Valvular heart disease and heart valve surgery pose unique clinical challenges compared with other cardiovascular diseases. As examples, the treatment of congestive heart failure and acute coronary syndromes have been revolutionized in the past few years by large, multi-center, randomized, placebo-controlled trials with statistically (and clinically) significant end points available in months if not in a few years. Valvular heart disease and heart valve surgery, however, do not lend themselves to similar investigations. Valvular heart disease is far less prevalent and its clinical end points far more indolent; clinical outcomes associated with prosthetic heart valves (PHVs) are of interest decades later, rather than acutely after intervention; and heart valve surgery does not lend itself to randomization. Other than two trials conducted nearly 30 years ago, there are no (and are unlikely to be any) large-scale randomized comparisons between valve prostheses. In addition, advances in valve technology make the determination of valve durability a moving target: by nature, 15-year durability data exist for valves that now may be obsolete. For these reasons, clinicians are forced to draw conclusions and make recommendations based on incomplete information, extrapolating from limited data, clinical experience, and common sense. In short, for PHV choices, randomized data alone are insufficient to dictate clinical practice.

The recent review in the Journal (1) concerning choices in PHVs raises important issues with respect to how physicians should inform patients and recommend therapy when existing data are incomplete or imperfect. The review draws heavily on the randomized trials performed in the late 1970s (2,3). Based on the assessment that outcomes are the same for newer prostheses, the review perpetuates traditional recommendations that tissue valves are appropriate for old patients and mechanical valves are appropriate for all others. But much in medicine has changed in the ensuing decades since these data were derived, raising questions of how to extrapolate data from these trials to modern practice.

**New valves versus old valves.** The Edinburgh heart valve trial (2) was conducted between 1975 and 1979; the Veterans Affairs trial (3) was conducted between 1977 and 1982. In these trials, late structural deterioration occurred more often with bioprostheses, which was felt to limit their usefulness among patients <65 years of age. Because there are no randomized trials comparing valve prostheses that enrolled patients after 1982, the clinician must draw conclusions about old versus new prostheses based on extrapolation of data.

In the decades since these trials were performed, the evolution of tissue technology has resulted in newer bioprostheses with superior durability. For example, the first-generation Hancock MO bioprosthesis (Medtronic, Inc., Minneapolis, Minnesota) was fixed at high pressure using no tissue treatment to mitigate calcification, whereas the second-generation Hancock II bioprosthesis used low-pressure fixation and an early form of tissue anticalcification treatment. The 15-year actuarial freedom from structural valve deterioration was $57 \pm 4\%$ for the first-generation
valve in a population 69 years of age at the time of implantation (4) and 81 ± 5% for the second-generation valve in a population 65 years of age at the time of implantation (5). Although long-term data are lacking for yet newer devices, data from animal studies (6) and early human experience (7–9) suggest that third-generation bioprostheses will be even more durable compared with earlier devices. Significantly, case-controlled studies of newer versus older bioprostheses demonstrate statistically significant survival advantages associated with some newer bioprostheses (10–13).

In addition to advances resulting in greater bioprosthesis durability and lower patient mortality, both tissue and mechanical prostheses have evolved to provide superior hemodynamics compared with older counterparts. For bioprostheses, this includes supra-annular seating and changes in sewing ring shape and strut materials that provide greater orifice area, as well as the development and ongoing refinement of stentless aortic bioprostheses (7,8). Similarly, mechanical prostheses have evolved to provide larger effective orifice area and superior hemodynamics compared with earlier valves (14). Although it remains debatable as to whether hemodynamics impact patient mortality (15,16), there is no question that superior prosthesis hemodynamics minimizes the occurrence of symptomatic prosthesis–patient mismatch. Albeit in the absence of randomized, double-blind trials, there are ample data to suggest that in hemodynamics, bioprosthesis durability, and mortality, good “new” valves are superior to good “old” valves.

