Mitral valve surgery: to repair or replace?

Heart valve prostheses remain imperfect substitutes for the native valve. There are problems of tissue failure in bioprostheses and the drawbacks of long term anticoagulation, predominantly with mechanical prostheses. There is a growing awareness that the native mitral valve annulus and chordae tendineae are essential components of normal ventricular morphology and function, and improved understanding of the pathological anatomy of mitral valve disease has encouraged the development of operations designed to repair the valve’s structural components, singly or in combination. Improvements in non-invasive echocardiography and Doppler ultrasound techniques allow more accurate preoperative assessment of diseased valves while refinements in techniques for myocardial preservation give more intraoperative time for precise evaluation and reconstruction. There is growing evidence that mitral valve repair improves short and long term survival and is associated with fewer thromboembolic complications than valve replacement.

The simplest conservative procedure, closed mitral valvotomy, was reported in 1925 and remains an important operation though it is not generally regarded as a "repair" procedure. The earliest reports of mitral valve repair procedures, suture annuloplasty under direct vision and on cardiopulmonary bypass, were in 1957. Initial enthusiasm for this procedure was dispelled, however, by reports of high operative mortality and loss of clinical improvement in up to 50% of patients within three years.

Valve replacement because of its more predictable results became the recommended procedure for mitral insufficiency. In the early 1970s several groups, most notably Carpentier and colleagues, reported that, compared with replacement, mitral valve repair improved survival and functional state and reduced thromboembolic complications. Recently, more support for repair rather than replacement has been generated by David and colleagues. They reported a postoperative increase in left ventricular end diastolic pressures and decreased ejection fractions when the chordae were disrupted in patients undergoing valve replacement, compared with repair or replacement when these structures were preserved. Others, however, dispute these findings.

Mitral valve reconstruction ranges from its simplest, commissurotomy, through annuloplasty, to complex repair procedures, in which the chordae tendineae are re-fashioned and transplanted. With surgical repair good results depend on the precise evaluation of the underlying morphology of the mitral valve—its leaflets, annulus, commissures, chordae tendineae, and papillary muscles. Carpentier et al produced a classification of leaflet abnormalities and emphasised the need to tailor the repair procedure to the exact pathological process; they attributed previous poor repair results to a failure to do this. Complex repair procedures have not been widely applied because they may be technically more difficult, demand greater subjective assessment, require increased intraoperative time, and have a longer learning curve and less predictable results than replacement (at least in the learning period). Intraoperative assessment of the efficacy of repair procedures is difficult and not entirely satisfactory. It has, until recently, relied on methods such as visual inspection for leakage after filling the ventricle, either during ventricular arrest or with an active beating heart. The increasing availability of transoesophageal or surface echocardiography at the time of surgery should, however, allow improved intraoperative assessment of the repair procedure and should reduce the likelihood of an unsatisfactory result.

Which valves are suitable for reconstruction? There are no absolute rules but, generally, regurgitant valves are more suitable than stenotic valves for reconstructive procedures. One exception is the purely stenotic valve with non-calcified and pliable leaflets, predominantly seen in younger patients in developing countries. In such countries closed valvotomy is the usual treatment because cost precludes open valvotomies, which require extracorporeal circulation. The cost of radiographic facilities and catheters also controls the use of percutaneous balloon dilatation of such valves in developing countries.

Few surgeons would disagree that the end stage rheumatic mitral valve (typified by retracted, fibrotic and/or calcified leaflets, and a fused subvalvar apparatus) requires replacement. In contrast, regurgitant mitral valves characterised by non-calcified floppy leaflets, annular dilatation, and elongated chordae are more amenable to repair—as are those with chordal rupture, which is frequently associated with floppy valves but may be idiopathic. Demographic changes and the decline in the incidence of rheumatic heart disease have increased the numbers of patients with floppy mitral valves that may be suitable for repair procedures. Indeed, pure mitral valve incompetence is now most commonly due to the floppy mitral valve syndrome (mitral valve prolapse), but such incompetence can also be congenital, idiopathic, ischaemic, infective, or cardiomyopathic.

Duran suggested that up to 40% of all mitral valves are suitable for reconstruction and Carpentier estimated that 5% of aortic valves, 50% of rheumatic mitral valves, and 90% of floppy mitral valves are suitable for repair. Floppy mitral valve disease was the reason for 40% of the mitral valve surgery performed in 1141 patients at the Cleveland Clinic between 1985 and 1988 and was usually suitable for operative correction. The procedures needed for surgical repair include ring annuloplasty, chordal shortening, resection and transposition of a normal leaflet to fill defects created by resection of flail leaflets, and occasionally transposition of the papillary muscle head.

Several groups reported better short and long term survival and functional state accompanied by a reduction in thromboembolic and anticoagulant-related complications, endocarditis, and the need for reoperation after mitral valve repair compared with replacement. Unfortunately, substantial differences in preoperative characteristics in the repair and replacement groups, which could themselves influence outcome, make it difficult to draw conclusions.
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from these studies. The possibility that improved survival in patients undergoing repair procedures may simply reflect selection of a population with less advanced disease is supported in a recent paper by Galloway and colleagues. This group compared operative and late results in patients with porcine valve replacements (975), mechanical valve replacements (169), and Carpentier type mitral valve reconstructions (280). Although operative and five year mortality were reduced in the repair group, multivariate analysis showed this to be an effect of younger age and better preoperative myocardial function rather than type of operation.

In view of the patient numbers and the expense and length of follow up that would be required, comparison of mitral valve reconstruction and replacement in a prospectively randomised trial is unlikely; indeed it is questionable whether such a trial would be ethical or practical. It is as inappropriate to attempt to repair an end stage, calcified, retracted mitral valve with a densely fused subvalvular apparatus as to replace a stenotic valve with fused, non-calcified, pliable leaflets with a prosthetic valve. The increasing prevalence of degenerative valve disease and growing dissatisfaction with currently available prostheses is likely to result in an increasing number of repair procedures despite their being more technically demanding and requiring greater expertise. It is likely, but as yet unproven, that reconstructive surgery of the mitral valve in appropriately selected patients will give better long term results and fewer complications than prosthesis insertion.

Improvements in non-invasive preoperative assessment of diseased valves allow a high degree of accuracy in predicting those in whom reconstructive procedures are likely to be successful. For rheumatic mitral disease repair is virtually always feasible (and highly desirable) for pure stenosis without significant calcification; and may be achieved by closed valvotomy, particularly if sinus rhythm is still present. Where regurgitation is a consequence of annular dilatation or the floppy valve syndrome, repair is also likely to be successful (as it is in rheumatic regurgitation if seen at an early stage). In contrast, the thickened, retracted, immobile, and calcified leaflets commonly seen in end stage rheumatic mitral valve disease in most Western countries are rarely suitable for repair.

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