

# MANAGEMENT OF THE SMALL AORTIC ROOT

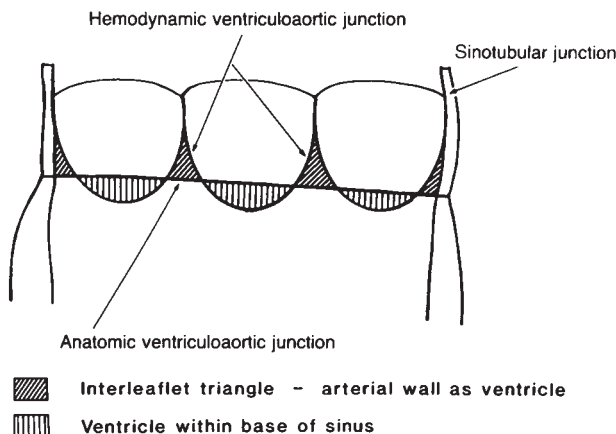
DAVID C. MCGIFFIN, MD, JAMES K. KIRKLIN, MD

The dilemma of the “small aortic root” in patients undergoing aortic valve replacement has permeated the surgical literature for many years. The crux of the issue is the adequacy of the aortic annulus to accept a valve replacement device that would not be “unacceptably obstructive,” implying a resultant reduction in duration of survival and/or exercise capacity. This chapter reviews the anatomy of the “aortic annulus” (an issue of relevance when considering enlargement of the aortic annulus), discusses issues involved in characterizing the small aortic root, and outlines one approach to the small aortic annulus, recognizing that many surgeons have equally justifiable approaches (together with surgical strategies).

## Anatomy of the Aortic “Annulus”

There are three important points:

1. There is an anatomic ventriculoaortic junction that is distinct from the hemodynamic ventriculoaortic junction (Figure 25-1) that is defined by the attachment of the leaflets of the aortic valve.

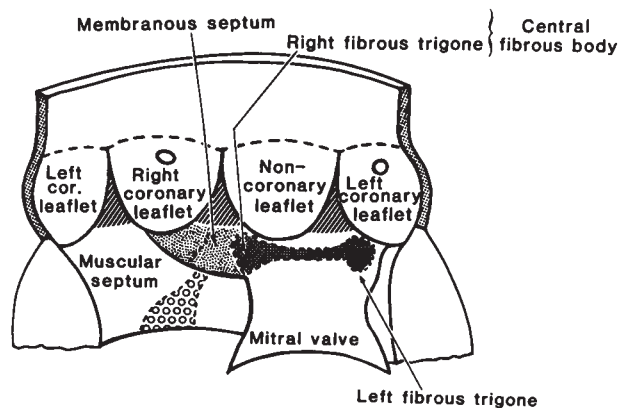


**FIGURE 25-1.** The anatomic and hemodynamic ventriculoaortic junctions are distinct and the hemodynamic ventriculoaortic function is defined by the attachments of the aortic valve leaflets. Reproduced with permission from Anderson RH et al.<sup>1</sup>

2. The impression that the fibroskeleton of the heart is a well-defined structure of the attachment of all four cardiac valves is erroneous.<sup>1</sup> The fibroskeleton really consists of the fibrous continuity between the aortic and mitral valves, each end being thickened to form the left and right fibrous trigones (Figure 25-2), the right fibrous trigone being continuous with the membranous component of the ventricular septum to form the central fibrous body.
3. Although the term *aortic annulus* is embedded in the surgical lexicon, it is the attachment of the aortic valve leaflets to both the aortic wall and left ventricular muscle that precludes the possibility of a true annular ring structure.

## Conditions Associated with a Small Aortic Annulus

The small aortic annulus is most likely to occur in elderly female patients with calcareous aortic stenosis of a trileaflet aortic valve and rheumatic aortic valve disease.



