International Journal of Healthcare Sciences ISSN 2348-5728 (Online)
Vol. 6, Issue 1, pp: (310-335), Month: April - September 2018, Available at: www.researchpublish.com

The Use Valve Prosthesis in case of Mitral Valve Stenosis and Regurgitation

[1]Abdulelah Naif Al-Enzi, [2]Waad Hassan Albadrani, [3]Rakan Saleh Alghumiz, [4]Abrar Ahmad Marei Asiri, [5]Abdulaziz Khaled Arar AlDosari, [6]Abdulaziz Ali Saleh AlMufadhi, [7]Yazeed Mohammed AlSulaimani

Abstract: **Considering that the initial implantation of an artificially developed valve in the human circulatory system by Hufnage in 1953, the range of prosthetic gadgets used for used by the cardiovascular plastic surgeon has actually broadened substantially. Technologic improvement has actually resulted not only in the development of brand-new prostheses, however similarly in serial adjustments in style and building of those presently in use. Bioprosthetic valves (see the image listed below) utilized in heart valve replacement normally provide functional homes (eg, hemodynamics, resistance to apoplexy) that are more similar to those of native valves. Implantation of prosthetic heart valves to deal with hemodynamically substantial aortic or mitral valve disease has actually become significantly common. This assessment will examine the development and differential advantages of replacement heart valves currently readily offered, in addition to use some perspective on those devices no longer is used.**

Keywords: **human circulatory system, prosthetic gadgets, plastic surgeon, Technologic improvement.**

1. INTRODUCTION

Since the very first implantation of a synthetically created valve in the human circulatory system by Hufnage[(1)] in 1953, the variety of prosthetic gadgets offered for used by the cardiovascular cosmetic surgeon has actually broadened considerably. Technologic enhancement has resulted not only in the advancement of new prostheses, however likewise in serial modifications in style and building of those currently in use. This evaluation will analyze the development and differential benefits of replacement heart valves presently readily available, in addition to offer some point of view on those devices no longer typically utilized.

2. HISTORY AND CLASSIFICATION

There are basically 4 practical categories of prosthetic heart valves: (1) caged ball, (2) caged disc, (3) tilting disc, and (4) tissue valve (Table 1).The initial Hufnagel prosthes is was of the caged ball variety and was implanted in the descending aorta in patients with serious aortic deficiency.[(2)] Not only was the valve unable to prevent regurgitant circulation from the aortic arch vessels, it was more compromised by a considerable occurrence of thromboembolic events. Loud prosthetic opening and closing sounds produced by a polypropylene ball in a methacrylate housing were adequately troubling to make the valve socially unacceptable.[(3)]

Harken[(4,5)]carried out the first subcoronary implantation of a caged ball valve in a group of patients with aortic regurgitation in 1960, however had the ability to accomplish survival for not than 3 months in 9 of 11 patients in his series. The very first insertion of a caged ball valve in the mitral position was performed by Starr, likewise in 1960.[(6)] The caged disc valve was introduced in between 1964 and 1965 by a number of detectives, amongst them Hufnagel,[(7)] J.H. Kay,[(8)] E.B. Kay,[(9)] and Cross,[(11)]each with little changes in design, but all trying to provide the benefits of a lower profile, enhanced hemodynamics, and lighter weight than the caged ball valve. Beall even more modified the style in 1967 to diminish the valve's thrombotic capacity. The tilting disc prosthesis was presented in 1967 by Wada,[(12)] Bjork,[(13)] and Lillehei,[(14)] supplying a higher approximation of laminar flow than did the cagedvalves.

The tissue valve made its first appearance in 1962 through a fresh aortic valve homograft usedsuccessfully for aortic valve replacement by Ross,[15] Duran,[16] and Barratt-Boyes[17] after previous trials in the coming down aorta by MurrayI[18] and Wilson.[19] Preliminary favorable results were tempered by reports of early degeneration,[20-23] rendering the bio prosthesis a less attractive option at the time. The clinical introduction of the aortic heterograft in 1967,[24-25] the subsequent development of a glutaraldehyde tanning method that substantially increased the valve's sturdiness promoted restored interest in thesevalves.[26]Glutaraldehyde preservation strategies were subsequently applied to valves built of fascia lata[27] and bovine pericardium[28] with satisfying results.First introduced by Gott in 1968, the bileaflet tilting disc valve was designed to supply totally central circulation. The most current bileaflet prosthesis was developed by St. Jude Medical, Inc., and has been studied in clinical trials given that 1977, although the valve remains restricted to investigational usage in the United States.[29].

3. CHARACTERISTICS OF A GOOD PROSTHETIC VALVE

In 1962, Harken (5) Spublishedwhat he called the "ten rules of satisfactory aortic valves." More just recently, Robert[30] has actually rearranged Harken's requirements into 6 characteristics that typify a great replacement valve in any position: (1) good hemodynamics, (2) minimal thrombogenesis, (3) durability, (4) very little hemolysis, (5) ease of implantation, and (6) patient acceptability. To these may likewise be included the following: (7) a low occurrence and lessened seriousness of late prosthetic endocarditis, and (8) anatomic suitability of the valve to its implanted place (Table 2). Roberts has actually suggested that there is currently little distinction in between any of the 4 kinds of prostheses in regards to the ease of surgical implantation. In addition, none of the valves now in use are adequately noisy regarding be unduly disturbing to the patient. The staying 6 characteristics work as a structure through which the different kinds of replacement valves might be examined.

4. THE CAGED BALL VALVE

EVOLUTION:

The Starr-Edwards valve prosthesis is most likely the most well-known and thoroughly studied caged ball valve. The original designs 6000 (mitral) and 1000 (aortic) were constructed of a heat cured silicone rubber ball housed in a cage made from Stellite 21 (an alloy of cobalt, chromium, molybdenum, and nickel) and anchored by a sewing ring of Teflon (polytetrathtorethylene) cloth[6] . While the valve provided adequate hemodynamics in both the aortic and mitral positions, hemolysis was a significant issue in the former. Anticoagulation was compulsory.

The model 1260 prosthesis was established in 1965, and stays in use today in numerous centers . In this valve, a Teflon fabric covering was extended over the entire seat as a means of promoting endothelialization about the orifice. This modification led to a significant decrease in the rate of thromboembolism from 34% in a 10-years follow-up of the design 1000 to 19% in a 7-years follow-up of the model 1260.[31] Ball variance in the models 1000 and 6000 led to both obstructive phenomena and fatal poppet embolization. This issue was countered in designs 1260 and 6120 by a very little decrease in temperature of the curing procedure along with the addition of an extra 10% silicone dioxide or fluoro silicone filler.[51,52] Additional alterations included an increase in the orifice to ball diameter ratio (88%), modification of the orifice configuration to lower opening pressure, and a decline in the total weight of the prosthesis by reduction in its metallic composition.[51 53].

Series 2300 aortic and 6300 mitral prostheses removed ball variance by replacing the silicone rubber poppet with a hollow Stellite 21 sphere[54] . Based on earlier observations by Braun-wald[55] and others that neointimal expansion along a fabric covered valve strut significantly minimized the danger of thrombus development,.

These valves utilized totally cloth covered struts. They were consequently customized to include a "composite seat" that included a ring of little metal studs predicting through the Teflon fabric of the valve orifice that both increased the orifice to ball size ratio and reduced wear on the orifice fabric. In clinical trials, thromboembolic phenomena were initially noted to be considerably reduced in both the aortic and mitral positions; however, constant anticoagulation remained mandatory.[56-58].

Reports of fabric wear and pronounced hemolysis with the cloth covered valve, especially in the aortic position,[47,59] motivated development of the series 2400 and 6400 composite strut "track valve" in 1972 . The valve was practically identical in construction to the composite seat cloth-covered series, but integrated strips of Stellite 21 on the inner surface of each strut, offering a metal track that safeguarded the Teflon covering from trauma resulting from ball impact. Early

studies on this design revealed substantial thrombogenesis in the absence of anticoagulation.[60] Later reports suggested that there was no higher thromboembolic potential related to this valve than with other valve prostheses if the patients were properly anticoagulated.[61,62] Hemolysis has actually stayed significant, nevertheless, leading to the desertion of fabric covered struts entirely in favor of the earlier non-cloth covered model 1260 aortic and design 6 120 mitral valves.

The Smeloff-Cutter valve was created mainly for the purpose of reducing outflow tract obstruction in the mitral position[63] .Its open cage design was engineered to decrease the frequency of thromboembolic occasions, while a smaller sized ball size was employed to minimize the gradient across the prosthesis.[64] Initially constructed in the early sixties of a silicone rubber ball housed in a titanium double cage with Teflon sewing ring, it was reissued in 1966 following adjustment of the silicone treating strategy (just like changes made in the 1260 and 6120 Starr models) as a result of numerous reports of ball variance[52,65]. Subsequent follow-up has actually revealed virtual elimination of ball difference as a complicating factor.[66].

The Braunwald-Cutter valve used functions much like those of the Starr-Edwards 2300 and 6300 series with some important differences [Fig. 6]. Previous evaluation of fabric covered caged ball valves in animals had actually revealed a preference for accumulation of thick fibrous tissue at particular websites, particularly the crossway of the struts. The prosthesis was subsequently produced with an open ended cage as well as Dacron fabric covered struts and a "mild treatment" silicone ball to prevent fabric wear.[67] Preliminary studies in calves and canines demonstrated the presence of almost complete neointimal pro- liferation along the struts 1mo following implantation. However, assessment of human pathologic specimens 1 year after valve replacement exposed the regular occurrence of insufficient endothelialization and ensuing fabric wear, especially rupture of the joint line at the pointers of the struts.[65-69] This change was probably responsible for persistent ball difference and embolization of the ball through the open cage in the aortic position. These issues eventually resulted in the withdrawal of the prosthesis from the marketplace.[70-71]

The Magovern-Cromie aortic prosthesis was first employed in human beings in March of 1962 with 4 subsequent alterations[72-76] . The fundamental style shows functions of both the Starr- Edwards 1260 and the Smeloff-Cutter valves employing a silicone rubber ball housed in an open ended non-cloth covered titanium cage. The innovative function of this valve was the presence of multiple pins about the external size of the seat that enabled rapid, sutureless insertion. These were initially organized horizontally in the model valve and were consequently mounted in 2 vertical rows in its 4 successors. In the most recent past, the prosthesis has actually been booked for usage in high threat patients for whom extended coronary perfusion could not be securely.

***Ensured*:** Reported cross clamp times of roughly 20 and 30 min for aortic insufficiency and aortic stenosis, respectively, rendered this valve of considerable value in this small subgroup.[75.76]. The introduction of cold cardioplegia as a means of intraoperative myocardial security has even more limited the number of patients who may require this prosthesis. This gadget has been associated with high postoperative event of atrioventricular dissociation, although this finding might show the patient population itself, which shows a high incidence of serious calcific aortic stenosis or pre-existing atrioventricular conduction disturbances, instead of an intrinsic problem of the prosthesis."

Results:

***Aortic placement*:** healthcare facility mortilaty for aortic valve replacement with all the presently readily available prostheses is reported to be between 5% and 8% in multiple series.[78-81]More current data using cold cardioplegia for myocardial preservation exposes healthcare facility death lower than 1% for isolated aortic valve replacement.[82] Death is not a function of the type of valve used, but rather of well-known perioperative issues such as perioperative myocardial infarction, left ventricular failure, and ventricular dysrhythmia. Late death ranges from 15% to 20% at 5 year [81] as well as appears independent of the type of prosthesis employed. Hemodynamics are typically similar in all designs with typical gradients varying from 10 to 19 mm Hg in multiple trials.[54,83]Thromboembolic conditions in anticoagulated patients likewise tend to be fairly comparable with 1.5% to 2% per patient-year reported for the Starr 2400 modelg4 and 5% over 4 year noted for the Smeloff-Cutter valve.[85] Bonchek and Starr observed no thromboembolic occasions in 116 anticoagulated patients followed for 3 year with cloth covered (2310-2320) valves. On the other hand, they found an 8.8% occurrence over 2 year in patients who were not anticoagulated, and 29.4% occurrence in 17 patients followed for 2 year who had actually ceased previous anticoagulation.[58] A dramatic increase in prosthetic thromboembolic occasions following discontinuation of anticoagulation compared to nonanticoagulated patients has actually likewise been observed by Isom et al. [31]

Hemolysis is noted with all kinds of mechanical prostheses, nevertheless frank hemolytic anemia has primarily been connected with cloth-covered aortic devices, and in the existence of perivalvular leakages. [59.86-90] Mitral replacement. Operative death for elective mitral valve replacement in New york city Heart Association (NYHA) class III patients is currently in between 1% and 3%,[81,91,92] although the figures end up being rather higher if NYHA class IV patients are included. Late mortality is essentially similar to that reported for aortic valve replacement[48,58,93] with only 10% to 15% of these related to problems of the prosthesis itself.[92] Mean resting valve gradients have actually varied from 5 to 9 mm Hg, although they have actually been observed to increase as much as threefold during workout. [54.84.94-96] Thromboembolic events are reported more frequently in the mitral position than in the aortic, although hemolysis is less noticable. The Starr-Edwards model 6400 composite strut valve was related to a 3% per patient-year rate of thromboembolism97 in constrast to 6.5% per patient-year observed with the Smeloff-Cutter prosthesis.[98].

Complications.

