Valve replacement techniques have been evolved because of dissatisfaction with the long-term results of previous operations for the correction of aortic stenosis and mitral regurgitation, and the absence of any other procedure for aortic regurgitation. It is now possible to insert prosthetic heart valves or homograft aortic valves with an acceptably low mortality, though little is known about their ultimate fate.

**Types of Prosthetic Valve**

(i) **Aortic.** Artificial cusps of teflon or terylene initially function satisfactorily (Bahnson, Spencer, Busse, and Davies, 1960; Muller, Warren, Dammann, Beckwith, and Wood, 1960; Hufnagel and Conrad, 1961; McGoon, 1961; McGoon and Moffitt, 1963; Kay and Suzuki, 1963), but follow-up has shown a disappointingly high incidence of valve failure; the cusps become thickened and rigid, due to fibrin deposition and the in-growth of connective tissue, and may fracture or prolapse and evert (Björk, Cullhed, and Lodin, 1963; Björk and Hultquist, 1964; Larson and Kirklin, 1964; Judson, Ardaiz, Strach, and Jennings, 1964; McGoon, Ellis, and Kirklin, 1965; Braunwald and Morrow, 1965; Bahnson, Lewis, Criley, and Ross, 1965). Bacterial endocarditis is frequent (McGoon and others, 1965), though thrombo-embolism is rare (Shanklin and Wheat, 1964).

The caged-ball valve (Starr, Edwards, McCord, and Griswold, 1963) is being widely used in America and Europe, and comprises a highly-polished metal cage with seating ring and three struts, silastic ball, and sewing ring of knitted teflon. The Magovern sutureless valve is similar, but is secured by a row of fixation pins (Magovern, Kent, and Cromie, 1963; Magovern and Cromie, 1963; Magovern, Kent, Cromie, Cushing and Scott, 1964); this valve can be inserted rapidly but may become displaced (Cooley, 1964).

(ii) **Mitral.** The mitral Starr valve differs in detail from the aortic prosthesis, and changes have been made in the original design so that there is a more streamlined inflow face, and the internal diameter of the seating ring has been increased relative to the external diameter (Starr and Edwards, 1961; Starr, Edwards, and Griswold, 1962; Starr, McCord, Wood, Herr, and Edwards, 1964b). The Magovern mitral prosthesis has two rows of interdigitating fixation pins. Melrose and others (1964) have introduced a self-retaining hingeless flap-valve made of polypropylene, which occupies less space than the ball-valve, avoids a fixed restraining mechanism, and is claimed to cause less obstruction to blood flow.

**Indications for Valve Replacement**

(i) **Aortic regurgitation.** Severe aortic regurgitation may be well tolerated for many years; operation should only be advised when increasing symptoms or increasing heart size on X-ray indicate worsening left ventricular function, but should not be delayed until there is frank left ventricular failure and the myocardium perhaps irretrievably damaged. Ejection fraction determined angiographically and rate of rise of left ventricular pressure give a measure of myocardial contractility (Miller, Kirklin, and Swan, 1965). Left axis deviation and intraventricular conduction defects usually denote a failing myocardium.

Bicuspidation gives poor results (McGoon and others, 1965), and pericardial grafts rapidly thicken and calcify (Björk and Hultquist, 1964); replacement with a Starr prosthesis is currently the procedure of choice. Ross (1962, 1964), Davies, Lessof, Roberts and Ross (1965), and Barratt-Boyes (1964) have had good results with homograft replacement but this is dependent on a supply of various sizes of homograft valves. Evidence so far suggests that homografts do not stiffen or calcify and do not elicit an immune response; infection and thrombosis are not a problem, and anticoagulants are not required; however, valvular competence cannot be guaranteed. Homograft replacement is unsuitable for patients with disease of the aortic wall leading to progressive dilatation, for whom a prosthesis must be used (Groves, Effler, Hawk, and Gulati, 1964).