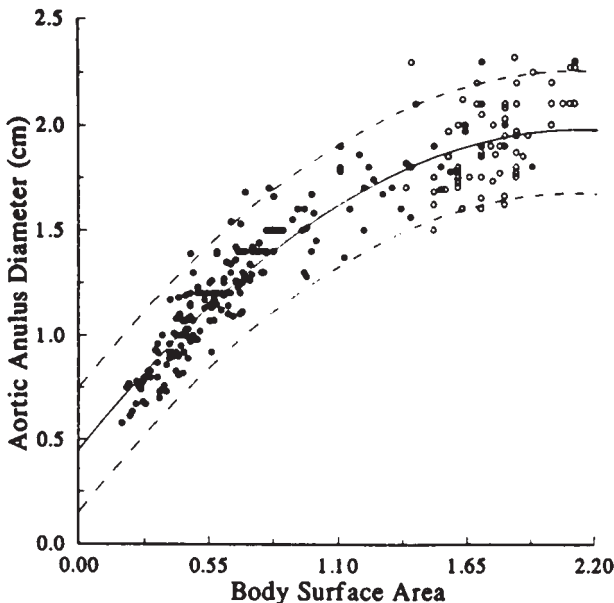
**FIGURE 25-2.** Relationships of the leaflets of the aortic valve to the components of the fibrous skeleton. The hatched areas under the apices of the commissures are a thin layer of arterial wall. Reproduced with permission from Anderson RH et al.<sup>1</sup>

In patients with congenitally bicuspid aortic valve disease the aortic annulus is frequently larger than normal. It is not uncommon for elderly patients with aortic stenosis to have associated subvalvar stenosis due to asymmetric septal hypertrophy.

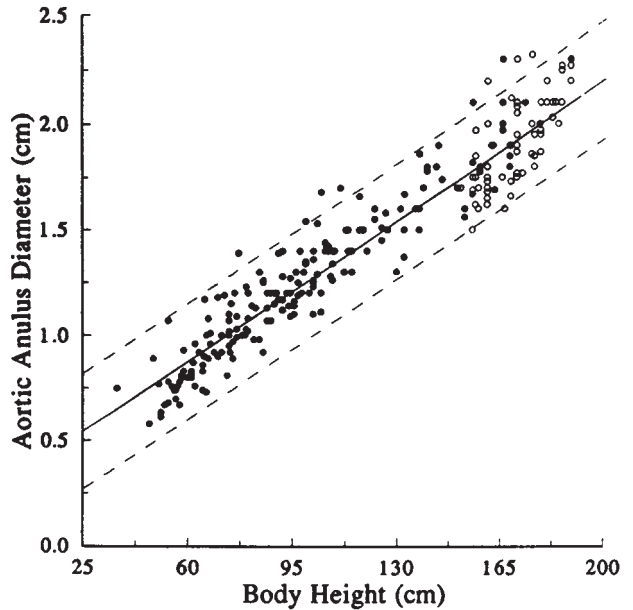
### Issues Inherent in Surgery Associated with a Small Aortic Root

#### The Relationship between Body Size and Aortic Valve Dimension

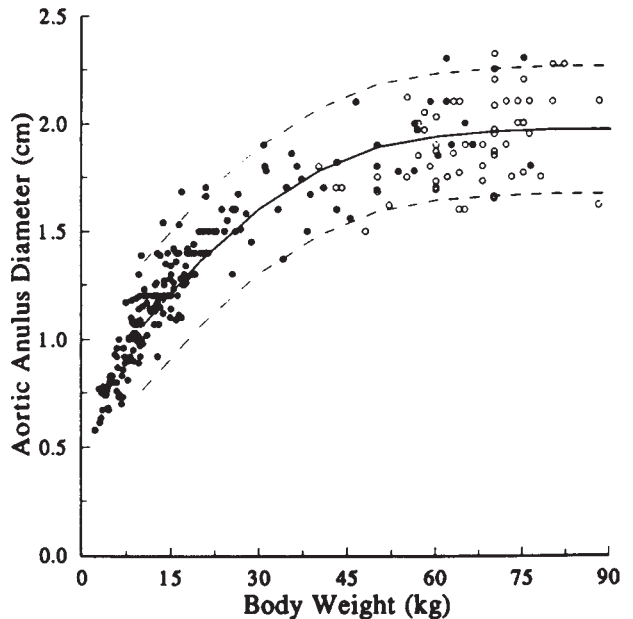
The practice of indexing valve area to body surface area is widely used in the angiographic and echocardiographic assessment of the degree of aortic stenosis to adjust for differences in body size, a technique that is also used for assessing prosthetic valve area. However, the use of body surface area as a means of allowing for difference in body size has been challenged, since the relationship between body surface area and aortic annulus diameter is nonlinear (Figure 25-3).<sup>2</sup> Although the relationship between aortic annulus diameter and body height is linear (Figure 25-4), it is the nonlinearity of the relationship between aortic annulus diameter and body weight (Figure 25-5) that is responsible for the nonlinear relationship between aortic annulus diameter and body surface area.<sup>2</sup> The linear relationship between aortic valve annulus and height suggests that during development, growth of the aortic annulus dimension is primarily due to skeletal growth.<sup>2</sup> With body surface areas greater than 1.0 m<sup>2</sup>, the major determinant of body surface area is weight as opposed to height (Figure 25-6); hence, it could be assumed that beyond a body surface area of 1.5 m<sup>2</sup>, increases in



