Editorial

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Valve replacement

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About 1950 it seemed likely that mitral valvotomy would prove a successful operation for mitral stenosis. Of course problems appeared, some valves resisted digital splitting, but a satisfactory mechanical dilator was devised; thrombus in the left atrium displaced at operation resulted in systemic embolism so that preoperative anticoagulant treatment is now widely practised; and it is still important to avoid producing significant mitral incompetence. Though an open operation has its advocates, a closed procedure can give a very satisfactory result at low risk, provided the patients are properly selected and this we should now be able to do. In the same period various ingenious operations for regurgitant or heavily calcified valves proved to be disappointing, and it was 10 years before the introduction of the Starr-Edwards prosthesis (Starr and Edwards, 1961), and improved techniques of extracorporeal circulation established valve replacement as a practical procedure. Shortly afterwards the free aortic homograft (Ross, 1962) offered an attractive alternative to the mechanical valves which now began to appear in so many different forms and designs, and not all were to prove successful. Valve replacement was an exciting development, a considerable advance in cardiac surgery, and offered for the first time a hope of recovery to many patients with hitherto uncorrectable lesions; it still remains the commonest open-heart operation in most centres. From a medical viewpoint it seems to me that despite these large numbers of operations and getting on for 10 years' experience, the position is very different from mitral valvotomy, where the operation has remained substantially the same for many years. There have been many difficulties, some were quite unexpected, and even now we should not underestimate the problems of valve replacement, the many possible complications that may occur at operation or appear in the years after it.

When assessing a surgical procedure one tends to think first of the operative mortality, and the risk of valve replacement does appear to be largely surgical, or technical, i.e. avoiding possible complications such as uncontrollable haemorrhage, coronary artery embolism with air or various particulate substances, damage to the coronary ostia during cannulation causing dissection or damage to the circumflex branch when suturing a mitral prosthesis in position. Traumatic damage to the myocardium may result from prolonged retraction, and there are patients where the cardiac state is entirely satisfactory but there is irrecoverable cerebral damage from one of several possible causes. Of course surgical skill is of paramount importance, and outstanding results such as a hospital mortality of 6 per cent for homograft replacement for aortic valve disease have been reported (Barratt-Boyes *et al.*, 1965), but most patients undergoing single valve replacement will even now face a higher risk than this. The type of case we refer for operation must be relevant: some patients are desperately ill when admitted to hospital and fail to respond to medical treatment. A surgical colleague can usually be persuaded to operate on such a high risk case as an emergency, but the results are not often as good as those reported by Emanuel (1968). Valve replacement has been reported in 'geriatric' patients, but for a number of reasons the mortality rises above the age of 60 years. In any series, therefore, there are patients where the individual risk should be very low, 5 per cent or less, and others where it must be much higher. We all know of patients who die on the waiting list while awaiting investigations or operation, but they do not appear in the published accounts, and for these and other reasons the mortality rate is of limited value in assessing this operation or in comparing the results from different centres.

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Accurate preoperative assessment is of the utmost importance in all forms of cardiac surgery: for example the site of a defect must be correctly identified and a precise enough measure made of the shunt and pulmonary vascular resistance. In patients presenting for valve replacement the questions posed are rather different and are sometimes not as easy to answer. The initial assessment is clinical: patients requiring mitral valve replacement will have disabling symptoms and a dependance on regular diuretic therapy, usually daily; patients requiring aortic valve replacement are often less disabled symptomatically but will present good evidence of severe left ventricular change. Further investigation commonly includes right and left heart catheter studies, and though there is no satisfactory method of measuring regurgitation accurately an estimate made by selective angiography or perhaps a dye dilution technique is certainly helpful; the presence and degree of valve calcification will be noted on screening. This information will usually give a clear indication of the operation required, but there may be difficulty in some cases, for example in a patient with severe aortic valve disease one may recognize that mitral regurgitation is present but be unable to be certain if it is organic, functional, or a combination of these. Sometimes in a patient requiring mitral valve replacement there is aortic regurgitation which seems at most to be moderate, and a decision as to whether this valve too should be replaced has to be left to the surgeon to decide at operation. Then at the tricuspid valve important stenosis can be detected but organic and functional regurgitation cannot always be distinguished; if the pulmonary vascular resistance is high regurgitation is more likely to be functional, but this is not wholly reliable. The cardiologist may think it unsatisfactory not to know whether these patients will need a single or a double valve replacement where the risk is generally higher.

