# The surgical management of aortic valve disease

JOANNA CHIKWE, AXEL WALTHER, JOHN PEPPER

**Abstract**

**We summarise the natural history and pathophysiology of aortic stenosis and regurgitation, the indications for surgery, the advantages and disadvantages of tissue, mechanical, homograft and autograft aortic valve replacement, and the prediction of operative mortality for individual patients.**

**Key words:** aortic valve, stenosis, regurgitation, surgery.

*Br J Cardiol* 2003;**10**:453–61

## Introduction

The prognosis of patients with valvular heart disease has improved dramatically in the last 15 years through major advances in a number of areas. These include our understanding of the natural course of valvular disease processes, monitoring of valvular and myocardial function, prosthetic valve technology, and the development of a large body of work which has helped to establish the optimum timing and nature of surgical intervention.

Without intervention all valvular heart disease eventually leads to the common end point of biventricular overload and the associated clinical features of congestive cardiac failure and an increased risk of sudden death. As for any operation, the key question is whether the predicted mortality and morbidity of surgery is less than that of the untreated lesion. This article describes the surgical approach to aortic stenosis and aortic regurgitation, and explains how a knowledge of the natural course of the lesion, together with improvements in our ability to predict operative risk for groups of patients, enable a surgeon to decide in whom, how and when to operate.

## Aortic stenosis

### Natural history

Aortic stenosis is the commonest acquired valvular lesion with a prevalence of 1–2% in the over 65s.[1] A bicuspid aortic valve is a risk factor for aortic stenosis and is present in up to 2% of the UK population. Calcific aortic stenosis, the commonest cause of aortic stenosis, shares the predisposing factors of coronary artery disease: age, male sex and hypercholesterolaemia.[2]

Major haemodynamic compromise does not occur until the aortic valve size is reduced to less than half the normal aortic valve area of 3–4 cm$^2$ (table 1) but, beyond this, left ventricular outlet obstruction rapidly increases.[3] Pressure overload of the left ventricle gradually leads to concentric hypertrophy of the ventricular wall. While this compensatory mechanism results in generation of the high interventricular pressures required to maintain cardiac output through the stenosis, there are a number of associated maladaptive features. Thickening of the ventricular wall and increased collagen content lead to diastolic dysfunction, with systolic dysfunction occurring eventually as a result of excess afterload and decreased contractility.[4,5]

Patients with aortic stenosis therefore present with the symptoms of congestive cardiac failure, angina and syncope. Angina occurs because of decreased coronary flow reserve and increased myocardial oxygen demand in a hypertrophic ventricle under increased afterload. The cause of exertional syncope is less well understood; it is thought to be due to the decrease in peripheral resistance observed during exercise in these patients combined with fixed left ventricular outflow.[6,7]

**Table 1.** Peak gradients across stenotic valve

| Aortic valve area (cm$^2$) | Mean gradient (mmHg) |
|---|---|
| 4 | 1.7 |
| 3 | 2.9 |
| 2 | 6.6 |
| 1 | 26 |
| 0.9 | 32 |
| 0.8 | 41 |
| 0.7 | 53 |
| 0.6 | 73 |
| 0.5 | 105 |

### Timing of surgery

*Symptomatic aortic stenosis*

The onset of symptoms in the patient with aortic stenosis is a poor prognostic sign: without valve replacement half of those presenting with angina will die within five years, half of those presenting with syncope will die within three years, and half of

Department of Cardiac Surgery, Royal Brompton Hospital, Sydney Street, London, SW3 6NP.
**Joanna Chikwe,** Specialist Registrar
**John Pepper,** Consultant Surgeon, Professor of Surgery
Royal Marsden Hospital, Fulham Road, London, SW3 6JJ.
**Axel Walther,** Specialist Registrar
**Correspondence to:** Dr J Chikwe
**(email: j.chikwe@medschl.cam.ac.uk)**

those presenting with dyspnoea will be dead within two years.[8] It is accepted that these symptoms are due to aortic stenosis if the valve area is less than 1 $cm^2$ or the gradient across the valve is greater than 50 mmHg, and this situation mandates prompt aortic valve replacement.[9]

There is a subgroup of symptomatic patients with transvalvular gradients of less than 30 mmHg. This arises, not because the aortic stenosis is mild, but as a result of left ventricular failure in the setting of severe aortic stenosis. These patients have severely depressed myocardial contractility and their prognosis after aortic valve replacement is extremely poor: less than 50% are alive four years after surgery.[10-12] The patients that survive beyond this may be those in whom the primary cause of the left ventricular dysfunction is excessive afterload, rather than irreversible structural changes. If interventricular pressures and, hence, transvalvular gradient increase in response to inotropic stimulation, it suggests that there is a true flow-limiting lesion in the setting of reversible ventricular dysfunction.[13] This is the principle of the dobutamine stress echo.[12] In the severely and irreversibly damaged ventricle there is no increase in transvalvular flow velocities in response to inotropes, and these patients are thought to make up the bulk of the post-operative mortality.[14,15]

*Asymptomatic aortic stenosis*

The management of the asymptomatic patient with severe aortic stenosis is less straightforward because, although the majority of such patients have almost normal life expectancy without a valve replacement, there is a 1–2% incidence of sudden death in this group.[16] The operative mortality and long-term morbidity of aortic valve replacement is, however, higher than this, making it important to select only those patients who are at high risk of sudden death without surgery.