**FIGURE 25-3.** Relationship between aortic annulus diameter and body surface area. (● = normal subjects younger than 18 years of age; ○ = normal subjects older than 18 years of age.) Reproduced with permission from Nidorf SM et al.<sup>2</sup>

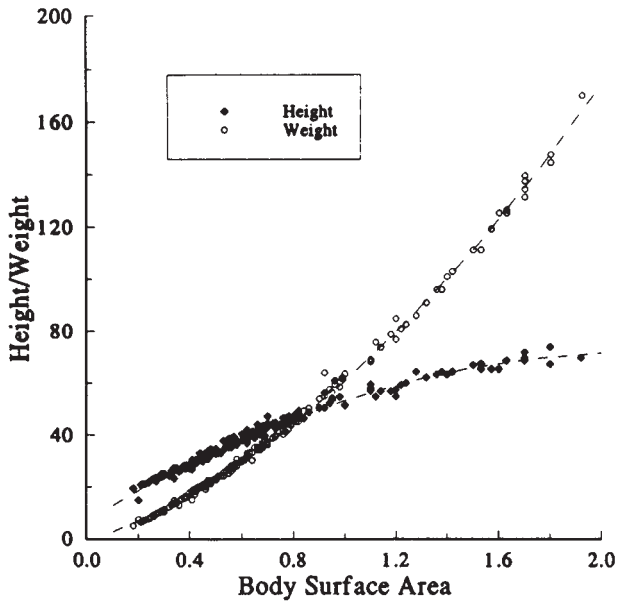


**FIGURE 25-4.** Relationship between aortic annulus diameter and body height. (● = normal subjects younger than 18 years of age; ○ = normal subjects older than 18 years of age.) Reproduced with permission from Nidorf SM et al.<sup>2</sup>



**FIGURE 25-5.** Relationship between aortic annulus diameter and body weight. (● = normal subjects younger than 18 years of age; ○ = normal subjects older than 18 years of age.) Reproduced with permission from Nidorf SM et al.<sup>2</sup>

body surface area due to an increase in weight may not necessarily be associated with increased aortic annulus dimensions.<sup>2</sup> When Figure 25-3 is examined, it appears that beyond a body surface area of 1.5 m<sup>2</sup>, the nonlinearity of the relationship may become important. An example of how this relationship might be important is, for



**FIGURE 25-6.** Relationship between body surface area and height and weight. As body surface area increases beyond 1.0, the major determinant of body surface area is weight rather than height. Reproduced with permission from Nidorf SM et al.<sup>2</sup>

example, in a short, obese patient (with a body surface area of 2.2 m<sup>2</sup>) who has a 21 mm mechanical valve implanted in the aortic position, and about whom there are postoperative concerns about a patient–prosthesis mismatch. If an effective orifice area was determined and then indexed to body surface area, it could be falsely assumed that the prosthesis was too small. This issue may also be of importance in predicting postoperative hemodynamic performance using indexed prosthetic valve area in an individual patient. It should be mentioned that the relationship between aortic valve dimensions measured at autopsy and body surface area is weak, which further suggests that relating valve area to body surface area (“valve area index”) is erroneous, particularly in an attempt to predict the adequacy of a prosthetic device in an individual patient given the wide individual variation in adult aortic valve diameters.<sup>3,4</sup>