***Circulation blockage*:** Circulation blockage is an intrinsic feature of the caged ball valve by virtue of its very design.[99] Three websites of obstructed flow are immediately evident: (1) the valve orifice itself, (2) the outflow orifice specified by the oblique range between the respective areas of the valve seat and the ball outdoors position, and (3) the centrally obstructed channel defined by the range between the circumference of the ball and the surrounding tissues. Centrally blocked circulation causes a considerable amount of turbulence, especially in the aortic position where flow speed is at least three times that across the mitral valve. Roberts[47] has actually shown considerable intimal proliferation in the aortic root of patients with caged ball aortic prostheses that has been presumed to be secondary to turbulent flow. Of these, more than 50% manifested evidence of fibrous tissue deposition in one or both of the coronary ostia. Considerable obstruction of the coronary ostia due to fibrous tissue was seen in 14% of the patients compared with 2% of patients who had been noted to have narrowing of the ostia at the time of surgery.

Likewise, Roberts and Morrow have documented left ventricular endocardial fibroelastosis as a late issue of mitral replacement with The caged ball valve.[100] As in the aortic root, the existence of fibrous expansion in the left ventricle was presumed to be an anatomic reflection of turbulent blood circulation. Not just is the endocardium subject to laterally displaced blood flow from the left atrium during ventricular diastole, it needs to likewise sustain a series of shock waves propagated throughout the residual end-systolic ventricular blood swimming pool by the forward motion of the ball throughout the fast filling phase . Substantial speculation still exists worrying the etiology of both primary and secondary endocardial fibroelastosis.[102,102]Among the more prominent alternative descriptions are endocardial tension irregularities, viral infection, autoimmune phenomena, and hypoxia. Short-term subendocardial ischemia secondary to lacking intraoperative myocardial defense has just recently been invoked as the reason for left ventricular fibroelastosis following valve replacement instead of the system proposed by Roberts. Regardless of, observations recently reported by Steele et al [103]. Pathologic specimens from patients with tilting-disc mitral valve prostheses also suggest that unstable blood flow is the prime cause of endocardial fibroelastosis in the adult. Endocardial fibroelastosis ends up being clinially significant when it is substantial, where instance it can be responsible for reduced ventricular compliance and congestive failure.

***Thrombogenicity*:** While necropsy research studies have actually shown the existence of prosthetic thrombi in the large majority of patients with caged ball valves, just a minority of these had displayed clinical proof of embolic occasions antemortem.[47] Area of the thrombus is variable and remains in part reliant upon the type of valve employed. There is predilection for thrombus development at the junction of the struts with the seat in non-cloth covered valves, while it is more commonly found at the apex of the cage in non-close-clearance fabric covered models. Thrombus formation in a little group of close-clearance cloth covered aortic prostheses appeared mostly at the midportion of the struts and was reported to be responsible for minimized forward excursion of the ball with consequent mortality.

A current reevaluation of actuarial thromboembolic rates by Macmanus et al [104] reveals that thromboemboli were substantially more typical from aortic and mitral silicone ball valves throughout the first decade of cardiac valve replacement than they have been for the second utilizing identical valve models. This difference probably shows decreased ball variance as a result of more recent alterations in silicone curing methods. Present thromboembolic rates in the anticoagulated patient with the Starr- Edwards silicone ball valve appear comparable to those observed for patients with both tilting disc and tissue valve prostheses.

Some proof has existed recommending that making use of acetylsalicylic acid in combination with anticoagulant therapy is more reliable than anticoagulants alone in the avoidance of thromboembolic episodes.[105] .Notwithstanding, making use of antiplatelet agents in the absence of anticoagulation has been revealed unequivocably to be not just inferior to anticoagulant therapy alone, but little better than no medication whatsoever for thromboembolism prophylaxis in patients with caged ball prostheses.[106,107].

Resilience:

Silicone ball variance was a consistent problem in prosthetic devices implanted prior to change of the treating method in 1965, especially those installed in the aortic position. The abnormalities were presumably ciency secondary to partial or complete detachment of the valve from the aortic root. In a larger series of 22 patients, prosthetic valve ring abscesses were recorded in all cases, regard- less of the site of the valve included."' Involvement of the entire area of the prosthesis causing valve detachment was observed in 80% of patients with aortic prosthetic endocarditis and in 28% of patient with prosthetic endocartitis mitral lesions. Conduction disturbances as a result of a ring abscess formation were observed in 47 % of patient with aortic involvement while on conduction abnorlmalities were related to mitral prosthetic infection. generated a variety of requirements for early operative intervention in late prosthetic valve endocarditis. Among these are relentless or reoccurring fever, non-streptococcal etiology, atrioventricular conduction irregularities, a murmur of prosthetic valvular deficiency, moderate to extreme congestive heart failure, systemic emboli, relapse, and fungal etiology.[18,120,125,127].

***Anatomic viability*:** The prime anatomic disadvantage of the caged ball valve is its size. The spatial requirements of the prosthesis have actually been responsible for a remarkable range of postoperative problems, more commonly seen in the mitral than in the aortic position. The absence of ventricular dilatation in the patient with pure mitral stenosis has both hemodynamic and electrophysiologic consequences following mitral valve replacement. Incomplete forward motion of the poppet has actually been reported as a result of ventricular myocardium interposed in between the struts of the cage, a phenomenon that is significantly improved by concentric ventricular hypertrophy in the presence of coexistent aortic stenosis.[47,8,29] In the presence of a little ventricular chamber, blockage to ventricular emptying can occur with the Starr-Edwards valve in the closed position because of its invasion into the ventricular outflow tract. This occurrence remains in part alleviated by the design of the Smeloff-Cutter prosthesis that houses half of the poppet in the left atrium throughout ventricular systole.

Disintegration of the ventricular septum in the vicinity of the conduction system along with injury to the ventricular complimentary wall have been accountable for deadly dysrhythmia."' Engagement of ventricular myocardium by the open cage of the Smel- off-Cutter valve has actually been reported, causing both injury and hemodynamic compromise.[13,132] The most commonly reported irregularity in the aortic position has actually been size inequality, leading to almost total outflow obstruction by a poppet whose diameter is only minimally less than that of the aortic root itself.[133,134] A fascinating, although uncommon, complication of double valve replacement was reported by Roberts et al. where a strut from the mitral prosthesis disrupted seating of the aortic ball, resulting in aortic deficiency.[135] Use of the valve in the tricuspid position has resulted in stenotic, obstructive, and thrombotic complications just like, if not more frequent than, those come across in mitral replacement.[136,138].

5. THE CAGED DISC VALVE

Evolution:

The caged disc valve was developed mostly to conquer the spatial drawbacks of the caged ball prosthesis, particularly in the atrioventricular position. Potential hemodynamic benefits were recommended by the minimized mass of the meniscoid poppet, prompting clinical application in the semilunar position also. To this end, a variety of different caged disc styles were developed nearly simultaneously in a variety of laboratories in the mid sixties. Hufnagel[7] made a prosthesisof plastic, including an unalloyed polypropylene housing and a biconcave disc built of silicone coated polypropylene . The real estate included a single unit with a diagonal seat and 4 rounded struts that converged centrally enabling a disc excursion in between 4.5 and 8 mm. General height of the valve was around 14 mm. Cross and Jones[10] devised a prosthesis with a 3 pronged, open-ended titanium housing, surrounded at the seat by a Teflon sewing ring, and a lenticular disc made completely of silicone rubber strengthened with a titanium ring . Height varied between 14.5 and 15.0 mm. The Kay- Suzuki valve[9] used a closed metal cage to house a silicone poppet, however extended the height of the valve to 19 mm permitting disc excursions approximately 10 mm. This height allowed increased flow rates, but increased the threat of disc cocking outdoors position and required using four extensionstruts in the valve seat to discourageit. The Kay-Shiley prosthesis[8] put a silicone disc in a Stellite housing consisting of two looped struts put parallel to one another rather than perpendicular as was the casewith the Hufnagel and Kay-Suzuki styles . The total height of the valve was lessthan 11 mm while disc adventure varied between 4.2 and 4.8 mm.

As might be gotten out of the experience with the caged ball valve, toughness of the silicone rubber discs, as well as the polypropylene real estate employed by Hufnagel, was a problem. The intro of the Beall prosthesis in 1967 represented the first effort to conquer this problem.[11] Originally constructed of a compression- molded Teflon disc housed in two parallel

Teflon-coated titanium struts installed on a Dacron velour covered titanium base, it was modified in 1972 to employ pyrolytic carbon-coated non- ferrous metal wire struts [139] . This valve is still readily available today in its modified type. In 1972, Cooley and associates [140] created a prosthesis constructed of a biconical pyrolytic carbon disc confined in an open ended, 4 strut titanium cage . Height differed from 11 to 14 mm consistent with other valves of its type, while the conical shape of the poppet was designed to increase circulation and reduce turbulence.

Results:

Operative and late death for insertion of the caged disc prosthesis is comparable to that reported for all other valve implants.[141-144] Hemodynamics differ from one series to another. E. B. Kay reported resting aortic gradients between zero and 10 mm Hg with the greater profile Kay-Suzuki valve. Additionally, he found no boost with exercise in 10 of 11 patients (the eleventh rose just 5 mm). [145] On the contrary, Bjork reported average peak aortic gradients of 27 mm Hg at rest and 38 mm Hg with exercise in the Kay-Shiley valve, which has less disc excrusion. [146] Mitral hemodynamics are more consistent, with typical mean gradients varying from 5 mm Hg for the Kay-Suzuki valve to 12 mm Hg for the Kay-Shiley and Beall valves. Average workout mean gradients varied between 8 and 24 mm Hg. [9,140,147-152] vitro studies of the Cooley- Cutter prosthesis show this prosthesis to have a substantially lower workout gradient than those of the other caged disc designs.[203]

Complications:

Flow obstruction: With the possible exception of the Kay-Suzuki and cooley-cutter protheses, the caged disc design is the most obstructive of all the prosthetic valves. Roberts [153] has actually reported intimal expansion in the aortic root of patients concerning autopsy with caged disc aortic replacement much like that observed in caged ball valve recipients. Considerable coronary ostial obstruction was observed in one of five patients . Likewise, left ventricular fibroelastosis was recorded in all patients with mitral prostheses. Roberts felt that the observation of large left atria1 thrombi in 3 of 10 late deaths was indirect evidence for substantial mitral blockage. Use of the caged disc valve in the aortic position assumes specific value in the face of substantial preoperative aortic insufficiency. The insertion of a centrally occluded prosthesis might raise ventricular end systolic pressure adequately to either speed up or enhance mitral regurgitation.

Although resting gradients in the mitral position have been mostly much like those tape-recorded for caged ball valves, exercise gradients have actually been substantially greater in all but the Kay-Suzuki higher profile design and the Cooley-Cutter prosthesis (vide supra). In reporting hemodynamic data in 15 patients with a Beall valve mitral replacement, Fernandez[154] notes that although the average mean lung artery pressure reduced considerably following surgery, the average mean mitral gradient did not.

***Valve incompetence*:** A strange feature of the caged disc valve in the atrioventricular position is its tendency to permit intermittent regurgitant flow as a result of cocking of the poppet during ventricular systole. This phenomenon was particularly encouraged by the three strut style of the Cross-Jones valve that not just allowed small leakages about the rim of the disc in the closed position, but also enabled the disc to catch listed below the valve orifice on regular manipulation.[155-156] Transient disc tilting was observed in patients with both 3 and 4 strut prostheses in the mitral position in the presence of aortic deficiency, probably as a result of a jet of regurgitant aortic blood flow causing an oblique orientation of the poppet at the end of ventricular diastole[156, 157]. Patients with valve replacements for mitral stenosis appeared more susceptible to this problem than did those with preoperative mitral incompetence, possibly reflecting distinctions in left ventricular chamber size and end diastolic configuration. Thrombogenicity. The occurrence of prosthetic thrombosis at necropsy is as high as 88%, although just a minority of these manifest either interference with valvular function or clinically noticeable embolic phenomena.[153] However, in a choose few cases, thrombus has actually been documented to cause both tilting and immobility of the disc resulting in deficiency and total blockage, respectively [158-160].

Early reported occurrence of thromboembolic occasions in anticoagulated patients varied between 1% and 2.5% annually in the aortic position and in between 1% and 3% each year in the mitral position.[146,161-163] The exception was Vogel's series of 21 cases of mitral replacement with the Kay-Shiley valve that exhibited an 18% annually occurrence of thrombotic problems over 2 year.[148] Enabling the fact that half of those impacted were not judged to be properly anticoagulated, the number still stays disturbingly high. Likewise, Wellons' work with the Kay-Shiley valve in 83 patients over 6 years of follow-up showed a 6% each year probability of embolic occasions. [164] A more current 1I-yr study of 63 patients with Kay-Shiley mitral prostheses by Edmiston et al exposed a thromboembolic incidence of 12.4% per patient year.[165]

Sturdiness:

There have actually been numerous reports of disc variation in all designs of the caged disc valve that include poppets constructed from Teflon, polypropylene, or silicone rubber.[166-170].