Two techniques are employed routinely to identify high-risk asymptomatic patients: echocardiography and exercise testing. Asymptomatic patients with a peak gradient across the aortic valve of 64 mmHg or above, measured by transthoracic echocardiography, were shown in a recent series to have a 70% chance of becoming symptomatic within two years.[17] Although exercise testing is contraindicated in patients with symptomatic aortic stenosis, it has been shown to be safe in asymptomatic patients and can identify the presence of exercise-induced haemodynamic compromise, which is a relative indication for elective valve surgery.[18] There is no literature on the natural history of the disease process in asymptomatic patients with a positive exercise test but it is probably very similar to that of patients presenting with exertional angina or dyspnoea.

## Aortic regurgitation

### Natural history

Aortic regurgitation is caused by dilation of the aortic root, or damage to the aortic valve leaflets, both of which may prevent leaflet coaptation. The latter process is most commonly caused by rheumatic valvular disease and infective endocarditis; the former results from collagen connective tissue disorders such as Marfan's syndrome and annulo-aortic ectasia, and infective endocarditis.

In chronic aortic regurgitation, left ventricular enlargement produces a large total stroke volume, all of which enters the aorta during ventricular systole. The resultant increase in pulse pressure produces systemic hypertension and large increases in left ventricular afterload.[15] Chronic, progressive pressure and volume overload of the left ventricle eventually lead to left ventricular dilatation and systolic dysfunction. Patients frequently remain asymptomatic until there is significant myocardial damage, finally presenting with dyspnoea, orthopnoea, fatigue and, occasionally, angina pectoris.

Acute aortic regurgitation, which is most frequently caused by infective endocarditis, results in pulmonary oedema because filling pressure is suddenly increased in a non-compliant left ventricle. Severe regurgitation means that cardiac output is immediately reduced, in contrast with the increase in cardiac output seen in chronic aortic regurgitation. This, together with the increase in left ventricular filling pressure, leads to reduced coronary blood flow and end-organ ischaemia.

### Timing of surgery

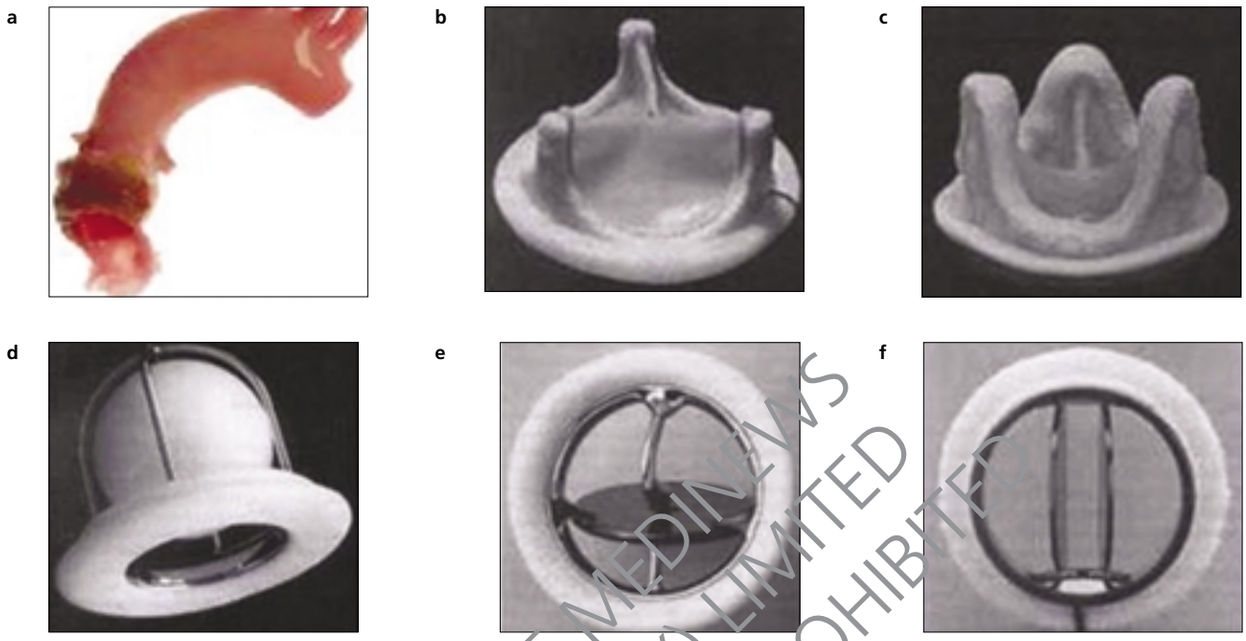
Acute aortic regurgitation is a surgical emergency. In the setting of infective endocarditis, the indications for early operation rather than prolonged intravenous antibiotics are the presence of septic emboli, growing vegetations or haemodynamic instability.[19] In chronic aortic regurgitation, the consensus opinion is that surgical intervention should take place before permanent left ventricular dysfunction results: this means that surgery is not infrequently indicated in asymptomatic patients. Aortic valve replacement should, therefore, be carried out before the ejection fraction falls below 55%, or the left ventricle dilates to more than 5.5 cm in systole.[20]

