**PROGRESS IN CARDIOVASCULAR SURGERY**

Prosthetic Aortic Valve: Current Status*

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Dramatic advances have taken place in the surgical treatment of aortic valve disease since 1951 when Hufnagel1 introduced the plastic ball valve for preventing backflow of blood from the descending thoracic aorta. Yet the basic principles of his valve prosthesis1 were those of the currently popular Starr-Edward valve that is now being implanted in the subcoronary portion of the excised anatomic valve. Much of the success of present techniques of implantation is derived from the experience gained in the early work of Bailey4,4 and Muller1 who attempted blind (closed) valvotomy for aortic stenosis, and in the later work of Hufnagel,4 Bahnson;7 Kay,9 Lillehei,9 and others who employed open-heart methods for operation on the diseased aortic valve. While encouraging results and an acceptable incidence of postoperative survival were reported by these authors, it was soon recognized that most lesions in the diseased valves consisted of extensive fibrosis and/or calcium deposits which all too often prevented commissurotomy from accomplishing more than the production of a “crack in the concrete.” Only rarely did the valvular structure lend itself to effective mobilization by simple incision, dilatation or suturing.

Mulder* reported success with subendothelial debridement of calcium deposits and mobilization of valvular leaflets in a limited number of patients, but follow-up studies suggest that residual hemodynamic abnormality is common and the recurrence of valvular disease is rapid.

Aortic insufficiency was also improved in some patients by plicating redundant and prolapsed valvular leaflets,10 patching defects of bacterial erosion,11 and obliterating the noncoronary sinus of Valsalva to produce a bicuspid valve.12 Rarely, however, have the long-term results of these procedures been satisfactory and most of them have been abandoned.

A number of factors complicate the surgical approach to the diseased aortic valve: (1) associated myocardial disease in the form of secondary hypertrophy and dilatation which prevents the myocardium from tolerating operative manipulation and ischemia; (2) the frequent presence of associated coronary artery disease, whose symptoms and manifestations may be indistinguishable from those of aortic disease; (3) the necessity of interrupting coronary flow during operative exposure of the valve; (4) the introduction of air or of calcium fragments from the diseased valve into the operative site with the hazard of subsequent embolization to the coronary or cerebral arteries; and (5) the multiple uncertainties associated with the currently available prosthetic devices. Despite their theoretical and practical flaws, however, it is apparent that these prostheses will ultimately provide the solution to the problems of treating most aortic valve disease.

For the past ten years, extensive effort and investment of money have been devoted to the development and perfection of prosthetic devices to replace the aortic valve. A few have withstood experimental testing and have undergone clinical trial although none has met all of the criteria for a completely satisfactory prosthesis. It is generally agreed that a proper replacement valve:

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(1) should employ a mechanism that is durable and reliable for an indefinite period of time;
(2) should not predispose to the redeposition of fibrin and calcium;
(3) should not lead to formation of clots;
(4) should be totally competent, but should not restrict forward flow;
(5) should be attached to the heart in such a way that it is incorporated into the tissue;
(6) should not interfere with flow in the coronary arteries;
(7) should not impair myocardial contraction or the function of other valves;
(8) should not destroy blood cells by its mechanical action.

Types of Valves

Many ingenious prosthetic devices have been developed experimentally (Fig. 1); many of them were fashioned for special circumstances and were applicable only in selected patients. Replacement of single cusps was attempted on a fairly extensive scale by Hufnagel, Bahnson, Muller, Morrow, and Kay. Prosthetic cusps were fashioned from woven Dacron and Teflon cloth (some impregnated with silastic or polyurethane) and modified variously to improve the strength of fixation to the aortic wall and the ingrowth of tissue. These same surgeons later accomplished replacement of the entire valve with three separate synthetic cusps sewn together. Although theoretically this type of replacement provided a mechanism that closely simulated the normal valvular action with little or no resistance, a high incidence of late complications has been reported: attachments have come loose with partial or total avulsion of the prosthesis; cloth leaflets have become stiffened with calcium deposits causing restenosis or regurgitation from actual cracking of the prosthetic cusp. Kay and Hufnagel report, however, that their silastic-impregnated cusps have maintained flexibility, and these two surgeons continue to implant this type of prosthesis.

This author has developed a single unit tricuspid valve that has a firm structural...
base for the cusps which are precision-molded for equalized strength and action. An early model implanted in a dog in 1957 is still flexible and competent by angiocardiography, as is the valve implanted in a patient in 1960. More recent models have been “stress tested” under high pressure gradients in a high frequency pulse duplicator with pulse equivalents of 19 years before showing evidence of damage. This experience suggests that a flexible prosthesis might yet prove to be the mechanism of choice, but problems of fixation and clotting are not completely solved.