**What Is a Small Aortic Root?**

The fundamental problem in the small aortic root is that the size of the device used to replace the aortic valve is inadequate rather than the aortic annulus being too small; consequently, the definition of the small aortic root is rather artificial and should be related to the valve replacement device to be implanted (because the aortic annulus is the size that it needs to be).<sup>5</sup> A small aortic root is implied by the suggestion that a 19 mm valve should not be implanted in a patient with a body surface area of 1.7 m<sup>2</sup>, effective orifice area of less than 1.2 cm<sup>2</sup>/m<sup>2</sup>, or 1.4 cm<sup>2</sup>/m<sup>2</sup>, or where an anticipated orifice area index of a prosthesis is below 0.80 cm<sup>2</sup>/m<sup>2</sup>.<sup>5-9</sup> These are but a few

of the many definitions of a small aortic root, most of which embody the prosthetic valve size of controversy, that is, insertion of a 19 mm or 21 mm mechanical or stented bioprosthesis (Figure 25-7).

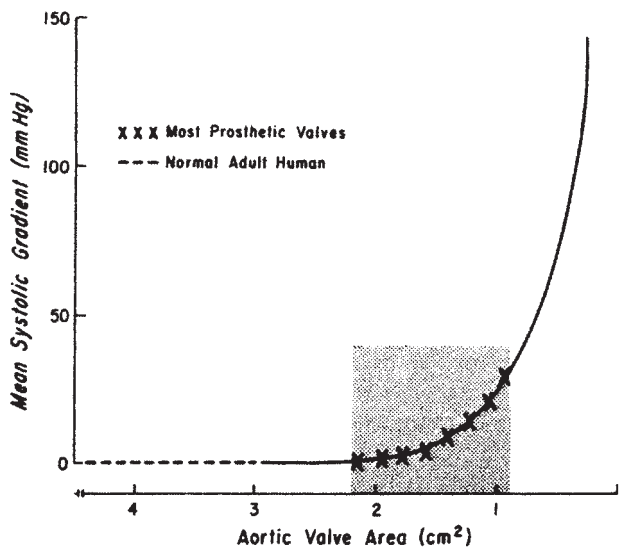
The crux of the issue is that all mechanical and stented bioprostheses have a smaller effective orifice area than that of a normal human aortic valve, and, as pointed out by Rahimtoola, the relationship between aortic valve area and the mean systolic gradient is exponential.<sup>10</sup> By using as examples several types of noncontemporary mechanical and bioprosthetic valves, it can be demonstrated that these valves are on the transition point of the curve, indicating that small decreases in valve area may result in large increases in gradient (Figure 25-8). More recently, the same exponential relationship between indexed effective orifice area and mean gradient for contemporary aortic bioprostheses was determined by using a pulse duplicator, this relationship being accentuated by increases in stroke volume (Figure 25-9).<sup>11</sup>

**Determining Whether an Aortic Root (and the Aortic Prosthesis) Is Too Small**

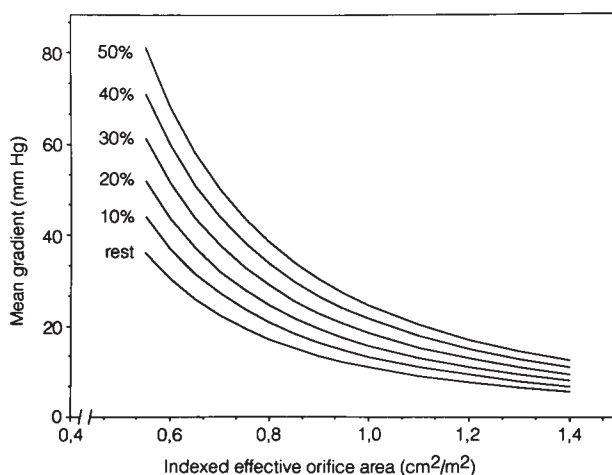
There are a number of ways in which the putative impact of valve replacement in the setting of small aortic root could be determined.



**FIGURE 25-7.** The definition of the small aortic root is not absolute and involves a size region of controversy, that is, insertion of a mechanical or stented bioprosthesis of size 19 or 21 mm.