## Aortic valve replacement

Aortic valve replacement is the definitive intervention in severe aortic stenosis and regurgitation. A brief flirtation with percutaneous balloon aortic valvuloplasty for aortic stenosis during the 1980s was unsuccessful – it only requires a brief look at a typically stenosed, calcified valve to explain why this was the case. Radial fractures of the thickened leaflets led either to incomplete relief of the stenosis, or to the creation of severe regurgitant lesions and fragments liable to embolise down the coronary, cerebral or celiac circulation.

Replacement aortic valves can be divided into tissue valves and mechanical valves. Tissue valves can be: autografts, where the patient's own pulmonary valve is dissected out and used to replace the aortic valve; homografts, where valves are harvested from donor hearts and preserved; or xenografts, which are made of porcine or bovine valvular or pericardial tissue. Autografts and homografts take the form of an aortic valve, *in situ* within the intact aortic root, complete with coronary ostia and a fringe of myocardium (figure 1a). Xenografts, which resemble the aortic leaflets in isolation, are usually mounted on a small metal support, or stent (figure 1b and 1c), but a new generation of stent-

**Figure 1.** Examples of replacement aortic valves: **a**) shows an aortic homograft, **b**) and **c**) show a xenograft, **d**) shows a ball and cage valve, **e**) shows a tilting-disk valve, **f**) shows a bi-leaflet valve






less bioprostheses are undergoing evaluation. Mechanical valves are now composed of carbon, or occasionally metal alloys, and are classified according to their structure into caged-ball (figure 1d) and tilting-disk valves, which may be single (figure 1e) or bi-leaflet (figure 1f).[21]

The perfect replacement valve would have a structural lifetime longer than the patient, an intrinsic gradient of 0 mmHg, a haemodynamic profile of the original valve, carry no infective or thrombotic risk, be cheap, readily available and technically easy to insert, and silent. Over 80 models of prosthetic heart valve have been developed since the 1950s, and the fact that scores are currently in use underlines the fact that no one design fulfils all the criteria of the perfect valve replacement.[21] There are advantages that can be exploited by each valve type in certain groups of patients – outside these groups it is likely that there is no long-term difference in outcome between patients with mechanical and those with biological valves.

## Mechanical valves

Mechanical valves are durable, lasting – with the exception of a few rare technical failures – for 20 to 30 years. Their main drawbacks include high transvalvular pressure gradients in the smaller sizes, an abnormal haemodynamic profile, thrombogenicity, infection rates, and haemolysis. A significant minority of patients find that the noise of the functioning mechanical valve is their greatest drawback.

In patients with small aortic roots the intrinsic transvalvular gradient for a mechanical valve is higher than that of an equivalently sized bioprosthesis (table 2).[22] In this situation, a replacement mechanical valve would offer an unacceptably high resistance to flow.[23] The effects on the left ventricle of changing the mobile three-dimensional structure of the aortic annulus to a fixed, two-dimensional structure is poorly understood.

