspid or pulmonary valve annulus.

At necropsy the finding of a sinus or pseudoaneurysm in the mitral annular region may confuse a pathologist if he has not encountered one previously. Both lesions are the result of a haemorrhage in the area with subsequent organization. Typically, the ostium of these lesions is small and has smooth edges. It is located in the posterior or posterolateral region of the annulus on the atrioventricular aspect and is intimately related to the rim of a mitral prosthesis. A sinus tract, lined by connective tissue and with thrombus in its lumen, extends from the ostium into the epicardial fat at the atrioventricular junction. If its distal end expands to produce a distinct bulge in the fat of the atrioventricular sulcus then the lesion may be called a pseudoaneurysm. Obviously these lesions have no myocardium in their walls. They must be distinguished from several others that may develop in this area following the insertion of a mitral valve prosthesis. Pseudoaneurysms caused by a weakening of the myocardium may also develop in the posterior left ventricular wall some distance from the atrioventricular canal. The first patient described by Spellberg and O'Reilly (1972) fits this category. Infection spreading from a prosthesis into the mitral annulus usually causes the prosthesis to dehisce. However, in rare instances, the infection may cause a sinus with a ragged lining that extends into the epicardial fat. Micro-organisms may be demonstrated in both the lining and any thrombus found in its lumen. True aneurysms of the posterior left ventricular wall may be caused by an iatrogenic occlusion of the circumflex coronary artery, another complication likely when a mitral prosthesis is inserted into a heavily calcified annulus (MacVaugh *et al.*, 1971). These aneurysms are found near the base of the heart, have a wide ostium, and have remnants of myocardium in their walls. True aneurysms may also occur if too much myocardium is removed when papillary muscles are excised during valve removal (Roberts *et al.*, 1973). Another lesion that must be differentiated may occur if there is disproportion between a mitral prosthesis and the left ventricular wall. This causes an erosion of the ventricular wall (Silver, 1968). Both the latter types of lesion are usually situated on the posterior wall but more towards the apex. Because sump wounds are usually made in the apex of the heart in patients having mitral valves replaced, aneurysms associated with surgical wounds are more likely to be found there.

Pseudoaneurysms resulting from annular tears may be associated with a loud apical systolic murmur. This will make the clinician suspect that the mitral prosthetic valve is regurgitant (Spellberg and O'Reilly, 1972). A definite diagnosis can be made by left ventriculography and the aneurysm can be excised successfully (MacVaugh *et al.*, 1969).

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