Do all patients require this routine assessment? It may be argued that in a middle-aged man with clear clinical evidence of severe aortic stenosis, appropriate symptoms, a heavily calcified valve, and an electrocardiogram indicating considerable left ventricular hypertrophy there is no necessity for further investigation particularly if this would delay surgical treatment. There has been, and still is, dispute about the incidence of important coronary disease in such patients; aortography to show the valve will usually outline the main coronary arteries. Perhaps this is not enough; there is a case for a more careful scrutiny of these vessels in many patients who are being considered for valve replacement. This is not to deny that valve disease is causing the presenting symptoms and signs but to enable a better assessment of the operative risk, and the enthusiast would add, to enable some form of coronary artery surgery to be performed at the time of valve replacement. Even when the coronary arteries are healthy, one can recall cases where one would like to have known that the left coronary artery was short with the risk that coronary perfusion would supply only the circumflex, or anterior descending branch, but not both. But the information most often absent from our cardiac assessment is some measure of the state of the myocardium and what could be more important? The patient's condition, age, heart size, and electrocardiogram are all very indirect; radiological assessment by cineangiography or measurement of ventricular volume changes may be useful but fibrillation or ectopics provoked by the injection of contrast can make interpretation difficult. Other techniques are being developed but at the moment are not very satisfactory for clinical practice. Lastly there are patients whose dyspnoea is partly due to abnormal pulmonary function, and it may be difficult to estimate this component when the cardiac lesion is causing a raised left atrial pressure.

Assessment should, ideally, identify those patients likely to obtain the greatest benefit from valve replacement and enable them to have their operation without undue delay. Of course we have all seen patients where despite age or severity of failure and valve disease, improvement has been so dramatic after successful operation that it has become almost impossible to refuse other similar patients the same opportunity. There is a dilemma when facilities are inadequate and waiting lists become long, for the average patient needing valve replacement must deteriorate to some degree if there is a long delay before operation.

The operation itself is naturally the domain of the cardiac surgeon with perfusionist, anaesthetist, and others and is outside the scope of this paper, but I believe the cardiologist must see enough of this type of work to be acquainted with the techniques used and also the difficulties in certain types of case. At the very least this knowledge will help him understand some problems in postoperative management when he surely has a place, though it is noticeable how infrequent complications now are if operation has been without incident. Haemopericardium is really quite uncommon, but deserves mention because the diagnosis may not be easy (Nelson,

Jenson, and Smoot, 1969) and exploration on suspicion seems to have no adverse effect on the patient. Arrhythmias continue to be troublesome, correction of potassium levels, or inadequate ventilation will deal with many, but the more intractable ones are often the result of serious myocardial damage produced at operation. A standard 12-lead electrocardiogram taken the day after operation, and in some patients subsequently, may show evidence of otherwise unsuspected localized damage; monitoring leads cannot be expected to do this. Valve detachment may occur in the first day or two after operation and present as hypotension or pulmonary oedema rather than as a murmur. Thrombosis on a valve cage is a later complication and by interfering with ball or disc movement may produce symptoms of a low output; it may be possible to recognize diminished intensity of the prosthetic valve sounds and restricted ball movement can be seen on screening. Antibiotic cover given for these operations has varied a great deal in the drugs used and the period for which they have been given, and perhaps we should try a little harder to get some objective assessment of their effect. There may be a relation between antibiotic cover and another uncommon complication which may appear before the patient leaves hospital, a prosthetic fungal endocarditis; the diagnosis may be difficult to prove but the prognosis is certainly bad.

A satisfactory assessment of the result of operation cannot be made for some months when the patient should be able to report an improved effort tolerance and a need for less or no diuretics. More often perhaps after valve replacement than after other forms of cardiac surgery some patients do very little because they are afraid to, and in this they may be supported by their family; here firm encouragement is necessary. Valve noise is seldom disturbing, though the Starr-Edwards mitral disc prosthesis is often audible several feet away and might seem unsuitable for a thin nervous patient. Careful follow-up examination is of great importance because late complications may arise which need urgent treatment and because we need to know the long-term results of valve replacement.