(ii) **Aortic stenosis, and combined aortic stenosis and regurgitation.** Open aortic valvotomy is indicated in congenital non-calcific aortic stenosis, and débridement operations still have a place in a few patients in whom it is possible to decalcify the valve adequately, and restore mobility to the cusps. Valve replacement is needed in other patients with calcific aortic stenosis, and in those with an important degree of regurgitation. Operation is advised when there is increasing effort or nocturnal dyspnoea, angina or syncope; these patients have a valve gradient of 60 mm. or more, a calculated valve area of less than 1 sq. cm., and well developed ST and T wave changes in the left ventricular leads.

Associated coronary artery disease may be
operation. Kloster, Bristow, and Griswold (1965) frequently perform coronary arteriography; others feel that this is unnecessary since operation is required when severe stenosis is confirmed, even though coronary disease may coexist.

(iii) Mitral regurgitation. Valve replacement should be advised when there is severe pulmonary venous hypertension, or clinical, haemodynamic, or angiographic evidence of impaired left ventricular function. The left atrial pressure may be only slightly raised, particularly if the atrium is large or compliant (Braunwald and Awe, 1963), unless there is associated stenosis or the left ventricle is failing. Severe pulmonary vascular disease increases the risk of operation; the high mortality in this group may be reduced by improved techniques in post-operative care.

Successful mitral replacement has been reported in corrected transposition (King, Kilman, Petry, and Shumacker, 1964), and in children with congenital or rheumatic mitral regurgitation (Linde, Harper, Chuang, and Mulder, 1965).

(iv) Mitral stenosis. In America, increasing numbers of predominantly stenotic mitral valves are being replaced. In this country, closed transventricular mitral valvotomy is still the procedure of choice for rigid and heavily calcified valves, for restenosis, and for dominant stenosis with mild to moderate regurgitation, since the operative mortality is less, and a high proportion of good results can be obtained even under these circumstances. When the valve cannot be split, or when valvotomy is followed by severe regurgitation, valve replacement is required, and it is likely that as operative mortality decreases more patients will be treated in this way.

(v) Tricuspid valve disease. Valvotomy for tricuspid stenosis often results in severe regurgitation. It is difficult to distinguish organic from functional tricuspid incompetence even when this is severe; attempts to repair the valve at the time of operation on the mitral valve may be successful but often fail. For these reasons, tricuspid replacement will be undertaken more often, with increasing experience of multiple valve replacement.

Ebstein's anomaly has been successfully treated by valve replacement (Barnard and Schrire, 1963; Lillehei and Gannon, 1965).

(vi) Multivalvular disease. Preoperative investigations should establish the nature and severity of each valve lesion, not only the one thought clinically to be the most important; associated valve disease may introduce difficulties in perfusion, increase the risk of operation and prejudice the final result. Successful insertion of two or three ball-valve prostheses has been reported (Starr and others, 1964b; Starr, Edwards, McCord, Wood, Herr, and Griswold, 1964a). Starr recommends aortic in addition to mitral replacement when aortic valve disease of any degree accompanies predominant mitral valve disease (Bristow, Farrell, McCord, and Griswold, 1965); other surgeons do not replace a second valve unless the lesion is a severe one. Aortic replacement may be combined with mitral valvotomy, but many surgeons still prefer double closed valvotomy for rheumatic mitral and aortic stenosis; when the severity of the aortic stenosis or regurgitation is only moderate, it is reasonable to treat the mitral lesion alone (by valvotomy or replacement) and reassess the situation later. Untreated mitral regurgitation should not be left after replacement for dominant aortic valve disease.

Operative Methods and Surgical Management

The technical details of valve replacement operations using the caged-ball prosthesis are described in the articles from Starr's group. The pre- and post-operative care of these patients does not differ in principle from that of others undergoing open-heart surgery (Kloster and others, 1965; Brandenburg, 1965). They are often very sensitive to digitalis after operation. Correction of potassium depletion and of respiratory or metabolic acidosis is particularly important, since these impair myocardial function and increase the tendency to arrhythmias. Multiple ventricular ectopic beats are better treated by intravenous isoprenaline than by procaine amide or lignocaine. Ventricular tachycardia or rapid atrial fibrillation require D.C. countershock.