Variance in the disc prosthesis takes the form of grooving of the poppet at each of its points of contact with the vertical struts, recommending that circular rotation of the poppet does not take place during the heart cycle. Discoloration of the silicone disc has actually been observed just like that discovered in the lipid loaded variant ball valves. Roberts' evaluation of the Hufnagel prosthesis revealed marginal disc grooves in the valves of eleven of 17 patients who concerned postmortem 2 or more months after implantation, the earliest manifestation of which occurred in the mitral position at 9 months.[153] Cracks also have actually been kept in mind in disc margins enabling lipid infiltration and limited cysts.

One of the outcomes of disc degeneration is acute prosthetic regurgitation as a result of disc impaction in an oblique or vertical orientation. Not all of a sudden, this complication has been associated with the Kay-Suzuki valve, whose big cage renders it particularly susceptible to this issue.[171] However, this issue has been noted in other designs also, both with and without the complicating element of strut thombus.[172-173].

The introduction of the Beall series 105 pyrolytic carbon disc mitral replacement appeared to resolve the issue of disc variance, however added among its own. Four cases of strut fracture with following embolization of the poppet and acute mitral regurgitation were reported within 1yr of implantation. [174] The series 106 Beall valve was designed to conquer this problem; nevertheless, long-lasting follow-up is not yet readily available.[175].

Hemolysis:

Similar to the caged ball valve, hemolysis is a basic problem of the caged disc design. The greatest occurrence has actually been reported in a series of patients with mitral valve replacement with the original Teflon disc Dacron velour-covered Beall valve, where 6 of 18 patients were found to have frank hemolytic anemia.[176] Clark observed symptomatic hemolytic anemia in 15%. of a 26 patient group with the same valve.[177] Findings of this magnitude have actually not been verified by all private investigators, perhaps due to the arrival of the pyrolytic carbon disc that has actually been connected with a less noticable occurrence of anemia.[139 ,178].

The high incidence of hemolysis has actually been associated by Roberts with a high occurrence of renal hemosiderosis. He demonstrated substantial renal hemosiderosis in 13 (81%) of 16 postmortem evaluations of patients with caged disc prostheses. [153] Two of the Three patients without renal iron deposition had separated tricuspid valve replacement, while all of the aortic receivers and 7 of 8 mitral recipients displayed substantial quantities of hemosiderin in the proximal tubule.

Anatomic viability:

In spite of the low profile of the majority of the caged disc valves, they have likewise been prone to some of the very same anatomic issues that besieged the caged ball prosthesis. Size disproportion was observed in 31% of early mortalities examined by Roberts.[153] The contributing aspects also appear to be similar. Nine out of ten cases with postoperative mitral prosthetic obstruction had actually severe preoperative isolated mitral stenosis. Postoperatively, obstructive contact occurred in between the ventricular myocardium and the valve struts along with the disc itself. Among the nine was noted to develop a left-bundle-branch block postoperatively and at necropsy showed burrowing of the prosthetic strut into the interventricular septum in addition to outflow tract blockage by the disc. This problem is especially troubling inasmuch as these were the patients for whom the valve was mainly created. The use of a pericardial spot as a way of broadening the aortic root and thus avoiding.

Prosthetic blockage has actually been related to fatal hemorrhage in as many as 50% of early operative mortalities in patients receiving the graft, [153] although this may show surgical method rather than a deficiency in either the prosthesis or the pericardial spot. The presence of associated valvular problems has actually likewise resulted in untoward consequences. Issues related to aortic caged disc implantation in patients with aortic deficiency and either pre-existing mitral incompetence or concomitant mitral commissurotomy have been explained above. Mitral prosthetic leakage in the face of aortic deficiency has likewise been observed (vide supra).

6. THE TILTING DISC VALVE

Evolution:

The concept of a tilting disc prosthesis was introduced by Juro Wada in 1968 when he developed a valve that allowed a Teflon disc in a titanium housing to pivot open up to an angle of 75 to 80 degrees, supplying semi centrallized circulation.

Although hemodynamically effective, the valve developed problems with early degeneration of the hinged Teflon disc resulting in its withdrawal from production. Problems consisted of regurgitant circulation through the prosthesis itself and severe disc variation with at least 13 reported cases of disc disengagement and embolization from both the aortic and mitral positions.[179,180]. In 1969, Bjork[13] reported preliminary studies on a new valve consisting of a free drifting Delrin disc suspended in between two eccentrically positioned struts in a Stellite housing designed to tilt available to a maximum angle of 60 degrees. Integrated into its design was the center of the disc to turn on its axis in a style much like that of a phonograph tape-recorded on a turntable as soon as every 180 to 200 cycles, consequently substantially decreasing the wear triggered by the hinge struts.[181,182] The Delrin disc proved to be relatively thrombo- resistant as well as durable; nevertheless, a tendency to take in wetness throughout steam autoclaving was discovered to lead to swelling of the occluder if it was not rigorously dried following sterilization. This prospective issue was fixed with the arrival of a pyrolytic carbon disc in 1972.[183,184] . More recently, a series of valves has actually been developed using a convex-concave pyrolytic carbon disc that increases out of the housing some- what when the valve is open, directing more flow through the lesser orifice . The style is planned to reduce thrombogenicity and resistance to stream, perhaps at the expenditure of a modest boost in regurgitation.[185,186].

Introduced scientifically in 1971, the Lillehei- Kaster prosthesis is constructed of a silicone alloyed pyrolytic carbon disc rotating freely in a titanium housing[14] . The valve has the ability of opening to an optimum angle of 80 ° while closing at an angle of 180 with the horizontal. Unlike the Bjork-Shiley gadget, in which the disc fits entirely within the valve orifice on closure, the Lillehei-Kaster disc is designed to overlap the valve ring in the closed position. There have been no reports of major disc difference in either valve to this day.

The Hall-Kaster pivoting disc aortic prosthesis was introduced into clinical study in 1977 . Including a pyrolytic carbon coated graphite disc in a titanium real estate, its distinguishing feature is an S-shaped disc guide strut that serves to pivot the disc through a maximum arc of 75 , in theory yielding much better hemodynamics than either the Bjork-Shiley hei-Kaster valves, although early reports have not demonstrated a considerable distinction.[187-188] St. Jude Medical, Inc. presented a bileaflet disc prosthesis for clinical trial in 1977. The valve incorporates 2 tungsten impregnanted pyrolytic carbon semicircular brochures in an orifice ring of pyrolytic carbon layered machined graphite.[189] The brochures are created to pivot from a resting angle of 30 ° to 35" with the horizontal. Through a maximum arc of 50 ° to 55' each, permitting nearly entirely centralized circulation . The pivoting system is devised to get rid of the requirement for assistance struts that exists with both the Bjork-Shiley and Lillehei-Kaster valves; nevertheless, it does not permit complimentary rotation of the discs that might increase the potential for prosthetic wear at the hinge sites.

Results:

Operative and late mortality for the tilting disc valve resembles that reported for other prosthetic implants.[14,190,191]Short-term follow-up information with the St. Jude prosthesis suggest a reduced late mortality, [192] although this distinction may show advances in surgical method rather than any advantage from the St. Jude prosthesis.

Hemodynamics differ in between the eccentric monocusp and the bileaflet tilting disc valves. Bjork-Shiley and Lillehei-Kaster prostheses typical resting mean gradients in between 12.5 and 17.4 mm Hg in the aortic position and between 4.5 and 7.6 mm Hg in the mitral position.[14,178,191-202].

Preliminary testing of the St. Jude prosthesis in little, selected series has yielded an average mean resting gradient of 4.2 mm Hg for aortic replacement and only 0.4 to 1.79 mm Hg for mitral implantation.[192,194] Workout gradients with the eccentric monocusp valves are not well recorded, however, Bjork has reported doubling of the aortic resting gradient and tripling of the mitral resting gradient on workout.[197,198] Adequate documentation of exercise gradients in large groups of patients with St. Jude valves is not currently available, although there is some recommendation that they compare positively with monocusp gradients in the mitral position.[202].

Problems:

***Flow obstruction*:** Circulation dynamics for the tilting disc prosthesis are better than for any other mechanical prosthesis. In vitro characteristics are better than those observed in the porcine xenograft at high flow[203,204] Incomplete opening of the mitral eccentric monocusp prosthesis can happen in the face of huge aortic deficiency; however, this issue is rather unusual unless motivated by inaccurate positioning of the valve (vide infra). There are no reports of the habits of the st. jude mitral prothesis in the face of aortic regurgitation.

***Valve incompetence*:** research studies of the initial Bjork mitral prothesis demonstraded regrutation flow of approximately 12% to 15 % in some patients, a phenomenon that was presumed to provide an antithrombotic "cleaning" system by its manufacturer. [205] Regurgitant flow was gotten rid of by the introduction of the pyrolytic carbon disc that fit

the valve orifice more snugly than did its predecessor. In vitro testing has recommended an almost similar amount of regurgitation in both the Bjork and the St. Jude valves of 4% 5% in 29-mm prostheses.(192).

***Thrombogenicity*:** The thrombotic potential of the Bjork valve in the absence of anticoagulation is popular. Although there is documents of reduced thrombogenesis with the use of pyrolytic carbon,(206) a significant risk of thrombotic and thromboembolic events stays in the patient who is not anticoagulated.

There are numerous reports of apoplexy of the prosthesis itself in both the aortic and the mitral position, usually in patients not getting anticoagulation therapy.(207-210) Necropsy research studies carried out by Roberts (209) demonstrated no obstructive prosthetic thrombosis in any patients treated with warfarin salt unless there was co-existing prosthetic endocarditis. Similarly, the general occurrence of prosthetic apoplexy associated Prosthetic heart valve With the Bjork valve was less than that obsereved in anticoagulated patients with caged ball valves. In our own experience, the Lillehei-Kaster valve has been associated with a considerable incidence of prosthetic thrombosis when implanted in the mitral position. Although no systematic postmortem studies have yet been published relating to the thrombotic potential of the St. Jude prosthesis, its clinical similarities recommend that the experience will be similar. Clinical incidence of prosthetic apoplexy with the St. Jude valve has averaged about 0.43% per patient-year and to date has been observed just in the mitral position in patients with double valve replacement and atrial fibrillation.(192,195).

Thromboembolic occasions have actually been reported to typical 1.9% per patient-year with the regular Bjork-SmiIey prosthesis and 1.0% per patient-year with the St. Jude device in groups of patients who have been variably anticoagulated (191.192.194).

***Toughness*:** There are no reports of pyrolytic carbon disc degeneration to date. Disc dislodgement from the Bjork-Smiley prosthesis has actually been reported in both mitral and aortic designs although the etiology appears to arise from damage throughout intraoperative manipulation of the valve instead of intrinsic defects in the device itself (66.211) There are no reports of disc escape from the Lillehei-Kaster valve.

It has been reported that the rate of wear of pyrolytic carbon is substantially less when it is brought into contact with other objects made from pyrolytic carbon than when it abuts titanium or Stellite. This element most likely represents the observation that the projected valve life of the St. Jude Medical prosthesis is 255 years when subjected to in vitro tiredness and tension testing, despite the fact that its semicircular discs can not rotate and equally disperse the shearing forces of the heart cycle.(191) As such it represents a significant improvement on previous attempts at style of bileaflet and trileaflet gadgets by Gott and Hufnagel.

***Hemolysis*:** Hemolysis with all tilting disc valves has actually been incredibly minimal with nearly no reports of substantial hemolytic anemia. (14,212,214) Postmortem examination has actually revealed the existence of kidney hemosiderosis in 3 of 9 patients, nevertheless only 1 of these was kept in mind to be significantly affected.(209).

Endocarditis: Endocarditis has actually been no more regularly observed in the tilting disc pros- thesis than in any other valve replacement,(191) although it has been related to an increased occurrence of prosthetic apoplexy.

Anatomic viability: Reports of anatomic disproportion of tilting disc valves are rare. Roberts(209) has observed 2 cases where the size of the Bjork-Shiley prosthesis picked for mitral valve replacement was too large for the left ventricular cavity leading to incomplete opening of the valve with resulting prosthetic stenosis. The benefit of the St. Jude prosthesis in both this circumstance and in patients with little aortic roots results from its low profile.

Positional relationships within the orifice may also be of value to the function of an eccentric monocusp prosthesis. In the mitral position, rotation of the valve seat such that the disc opens toward the high ventricular septum instead of toward the ventricular peak not only might lead to suboptimal flow dynamics and insufficient ventricular filling, but likewise might increase the capacity for disc contact with the interventricular septum and its attendant stenotic and thrombotic problems. This phenomenon has actually been suggested to be the cause of death in necropsy research studies.(30) Similarly, in the aortic position, opening of the disc towards the noncoronary cusp not just might lessen intimal proliferation about the coronary ostia, but may likewise promote laminar circulation by permitting the valve to open parallel to the tilt of the aortic root instead of at an angle to it(215).

Migration of foreign material between the disc and the valve ring is an unusual, however possibly catastrophic event, especially for prostheses situated in the mitral position. Recently, 2 cases have been explained wherein intraoperative entrapment of chordal remnants in a mitral prosthesis necessitated reinstitution of cardiopulmonary bypass and cutting of the chordae(216).