It is of interest that although the over-all experience with many of these early prosthetic valves was poor, many surgeons report that their first or second patient is still living with a functioning prosthesis: Lillehei’s patient with a spring flap valve inserted in October 19, 1958, is still alive and active; Hufnagel, Magovern, Harkin, Muller, Kay, McGoon, Bahnsen, and this author each have patients living now in whom different total valve replacements were performed between March and October of 1960.

Caged ball valves were introduced by Harken in 1960, and improved by Starr and Edwards with minor variations adopted by Cartwright and others. The mechanism is simple with an effective rugged action that is not dependent on indefinite flexibility. It does, however, require a limited orifice on which the ball is seated in the closed position. The presence of the ball in the path of ejection during outflow results in a 10 to 30 millimeter systolic gradient. Formation of thrombi on the metal cage with propagation down the aorta has been seen occasionally. Despite these disadvantages, however, the majority of surgeons have abandoned other devices in favor of the ball valve because of its reliability.

Magovern has developed an ingenious modification of the caged ball valve resulting in a mechanism whose implantation without sutures requires only a few seconds. His early experience has been favorable, although the ultimate success of this type of fixation remains to be determined.

Problems of Prosthetic Implantation

A number of formidable hazards accompany the replacement of an aortic valve, and the good results now being reported necessarily reflect our ability to surmount most of these difficulties.

Selection of patients. It would appear from the current experience reported by Efler and McGoon that replacement of an aortic valve can be successful even in patients with functional class 4 aortic valve disease. This would suggest that there are no reasonable criteria for the exclusion of patients on this basis. Undoubtedly, a small number of patients with significant irreversible myocardial damage will not survive the procedure, but their exclusion is extremely difficult except in the absence of significant mechanical valvar dysfunction. Similarly, patients with concomitant coronary artery disease represent a far higher surgical risk than do those with aortic valve disease alone, but there is evidence to suggest that such patients should not necessarily be excluded from corrective valvular surgery.

Since the symptoms of coronary insufficiency are frequently manifest in both aortic valve disease and coronary occlusion, one should attempt to establish that the cause of the patient’s disability is primarily valvular, although this is sometimes difficult in borderline cases. Coronary angiocardiography is helpful in demonstrating areas of partial or complete occlusion but it does not necessarily define the role of occlusion in the handicap of any given patient. For that reason, patients are now being accepted for valvular replacement in the presence of known coronary artery disease when significant stenosis or regurgitation is demonstrated. Experience would tend to support the reasoning that a patient who continues to live with coronary artery disease in the presence of an overloaded myocardium will derive so much immedi-
ate benefit from an effective valvular mechanism that he will survive the procedure. His impaired coronary circulation, relieved of its extra demands, will be more nearly adequate.

**Causes of complications.** (A) Ventricular distention. Overloading of the left ventricle by retrograde flow from the extracorporeal perfusion can occur during even very brief periods of ineffective myocardial contraction. Cardiac arrest or ventricular fibrillation may be induced by cross-clamping of the ascending aorta, by cooling, or by surgical manipulation. The previously arrested heart may not be able to eject blood against the perfusion pressure after completion of valvular replacement, or some incompetence of the prosthesis may lead to distention at this stage. Avoidance of this potentially fatal mishap is accomplished by meticulous decompression of the left ventricle, either transatrially or through the apex, until normal function is restored.

(B) Myocardial ischemia. The hypertrophied and/or distended myocardium will not tolerate prolonged ischemia without serious impairment of ventricular function. The normal coronary perfusion that is necessarily interrupted during operation on the aortic valve must be replaced by some form of intermittent or constant artificial perfusion. Hufnagel has proposed, instead, the use of profound cardiac hypothermia (by immersion in saline slush), but most surgeons favor intermittent or constant coronary perfusion with or without hypothermia. McGoon favors constant normothermic perfusion with separate pumps to control the flow to each coronary artery. A wide assortment of indwelling coronary artery cannulae have been developed for this purpose, self-retaining and watertight features are desirable in maintaining constant perfusion.

(C) Damage to the coronary arteries. Injury or occlusion of a coronary artery may occur during dissection, cannulation, or closure of aortotomy. This hazard can usually be avoided by careful surgical techniques and judicious use of the coronary cannulae. Damaged arteries, if recognized, can be repaired.