Mechanical versus tissue. In the randomized trials between mechanical and tissue valves that were performed in the late 1970s, there was either a trend (2) or statistical significance (3) favoring improved survival in association with a mechanical prosthesis. Freedom from re-operation was superior with a mechanical prosthesis, with higher rates of bioprosthesis failure among patients <65 years of age. However, freedom from all valve-related complications for tissue and mechanical prostheses was indistinguishable at 12 years (Fig. 1) (3). (Morbidity among patients with a bioprosthesis was composed predominantly of late structural failure, whereas morbidity among patients with a mechanical prosthesis occurred largely as the result of a constant, cumulative occurrence of major hemorrhage.) Although physicians concentrate on mortality and freedom from re-operation as dominant end points, patients may be more interested in avoiding all valve-related morbidity after surgery. Using this end point, bioprostheses were superior to mechanical valves prior to 12 years after surgery and were equivalent thereafter. In the decades since these trials were performed, anticoagulation therapy has become more refined, with closer control of anticoagulation raising the possibility that bleeding rates in a similar trial would now be lower. Concurrently, as previously described, freedom from structural valve deterioration is superior for newer compared with older bioprostheses owing to advances in tissue technology that result in mitigation of calcification. Since the randomized trials were performed, there have been changes that favorably affect morbidity and mortality for both tissue and mechanical prostheses. Extrapolating from available data to modern practice, it is likely that freedom from valve-related complications continues to favor bioprostheses early after surgery, that total complications are similar for mechanical and tissue valves late after surgery, and that the curves would likely now superimpose later than 12 years after surgery.

Differences between tissue prostheses. There are no large prospectively randomized comparisons among tissue prostheses, and published comparisons often involved dissimilar populations. In such circumstances, the devil is in the details. The conclusion that pericardial valves have superior durability to porcine tissue valves (1,17) may be flawed when the pericardial valve was studied in a population substantially older than that for the porcine valve and the porcine valve used in the comparison was a first-generation valve no longer in clinical use. As already described, second-generation porcine valves have greater 15-year freedom from structural deterioration compared with first-generation prostheses, and there is reason to believe that there is
incremental improvement associated with third-generation bioprostheses. In short, there are no available data to suggest that modern pericardial bioprostheses have any advantage over modern porcine bioprostheses. To the contrary, data reported in similar populations suggest equivalent actuarial and actual freedom from structural valve deterioration at 15 years for pericardial and porcine bioprostheses (Table 1) (5,18).

**Patient quality of life.** There are ample data to show that, regardless of prosthesis choice, mortality is higher among patients after valve replacement than among age-matched controls. Further, any differences in mortality found among prostheses are of small magnitude and of arguable clinical relevance. As such, in addition to end points of mortality and valve-related morbidity, prosthesis choices should take into consideration the difficult-to-quantify end point of patient quality of life after valve replacement. Individual patients may place different emphasis on mortality, freedom from re-operation, risk of thromboembolism (stroke), risk of anticoagulation-related hemorrhage, and the lifestyle modification required with chronic anticoagulation. When given the chance, some patients choose to accept a near certainty of requiring re-operation 10 to 20 years in the future in exchange for the benefit of avoiding anticoagulation, whereas others opt to minimize the likelihood of re-operation.

As physicians who counsel and help educate our patients before valve surgery and who continue to care for them after, we should allow greater latitude in the options that we present. Simple algorithms that assign patients under an arbitrary age to receive a mechanical prosthesis minimize our role to help optimize patient quality of life. If there is a new “state-of-the-art” for internists and cardiologists in valve surgery and prosthesis choices, it lies in understanding the developments in the field and how these changes can favorably impact our patients’ lives.

**Table 1. 15-Year Freedom From SVD of Porcine and Pericardial Aortic Valve Bioprostheses**

<table>
<thead>
<tr>
<th>Author (Ref)</th>
<th>Valve Prosthesis</th>
<th>Implant Years</th>
<th>n</th>
<th>Patient Age at Implant</th>
<th>Actuarial</th>
<th>Actual</th>
</tr>
</thead>
<tbody>
<tr>
<td>David et al. (5)</td>
<td>Hancock II (porcine)</td>
<td>1982–1994</td>
<td>670</td>
<td>65 ± 12</td>
<td>81 ± 5%</td>
<td>90 ± 3%</td>
</tr>
<tr>
<td>Banbury et al. (18)</td>
<td>Carpentier-Edwards (pericardial)</td>
<td>1981–1984</td>
<td>267</td>
<td>65 ± 12</td>
<td>77%*</td>
<td>90%*</td>
</tr>
</tbody>
</table>

*Standard deviation not reported.
SVD = structural valve deterioration.

**REFERENCES**