7. THE TISSUE VALVE

Evolution:

The obvious prospective advantages of absent or substantially lessened thrombogenicity and unobstructed laminar flow render the tissue valve an extremely preferable alternative to other prosthetic devices. Following the publication of Hufnagel's original deal with intravascular valve implantation, Murray and associates attained some success positioning homografts in the coming down aorta as surgical therapy for aortic regurgitation. Strategies for subcoronary implantation were developed by Ross, Barratt-Boyes, and Duran and Gunning with early beneficial outcomes reported by several laboratories.(15,17,217,218) A frame mounting was included by Angel1 to stabilize and support the prosthesis and applied to bovine heterografts by Carpentier, rendering the valve suitable for atrioventricular replacement.(219,220) Sanitation and preservation of the very first heterografts was accomplished at first with a buffered mercurial option and subsequently with formalin, nevertheless early wear and tear was quickly reported.(221) The use of 1% glutaraldehyde as a preservative resulted in stiffer, less mobile valve leaflets, but with the subsequent exemption of salt metaperiodate oxidation from the tanning process and using a 0.6% glutaraldehyde option, the Carpentier valve ended up being both long lasting and functiona1.(222) Xenograft durability was further improved by the intro of an asymmetric versatile stent that was shown to promote significantly less leaflet strain than the original frame mounting procedure.(223).

At present, there are 2 heterograft valves. characteristics for the Sauvage: Recreated by approval.) readily available for large industrial distribution: the Hancock and the Carpentier-Edwards. Both are of porcine origin and differ in the concentration of glutaraldehyde utilized in the tanning process (0.2% from the Hancock and 0.625% for the Carpentier-Edwards) and stent structure (polypropylene in the Hancock and Elgiloy in the Carpentier-Edwards). The right coronary cusp of the porcine aortic valve is supported by the interventricular septum. Thus, both the Hancock and Edwards valves integrate a muscular shelf attached to the right coronary cusp that narrows the inlet orifice. Narrowing appears more popular in the Hancock prosthesis, which is installed on an in proportion base. The Carpentier-Edwards design tries to minimize the amount of septal muscle incorporated in the prosthesis by mounting it on an unbalanced orifice that is contoured to the shape of the porcine valve. Both resilience and occurrence of problems seem similar.

for both prostheses, although the Carpentier prosthesis might be hemodynamically remarkable.(224,225) Reports of unacceptably high trans- valvular gradients in smaller aortic heterografts have led to the development of a modified Hancock prosthesis that replaces a cusp without a muscular ridge for the ideal coronary cusp as a means of broadening the valve orifice.(226)Early reports of postoperative follow-up in an elderly patient population reveal appropriate hemodynamics as well as a low occurrence of thromboembolic events.(227,228).

The desire for valves that could be specifically tailored to even the tiniest size requirements prompted the development of frame mounted autologous fascia lata and heterologous (bovine) pericardial prosethesis by Ionescu and associates.(27-28)The valve is presently constructed of glutaraldehyde treated bovine pericardium installed on a Dacron covered titanium frame with a Dacron sheathed silicone sewing ring and includes a bigger outlet size to inlet diameter (OD/ID) ratio then the heterograft(229-232).

Results:

Operative death has actually varied in between 3% and 10% for aortic replacement and between 1% and 13% for mitral replacement in a lot of series.(233,234) Late mortality has varied between 15% and 30% at 4 years. The average resting mean gradient throughout the aortic prosthesis is 16.8 mm Hg for the porcine xenograft and 7-10 mm Hg for the Ionescu-Shiley in several series.(230-235) The typical resting mitral mean gradient has actually been reported as 6.9 mm Hg for the xenograft and 5.3-6.4 mm Hg for the pericardial prosthesis.

Hemolysis is not medically noticeable in the absence of perivalvular leak with the exception of one current report recording increased LDH and reduced haptoglobin levelsin a small population of patients with the Ionescu-Shiley prosthesis.(236).

Complications:

Obstructed flow: In spite of the absence of a central occluder, porcine heterograft flow characteristics have been frustrating in vitro. Studies carried out by Gabbay,(203,204) in a pulse duplicator system have actually demonstrated pressure gradients in between 3.2 and 12.6 mm Hg at a flow rate of 5 liters per minute relying on the size of the prosthesis. At a circulation rate of 9 liters per minute, the gradient climbed to between 10.3 and 40.8 mm Hg, approximating that noted for

the centrally occluded Starr-Edwards valve and significantly higher than that generated by the tilting disc gadgets. By contrast, gradients across the Ionescu-Shiley bio prosthesis varied between 1.5 and 7.7 mm Hg at 5 liters per minute and in between 4.8 and 24.9 mm Hg at 9 liters per minute, comparing positively with the Bjork-Shiley and Lillehei-Kaster mechanical replacements. In vivo resting information do not reveal any significant distinctions between the porcine heterograft and the tilting-disc prosthesis. Although there is a significant advantage claimed for the pericardial prosthesis in the aortic position, there are not yet appropriate numbers of patients to confirm Ionescu's findings.[230,235].

In vivo, the outcomes have actually been significantly more encouraging. Mitral valvular gradients reported by Horowitz et a1 [237] averaged 5.7 + 2 mm Hg at rest increasing just to 15 +- 10 mm Hg on exercise. Patients in this group who established symptomatic failure postoperatively did quickly the basis of left ventricular dysfunction instead of valvular blockage (preoperative mitral deficiency was the most typical predisposing sore). Resting mitral mean gradients observed by Johnson et al [238] varied between 4 and 14 mm Hg with the largest gradient produced by thrombotic limitation of xenograft leaflet movement rather than by inherent prosthetic obstruction. Hannah and Reis [239] also recorded outstanding mitral function of the heterograft, noting typical resting mean gradients of 3.6 mm Hg increasing to 7.6 mm Hg on exertion.

Aortic hemodynamics were not as encouraging with the peak resting gradient averaging 16 mm Hg increasing to 70 mm Hg on effort. The large gradients created throughout the heterograft in the aortic position on workout as well as those kept in mind in vitro at high circulation most likely reflect a decreased outlet size to inlet size (OD/ID) ratio when compared to either the Ionescu-Shiley bio prosthesis or the tilting-disc mechanical valves. Nevertheless, in the mitral position, the increased valve location of the Ionescu device appears compromised by its tapered outlet that has the propensity to funnel blood circulation directly at the interventricular septum rather than allowing a more typical diffusion throughout the ventricle. As a result, the diastolic mitral gradients throughout all tissue valves are practically identical, while the heterograft most likely offers a more physiologic mechanism for left atria1 emptying.

Thrombogenicity: Thromboembolic phenomena occur considerably less often with tissue valves than with mechanical prostheses. The rate of thromboembolism has actually been variably reported, in part as a result of inconsistent anti-coagulation procedures. Incidence has actually varied in between zero and 2.8% per patient-year for aortic replacement and between 0.6% and 10.8% per patient-year (with a fixed average of 2.9% per patient-year) in the mitral position for all tissue valves[230,233].

Most of the total thromboembolic episodes occur within the very first 3 postoperative months for both mitral and aortic replacement, with the total occurrence greater in atrioventricular replacement.[240-244] The regular occurrence of early postoperative thrombosis most likely shows insufficient endothelial proliferation along the valve stent and sewing ring. If the instant postoperative duration is omitted, the occurrence of thromboembolism from mitral heterograft prosthesis is up to 0.7% per patient- year.[233].

Whether to anti coagulate patients with tissue valves is a matter of continuous discussion. Comparison of the occurrence of thromboembolic events in patients who have porcine mitral prostheses with the occurrence of substantial hemorrhage in anticoagulated patients with mechanical valves (0.4% to 7.8% per patient-year) raises serious doubt about the effectiveness of long-term warfarin therapy for tissue valve recipients.[233] In Stinson's series of aortic valve replacements, using warfarin in the postoperative duration did not substantially alter the occurrence of early thromboembolic occasions, although other investigators have actually not reported similar findings.[240].

More recent work by Jamieson et a1. [242] and Geha et a1[242] suggest that anticoagulation is required mostly in patients with mitral valve replacement who remain in atria1 fibrillation postoperatively. A report by Platt et al [243] documenting 6 cases of Hancock prosthetic thrombosis is remarkable because 3 of the 6 suffered prosthetic degeneration prior to apoplexy. Prosthetic degeneration was either connected with young age of the recipient (vide infra) or was secondary to prosthetic endocarditis. 2 of the remaining three thrombi happened on prosthetic valves of patients with rheumatic cardiovascular disease. Heart rhythm was not defined for any in this series.

There is no validated proof that aspirin or dipyridamole are of value in reducing the thromboembolic potential of tissue valve replacement.

Resilience: Degeneration of glutaraldehyde-tanned bioprostheses has actually been minimal in reported mean follow-ups of approximately 6 hours [244,245] Nevertheless, recent information suggest a dramatically increased incidence of xenograft failure in between 5 and 7 years following implantation.[233,246-248] Necropsy data assembled by Spray and Roberts [249]

exposed the presence of microscopic focal cusp interruption in 45% of cases studied, although gross cusp degeneration was only seen in 3 patients (17%). 2 of the three had mitral prostheses in place for 4.5 and 6.5 year each. Fibrin deposition on cusp out- circulation surfaces appeared to take place early, while accumulation of histiocytes and huge cells on cusp surface areas with focal invasion was a late incident.

In vitro tiredness screening has actually likewise documented separation and fracture of the collagen bundles of glutaraldehyde treated tissue subjected to tension equivalent to 12 year of in vivo function[250] Work by Barratt-Boyes[251] and others recommends that prosthetic leaflet stiffening and changes in collagen architecture result in part from glutaraldehyde fixation of the bio prosthesis at brochure closing pressures of in between 4 and 100 mm Hg. In vitro assessment of tissue prostheses subjected to glutaraldehyde fixation at absolutely no pressure shows both hemodynamics and collagen microstructure that more closely approximate that of native valvular tissue,[251,252] however confirmation of enhanced prosthetic survival time is doing not have.

Multiple reports of early prosthetic calcification in children and young adults, in addition to patients with chronic renal failure have actually recently prevented making use of xenografts in these patient populations.[253-260] The etiology of this phenome- non is unidentified, although current data recording infiltration of plasma cells and histiocytes into the prosthetic brochures of children with tissue valves suggest an immunologically moderated mechanism.[261].

Trace quantities of glutaraldehyde remaining in implanted valves have been shown to promote perivalvular leakage and thrombotic phenomena.[230].

***Endocarditis*:** Occurrence of late prosthetic endocarditis in patients with a porcine xenograft differs bit from that reported from mechanical values, averaging 1.1% per patient-year.[233] However, the website of infection in the bio prosthesis seems mostly in fibrin deposits discovered on valve brochures rather than along the sewing ring as is the case with mechanical valves. This possibly accounts for the increased effectiveness of medical therapy in the treatment of bioprosthetic endocarditis observed by Rossiter.[262] There is no apparent difference in the histologic discussion of prosthetic endocarditis between the porcine xenograft and the bovine pericardial bioprosthesis.[263].

Although irregular mycobacteria have been reported in cultures of aortic wall that is customarily shipped with the commercially readily available porcine valve, there have just been two reported clinical cases of bioprosthetic endocarditis secondary to acid quick organisms.[264,265].

Anatomic suitability. Reference has been made formerly to the anatomic benefits of the porcine xenograft in contrast to the pericardial bio prosthesis in the mitral position. Spray and Roberts [249] have reported 3 instances in which positioning of among the struts of a mitral prosthetic flexible stent in the ventricular outflow system caused a low output syndrome and death. Compression of the aortic root by a big mitral bioprosthetic sewing ring has also been observed.

In the aortic position, procine xenograft implantation has been reported to lead to both considerable compromise of aortic root size by struts and total occlusion of both coronary ostia by the struts of an improperly located prosthesis.[249,266] The Ionescu-Shiley valve, on the other hand, has been revealed to be especially appropriate for aortic valve replacement in patients with a small aortic root.[267] Regardless of, its excellent hemodynamics must be weighed versus the technical problem of insertion of the Ionescu-Shiley prosthesis. This prosthesis is bulkier than the porcine xenograft and regularly requires the use of a pericardial gusset in the aortic root.

8. SUMMARY AND CONCLUSION

There are a number of difficulties inherent in the analysis of such a big and diverse amount of information. In a significant variety of clinical trials, there is no significant effort made to examine prosthetic performance as a function of preoperative heart anatomy. Hemodynamics have actually not been methodically studied in relation to preexisting left ventricular size, shape, or setup, mitral annular orientation, or left atria1 size. Postoperative anticoagulation procedures differ from one institution to another and sometimes within study hall themselves. Less tangible variables such as change in surgical technique with time and differential familiarity of cardiovascular surgeons with the prostheses employed are persistent issues in any research study of this sort. Perhaps the greatest variable in assessing the postoperative performance of valvular prostheses over the past Twenty Years is the extreme improvement in methods of intraoperative myocardial preservation. Regardless of, contrasts are possible within the confines of specific requirements.

The caged ball value remains in use after 20 years of clinical experience. It has sustained the greatest variety of adjustments, probably due to the fact that it has been the most extensively studied. Hemodynamics are adequate although its centrally blocked style is responsible for increased turbulence, hemolysis, and neointimal proliferation, particularly in

the aortic position. The device has actually been shown to be resilient with virtually no reports of ball variance since the modification of the silicone treating treatment in1965. Thromboembolic rates are acceptable in the anticoagulated patient while prosthetic thrombosis is not a severe hazard in the non-close clearance gadget. Occurrence of endocarditis is not particularly different from that related to all nonbioprosthetic valves, although there is a much greater published volume of clinical experience worrying acknowledgment and treatment of late prosthetic valve endocarditis in patients with caged ball valves than there is for other replacement gadget. Perhaps the most severe drawback to caged ball style is its size. Its big spatial requirements have actually resulted in anatomic complications in patients with small aortic roots, separated mitral stenosis, left ventricular hypertrophy, and double valve replacement, to name a few. Nonetheless, this is still the valve of choice in some centers.