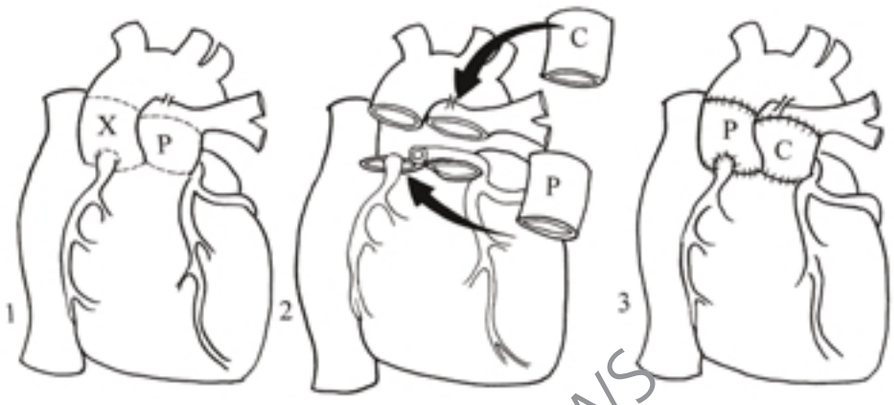
**Table 2.** Peak gradients across prosthetic 21 mm valves

| Valve type | Valve name | Systolic left ventricular gradient (mmHg) | |
|---|---|---|---|
| | | Rest | Exercise |
| Ball and cage | Starr-Edwards 6120 | 11 | 63 |
| Tilting disc | Medtronic Hall | | 31 |
| Stented bioprosthesis | Carpentier Edwards | 13 | 16 |
| Stented bioprosthesis | Medtronic Hancock modified orifice bioprosthesis | 9 | 18 |

Adapted from: Kirklin JW, Barret-Boyes BG.[22]

In an adequately anticoagulated patient (International Normalised Ratio [INR] 2.5–3), the incidence of valve thrombosis is the same as for a bioprosthetic valve: 0.2–5.7% per patient-year.[24] Patients with prosthetic valve thrombosis may present with pulmonary oedema, poor peripheral perfusion and systemic embolisation but, more commonly, the scenario is acute haemo-

**Figure 2.** The Ross procedure. The diseased aortic valve and a segment of the aortic artery (X) are removed. A segment of the pulmonary artery containing the pulmonary valve (P) is excised and then placed in the aortic position, replacing the diseased tissue. A cadaveric pulmonary valve (C) is then placed in the pulmonary position




dynamic compromise. Thrombi of less than 5 mm that are not obstructing the valve orifice or mechanism may be treated with formal anticoagulation alone but larger thrombi require fibrinolysis or valve replacement.

Mechanical valves may be at higher risk of infection than tissue valves during the first three post-operative months but infection rates converge and are similar at five years.[19] The risk of a prosthetic valve infection is up to 3.1% at one year and 5.7% at five years.[21] Skin commensals such as *Staphylococcus epidermidis* and *S. aureus* are usually identified as the cause of peri-operative prosthetic endocarditis – associated mortality is between 30 and 80%.[19] Late prosthetic endocarditis, which is caused by the same organisms that are responsible for native valve endocarditis, i.e. streptococci, has an associated mortality of between 20 and 40%. Patients with valve obstruction, new or deteriorating heart failure, conduction abnormalities, septic emboli, myocardial abscess or persistent bacteraemia despite intravenous antibiotics, should be managed with prompt valve replacement, rather than four to six weeks of intravenous antibiotics which effects a cure in 50% of patients presenting with uncomplicated streptococcal infections. Prevention is imperative. Compliance with advice regarding antibiotic prophylaxis and dental care is essential for patients with prosthetic heart valves.

## Tissue valves

Some 10–20% of homograft and 30% of xenograft valve replacements fail within 10–15 years of implantation and require replacement.[25,26] Tissue valves last longer in the elderly population because the haemodynamic demands on the valve are less: the freedom from operation for homograft valve failure is 47% in patients aged 0–20 years, 85% in the 21–60 years age group, and 94% in patients aged over 60 years of age.[26] Tissue valves last much longer in the aortic position compared to the mitral (the mitral valve must remain closed against peak ventricular systolic pressures, whereas the aortic valve opens passively during ventricular systole), which is why tissue valves are rarely, if ever, used in the mitral position. Tissue valves do, however, have certain features that make them ideal for aortic valve replacement in selected patients.

Tissue valves are far less thrombogenic than mechanical valves and anticoagulation is not required. Unless an elderly patient is already undergoing anticoagulation for some pre-existing condition, a tissue valve would be the prosthesis of choice as it affords 10–15 years of warfarin-free life. Similarly, in patients with bleeding diathesis, who could not be maintained safely on warfarin, most surgeons would select a bioprosthesis over a mechanical valve replacement. Pregnant women have an increased risk of prosthetic valve-related thromboembolic complications and adequate anticoagulant therapy is particularly important in this group if a mechanical valve has been implanted. Warfarin is contraindicated during the first trimester as it is teratogenic – twice-daily subcutaneous heparin or intravenous heparin should be administered instead of warfarin from initial attempts to conceive up until either the beginning of the second trimester or until delivery. The understandable preference of many women is, therefore, for a bioprosthetic valve. Where the expertise is available, a homograft, which offers the longest lifespan, may be the preferred option.