Results of Operation

Operative mortality depends on the type of patient submitted to operation, and on the surgical team's experience of valve replacement technique. The best results in America are those of Starr (Kloster and others, 1965), Cooley (Nelson and Cooley, 1964), the Mayo Clinic (McGoon and others, 1965), and the Cleveland Clinic (Effler, Groves, and Favorolo, 1964), where mortality rates vary from 4-23%. Few series have been reported from Europe where the mortality rate is probably higher (e.g., Björk, 1964; Björk and Malers, 1964).

Complications. Complications which may occur during or soon after operation have been reported by Hughes (1965) and include cutting out or breakage of fixation sutures, air embolism, "stuck ball", perforation of the left ventricle, and injury to the coronary arteries or coronary sinus.

Blood-stream infections are disastrous; cure is exceptional, and replacement of the infected prosthesis is essential. Meticulous aseptic technique is vital, and antibiotic prophylaxis should include cloxacillin. Late bacterial endocarditis is disturbingly frequent (McGoon and others, 1965; Brandenburg, 1965).

The formation of thrombus on the cage and sewing-ring may result in systemic embolism, interference with free movement of the ball, or occlusion of a coronary ostium. There is still a high incidence of thrombo-embolic complications, despite improvements in valve design. In most centres, anticoagulant treatment is continued indefinitely after both mitral and aortic valve replacement (Kloster and others, 1965), though
difficult to diagnose, and increases the risk of so far there is no proof of its effectiveness.

Regurgitation around the prosthesis occurs when sutures break or cut out, and is more likely to occur in the presence of infection. Aortic leaks produce a typical regurgitant murmur, but are often trivial. Even serious leaks around a mitral prosthesis occur in the absence of a pansystolic murmur, and may be recognized only by the occurrence of left ventricular failure or when the heart size fails to decrease after operation (Morrow, Clark, Harrison and Braunwald, 1964).

Haemolytic anaemia occurred in 5% of Starr's aortic patients (Kloster and others, 1965), and has also been reported after aortic valve replacement by Reed and Dunn (1964), Marsh (1964), and Stevenson and Baker (1964); it apparently occurs only when the valve is incompetent.

Clinical and haemodynamic results. The relief of symptoms following valve replacement is usually impressive (Bristow, McCord, Starr, Ritzmann, and Griswold, 1964; Bristow and others, 1965; Morrow and others, 1964; Judson and others, 1964; Brandenburg, 1965), and results from the complete restoration of valvular competence, with no more than mild residual stenosis. A poor clinical result may be due to regurgitation around the prosthesis, to irreversible damage to the left ventricular myocardium, to uncorrected associated valve lesions, or to severe pulmonary vascular disease. Lillehei, Levy, and Bonnabeau (1964) suggest that post-operative left ventricular function may be better if chordae and papillary muscles are not excised.

There is a mitral diastolic mean gradient of 2-6 mm. at rest in all patients after mitral replacement, and this increases to 6-11 mm. on exercise (Judson and others, 1964; Morrow and others, 1964: Bristow and others, 1965). After aortic replacement, there is a small systolic gradient in some patients (Judson and others, 1964; Bristow and others, 1964) though this occurs less frequently since the design of the prosthesis has been improved (Bristow and others, 1965). Kezdi, Head, and Buck (1964) have shown in the experimental model and in the human patient that the effective orifice area of a Starr prosthesis is only 80-90% of the area of the ring at the base of the valve. There is thus mild obstruction to blood-flow, either between the ring and the ball in the open position, or between the ball and the aortic or ventricular wall (Bristow and others, 1964); an additional factor may be the inertia of the ball, particularly at rapid heart rates (Morrow and others, 1964).

Left atrial pressure usually returns to normal, despite the persistence of small mitral gradients, but may not do so if there is impaired left ventricular function. Cardiac output at rest usually improves and may return to normal, though the response to exercise more often remains sub-normal.

Conclusion

The results of valve replacement operations are now good enough to justify recommending them to suitable patients with valvular heart disease and increasing disability. In certain cases of aortic valve disease, a homograft may have advantages over a prosthesis. Advances will come from improvements in valve design so as to reduce the incidence of thrombo-embolism, and increase the effective orifice area.

REFERENCES


with Total Mitral Valve Replacement with Prosthetic Valves, J. thorac. cardiovasc. Surg., 46, 482.


Prosthetic heart valves.

M. Honey

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