In patients with mechanical valves, the need for continuing anticoagulant treatment is in itself a problem even when such treatment is uneventful. In this respect homograft valves have a decided advantage, though Carpentier and his co-workers (1969) give anticoagulants to patients with mounted heterografts in the mitral ring who are fibrillating. For geographical reasons, supervision has often to be delegated to a clinic nearer home, some patients prove to be unsatisfactory subjects for this type of treatment, others develop peptic ulcer or require further operation so that treatment has to be stopped, and some clinics are not as good as others. Yet the effort and care devoted to correct assessment and surgery of this order surely demand a comparable attempt at a satisfactory anticoagulant control, for there seems little doubt about its continuing importance. Bloodwell and his associates (1969) found thromboembolism a major complication even three years after operation, and in their series half the patients with emboli died as a result. Even good control does not provide complete protection from apparent embolic episodes, and Sullivan, Harken, and Gorlin (1969) found some evidence of increased protection from dipyridamole, a drug that reduces platelet aggregation and adhesiveness.

Detachment of a prosthetic valve may be evident early after operation but more commonly appears after a few weeks or months; I believe this complication is commoner than one would think from published reports. Acute detachment may cause sudden death or pulmonary oedema, but it is usually less dramatic with a return of dyspnoea and failure. It may be progressive, for there is increased tension on the remaining sutures, but sometimes detachment is slight and remains so.

When an aortic prosthesis is affected, regurgitation becomes audible, there is no difficulty in hearing the murmur but the peripheral signs may be a better guide to severity. Detachment of a mitral prosthesis causes a systolic murmur which may not be truly pansystolic and which may be maximal rather medially near the lower sternum, perhaps because of the angle of the regurgitant jet. The murmur may be attributed to continuing tricuspid regurgitation which indeed is quite likely to be present as well if the detachment is severe, but the early timing of the opening sound of the prosthesis should be helpful. Clinical diagnosis may be less certain in patients where both mitral and aortic valves have been replaced, the systolic murmur of the aortic prosthesis making it difficult to determine if there is a mitral murmur as well. An abnormal rocking movement seen on screening is diagnostic but is exceptional nowadays, even when regurgitation is severe because of the larger number of retaining sutures being inserted. Most patients are not restudied routinely after major cardiac surgery but certainly in any case where there has been no improvement and detachment is a possible reason for this, a fresh assessment by

cardiac catheterization and angiography is necessary. The state of the tissues through which the retaining sutures are placed must be one factor leading to this complication; heavy calcification in the valve ring, recent bacterial endocarditis, non-rheumatic mitral incompetence, and rheumatic mitral valve disease with spontaneously ruptured chordae are thought to be predisposing factors. The results of second operations – either the insertion of further sutures or removal of the prosthesis and its complete replacement – remain unsatisfactory in some cases. There is a continuing leak or a recurrence in a short space of time. If we could recognize those patients where there is a special risk of detachment perhaps our surgical colleagues could devise a fixation procedure which would prevent it. Of course not all detachment is of great haemodynamic importance or progressive, but slight degrees of haemolysis are common with all mechanical valves, and detachment, particularly of an aortic prosthesis, may cause important anaemia. The model 2300 Starr-Edwards prosthesis has been particularly troublesome in this respect, so that in some patients cardiac symptoms have been relieved but have been replaced by symptoms of anaemia.

The diagnosis of important haemolysis is made on finding an anaemia with a reticulocytosis, and there are usually fragmented red cells in the peripheral blood, the platelet count is normal, and the Coombs test negative. Biochemically the serum bilirubin may be raised and there is excess urobilinogen in the urine, the serum haptoglobin is low, and the lactic acid dehydrogenase is raised. The loss of haemosiderin in the urine can lead to secondary iron deficiency aggravating the anaemia, and theoretically a folic acid deficiency could appear.

After replacement there is a continuing risk of bacterial endocarditis, so that antibiotic cover for dental treatment is as necessary as before operation, as is attention to infection elsewhere.