Homografts carry a small risk of infection linked to donor and storage mechanisms, although this is offset many times over by their low susceptibility to post-operative prosthetic endocarditis. As a result, these are the valve replacement of choice in fulminant bacterial endocarditis.

The increased longevity and reduced incidence of prosthetic endocarditis in homografts compared to xenograft valves begs the question of why they are not more frequently used. Harvesting, selecting and preparing homografts requires a degree of expertise and resources that are not available to many centres. Implantation is technically more demanding, requiring experience in positioning the homograft in the correct anatomi-

cal position, dissecting out the coronary ostia and re-implanting them, and additional suture lines. All of these carry an increased risk of post-operative bleeding and prolong the time for which the patient is on bypass, leading to an increased post-operative incidence of renal failure, pulmonary dysfunction, myocardial dysfunction and neurological sequelae. In inexperienced hands, early and late complications render this a less favourable option.

## Pulmonary autograft

Using the patient's own pulmonary valve to replace the aortic valve is known as the Ross procedure, after Sir Donald Ross, the British surgeon who pioneered its use. The pulmonary valve is replaced with a pulmonary homograft, which has a life expectancy of 20–30 years[27] (figure 2). The patient's own pulmonary valve in the aortic position has a similar life expectancy, is resistant to infection and does not warrant formal anticoagulation. The drawbacks are primarily related to the technical difficulty of both procedure and reoperation. Many more surgeons have been discouraged by the steep learning curve and abandoned the operation, than those who have managed to successfully adopt it as part of their repertoire.

## Operative risk

The operative mortality associated with aortic valve replacement varies between 2% for an otherwise fit patient in the elective setting, to over 30% for a combined or emergency procedure in a patient with multiple co-morbidity. This is a useful point to introduce a powerful tool used by cardiac surgeons to predict mortality of a given procedure for a particular patient. The EuroSCORE system is one of a number of scoring systems derived from studies of large populations of patients (table 3). With the knowledge of 12 clinical variables such as age, sex, serum creatinine and left ventricular dysfunction, a percentage operative risk can be quoted for that patient, which has been shown to be accurate in all but the highest risk cases where mortality is generally underestimated. The best operative mortality for aortic stenosis according to EuroSCORE would be approximately 3%. UK registry data give the mortality for isolated aortic valve replacement at 2.2% for mechanical valves, 4% for bioprosthetic valves and 5% for homograft replacement.[29] This may reflect the higher proportion of young, low-risk patients undergoing mechanical valve replacement. Surgeon-specific data in the UK suggest that variability between individual surgeons accounts for less than 0.4%.[29] The operative risk of a mechanical valve replacement is similar to that for a tissue valve. The Ross procedure carries an increased risk of up to 7.4%.[30] It is also apparent from EuroSCORE that concomitant coronary artery bypass grafting caries a substantial additional risk.[31]

The risk of stroke (because of emboli from the calcified valve, the aortic cannulation site, hypoperfusion or haemorrhage) is 3% in patients without other risk factors for cerebrovascular events. In addition to the problems with post-operative management described above, patients are warned that aortic valve replacement carries a risk of conduction abnormalities requiring antidysrhythmics, or occasionally permanent pacemaker insertion. The other main peri-operative complications are – as for any open heart surgery – chest infection, pleural effusion, post-operative haemorrhage requiring resternotomy, wound infection which may require further surgery, and acute renal failure.

**Table 3.** The EuroSCORE (European system for Cardiac Operative Risk Evaluation Score). Points add up to an approximate percentage predicted mortality risk

| Factor | Definition | Score |
|---|---|---|
| Age | Per 5 years or part thereof over 60 years | 1 |
| Gender | Female | 1 |
| COPD | Long-term bronchodilators or steroids for lung disease | 2 |
| Arteriopathy | Any one or more of: claudication, carotid occlusion or > 50% stenosis, previous or planned surgery on abdominal aorta, limb arteries or carotids | 2 |
| Neurological | Disease severely affecting ambulation or activity | 2 |
| Previous cardiac surgery | Previous surgery requiring opening of pericardium | 3 |
| Serum creatinine | > 200 μmol $l^{-1}$ pre-operatively | 3 |
| Active endocarditis | Patient still under antibiotic treatment for endocarditis | 3 |
| Critical pre-operative state | Ventilation before arrival in anaesthetic room, preop inotropes, IABP, acute renal failure | 3 |
| Unstable angina | Angina requiring i.v. nitrates until operation | 2 |
| LV dysfunction | Moderate (ejection fraction 30–50%)<br>Poor (ejection fraction < 30%) | 1<br>3 |
| Recent MI | < 90 days | 2 |
| Pulmonary hypertension | Systolic PA pressure > 60 mmHg | 2 |
| Emergency | Carried out on referral within 24 hours | 2 |
| Other than CABG | Major cardiac operation other than or in addition to CABG | 2 |
| Surgery on thoracic aorta | Ascending, arch or descending aorta | 3 |
| Post-infarct septal rupture | | 4 |