(D) Air emboli. The introduction of even very small amounts of air into the coronary circuit has been shown to cause serious impairment of ventricular function. This complication may arise during replacement of aortic valves, from faulty techniques of coronary perfusion or, more likely, from the incomplete evacuation of air from the left heart at the end of the procedure. A careful filling of the operative site and meticulous aspiration of air from the ascending aorta and the apex of the upturned ventricle before restoration of ventricular beat are essential to prevent air emboli.

(E) Calcium emboli. The sand-like quality of the calcium deposits frequently encountered in diseased aortic valves makes it necessary to exercise great care in debridement during valvular excision. A sponge may be introduced into the ventricle to catch these fragments, and repeated irrigation will help to wash them away.

F. Total heart block. Temporary or permanent injury to the atrioventricular conduction bundle may be caused by dissection or suturing in the region of the base of the noncoronary cusp. This troublesome complication may usually be avoided by cautious technique, but when it does occur, a pacemaker should be employed during the postoperative period to prevent low cardiac output or cardiac arrest.

(G) Malfitting prosthesis. A prosthetic device which is so constructed that it interferes with flow to the coronary ostia may embarrass the coronary circulation. A valve that does not fit snugly in the aortic annulus may undergo sufficient motion during the cardiac cycle to pull out its suture attachments. Improperly balanced or sized leaflet valves may result in regurgitation. Ball valves have been known to get stuck or to become dislodged from their cages. These problems have largely been avoided by improved design and manufacture of
the prostheses. A wide assortment of valve sizes is now available for proper fitting.

(H) Failure of fixation. This author and others have encountered failures when surgical knots in synthetic suture material have come untied or have cut through the aortic wall. This experience has revealed the necessity of having multiple knots properly set for each suture and of placing a sufficient number of sutures to distribute the stress.

(I) Intractable ventricular fibrillation. While most hearts that have been satisfactorily perfused during prosthesis and that have not been subjected to chamber distention will defibrillate without difficulty, there have been hypertrophies and dilated hearts that resisted multiple attempts at defibrillation with alternating current devices. A direct current defibrillator is essential for this group of patients.\(^{(24)}\)

(J) Thrombosis and embolization. Embolization from the prosthesis has been commonly experienced with laboratory animals and with a few patients. On this basis, it is generally accepted that implantation of a prosthetic valve carries a significant risk of producing emboli, and indefinite prophylactic anticoagulative therapy is thereby recommended by some authors.\(^{29,30,43}\)

Recent experience suggests, however, that, in the aortic area at least, the velocity of flow is sufficient to prevent the formation of most clots and this hazard seems to be less of a problem now than it was in our early experiences.

CONCLUSION

The problems associated with the inherent character of aortic valve disease and the surgical complexities involved in aortic valvular prosthesis have by no means been totally solved, but it is evident from the experiences reported by many leading cardiac surgeons that satisfactory operative results are now being obtained with an acceptably low mortality rate of 10 per cent or less. The long-term results with the recently-developed caged ball valve remain to be evaluated and late problems are bound to develop. At the present time, the results appear to be good enough to justify subjecting a large number of patients with symptomatic aortic valve disease to valvular prosthesis.

References will appear in the reprints.

For reprints, please write Dr. Roe, Department of Surgery, University of California Medical Center, San Francisco.

Readers are invited to submit articles for Progress in Cardiovascular Surgery. Please submit material to David P. Boyd, M.D., 605 Commonwealth Avenue, Boston, Massachusetts.

HISTOPLASMOSIS IN MALAYA

A survey of 227 patients from five to 60 years of age revealed the presence of positive histoplasmin skin tests in 10.5 per cent and positive complement fixation tests in 19.6 per cent. Sputum from 13 of the 37 patients who had positive complement fixation test were cultured for H. capsulatum, but with negative results. Exposure to infection by the fungus is equal distributed among the different race and age groups.


WIDENING OF MEMBRANOUS WALL AND FLATTENING OF TRACHEA

In a postmortem series of 61 older men, the width/depth ratio of the airways was measured as an index of flatness. Rather unexpectedly, it was found that the trachea and major bronchi in cross-section not infrequently varied from the classic U or C shape and were flat and trough-shaped with widening of the posterior wall. Definite flattening of the airways was found in 21 per cent of the cases and in 5 per cent this was gross with a maximum width/depth ratio exceeding 3.0. Flattened airways with wide membranous walls are less capable of resisting external pressure than the classically shaped airways. Consequently, they are potential cause of tracheobronchial collapse.