Mechanical heart valves. Conclusions from long-term follow-up

S. Nitter-Hauge

Medical Department B, Rikshospitalet, Oslo, Norway

Introduction

In this survey about the long-term follow-up of patients with valvular prosthesis, I have restricted myself to discussing experiences and conclusions that can be drawn from the use of mechanical heart valve prostheses. This is because a mechanical heart valve prosthesis is more durable than a bioprosthesis. A bioprosthesis has a limited life-span and degeneration starts about 6–7 years after implantation. Thus, if one wants to study the long-term course, i.e. more than 10 years after valvular replacement, only cohorts of patients with mechanical prostheses should be included. About 200 000 valves have been implanted world-wide. Mechanical valves are the most commonly used, and have about 70% of the market, although large variations exist between countries.

The Starr-Edwards caged-ball valve prosthesis, introduced in the 1960s, is usually referred to as the 'first' generation of mechanical prostheses in practical use. Investigators soon replaced the ball valve with the 'second' generation of mechanical prosthesis, the so-called low profile mono-leaflet disc valves, which became available in the 1970s, exemplified by the Bjork–Shiley, Lillehei–Kaster and Medtronic–Hall valves. In 1977 low profile bi-leaflet valves, such as the St. Jude Medical, Carbo-Medics and Omni-Science prostheses became available, representing the so-called 'third' generation. Today, five mechanical valve prostheses, representing all three generations, have been approved by the FDA in the United States: Starr–Edwards, Medtronic–Hall, St. Jude Medical, Omni-Science and Carbo-medics. These mechanical heart valve models have a high degree of structural integrity and durability in common. However, they differ in other aspects, above all in haemodynamics and thrombogenicity, both important factors presumed to affect long-term prognosis. These differences will be commented upon more closely, in particular by comparing the first vs the second and third generation valves.

Haemodynamics

It is generally accepted that the first generation of mechanical valves is inferior to the second and third generations, as far as flow profile and haemodynamics are concerned. Based on resting observations, the ball valve is characterized by a relatively high transvalvular pressure gradient, in particular with the smaller valve sizes, where gradients have been reported to be around 13–54 mmHg in the aortic and up to 10 mmHg in the mitral position[11]. In contrast, the mono- as well as the bi-leaflet disc valves are characterized by significantly lower gradients. The gradients are almost zero with the large sized valves and around 12 mmHg in the aortic and 3 mmHg in the mitral position with the smallest sized valves[12]. Furthermore, while exercise in patients with ball valves causes a significant increase in pressure gradients, patients with a mono- or bi-leaflet disc valve can perform moderate exercise without unacceptable increases in pressure gradients, patients with a mono- or bi-leaflet disc valve can perform moderate exercise without unacceptable increases in pressure gradients[13], although relatively high gradients have been reported with the smallest sized valves[14]. Any adverse effects by exercise-induced high pressure gradients across these small sized valves have been difficult to demonstrate, but might have contributed to the somewhat poorer long-term prognosis in this subgroup of patients, compared with those with the large sized valves[15]. With this exception, no-one has been able to demonstrate any significant relationship between the transvalvular pressure gradient in mechanical heart valves in use today and long-term prognosis.

Thrombogenicity

Although degeneration affects biological valves, the main hazard with the mechanical valves is their thrombogenicity. It is agreed that the incidence of thromboembolic complications in patients with the first generation prosthetic valves was high. According to Miller et al.[16], thromboembolic complications with Starr-Edwards mitral valve prostheses was the cause of valve failure in close to 40% of cases, and was fatal in about 50%. The second and third generation valves brought technical improvements in valve design, and
better quality anticoagulation therapy. However, the most significant improvement was as regards the greatly reduced incidence of thromboembolic complications and fatalities from thrombotic obstructions. Mono- and bi-leaflet valves positioned in the aorta have a reported linearized rate of thromboembolic complications of around 1–2%, compared with 4% with ball valves. In patients with a mitral valve prosthesis, the causative factors of thromboembolism are more numerous, and the number of thromboembolic complications therefore tends to be higher than in the aortic position. However, in the mitral position, the incidence of thromboembolic complications have been reported as significantly lower with second and third generation mechanical valves as compared with the ball valves.