**Key:** COPD = chronic obstructive pulmonary disease; LV = left ventricular; IABP = intra-aortic balloon pump; MI = myocardial infarction; CABG = coronary artery bypass graft; PA = pulmonary artery

Adapted from Nashef SAM *et al*.[28]

## Summary

Aortic valve replacement is the definitive therapy in severe aortic stenosis and regurgitation. This reflects the grim natural history of these lesions, the disastrous consequences of alternative, less invasive options such as balloon valvotomy, and the comparatively low mortality and morbidity associated with surgery.

### Key messages

- Symptomatic aortic stenosis (AS) has a poor prognosis: the two-year survival of dyspnoeic patients with AS is less than 50%
- Aortic valve replacement (AVR) is the definitive therapy in symptomatic AS and severe aortic regurgitation
- UK mortality rates for first time surgery range from 2.2% for mechanical AVR up to 5.0% for homograft AVR. Concomitant CABG increases these risks from 4.6 to 23.5% respectively
- Aortic valve replacement for aortic stenosis improves outcome even in high-risk patients
- Tissue valves are indicated in patients over 65 years of age, and for those with contraindications to permanent anticoagulation
- Mechanical valves are indicated for younger patients, but lifelong warfarinisation is required. Increasing experience with aortic homografts and the Ross procedure are making these a viable alternative to mechanical aortic valves in younger patients

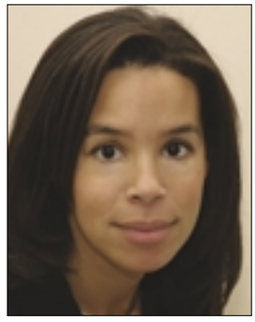
Advanced age and significant comorbidity have not been shown to be contraindications to surgery because aortic valve replacement is likely to improve outcome even in high-risk patients. The choice between mechanical and tissue valve in the elective setting is dictated by balancing the risks of long-term anticoagulation against the risks of re-do surgery for structural valve degeneration: a tissue valve should be selected for patients over 65 years of age, and those with contraindications to permanent anticoagulation. Increasing experience with aortic homografts and the Ross procedure are making these a viable alternative to mechanical aortic valves in younger patients. While minimal access, self-stapling devices currently look attractive in normal animal models, the technical challenge presented by the diseased human aortic valve strongly suggests that conventional surgery will remain the definitive treatment for many years to come.