Caged disc design would seem the least appealing of all. It is definitely the most obstructive, particularly in the aortic position where it has actually been plainly shown to increase preexisting mitral insufficiency when implanted in patients with aortic regurgitation. Although the general incidence of thromboembolism is not particularly greater than that priced estimate for other mechanical valves, the potential for disastrous result in the existence of small prosthetic thrombi is higher. The occurrence and intensity of hemolysis is at least equal to that reported for the caged ball valve, while the occurrence of frank hemolytic anemia is higher in high circulation states. The incidence of degeneration prior to introduction of the pyrolytic carbon disc was extraordinary. Clinical experience with the toughness of the most current Beall prosthesis (design 106) stays limited. More than other changement valve, the caged disc appears vulnerable to any variety of anatomic or hemodynamic alterations, particularly in the mitral position. Disc cocking, and resultant prosthetic regurgitation has been often reported in association with prosthetic apoplexy, left ventricular hypertrophy, and aortic incompetence.

Design of the tilting disc valve appears to overcome most of these problems. Hemodynamics are excellent if effectively positioned. Circulation characteristics throughout the St. Jude valve are most likely the very best of all, although substantially more clinical data is required, particularly on exercise. The valves are mostly proficient, show practically no considerable hemolysis, and have demonstrated remarkable resilience with rare exceptions (which might be attributed to implantation method). Reported anatomic complications have been very little and have actually often been a function of orientation of the orifice of the eccentric monocusp valve. Although thromboembolic episodes are irregular in the anticoagulated patient, the occurrence of deadly prosthetic apoplexy is considerable in the patient who ceases anticoagulation. Prosthetic endocarditis has been observed to promote the occurrence of valvular apoplexy, even in the existence of warfarin.

The introduction of the glutaraldehyde-treated tissue valve solves some problems and develops others. The porcine xenograft appears variably obstructive at high circulation rates although this observation has actually not posed a significant problem. The valves are skilled, nonhemolytic, and essentially no thrombogenic. There is need to believe that late tissue valve endocarditis, although no less common than that connected with mechanical valves, may be more vulnerable to medical instead of surgical treatment. A clear risk of mycobacterial infection in association with the porcine xenograft has not been satanic force- striated. Couple of anatomic issues have actually been described although the pericardial bio prosthesis may be more suitable for aortic replacement as a result of its greater flow capability, while the porcine heterograft might be more appropriate in the mitral position because of anatomic factors to consider. All of these valves are bulkier than the tilting-disc prosthesis which may increase the technical trouble of insertion in some patients. Sturdiness remains the significant issue. Known incidence of early calcification and degeneration of the porcine valve in children and teenagers in addition to recent reports of increasing prosthetic degeneration 5-7 years following implantation would have the tendency to restrict making use of these prosthesis in specific patient populations. There is proof that prosthetic lifespan may be extended in some patients by an alteration in the valve closing pressure throughout glutaraldehyde fixation.

In conclusion, it would seem that at present prosthetic innovation has mainly rendered the caged disc valve obsolete. Choices regarding which of the other replacement valves are most proper need to be made with respect to the individual patient and think about elements such as age, activity, medication compliance, tolerance and relative safety of anticoagulation, psychologic status, associated disease entities, practical cardiac anatomy, and the experience of the operating surgeon.

The authors gratefully acknowledge Michael le porta of Merck, Sharp and Dohme for his vital aid in evaluation of the literature, and Kathleen Thomas for her patient and skilled help in the preparation of the manuscript.

REFERENCES

[1] Hufnagel CA, Harvey WP: The surgical correction of aortic regurgitation: Preliminary report. Bull Georgetown U Med Ctr 6:60, 1953

[2] Hufnagel CA, Harvey WP, Rabil PJ, et al: Surgical correction of aortic insufficiency. Surgery 35:673, 1954

[3] Halkier E. Hansen PF, Anderson I: Aortic incompetence: The eventual outcome in a small series treated with Hugnagel's descending aorta ball-valve. Stand J Thorax Cardiovasc Surg 452, 1970

[4] Harken DE, Soroff HS, Taylor WJ, et al: Partial and complete prostheses in aortic insufficiency. J Thorax Cardio-vast Surg 40:744, I960

[5] Harken DE, Taylor WJ, LeFemine AA, et al: Aortic valve replacement with a caged ball valve. Am J Cardiol 91292, I962

[6] Starr A, Edwards ML: Mitral replacement: Clinical experience with a ball-valve prosthesis. Ann Surg lS4:726. 1961

[7] Hufnagel CA, Conrad PW: Comparative study of some prosthetic valves for aortic and mitral replacement surgery 57;205,1965 .

[8] kay JH, Kawashima Y, Kagawa Y: Experimental mitral valve replacement with a new disc valve. Ann Thorax sur 2:485, 1966

[9] Kay EB, Suzuki A, Demaney M, et al: Comparison of ball and disc valves for mitral valve replacement. Am J Cardiol l8:504, 1966

[10] Cross FS, Jones RD: A caged-lens prosthesis for replacement of the aortic and mitral valves. Ann Thorax Surg 21499, I966

[11] Beall AC, Bloodwell RD. Liotta D, et al: Clinical experience with a Dacron velour-covered Teflon-disc mitral valve prosthesis. Ann Thorax Surg 5:402, 1968

[12] Wada J, Jomatsu S,Kamata K; cardiac valve replacment with wada-cutter prosthesis.Ann thorac surg 14:38,1972

[13] Bjork VO: A new tilting disc valve prosthesis. Stand J Thorax Surg 3: I, 1969

[14] Lillehei CW, Kaster RL, Coleman M, et al: Heart valve replacement with Lillehei-Kaster pivoting disc prosthesis. NY State Med J 74:1426, 1974

[15] Ross DN: Hemograft replacement of the aortic Valve. Lancet 2:487, 1962

[16] Duran CC, Gunning AJ: A method for placing a total homologous aortic valve in the subcoronary position. Lancet 2:488, 1962

[17] Barratt-Boyes BG: Homograft aortic valve replacement in aortic incompetence and stenosis. Thorax 19:131, 1964

[18] Murray G: Homologous aortic-valve segment trans- plants as surgical treatment for aortic and mitral insufficiency. Angiology 71466, 1956

[19] Kerwin A, Lenkei SC, Wilson DR: Aortic-valve homograft in the treatment of aortic insufficiency: Report of nine cases, with one followed for six years. N Engl J Med 266:852, 1962

[20] Ross D: Biologic valves: Their performance and prospects Circulation 45:1259, 1972

[21] Angel WW, Shumway NE, Kosek JC: A five-year study of variable aortic valve homografts. J Thorax Cardio- vast Surg 641329, 1972

[22] Wallace RB: Tissue valves. Am J Cardiol 35:866, 1975

[23] Moore CH, Martelli V, Al-Janabi N, et al: Analysis of homograft valve failure in 31 I patients followed up to IO years. Ann Thorax Surg 20:274, 1975

[24] lonescu MI, Wooler GH, Smith DR, et al: Mitral valve replacement with aortic heterografts in humans. Thorax 22:305, 1967

[25] Carpentier A, Blondeau P, Laurens B, et al: Mitral valve replacement with frame-mounted aortic heterografts. J Thorac Cardiovasc Surg 56388, 1968

[26] Carpentier A, Lemaigre Cl, Robert L, et al: Biological factors affecting long-term results of valvular heterografts. J Thorax Cardiovasc Surg 58:467, 1969

[27] Ionescu MI, Ross DN : Heart-valve replacement with autologous fascia late.lancet 2:335,1969

[28] Ionescu MI,Pakrashi BC , Mary DAS , ET AL ; Results of aortic valve replacement with frame-supported fascia lata and pericardial grafts .J thorac cardiovasc surg 64:240, 1972

[29] Nicoloff DM, Emery RW: Current status of the St. Jude cardiac valve prosthesis. Contemp Surg 15:11, 1979

[30] Roberts WC: Choosing a substitute cardiac valve: Type, size, surgeon. Am J Cardiol 38:633, 1976

[31] Starr A, discussion of Isom, et al: Long-term evaluation of cloth-covered metallic ball prostheses. J Thorax Cardiovasc Surg 64:354, 1972

[32] Krosnik A: Death due to mitration of the ball from an aortic valve prosthesis. JAMA 191:1083, 1965

[33] Starr A, Pierie WR, Raible DA, et al: Cardiac valve replacement: Experience with the durability of silicone rubber. Circulation 33/34 (Suppl I): 115, 1966

[34] McKenzie JW, Almond CH: Expulsion of ball from aortic valve prosthesis. Ann Thorac Surg 2:435, 1966

[35] Newman MM, Hoffman MS , Gesink MH : Mechanical failure of starr-Edwards aortic prosthesis due to ball fracture .J Thorax cardiovascs surg 53:398, 1967

[36] Hairston P,Summerall CP, Muller WH : Embolization of silastic ball from starr-edwards prosthesis : case report and comments . ann surg 166:817, 1967

[37] Roberts WC , Morrow AG : late postoperative pathological findings after cardiac valve replacement . circulation 35/26 (suppl 1):48, 1967 .

[38] La Foret EG: Death due to swelling of ball component of aortic ball-valve prosthesis. N Engl J Med 276:1025, 1967

[39] Roberts WC, Morrow AG: Anatomic studies of hearts containing caged-ball prosthetic valves. Johns Hopkins Med J 121:271, 1967

[40] Bonnabeau RC, Lillehei CW. Mechanical “ball failure” in Starr-Edwards prosthetic valves: A report of three cases. J Thorax Cardiovasc Surg 56:258, 1968

[41] Roberts WC, Morrow AG: Fatal degeneration of the silicone rubber ball of the Starr-Edwards prosthetic aortic valve. Am J Cardiol 22:614, 1968

[42] Garcia MC, Clarysse AM, Alexander CS, et al: Hemolytic anemia due to progressive enlargement of Silastic ball component of aortic prosthesis. Circulation 38:505, 1968

[43] Cohen M, Barwinsky J, Mymin D: Late failure of the ball valve (Starr) aortic prosthesis. Can J Surg 11: 144, 1968

[44] Leatherman LL, Leachman RD. McCann RG, et al: malfunction of mitral ball-valve prostheses due to a swollen poppet. J thorac cardiovascs surg 57:160, 1969 .

[45] Roberts WC, Levison GE, Morrow AG: Lethal ball variance in the Starr-Edwards prosthetic mitral valve. Arch Intern Med 126:517, 1970

[46] Hylen JC, Closter FE, Starr A, et al: Aortic ball variance: Diagnosis and treatment. Ann Intern Med 72:l. 1970

[47] Roberts WC, Bulkley BH, Morrow AG: Pathologic anatomy of cardiac valve replacement: A study of 224 necropsy patients. Prog Cardiovasc Dis I5:539, 1973

[48] Barnhorst DA, Oxman HA, Connolly DC, et al: Long-term follow-up is isolated replacement of the aortic or mitral valve with the Starr-Edwards prosthesis: An eleven- year review. Am J Cardiol 35:228, 1975

[49] Barnhorst DA, Oxman HA , connolly DC , et al : Long-term follow-up is isolated replament of the aortic or mitral valve with the starr-edwards prosthesis :An eleven-year review ,J Thorax cardiovascs surg 71:230,1976

[50] Joyce LD, Emery RW, Nicoloff DM: Ball variance and fracture of mitral valve prosthesis causing recurrent thromboemboli. J Thorax Cardiovasc Surg 75:309, 1978

[51] Herr RH, Starr A, Pierie WR, et al: Aortic valve replacement: A review of six years experience with the ball valve prosthesis. Ann Thorax Surg 6: 199, 1968

[52] McHenry MM, Smeloff EA, Fong WY, et al: Critical obstruction of prosthetic heart valves due to lipid absorption by Silastic. J Thorax Cardiovasc Surg 59:413, 1970

[53] Starr A, Herr RH, Wood JA: Mitral replacement: Review of six years experience. J Thorax Cardiovasc Surg 54~333, 1967

[54] Hodam R, Starr A, Raible D, et al: Totally cloth- covered prostheses: A review of two years clinical experience. Circulation 41/42 (Suppl 2):33, 1970

[55] Braunwald NS, Boncheck LI: Prevention of thrombus formation on rigid prosthetic heart valves by the ingrowth of autogenous tissue. J Thorax Cardiovasc Surg 54:630, 1967

[56] Hodam R, Anderson R, Starr A, et al: Further evalua- tion of the composite seat cloth covered aortic prosthesis. Ann Thorax Surg 12:621, 1971

[57] Isom OW, Williams CD, Falk EA. et al: Long term evaluation of cloth covered metallic ball prostheses. J Thorax Cardiovasc Surg 64:354, 1972

[58] Bonchek LI, Starr A: Ball valve prostheses: Current appraisal of late results. Am J Cardiol 35:843, 1975

[59] Santinga JT, Kirsh MM: Hemolytic anemia in series 2300 and 23 10 Starr-Edwards prosthetic valves. Ann Thorax Surg 14:539, 1972

[60] Stein DW, Rahimtoola SH, Kloster FE, et al: Thrombotic phenomena with nonanticoagulated composite-strut aortic prostheses. J Thorax Cardiovasc Surg 7 1:680, 1976

[61] Starr A, Grunkemeier GL, Lambert L, et al: Clinical results with composite strut (track valve) prostheses. Circulation 55/56 (Suppl3):29, 1977

[62] Macmanus Q, Grunkemeier G, Housman L, et al: Early results with composite strut caged ball prostheses. Am J Cardiol46:566, 1980

[63] Oxman HA, Connolly DC , Ekkis FH : mitral valve replacement with the smeloff-cutter prosthesis . J thorac cardiovascs surg 69:247, 1975 .