Changes in the valve, homograft or mechanical, may lead to mortality and morbidity in patients years after operation. Some homografts calcify, but we cannot assume that they all will, and there is a continuing search for methods of storage and sterilization that will delay or prevent this change. A very important late complication appearing in patients with a model 1000 Starr-Edwards aortic prosthesis is ball variance where lipoid infiltration has caused swelling of the silicone rubber ball so that damage from the cage causes grooving, cracking, fragmentation, and impaction; the ball may even be dislodged from the cage. These valves have been used for a longer period in Starr's unit than in other centres, and of 40 patients operated on there in a 12-month period from July 1963, variance has been found at reoperation or necropsy in 30 (Hylen *et al.*, 1970). The average time to reoperation was 47 months but the range was wide, from 16 to 67 months. The commonest symptoms, fatigue and dyspnoea, may be less helpful in diagnosis than a recent history of syncope or embolism, and early recognition of the condition is essential for it may be rapidly fatal. The most useful sign is reported to be a decrease in the intensity of the aortic opening sound to less than half the closing sound, as measured on a phonocardiogram or a sound spectrogram. This sign is not always present, false positives may occur, and multiple valve replacement makes evaluation more difficult. The late onset of aortic regurgitation is suspicious, and in routine examination of patients some years after operation evidence of this type of valve failure should be sought. Other types of valves using silicone rubber have been affected, and there are also recent reports of a similar complication affecting mitral prostheses, but changes here may well take longer to appear.

Pregnancy remains a troublesome problem, I suppose that most patients with hearts bad enough to demand valve replacement should be advised against it, but some seek operation in the hope that pregnancy may be possible later. There are single case reports of success and some of mortality during pregnancy, and at the moment it is difficult to estimate the degree of risk.

This then is a medical view of some of the problems of patients facing valve replacement. Because of late mortality, 5-year survival rates of 55 to 70 per cent for aortic valve replacement and less than this for mitral valve replacement have been reported (Bloodwell *et al.*, 1969; Duvoisin *et al.*, 1969). Our attention should now be on these late results rather than the immediate operative mortality, and a careful assessment of the incidence and causes of late complications is required. The defects of the valves used in the past are readily accepted, new modifications of the mechanical ones appear regularly, and the preparation, sterilization, and storage of the free homograft is under constant review; mounted homografts and heterografts (Ionescu *et al.*, 1967) are currently in vogue, as are valves fashioned from the patient's fascia lata (Senning, 1967). With all this endeavour we can hope that the thrombotic complications will diminish and that long-term mechanical

efficiency will improve; but progress is likely to be slow, and there will be no sudden breakthrough.

But it is evident that not all late deaths are explained by some fault at the valve; appreciable numbers of patients are reported to have died of arrhythmia, myocardial factors, and coronary insufficiency, with the implication that the underlying rheumatic disease or the myocardial effects of long-standing haemodynamic upset or related coronary atherosclerosis are responsible. Of course this is sometimes the case, but it demands examination because the operation itself may cause damage to the myocardium and changes in the coronary arteries. Niles and Sandilands (1969) reported myocardial damage in 57 of 62 patients dying up to 76 months after valve replacement; in about half there was thrombosis on the prosthesis and coronary embolism was identified in 7 of them. But in 13 others without thrombosis, there was a distinctive lesion, a widespread ischaemic necrosis, and scarring mainly of the inner layers of the left ventricular wall, and the pathogenesis they regarded as both unsettled and important. Roberts and Morrow (1969) found fibroelastosis of the left ventricle after mitral valve replacement in each of 22 late deaths; turbulent blood flow was suggested as the cause, and in 2 of these cases they believed the degree to have been of functional significance. The same authors found intimal thickening of the aortic root and proximal coronary arteries due to the deposition of fibrous tissue after aortic valve replacement. In one patient who developed angina after operation, intimal thickening was so severe that coronary cannulae could not be inserted into the ostia at necropsy, though there had been no difficulty at operation some months earlier; they speculated about possible causes.

The issue now is that cardiac operations, once the initial mortality begins to fall, are progressively extended to less disabled patients; indeed this is one of the reasons for improvement in operative mortality. To what extent should this be done with valve replacement? It is possible to identify patients who are unlikely to survive more than a few years without operation, but, as Morrow *et al.* (1967) have pointed out, there is as yet no evidence that total life expectancy would be extended if the operation were carried out any earlier than this. With the expected improvement in replacement valves therefore, the selection of patients for operation and the timing of the operation remains a heavy responsibility.

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