## References


1. Otto CM. Aortic stenosis - listen to the patients, look at the valve. *N Engl J Med* 2000;**343**:652-4.
2. Carabello BA, Crawford FA, Jr. Valvular heart disease. *N Engl J Med* 1997;**337**:32-41.
3. Passik CS, Ackermann DM, Pluth JR, Edwards WD. Temporal changes in the causes of aortic stenosis: a surgical pathologic study of 646 cases. *Mayo Clin Proc* 1987;**62**:119-23.
4. Marcus ML, Doty DB, Hiratzka LF, Wright CB, Eastham CL. Decreased coronary reserve: a mechanism for angina pectoris in patients with aortic stenosis and normal coronary arteries. *N Engl J Med* 1982;**307**:1362-6.
5. Huber D, Grimm J, Koch R, Krayenbuehl HP. Determinants of ejection performance in aortic stenosis. *Circulation* 1981;**64**:126-34.
6. Schwartz LS, Goldfischer J, Sprague GJ, Schwartz SP. Syncope and sudden death in aortic stenosis. *Am J Cardiol* 1969;**23**:647-58.
7. Richards AM, Nicholls MG, Ikram H, Hamilton EJ, Richards RD. Syncope in aortic valvular stenosis. *Lancet* 1984;**2**:1113-16.
8. Ross J, Jr., Braunwald E. Aortic stenosis. *Circulation* 1968;**38**:61-7.
9. Carabello BA. Timing of valve replacement in aortic stenosis. Moving closer to perfection. *Circulation* 1997;**95**:2241-3.
10. Carabello BA. Ventricular function in aortic stenosis: how low can you go? *J Am Coll Cardiol* 2002;**39**:1364-5.
11. Connolly HM, Oh JK, Schaff HV *et al*. Severe aortic stenosis with low transvalvular gradient and severe left ventricular dysfunction: result of aortic valve replacement in 52 patients. *Circulation* 2000;**101**:1940-6.
12. Lalka SG, Sawada SG, Dalsing MC *et al*. Dobutamine stress echocardiography as a predictor of cardiac events associated with aortic surgery. *J Vasc Surg* 1992;**15**:831-40; discussion 841-2.
13. Carabello BA. Evaluation and management of patients with aortic stenosis. *Circulation* 2002;**105**:1746-50.
14. deFilippi CR, Willett DL, Brickner ME *et al*. Usefulness of dobutamine echocardiography in distinguishing severe from nonsevere valvular aortic stenosis in patients with depressed left ventricular function and low transvalvular gradients. *Am J Cardiol* 1995;**75**:191-4.
15. Taniguchi K, Nakano S, Kawashima Y *et al*. Left ventricular ejection performance, wall stress, and contractile state in aortic regurgitation before and after aortic valve replacement. *Circulation* 1990;**82**:798-807.
16. Rosenhek R, Binder T, Porenta G *et al*. Predictors of outcome in severe, asymptomatic aortic stenosis. *N Engl J Med* 2000;**343**:611-17.
17. Otto CM, Burwash IG, Legget ME *et al*. Prospective study of asymptomatic valvular aortic stenosis. Clinical, echocardiographic, and exercise predictors of outcome. *Circulation* 1997;**95**:2262-70.
18. Linderholm H, Osterman G, Teien D. Detection of coronary artery disease by means of exercise ECG in patients with aortic stenosis. *Acta Med Scand* 1985;**218**:181-8.
19. Mylonakis E, Calderwood SB. Infective endocarditis in adults. *N Engl J Med* 2001;**345**:1318-30.
20. Carabello BA, Usher BW, Hendrix GH, Assey ME, Crawford FA, Leman RB. Predictors of outcome for aortic valve replacement in patients with aortic regurgitation and left ventricular dysfunction: a change in the measuring stick. *J Am Coll Cardiol* 1987;**10**:991-7.
21. Vongpatanasin W, Hillis LD, Lange RA. Prosthetic heart valves. *N Engl J Med* 1996;**335**:407-16.
22. Kirklin JW, Barret-Boyes BG. *Cardiac surgery morphology, diagnostic criteria, natural history, techniques, results and indications.* Volume 2. 2nd edition. Churchill Livingstone, 1993.
23. Pibarot P, Dumesnil JG. Hemodynamic and clinical impact of prosthesis-patient mismatch in the aortic valve position and its prevention. *J Am Coll Cardiol* 2000;**36**:1131-41.
24. Butchart EG, Lewis PA, Grunkemeier GL, Kulatilake N, Breckenridge IM. Low risk of thrombosis and serious embolic events despite low-intensity anticoagulation. Experience with 1,004 Medtronic Hall valves. *Circulation* 1988;**78**:166-77.
25. Hammermeister K, Sethi GK, Henderson WG, Grover FL, Oprian C, Rahimtoola SH. Outcomes 15 years after valve replacement with a mechanical versus a bioprosthetic valve: final report of the Veterans Affairs randomized trial. *J Am Coll Cardiol* 2000;**36**:1152-8.
26. O'Brien MF, Harrocks S, Stafford EG *et al*. The homograft aortic valve: a 29 year, 99.3% follow up of 1022 valve replacements. *J Heart Valve Dis* 2001;**10**:334-44.
27. Oury JH, Hiro SP, Maxwell JM, Lamberti JJ, Duran CM. The Ross Procedure: current registry results. *Ann Thorac Surg* 1998;**66**:S162-S165.
28. Nashef SAM, Roques F, Michael P, Ghauducheau E, Lemeshow S, Salamon R. The EuroSCORE Study Group. European system for cardiac risk evaluation (EuroSCORE). *Eur J Cardiothorac Surg* 1999;**16**:9-13.
29. *The National Audit Cardiac Surgical Database Report 1999-2000*. The Society of Cardiothoracic Surgeons of Great Britain and Ireland.
30. Taggart D. Replacement of the aortic root with a pulmonary autograft. *N Engl J Med* 1994;**330**:1615-16.
31. Cohen G, David TE, Ivanov J, Armstrong S, Feindel CM. The impact of age, coronary artery disease, and cardiac comorbidity on late survival after bioprosthetic aortic valve replacement. *J Thorac Cardiovasc Surg* 1999;**117**:273-84.

# Joanna Chikwe answers frequently asked questions on aortic valve replacement



**When does a surgeon decide which type of valve will be deployed?**
The type of valve replacement will be decided at the initial consultation with the patient, when factors such as the patient's age, co-morbidity, lifestyle and preferences can be taken into account. It is uncommon for this decision to be changed by intra-operative findings.