[64] Lee SJK, Haraphongse M, Callaghan JC , et al : Hemodynamic changes following correction of severe aortic stenosis using the cutter-smeloff prosthesis . circulation 42:719, 1970 .

[65] Lee SJK, Zaragoza AJ, Callaghan JC, et al: Malfunction of the mitral valve prosthesis (Cutter-Smelloff). Circulation 41:479, 1970

[66] Brawley RK, Donahoo JS, Gott VL: Current status of the Beall, Bjork-Shiley, Braunwald-Cutter, Lillehei-Kaster, and SmelolT-Cutter cardiac valve prosthesis. Am J Cardiol 35:855, 1975

[67] Braunwald NS, Tatooles C, Turina M, et al: New development in the design of fabric-covered prosthetic heart valves. J Thorax Cardiovasc Surg 62:673. I97 1

[68] Bull B, Braunwald NS: Human histopathologic response to a completely fabric-covered prosthetic heart valve. Ann Surg 174:755, 1971

[69] O'Rourke RA. Peterson KL, Braunwald NS: Postoperative hemodynamic evaluation of a new fabric-covered ball valve prosthesis. Circulation 47/48 (Suppl 3): 74, 1973

[70] Pluth JR, Broadbent JC, Danielson GK, et al: Aortic and mitral valve replacement with the cloth-covered Braunwald-Cutter valve. Mayo ClinProc 48:722, 1973

[71] Pluth JR, Broadbent JC, Barnhorst DA, et al: Aortic and mitral valve replacement with clot-covered Braunwald-Cutter prosthesis: A three year follow-up. Ann Thorax Surg 20:239, 1975

[72] Magovern GJ, Kent EM, Cromie HW : sutureless prosthetic heart valves , circulation 27:784, 196.

[73] Magovern GJ,Cromie HW: sutureless prosthetic heart valves. J thorac cardiovasc surg 46:726,1963

[74] Magovern GJ, Kent EM, Cushing WJ, et al: Sutureless aortic and mitral prosthetic valves: Clinical results and operative technique on sixty patients. J Thorax Cardiovasc Surg 48:346, 1964

[75] Reed GE, Clauss RH, Tice DA, et al: Five-year experience with Magovern aortic prostheses. Circulation 43/44 (Suppl 1):73,1971

[76] Magovern GJ, Liebler GA, Cushing WJ, et al: A thirteen-year review of the Magovern-Cromie aortic valve. J Thorax Cardiovasc Surg 73:64, I977

[77] Sanoudos G, Reed GE: Late heart block in aortic valve replacement. J Cardiovasc Surg (Torino) 15:475, 1974

[78] Anderson RP. Bonchek LI. Wood J, et al: The safety of combined aortic valve replacement and coronary bypass grafting. Ann Thorax Surg I5:249. 1973

[79] Thompson R, Ahmed M, Seabra-Gomes R, et al: Influence of preoperative left ventricular function on results of homograft replacement of the aortic valve for aortic regurgitation. J Thorax Cardiovasc Surg 77:41 I, 1979

[80] Forman R, Firth BG, Barnard MS: Prognostic significance of preoperative left ventricular ejection fraction and valve lesion in patients with aortic valve replacement. Am J Cardiol 45: I 120, 1980

[81] Bonchek LI: Current status of cardiac valve replacement: Selection of a prosthesis and indications of operation, Am Heart J 101:96, 198 I

[82] Jacobs ML. Fowler BN, Vezeridis MP, et al: Aortic valve replacement: A nine-year experience. Ann Thorax Surg 30:439, 1980

[83] McHenry MM: Smeloff EA, Davey TB, et al: Hemodynamic results with full-flow orifice prosthetic valves. Circulation 35/36 (Suppl I): 24, 1967

[84] Starr A, Grunkemeier GL, Lambert LE, et al: Aortic valve replacement: A ten-year follow-up of noncloth-covered vs. clot-covered caged- ball prostheses. Circulation 56 (Suppl 2):133, 1977

[85] Bloodwell RD, Okies JE, Hallman GL, et al: Aortic valve replacement: Long-term results. J Thorax Cardiovasc Surg 58:457, 1969

[86] Brodeur MTH, Sutherland DW, Koler RD. et al: Red blood cell survival in patients with aortic valvular disease and ball valve prostheses. Circulation 32:570, 1965

[87] Seans AD, Crosby WH: Intravascular hemolysis due to intracardiac prosthetic devices: Diurnal variations related to activity. Am J Med 39:34 I, 1965

[88] DeCesare W, Rath C, Hufnagel CA: Hemolytic ane- mia of mechanical origin with aortic-valve prostheses. N Engl J Med 272:1045, 1965

[89] Walsh JR, Starr A, Ritzman LW: Intravascular hemolysis in patients with prosthetic valves and valvular heart disease. Circulation 39/40 (Suppl 1):135, 1969

[90] Wallace HW, Kenepp DL, Blakemore WS: Quantitation of red blood cell destruction associated with valvular disease and prosthetic valves. J Thorax Cardiovasc Surg 60:842, 1970

[91] Bonchek LI, Anderson RP, Starr A: Mitral valve replacement with cloth-covered, composite seat prostheses: The case for early operation. J Thorax Cardiovasc Surg 67:93, 1974

[92] Appelbaum A, Kouchoukos NT, Blackstone EH: Early risks of open heart surgery for mitral valve disease. Am J Cardiol 37:201, 1976

[93] Allen WB, Karp RB, Kouchoukos NT: Mitral valve replacement. Arch Surg 109:642, 1974

[94] Morrow AG, Oldham HN, Elkins RC, et al: Prosthetic replacement of the mitral valve: Preoperative and postoperative clinical and hemodynamic assessments in 100 patients. Circulation 35:962, 1967

[95] Kloster FE, Herr RH, Starr A, et al: Hemodynamic evaluation of a cloth-covered Starr-Edwards valve prosthesis. Circulation 39/40 (Suppl I):I 19, 1969

[96] Winter TQ, Reis RL. Glancy DL, et al: Current status of Starr-Edwards cloth-covered prosthetic cardiac valves. Circulation 45/46 (Suppl 1):14. 1972

[97] Starr A, Grunkemeier GL, Lambert L, et al: Mitral valve replacement: A ten-year follow-up of non-cloth-covered vs. cloth-covered caged-ball prostheses. Circulation 54 (Suppl 3):47, 1976

[98] Fishman NH, Edmunds LH, Hutchinson JC, et al: Five-year experience with the Smeloff-Cutter mitral prosthe- sis. J Thorax Cardiovasc Surg 62:345, 1971

[99] Behendt DM, Austen WG: Current status of pros- thetics for heart valve replacement. Prog Cardiovasc Dis 15:369, 1973

[100] Roberts WC, Morrow AG: Secondary left ventricular endocardial fibroelastosis following mitral valve replacement: Cause of cardiac failure in the late postoperative period. Circulation 37/38 (Suppl 2):101, 1968

[101] Schryer MJP, Karauchow PN: Endocardial fibro- elastosis: Etiologic and pathogenetic considerations in children. Am Heart J 88:557. 1974

[102] Fishbein MC, Ferrans VJ, Roberts WC: Histologic and ultrastructural features of primary and secondary endocardial fibroelastosis. Arch Pathol Lab Med lOl:49, 1977

[103] Steele A, Tsao M, Finlayson M: Endocardial fibroelastosis after replacement of mitral valves with tilting disc prostheses. Arch Pathol Lab Med 104:422, 1980

[104] Macmanus Q, Grunkemeier GL, Lambert LE, et al: Year of operation as a risk factor in the late results of valve replacement. J Thorax Cardiovasc Surg 80:834-841, 1980

[105] Dale J, Myhre E, Storstein 0, et al: Prevention of arterial thrombocmbolism with acetylsalicyclic acid: A con- trolled clinical study in patients with aortic ball valves. Am Heart J 94:101, 1977

[106] Dale J, Myhre E: Can acetylsalicyclic acid alone prevent arterial thromboembolism? A pilot study in patients with aortic ball valve prostheses. Acta Med Stand (Suppl 645):73, 1981

[107] brott WH, Zajtchuk R, Bowen TE, et al: Dipyridamole-aspirin as thrombormbolic prophylaxis in patients with aortic valve prosthesis. Prospective study with the model 2320 starr-edwards prosthesis. J thorac cardiovascs surg 81:632,1981.

[108] Carmen R, Kahn P: In vitro testing of silicone rubber heart valve poppets for lipid absorption. J Biomed Mater Res 2:457, 1968

[109] Hjelm M, Hogma CF. Finnson M, et al: Transient autoantibody formation in a case of open heart surgery with no signs of increased red-cell destruction. VOX Sang 9:505, 1964

[110] Pirofskey B, Sutherland DW, Starr A, et al: Hemolytic anemia complicating aortic valve surgery: An autoimmune syndrome. N Engl J Med 272:235,1965

[111] Stein PD, Harken DE, Dexter L: The nature and prevention of prosthetic valve endocarditis. Am Heart J 71:393, 1966

[112] Killen DA, Collins HA, Koenig MG, et al: Prosthetic cardiac valves and bacterial endocarditis. Ann Thorax Surg 9:238, 1970

[113] Block PC, DeSanctis RW, Weinberg AN, et al: Prosthetic valve endocarditis. J Thorax Cardiovasc Surg 60540, 1970

[114] Slaughter L, Morris JF, Starr A: Prosthetic valvular endocarditis. Circulation 47: I3 19, 1973.

[115] Dismukes WE, Karchmer AW, Buckley MJ, et al: Prosthetic valve endocarditis. Circulation 48:365, 1973

[116] Weinstein L, Rubin RH: Infective endocarditis- 1973. Prog Cardiovasc Dis 16:239, 1973

[117] Kloster FE: Diagnosis and management of complications of prosthetic valves. Am J Cardiol 35:872, 1975

[118] Wilson WR, Jaumin PM, Danielson GK. et Prosthetic valve endocarditis. Ann Intern Med 82:75 1, 1975

[119] Madison J, Wang K, Gobel FL, et al: Prosthetic aortic valvular endocarditis. Circulation 51:940, 1975

[120] Karchmer AW, Dismukes WE, Buckley MJ, et al: Late prosthetic valve endocarditis: Clinical features influencing therapy. Am J Med 64:199, 1978

[121] Arnett EN, Roberts WC: Prosthetic valve endocarditis: Clinicopathologic analysis of 22 necropsy patients with comparison of observations in 74 necropsy patients with active infective endocarditis involving natural left-sided cardiac valves. Am J Cardiol 38:281, 1976

[122] Arnett EN, Roberts WC: Valve ring abscess in active infective endocarditis: Frequency, location, and clues to clinical diagnosis from the study of 95 necropsy patients. Circulation 54:140, 1976

[123] Watanakunakorn C: Prosthetic valve infective endocarditis. Prog Cardiovasc Dis 22:181, 1979

[124] Harford CJ: Postoperative fungal endocarditis. Arch Intern Med 134: 116, 1974

[125] Norenberg RG, Sethi GK, Scott SM, et al: Opportunistic endocarditis following open heart surgery. Ann Thorax Surg 19:592, 1975

[126] Turnier E, Kay JH, Bernstein S, et al: Surgical treatment of candida endocarditis. Chest 67:262, 1975

[127] Rubenstein E, Noriega ER, Simberkoff MS: Fungal endocarditis. Medicine 54:331, 1975

[128] Roberts WC, Morrow AG: Mechanisms of acute left atrial thrombosis after mitral valve-replacement: Pathologic findings indicating obstruction to left atrial opening. Am J Cardiol 18:497, 1966

[129] Roberts WC, Morrow AG: Causes of early postoperative death following cardiac valve replacement: Clinicopathologic correlations in 64 patients studied at necropsy. J Thorax Cardiovasc Surg 54422, 1967

[130] Robicsek F, Sanger PW, Daugherty HK, et al: Fatal arrhythmias caused by the pressure of the ball-valve prosthesis upon the left ventricular myocardium. Dis Chest 52:813, 1967

[131] Kalke B, Korns ME, Goott B, et al: Engagement of ventricular myocardium by open-cage atrioventricular valvular prosthesis. J Thorax Cardiovasc Surg 58:92, 1969

[132] Ibarra-perez C, rodriguez-trujillo F,PEREZREDONDO H:Engagement of ventricular myocardium by struts of mitral prosthesis: fatal complications of use of open-cage cardiac valves.J thorac cardiovascs surg 61:403,1971

[133] Quattlebaum FW, Kalke B, Edwards JE, et al: Obstruction of the aorta by prosthetic aortic valve. J Thorax Cardiovasc Surg 55:23 1, 1968

[134] Seningen RP, Bulkley BH, Roberts WC: Prosthetic aortic stenosis: A method to prevent its occurrence by measurement of aortic size from preoperative aortogram. Circulation 49:921, 1974