As all patients with mechanical valve prostheses need anticoagulation, they are also predisposed to anticoagulation-related bleeding. In deciding the intensity of anticoagulation, the ideal level should be adequate to prevent the formation of thromboembolic as well as bleeding complications. In a 15-year follow-up study with the Medtronic–Hall valve, the incidence of thromboembolic episodes was 1.5% per patient-year in those with an ideal target therapeutic level (between 10% and 5%), which corresponds to an arterial anticoagulation level, as an international normalized ratio, of between 2.8 and 4.8, vs 4.1% per patient-year in those under inadequate therapy. With an ideal therapeutic level, the rate of bleeding was 1.02% per patient-year, compared with 2.18% per patient-year in those under inadequate therapy. This indicates that high-dose anti-coagulation therapy under strict control can prevent thromboembolic complications as well as anticoagulated-related bleeding.

Long-term survival

As can be seen, improved haemodynamics and a reduced incidence of thromboembolic complications can be achieved with the second and third generations of mechanical prostheses. In the light of this, a comparison between the long-term results reported with the various generations of valve models should be of interest. However, it has to be taken into account that if patients with heart valve disease do not undergo an operation, their life span will be limited. From past studies on the ‘natural’ course of disease based on non-operated patients, it is known that after symptoms onset, patients with aortic stenosis have a less than 20% 10-year survival, and in patients with mitral regurgitation/stenosis or aortic regurgitation the 10-year survival varies between 50 and 60%.

The introduction of surgical replacement of a diseased valve with a valve prosthesis caused a dramatic change in the prognosis of patients with heart valve disease, although not to the same extent in all categories of patients. In 1982, Miller and co-workers reported that 5-year survival with the Starr–Edwards mitral valve prosthesis was 71%, and 10-year survival 47%. The survival curve indicated a gradual decrease in survival the first years after operation, a pattern which later turned out to be similar in other long-term survival report. Akins et al. reported that 10-year survival with the Starr–Edwards prosthesis in the aortic position was 61%, and 57% in the mitral position. The Bjork–Shiley standard valve, Lindblom reported that 10-year survival was 66% in the aortic, 58% in the mitral and 57% after double-valve replacement (aortic and mitral). With the Medtronic–Hall valve, 10-year survival was 72% after aortic valve replacement, 56% after mitral and 60% after double-valve replacement. All the reports referred to indicate that survival was better in patients with an aortic prosthesis than in those with a mitral or double-valve prosthesis. Khan et al. recently concluded that with the St. Jude Medical 10-year survival after aortic valve replacement was 43%, after mitral valve replacement 42%, and after double-valve replacement 43%. The data are summarized in Table 1.

There are many comparisons of results of long-term survival studies after valve replacement. The majority will present survival figures similar to those referred to above, and some will show better survival. However, survival after major heart surgery will be largely influenced by many factors, such as indications for surgery, the surgical technique, and the age of the patients under study. Taking all these factors into account, it can be concluded that the data referred to in this overview are representative, and that there is a remarkable similarity between long-term survival data when results with the various generations of mechanical prostheses are compared. When patients are grouped according to their pre-operative valve lesions, it is obvious that those operated on for aortic stenosis have the best long-term prognosis. Figures for ‘relative survival’ indicate that surgery has been close to curative

---

Table 1: Actuarial 10-years survival after valvular replacement with various mechanical heart valve prostheses

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>S-E = Starr-Edwards; M-H = Medtronic-Hall; AVR = aortic valve replacement; B-S = Bjork-Shiley; SJM = St. Jude Medical; MVR = mitral valve replacement.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S-E</td>
<td>S-E</td>
<td>B-S</td>
<td>M-H</td>
<td>SJM</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVR</td>
<td>AVR</td>
<td>47%</td>
<td>57%</td>
<td>58%</td>
<td>54%</td>
<td>42%</td>
<td>43%</td>
</tr>
</tbody>
</table>

Table 1: Actuarial 10-years survival after valvular replacement with various mechanical heart valve prostheses

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>S-E = Starr-Edwards; M-H = Medtronic-Hall; AVR = aortic valve replacement; B-S = Bjork-Shiley; SJM = St. Jude Medical; MVR = mitral valve replacement.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S-E</td>
<td>S-E</td>
<td>B-S</td>
<td>M-H</td>
<td>SJM</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVR</td>
<td>AVR</td>
<td>47%</td>
<td>57%</td>
<td>58%</td>
<td>54%</td>
<td>42%</td>
<td>43%</td>
</tr>
</tbody>
</table>
Table 2 Ultrasound data in two cases with chronic aortic regurgitation