**As a homograft involves introducing 'foreign' tissue, does this ever or rarely provoke an immune response or cause rejection?**
There is no evidence to show that significant homograft rejection takes place. Modern storage techniques aim to preserve the fibroblasts and endothelial cells that help to maintain a homograft's long-term structural viability, but viable endothelial cells disappear within hours of homograft implantation. Major histocompatibility complex (MHC) antigens, which are the primary target of the immune response in allograft rejection, are therefore not expressed by the graft.

**In the Ross procedure, is the patient's own pulmonary valve – which is used as an aortic valve replacement – replaced by a mechanical valve?**
No. A mechanical valve offers too much resistance to flow to be placed in the low pressure right ventricular outflow tract system. As the patient's entire pulmonary root (annulus, valve leaflets, sinuses and initial portion of the pulmonary artery) has been excised in order to replace their aortic root, the replacement prosthesis of choice is a pulmonary homograft root.

**Is aspirin an effective antithrombotic/anticoagulant in patients with tissue valves who do not require warfarin?**
Yes, and tissue valves are frequently used for this reason in patients who have contraindications to life-long warfarinisation.

**Are warfarin and aspirin ever combined in patients with mechanical valves?**
There is some evidence that high-dose warfarin (target INR 3.0–4.0) combined with low-dose aspirin (75–100 mg) in patients with a left-sided mechanical valve reduces mortality and morbidity from all causes, despite an increased risk of bleeding episodes, but this has not been supported by all studies. In our practice, therefore, patients are not routinely placed on combined therapy.

**How long a duration is an aortic valve operation?**
An uncomplicated aortic valve replacement takes approximately three hours.

**How does the surgeon test the operative result, i.e. what are the procedures for valve testing (function) before chest closure and is intra-operative transoesophageal echocardiography used during most valve replacement operations?**

Intra-operative transoesophageal echocardiography (TOE) is increasingly used prior to chest closure to assess prosthetic aortic valve function and left ventricular function, as well as assessing how completely the heart has been de-aired before coming off cardiopulmonary bypass. Intra-operative TOE is not mandatory, however, because problems with a correctly sited mechanical valve are unusual: for some surgeons the main intra-operative test of the function of the aortic valve is whether the patient weans from bypass satisfactorily. Most patients with left ventricular hypertrophy secondary to aortic stenosis have a poorly compliant (stiff) left ventricle. Adequate filling of the left ventricle is critical in the early post-operative hours and TOE is a very useful guide.

**Is biological glue ever used in valve implantation?**
Good surgical technique means that haemostasis can be achieved without the use of biological glue in most valve implantations. Friable tissue, such as that present in endocarditis and some homografts, may necessitate the use of biological glue over the aortotomy suture line. Glue is rarely used around the valve sewing ring because of the risk of impairing leaflet motion and embolisation.

**What is the authors' opinion of percutaneous valve replacement? Are there occasions when such an operation might be advantageous?**
The complete excision of native leaflets and satisfactory implantation of a prosthesis in the setting of calcific aortic stenosis is challenging even under the excellent view afforded by the conventional open approach. Percutaneous valve replacement, which is more likely to be employed for mitral and pulmonary valve incompetence, may eventually be feasible in aortic regurgitation where the leaflets and annulus are relatively disease free: whether this approach would have comparable outcomes to an open one, is debatable.

**What is the percentage increase in operative risk with aortic valve re-dos?**
The Society of Cardiothoracic Surgeons of Great Britain and Northern Ireland 2001–2002 database shows an additional 0.4% operative mortality for re-do surgery over first-time aortic valve replacement for patients receiving mechanical valves (2.4% and 2.0% respectively), and an additional 5% risk for the cohort receiving tissue valves (9.4% and 4.4% respectively). This reflects the increased age and co-morbidity of patients selected to receive tissue valves. Concurrent coronary artery bypass grafting increases the operative risk to over 15%.

**Is retrograde catheterisation of the aortic valve a common investigation? The potential for neurological complications and silent embolism has been reported. Since the study by Oman *et al*. (*Lancet* 2003), do you think that this invasive investigation would be almost entirely replaced by non-invasive assessment by echocardiography?**
Retrograde catheterisation of the aortic valve is commonly performed during the work-up of patients for surgery. This study showed that patients with calcific aortic stenosis who underwent coronary angiography and retrograde catheterisation of the aortic valve for assessment of trans-valvular gradient had a 3% incidence of neurological deficit post-procedure (99% CI 0–13%) as well as a 22% incidence of clinically silent cerebral events (99% CI 13–30%). This compared unfavourably to a similar group of patients that underwent coronary angiography alone. The main risk of stroke arises when crossing the aortic valve to obtain a ventriculogram and a pull-back gradient. Both pieces of data can be obtained very easily from echocardiography or, in echo-poor subjects, from magnetic resonance imaging (MRI). There is, therefore, no indication for routine retrograde catheterisation of the heavily calcified aortic valve.