[135] Roberts WC, Lambird PA, Gott VL, et al: Fatal aortic regurgitation following replacement of the mitral and aortic valves: A mechanical complication of double valve replacement. J Thorac Cardiovasc Surg 52: 189, 1966

[136] Tatooles CJ, Braunwald NS, Morrow AG: Prosthetic replacement of the tricuspid valve. Surg Forum 16:171, 1965

[137] Grondin P, Lepage G, Costonguay Y, et al: The tricuspid valve: A surgical challenge. J Thorax Cardiovasc Surg 53:7, 1967

[138] VanderVeer JB, Rhyneer GS, Hodam RP, et al: Obstruction of tricuspid ball-valve prostheses. Circulation 43/44 (Suppl 1):62, 1971

[139] Beall AC, Morris GC, Noon GP, et al: An improved mitral valve prosthesis. Ann Thorax Surg 15:25, 1973

[140] Cooley DA, Okies JE, Wukasch DC, et al: Ten-year experience with cardiac valve replacement: Results with a new mitral prosthesis. Ann Surg 177:818, 1973

[141] Beall AC, Bricker DL, Messmer BJ: Results of mitral valve replacement with Dacron velour-covered Teflon-disc prosthesis. Ann Thorax Surg 9:195, 1970

[142] Javier RP, Hilkner FJ, Berry W, et al: Systemic emvolism and the beall mitral valve prothesis . Ann thorac surg 10:20,1970

[143] Nicholas HT, Fernandez J, Morse D, et al: Improved results in 336 patients with the isolated mitral Beall valve replacement. Chest 62:266, 1972

[144] Cooley DA, Sandiford FM, Wukasch DC, et al: Mitral valve replacement with a new prosthesis: Experience with 366 patients. J Cardiovasc Surg I5 (Suppl):378, 1974

[145] Kay EB, Suzuki A, Tambi A, et al: Comparative analysis of the Starr-Edwards ball valve and Kay-Suzuki disc valve and transplant valves in the surgical correction of aortic valve diseases. Am J Cardiol 25:535, 1970

[146] Bjork VO, Olin C, Astrom H: Results of aortic valve replacement with the Kay-Shiley disc valve. Stand J Thorax Cardiovasc Surg 3:93, 1969

[147] Linhart JW, Barold SS,Hildner FJ, et al : Clinical and hemodynamics findings following replacement of the mitral valve with a beall valve prosthesis (Dacron velour covered Teflon disc valve). Circulation 39/40 (suppl 1):127,1969

[148] Vogel JRK, Paten BC, Overy HR, et al: Abnormal hemodynamic function after disc mitral valve replacement. Circulation 39/40 (Suppl 1):141, 1969

[149] Ramsey RW, Williams JC Jr, Vernon CR: Hemodynamic findings following replacement of the mitral valve with the Beall valve prosthesis. J Thorax Cardiovasc Surg 62:624, 1971

[150] Reid JA, Stevens TW, Sigwart V, et al: Hemodynamic evaluation of the Beall mitral valve prosthesis. Circulation 45/46 (Suppl. I): I. 1972

[151] Shepherd RL, Glancy DL, Reis RC, et al: Hemodynamic function of the Kay-Shiley prosthetic cardiac valve. Chest 63:323, 1973

[152] Hawe A, Frye RL , Ellis FH Jr: Late hemodynamic studies after mitral valve surgery .J thorac cardiovasc surg 65:351:1973 hology after valve replacement by disc prosthesis: A study of 61 necropsy patients. Am J Cardiol 35:740, 1975

[153] Fernandez J, Morse D, Spagna P, et al: Results of mitral valve replacement with the Beall prosthesis in 209 patients. J Thorax Surg 71:218, 1976

[154] Cross FS, Jones RD: Caged-lens prosthesis for replacement of mitral and aortic valves. Circulation 32 (Suppl 2):72, 1965

[155] Edgett J W, Nelson WP, Hall RJ, et al: A complication of valve replacement by a caged-lens prosthesis. Circulation 36:422, 1967

[156] Hammermeister KE. Dillard DH, Kennedy JW: Severe, intermittent mitral regurgitation in a Cross-Jones valve: Report of a case with angiographic and hemodynamic observations. J Thorax Cardiovasc Surg 58:575, 1969

[157] Paton BC: Reoperation for fibrinous stenosis of disc mitral valve prosthesis. J Thorac Cardiovasc Surg 57:726, 1969

[158] Demaney MA, Zimmerman HA: Thrombosis of a mitral disc-valve prosthesis: Diagnostic importance of the absent opening click. Am Heart J 80:816, 1970

[159] Mallen DC, Shull WH, Daugherty HK, et al: Emer- gency thrombectomy of the prosthetic mitral valve. Vas Surg 4:137,1970

[160] Kay EB, Suzuki A, Demaney M, et al: Surgical correction of rheumatic valvular disease: Late survival stud- ies with the Kay-Suzuki disc valve. Ann Thorax Surg 13: 110, 1972.

[161] Beall AC Jr: Late results with cardiac valve replacement: Reduction of thromboembolic complications of mitral valve replacement. J Cardiovasc Surg 13:261, 1972

[162] Beall AC Jr, Morris GC, Howell JF Jr, et al: Clinical experience with an improved mitral valve prosthesis. Ann Thorax Surg 15:601, 1973

[163] Wellons BA, Strauch RS, Nolan SP, et al: Isolated mitral valve replacement with the Kay-Shiley disc valve: Structural analysis of the long-term results. J Thorax Cardio- vast Surg 70:862, 1975

[164] Edmiston WA, Harrison EC, Betista E, et al: Clini- cal experience with the Kay-Shiley mitral valve prosthesis: An eleven-year follow-up study. Stand J Thorax Cardiovasc Surg 14:241, 1980

[165] Carey JS, Hughes RR: A complication of mitral- valve replacement with caged-lens prosthesis. Ann Thorax Surg 6177. 1968

[166] Hopeman AR, Treasure RL, Hall RJ: Mechanical dysfunction in caged-lens prostheses. J Thorax Cardiovasc Surg 60:51, 1970

[167] Robinson MJ, Hildner FJ, Greenberg JJ: Disc vari- ance of Beall mitral valve. Ann Thorax Surg I I : I I, 197 I

[168] Detmer DE, McIntosh CL, Boretos JW, et al: Poly- propylene poppets for low-profile prosthetic heart valves. Ann Thorax Surg 13: 122, 1972

[169] Buroya RB, Escano FB Jr: Mitral disc valve variance. Thorax 27:87, 1972

[170] Law HBC, Lefmine AA: Acute mitral insufficiency due to jamming of a disc-valve prosthesis. Ann Thorax Surg 4:7l. 1970

[171] Vasko JS, Leighton RF: Acute massive mitral regur- gitation resulting from disc-valve malfunction. Ann Thorax Surg 6:564, 1968

[172] Hammer WJ, Hearne MJ, Roberts WC: Cocking of a poppet disc prosthesis in the aortic position: A cause of intermittent aortic regurgitation. J Thorac Cardiovasc Surg 71:259. 1976

[173] Nathan MJ: Strut fracture: A late complication of Beall mitral valve replacement. Ann Thorax Surg 16:610, 1973

[174] Beall AC Jr, discussion of Starek FJK, et al: Clinical evaluation of the Lillehei-Kaster pivoting-disc valve. Ann Thorax Surg 22:362, 1976

[175] Williams JC Jr, Vernon CR, Dacoff GR, et al: Hemolysis following mitral valve replacement with the Beall valve prosthesis. J Thorax Cardiovasc Surg 61:393, 1975

[176] Clark RE, Grubbs FL, McKnight RC, et al: Late clinical problems with Beall model 103 and 104 mitral valve prostheses. Hemolysis and valve wear. Ann Thorax Surg 211475, 1976

[177] Vogel JHK, Paton BC, Overy HR, et al: Advantages of the Beall valve prosthesis. Chest 59:249, 1971

[178] Bjork VO: Experience with the Wada-Cutter valve prosthesis in the aortic area: One year follow-up. J Thorax Cardiovasc Surg 60:26, 1970

[179] Roe BB, Fishman NH, Hutchinson JC, et al Occluder disruption of Wada-Cutter valve prosthesis. Ann Thorax Surg 20:256, 1975

[180] Bjork VO, Ovenfers CO: The disc rotation in patients operated upon with the Bjork-Shiley tilting disc valves with radiopaque markers. Stand J Thorax Cardiovasc Surg 5:83, 1971

[181] Bjork VO: Disc wear in the Bjork-Shiley tilting disc valve: Experimental and clinical observations. Stand J Tho- rat Cardiovasc Surg 5:87, 1971

[182] Bjork VO: Delrin as implant material for valve occluders . Scand J thorac cardiovasc surg 6:103,1972

[183] Bjork VO : The pyrolytic carbon occlude for the Bjork-shiley tilting disc valve . J thorac cardiovasc surg 78:331,1979 .

[184] Bjork VO, Henze A: Ten years experience with the Bjork-Shiley tilting disc valve. J Thorax Cardiovasc Surg 78:331,1979

[185] Aberg B, Henze A: Comparison between the in vitro flow dynamics of the standard and the convexo-concave Bjork-Shiley tilting disc valve prostheses. Stand J Thorax Cardiovasc Surg 13: 177, 1979

[186] Nitter-Hauge S, Enge I, Sembe BK, et al: Primary clinical experience with the Hall-Kaster valve in the aortic position. Results at 3 months including hemodynamic stud- ies. Circulation 60 (Suppl 1):55, 1979

[187] Sembe BK, Nitter-Hauge S, Hall KV: The Hall- Kaster disc valve prosthesis. Clinical and hemodynamic observations in patients undergoing aortic valve replacement. J Cardiovasc Surg (Torino) 21:387, 1980

[188] Emery RW, Nicoloff HM: St. Jude Medical cardiac valve prosthesis: In vitro studies. J Thorax Cardiovasc Surg 781269, 1979

[189] Henze A, Carlsson S, Bjork VO: Mortality and pathology following aortic valve replacement with the Bjork-Shiley tilting disc valve. Stand J Thorax Surg 7:7, 1973

[190] Lepley D, Flemma RJ, Mullen DC, et al: Late evaluation of patients undergoing valve replacement with the Bjork-Shiley prosthesis. Ann Thorax Surg 24: 13 1, 1977

[191] Lillehei CW: Worldwide results with the St. Jude cardiac prosthesis. St. Jude Medical, Inc: Paris, First European Symposium, 1980, p 49

[192] Hehrlein FW, Gottwik M, Mulch J, et al: Heart valve replacement with the new all pyrolytic bi-leaflet St. Jude Medical prosthesis. J Cardiovasc Surg (Torino) 21:395, 1980

[193] Chaux A, Gray RJ, Matloff JM, et al: An apprecia- tion of the new St. Jude valvular prosthesis. J Thorax Cardiovasc Surg 8 1:202, 198 I

[194] Nicoloff DM, Emery RW, Arom KV, et al: Clinical and hemodynamic results with the St. Jude Medical cardiac valve prosthesis. A three-year experience. J Thorac Cardio- vast Surg 82:674, 198 1

[195] Bjork VO, Holmgren A, Olin C, et al: Clinical and hemodynamic results of aortic valve replacement with the Bjork-Shiley tilting disc valve prosthesis. Stand J Thorax Cardiovasc Surg 5:177, 1971

[196] Bjork VO, Henze A, Holmgren A: Central hemody- namics at rest and during exercise before and after aortic valve replacement with the Bjork-Shiley tilting disc valve in patients with isolated aortic stenosis. Stand J Thorax Cardio- vast Surg 7:111, 1973

[197] Bjork VO, Book K, Cernigliaro C, et al: The Bjork- Shiley tilting disc valve in isolated mitral lesions. Stand J Thorax Cardiovasc Surg 7: 13 1, 1973

[198] Schaff HV, Borken AM, Hughes C, et al: Clinical and hemodynamic evaluation of the 19 mm Bjork-Shiley aortic valve prosthesis. Ann Thorax Surg 32:50, 1981

[199] Lillehei CW, Kaster RL, Starek PJ, et al: A new central flow pivoting disc aortic and mitral prosthesis: Initial clinical experience. Am J Cardiol 26:688, 1970

[200] Pyle RB, Mayer JE, Lindsay WC, et al: Hemody- namic evaluation of Lillehei-Kaster and Starr-Edwards pros-theses. Ann Thorax Surg 26:336, 1978

[201] Horstkotte D, Haerten K, Herzer JA, et al: Prelimi- nary results in mitral valve replacement with St. Jude Medi- cal prosthesis: Comparison with the Bjork-Shiley valve. Cir- culation 64 (Suppl 2):203, 1981

[202] Gabbay S, McQueen DM, Yellin FI, et al: In vitro hydrodynamic comparison of mitral valve prostheses at high flow rates. J Thorac Cardiovasc Surg 76:77 1. 1978

[203] Gabbay S, McQueen DM, Yellin EL, et al: In vitro hydrodynamic comparison of mitral valve bioprostheses. Cir-culation 60 (Supp12):62, 1979

[204] Bjork OV, Olin 0: A hemodynamic evaluation of the new tilting disc valve (Bjork-Shiley) for mitral valve replace-ment. Stand J Thorax Cardiovasc Surg 4:37, 1970

[205] Bokros J, Cott V, LaGrange L, et al: Heparin absorptivity and blood compatibility of carbon surfaces. J Biomed Mat Res 4:145, 1970

[206] Bjork VO, Henze A: Encapsulation of the Bjork- Shiley aortic disc valve caused by the lack of anticoagulation treatment. Stand J Thorax Surg 7:17, 1973

[207] Byrd CC, Yahr WZ, Greenberg JJ: Long-term results of “simple” thrombectomy for thrombosed Bjork- Shiley aortic valve prostheses. Ann Thorax Surg 20:265, 1975

[208] Roberts WC, Hammer WJ: Cardiac pathology after valve replacement with a tilting disc prosthesis (Bjork-Shiley type): A study of 46 necropsy patients and 49 Bjork-Shiley prostheses. Am J Cardiol 37:1024, 1976

[209] Karp RB, Cyrus RJ, Blackstone EH, et al: The Bjork-Shiley valve: Intermediate-term follow-up. J Thorax Cardiovasc Surg 81:602, 1981

[210] Messmer BJ, Rothlin M,Senning A: Early disc dislodgement : An unusual complications after insertion of a Bjork-shiley mitral valve prosthesis . J thorac cardiovascs surg 65:386,1973.