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular end-diastolic diameter</td>
<td>6.4 cm</td>
<td>6.3 cm</td>
</tr>
<tr>
<td>Fractional shortening</td>
<td>35%</td>
<td>40%</td>
</tr>
<tr>
<td>Diastolic retrograde velocity in aorta descendens</td>
<td>0.3 ms⁻¹</td>
<td>0.4 ms⁻¹</td>
</tr>
<tr>
<td>Diastolic pressure half-time</td>
<td>340 ms</td>
<td>160 ms</td>
</tr>
<tr>
<td>IVR</td>
<td>80 ms</td>
<td>20 ms</td>
</tr>
<tr>
<td>Mitral inflow: e-wave</td>
<td>small, broad</td>
<td>large, high, narrow</td>
</tr>
<tr>
<td>e-wave deceleration time</td>
<td>240 ms</td>
<td>120 ms</td>
</tr>
<tr>
<td>a-wave</td>
<td>high, narrow</td>
<td>lacking</td>
</tr>
</tbody>
</table>

IVR = isovolumic relaxation time.

in certain subgroups of patients with aortic stenosis (very young age or age >70 years). Otherwise, our survival data indicate that surgical intervention in patients with valvular heart disease has been more of a palliative than a curative intervention, irrespective of which valve models implanted.

**How to improve our results?**

If we analyse the causes of death in the operated patients, many reports show that the list includes both cardiac and non-cardiac diseases. However, in the early studies of patients with the Starr–Edwards valve⁶⁰, the most important cause, in addition to valve-related malfunction failures, was congestive heart failure accounting for about one third of all early and late deaths. With the introduction of the second and third generation prostheses, valve malfunctions are no longer seen (except for the special problems related to the Björk–Shiley CC model), while congestive heart failure still appears as the dominant cause of death. In my study⁶¹, congestive heart failure was the cause of death in 38% of all late cases, and similar figures are also reported by others. It is unlikely that the operation itself or the prostheses was responsible for this. Thus, the occurrence of congestive heart failure, as a major cause of late death, indicates that significant left ventricular dysfunction developed before the patient was referred for surgery. In the future, we should recommend surgery to our patients at an earlier stage of disease than hitherto practised, and preferably before myocardial failure has developed. The durability and low thrombogenicity of the modern mechanical valve prostheses also justify such a recommendation. Nevertheless, it is still difficult to choose the correct time for surgery in individual patients, at least in some patient subgroups.

Today, the most challenging group of patients are those with chronic aortic regurgitation, known to have an excess mortality in long-term studies. This excess mortality is assumed to reflect left ventricular dysfunction at the time of operation. This has been demonstrated in many studies, when various echocardiographic parameters for left ventricular function have been correlated to long-term survival after valve replacement. In a recent report from Acar et al⁷⁷, patients with pre-operative left ventricular dysfunction (ejection fraction <40%) had a 10-year survival of 54% vs 67% in those with normal left ventricular function while there was no difference at 5 years follow-up.

In addition to determination of left ventricular ejection fraction, the most common routinely used echocardiographic parameters to identify left ventricular dysfunction are the left ventricular end-systolic and end-diastolic diameters, and serial examinations have therefore been recommended. Rather fixed figures for left ventricular dimensions have been established to define the optimal time of surgery in these patients. Pre-operative follow-up registrations with echocardiography in individual patients can often be a long and time-consuming process. This is demonstrated in Fig. 1, which shows how left ventricular end-systolic and end-diastolic diameters can be essentially unchanged during a 13-year observation period in patients with chronic aortic regurgitation.

The question may also be raised as to what extent such serial measurements of left ventricular dimensions provide all the data needed to understand the complexity of left ventricular function in chronic aortic regurgitation. In this connection, I will comment shortly upon observations (personal communication) in two patients with long-standing chronic aortic regurgitation (Table 2). They had identical left ventricular end-diastolic dimensions and identical fraction shortening.
By adding measurements of left ventricular diastolic function, expressed as the diastolic pressure half time, isovolumic relaxation time and mitral inflow velocities, certain differences between the two cases were noticed; in case 1, diastolic pressure half-time and isovolumic relaxation were significantly longer than in case 2; the E-wave was high and narrow in case 1, and was lacking in case 2; and the E-wave deceleration time was twice as long in case 1 as in case 2.

The interpretation of these data is that in case 1 there was a delayed filling of the left ventricle, largely dependent on the atrial contraction. Case 2 showed a more restrictive filling pattern, with a rapid increase in left ventricular pressure. The haemodynamic situation was more serious in case 2 than in case 1. The full meaning of these observations has yet to be evaluated, but may indicate that measurements of various parameters of left ventricular diastolic function, in addition to the determination of left ventricular dimensions, should be taken into account when determining the timing of surgery in patients with chronic aortic regurgitation.

References