[211] Slater SD, Sallam IA, Bain WH, et al: Haemolysis with Bjork-Shiley A comparative study. Thorax 29:624,1974

[212] Emery Clinical and hemodynamic results with the St. Jude Medical aortic valve prosthesis. Surg Forum 30:235, 1979

[213] Rao KM, Learoyd PA, Rao RS, et al: Chronic haemolysis after Lillehei-Kaster valve replacement. Compar- ison with the findings after Bjork-Shiley and Starr-Edwards mitral valve replacement. Thorax 35:290, 1980

[214] Sauvage LR: Prosthetic valves 1977: A retrospective analysis and a look to the future. Med Instrum 11:107, 1977

[215] Williams DB, Pluth JR, Orszulak TA: Extrinsic obstruction of the Bjork-Shiley valve in the mitral position. Ann Thorac Surg 32:58, 1981

[216] Nelson RJ , Mohri H, Winterscheid LC, et al : Early clinical experience with homotransplantation oft he aortic valve . circulation 35/36 (suppl 1):9, 1967

[217] Hoeksema TD, Titus JL, Giukiani ER, et al: early results of use of hemografts for replacement of the aortic valve in man . Circulation 35/36 (suppl 1):9,1967 .

[218] Angel1 WW, Iben AB, Shumway NB: Fresh aortic homografts for multiple valve replacement. Arch Surg 97~826, I968

[219] Carpentier A, Blondeau P: Mitral and tricuspid valve replacement with frame-mounted aortic heterografts. J Tho-rat Cardiovasc Surg 56:388, 1968

[220] Buch WS, Kosek JC, Angel1 WW: Deterioration of formalin treated aortic valve heterografts. J Thorax Cardiovast Surg 60:673, 1970

[221] Carpentier A, Deloche A, Relland J, et al: Six-year follow-up of glutaraldehyde-preserved heterografts. J Thorax Cardiovasc Surg 68:77 1, 1974

[222] Reis RL, Hancock WD, Yarbrough JW, et al: The flexible stent: A new concept in the fabrication of tissue heart valve prostheses. J Thorax Cardiovasc Surg 62:683, 1971

[223] Isom CW, Culliford A, Colvin S, et al: Porcine valves: Is there a difference? Circulation 62 (Suppl 3):156, 1980

[224] Levine FH, Carter JE, Buckley MJ, et al: Hemodynamic evaluation of Hancock and Carpentier Edwards bio-prostheses. Circulation 64 (Suppl 2):192, 1981

[225] Rossiter SJ, Miller DC, Stinson ER, et al: Hemodynamic and clinical comparison of the Hancock modified orifice and standard orifice bioprostheses in the aortic posi- tion. J Thorax Cardiovasc Surg 80:54, 1980

[226] Craver JM, King SB, Douglas JS, et al: Late hemodynamic evaluation of Hancock modified orifice aortic bio-prosthesis. Circulation 60 (Suppl 1):93, 1979

[227] Zusman DR, Levine FH, Carter JE, et al: Hemodynamic and clinical evaluation of the Hancock modified orifice aortic bio prosthesis. Circulation 64 (Suppl 2):189, 1981

[228] Bartek IT, Holden MP, lonescu Ml: Frame-mounted tissue heart valves: Technique of construction. Thorax 29:51, I974

[229] Ionescu MI, Tandon AP, Maury DA, et al: Heart valve replacement with the lonescu-Shiley pericardial xeno-graft. J Thorax Cardiovasc Surg 73:3 1, 1977

[230] Becker RM, Strom J, Frishman W, et al: Hemodynamic performance of the Ionescu-Shiley valve prosthesis. J Thorax Cardiovasc Surg 80:613, 1980

[231] Tandon AP, Whitaker W, Ionescu Ml: Multiple valve replacement with pericardial xenograft. Clinical and haemodynamic study. Br Heart J 44:534, 1980

[232] Angel1 WW, Angel1 JD: Porcine Valves. Prog Cardiovasc Dis 23: 141, 1980

[233] Davilla JG, Magilligan DJ, Lewis JW: Is the Han- cock porcine valve the best cardiac valve substitute today? Ann Thorax Surg 26:203, 1978

[234] Rivera R, Infantes C, Delean JL, et al: Clinical and hemodynamic assessment of the Angell-Shiley porcine xeno- graft. Ann Thorax Surg 30:455, 1980

[235] Febres-Roman PR, Bourg WC, Crone RA, et al: Chronic intravascular hemolysis after aortic valve replace- ment with Ionescu-Shiley xenograft: Comparative study with Bjork-Shiley prosthesis. Am J Cardiol46:735, 1980

[236] Horowitz MS, Goodman DJ, Fogarty TJ, et al: Mitral valve replacement with the glutaraldehyde-preserved porcine heterograft: Clinical, hemodynamic, and patholog- ical correlations. J Thorax Cardiovasc Surg 67:885, 1974

[237] Johnson AD, Daily PO, Peterson KL, et al: Functional evaluation of the porcine heterograft in the mitral position. Circulation 51/52 (Suppl 1):40, 1975

[238] Hannah E, Reis KL: Current status of porcine heterograft prostheses. Circulation 53/54 (Suppl 3):27, 1976

[239] Stinson 99. Griepp RB, Oyer PE, et al: Long term experience with porcine aortic valve xenografts. J Thorax Cardiovasc Surg 73:54, 1977

[240] Jamieson WR, Janus MT, Minagishima RT, et al: Embolic complications of porcine heterograft cardiac valves. J Thorax Cardiovasc Surg 8 1:626, 198 1

[241] Geha AS, Holter AR, Langou RA, et al: Dysfunction and thromboembolism associated with cardiac valve xenografts in adults. Circulation 64 (Suppl 2):172, I98 1

[242] Platt MR. Mills LJ, Estrera AS, et al: Marked thrombosis and calcification of porcine heterograft valves. Circulation 62:862, 1980

[243] Oyer PE, Stinson EB, Reitz BA, et al: Long term evaluation of the porcine xenograft bio prosthesis. J Thorax Cardiovasc Surg 78:343, 1979

[244] Cohn LH, Mudge GH, Pratter F, et al: Five to eight year follow-up of patients undergoing porcine heart-valve replacement. N Engl J Med 304:258, 198 1

[245] Magilligan DJ Jr, Lewin JW Jr, Jara FM, et al: Spontaneous degeneration of porcine bioprosthetic valves. Ann Thorax Surg 30:259, 1980

[246] Oyer PE, Miller DC, Stinson EB, et al: Clinical durability of the Hancock porcine bioprosthetic valve. J Thorax Cardiovasc Surg 80:824, 1980

[247] Lipson LC, Kent KM, Rosins DR, et al: Long-term hemodynamic assessment of the porcine heterograft in the mitral position. Late development of valvular stenosis. Circu- lation 64:397. 1981

[248] Spray TL, Roberts WC: Structural changes in porcine xenografts used as substitute cardiac valves: Gross and histologic observations in 5 I glutaraldehyde preserved Han- cock valves in 41 patients. Am J Cardiol40:3 19, 1977

[249] Broom ND: Fatigue-induced damage in glutaraldehyde-preserved heart tissue. J Thorax Cardiovasc Surg 761202, 1978

[250] Barratt-Boyes BG: Cardiothoracic surgery in the Antipodes. Including a report of a randomized trial of medical and surgical treatment of asymptomatic patients with severe coronary artery disease; a long-term follow-up of both fresh and antibiotic treated hemograft valves; and some observations on glutaraldehyde preserved valve tissue. J Thorax Cardiovasc Surg 78:804, 1979

[251] Imamura E, Wada J: Open aortic bio prosthesis at zero pressure. A new concept in glutaraldehyde fixation of tissue valve. J Cardiovasc Surg (Torino) 21:6 17, 1980

[252] Rose AG, Forman R, Bowen RM: Calcification of glutaraldehyde-fixed porcine xenograft. Thorax 33:111, 1978

[253] Kutsche LM, Oyer PE, Shumway N, et al: An important complication of Hancock mitral valve replacement in children. Circulation 60 (Suppl 1):98, 1979

[254] Geha AS, Stansel RC Jr, Cornhill JF, et al: Late failure of porcine valve heterografts in children. J Thorax Cardiovasc Surg 7835 1, 1979

[255] Silver MM, Pollock J, Silver MD, et al: Calcification in porcine xenograft valves in children. Am J Cardiol45:685, 1980

[256] Thandroyen FT, Whitton IN, Pirie D, et al: Severe calcification of glutaraldehyde preserved porcine xenografts in children. Am J Cardiol45:690, 1980

[257] Sanders SP, Levy RJ. Freed MD, et al: Use of Hancock porcine xenografts in children and adolescents. Am J Cardiol 45:429, 1980

[258] Ferrans VJ, Boyce SW ,Billinham ME, et al : calcific deposits . Am J Calcific deposits in porcine bioprostheses : structure and pathogenesis . Am J cardiol 46:721,1980 .

[259] curcio CA , commerford PJ, Rose AG , et al : calcifications of glutaraldehyde-preserved precine xenografts in young patients . J thorac cardiovascs surg 81:621,1981

[260] Rocchini AP, Weesner KM, Heidelberger K, et al : procine xenograft valve failure in children . An immunologic response . Circulation 64 (suppl 2):162,1981 .

[261] Rossiter SJ, Stinson EB. Oyer PE, et al: Prosthetic valve endocarditis: Comparison of heterograft tissue valves and mechanical valves. J Thorax Cardiovasc Surg 76:795, 1978

[262] Williams EH, Conti VR, Nishimura A, et al: Early calcific stenosis of aortic and mitral Ionescu-Shiley valves in a patient with bioprosthetic infection. J Thorax Cardiovasc Surg 82:391, 1981

[263] Tyras DH, Kaiser CC, Barner HB: Atypical myco- bacteria and the xenograft valve. J Thorax Cardiovasc Surg 75:331, 1978

[264] O'Rourke MF, Shanahan MX, Harkness JL: Endo- carditis with an acid-fast organism after porcine heart valve replacement. Lancet 2:686, 1978

[265] Fishbain MC, Gissen SA, Collins JJ Jr, et al: Patho- logic findings after cardiac valve replacement with glutaral-dehyde-fixed porcine valves. Am J Cardiol40:331, 1977

[266] Tandon AP, Thameur HM, lonescu MI: The small aortic root and the lonescu-Shiley pericardial xenograft. Circulation 62 (Suppl 3):155, 1980

APPENDIX - A

List of Table:

Table 1: Brief Chronology of Prosthetic valve Development

1953	Hufnagel	Caged ball valve	Descending aorta
1956	Murray	Tissue valve homograft	Descending aorta
1962	Harken Starr	Caged ball valve Caged ball valve	AVR* MVR*
1965	Ross Duran Gunning Barratt-Boyes	Tissue valve homograft	AVR
1966	Smeloff	Double cage ball valve	AVR AND MVR
1967	Wada Bjork	Tilting dics valve	AVR AND MVR
1971	Braunwald	Cloth-Covered caged ball valve	NVR
1977	Hall	Rod-huige tilting dics valve	AVR

l AVR = aortic valve replacement valve replacement (Subcoronary) ; MVR = mitral valve replacment .

Table 2: Characteristics of a good prosthetic cardiac Valve

Good hemodynamic characteristics (non-obstructive) (Competent)
Nonthrombogenic
Does not generate
Does not significantly alter blood components
Can be inserted without undue difficulty
Does not disturb the patient
A low incidence and diminished severity of late prosthetic endocarditis
Anatomic suitability of the valve to its implanted location

Table 3: Prosthetic Valve Hemodynamics Mitral Position

Valve	No. Subjects studied	invetigator	Rest Exercise
Starr-Edwards 6120	20	Winter et al .[96]	5.2
Star-edwards 6300	12	Kloster et al.[95]	9.4
Star-edwards 6310	21	Winter et al.[96]	5.4
Star-edwards 6310	6	Hodam et al.[143]	4.9
Smeloff-cutter	7	Hawe et al.[143]	6 12
Smeloff-cutter	8	McHenry et al.[83]	6 15
Braunwald-cutter	8	O'Rourke et al.[96]	4.8