**FIGURE 25-8.** Representation of the relationship of mean systolic gradient to the aortic valve area, assuming the cardiac output and velocity of flow are constant. Reproduced with permission from Rahimtoola SH.<sup>10</sup>



**FIGURE 25-9.** Relationships between mean transprosthetic pressure gradients and indexed effective orifice areas for aortic bioprostheses studied *in vitro* in a physiologic pulse duplicator system, assuming a normal cardiac index of 3.0 L/min/m<sup>2</sup> at rest (systolic ejection period 285 ms) and 10 to 50% increases in stroke volume, as may occur during maximal upright exercise. Reproduced with permission from Dumesnil JG et al.<sup>11</sup>

A number of studies attempted to investigate whether insertion of a “small prosthesis” (most studies focused on a 19 mm or 21 mm mechanical valve or stented bioprosthesis) influences short- and long-term *survival*. However, survival is a very insensitive determinant of prosthetic valve performance. Early and late survival after aortic valve replacement is principally determined by the structure and function of the left ventricle and the presence of comorbidity (including coronary artery disease).<sup>12</sup> Given this important caveat, it is not surprising that most studies have not found a relationship between early and late survival and aortic valve prosthesis size.<sup>13–15</sup> The study by Medalion and colleagues, with survival as the end point and using multivariable methods to allow for valve device selection factors (mechanical, stented xenograft, and allograft valves were used) and prevalent risk factors for death, was unable to detect an adverse impact on survival of moderate patient–prosthesis mismatch, and in a few patients, even at a Z-value (number of standard deviations the diameter of the device departed from the mean normal native valve size for a given body surface area) of  $-4$ .<sup>16</sup> Despite this finding and the sophistication of the methods, this study should not be regarded as an endorsement of a disregard for the postoperative prosthetic gradient, given the insensitivity of survival as an end point. However, there are studies that did find that aortic valve prosthesis size is a determinant of survival. The study by Kratz and colleagues suggested that patients with a body surface area of greater than 1.9 m<sup>2</sup> who received St. Jude valves sized 19 mm or 21 mm had a greater probability of late sudden death.<sup>17</sup> The study of He and colleagues found that (by multivariable methods) patients with a body sur-

face area who received a small prosthesis (defined as 21 mm or less) who also underwent a concomitant coronary bypass procedure had decreased long-term survival.<sup>18</sup> The study by Adams and colleagues found that insertion of a 19 mm stented bioprosthesis or mechanical valve in elderly men with aortic stenosis was associated with a higher operative mortality and on that basis had recommended that an aortic root enlargement procedure should be performed in these patients.<sup>19</sup> However, it is very possible that these findings are spurious. In this context, the study by Morris and colleagues is germane.<sup>20</sup> They found by both univariate and multivariate methods that, in a series of patients undergoing aortic valve replacement, smaller prosthetic valve size was associated with increased risk of mortality. However, on closer examination, as the authors of this study quite correctly point out, survival was better not only in those patients receiving a 23 mm prosthesis as compared to those receiving a 21 mm prosthesis or smaller, but was also better in patients receiving a prosthesis larger than 25 mm as compared to those patients receiving a 23 mm prosthesis, a situation in which unacceptable postoperative prosthetic valve gradients is highly unlikely to be an issue. Consequently, using survival as an end point for studying the impact of small prosthetic valves does carry the risk of producing spurious findings.

The degree of *regression of left ventricular hypertrophy* may be a more sensitive indication of the adequacy of an implanted aortic valve prosthesis. Preoperative left ventricular hypertrophy is a well-validated risk factor for decreased long-term survival after aortic valve replacement.<sup>12,20</sup> Regression of left ventricular mass has been demonstrated, with most of the regression occurring within the first few weeks or months after surgery.<sup>21–25</sup> It is unproven, although highly likely, that regression of left ventricular hypertrophy after aortic valve replacement is associated with improved long-term survival. It is interesting to note that this regression is incomplete, which could be the result of residual postoperative aortic valve prosthetic gradients, as well as of a hypertrophy process and an accompanying fibrosis that may be immutable.<sup>24,25</sup> Of particular interest is the degree of regression of left ventricular hypertrophy associated with different valve sizes; however, the information is unclear. Some authors found that regression associated with 19 mm and 21 mm stented and mechanical valves was similar to that of patients receiving larger-sized valves.<sup>25,26</sup> On the other hand, the data of Sim and colleagues suggested that the regression of left ventricular hypertrophy was less in patients receiving a 19 mm stented bioprosthesis or mechanical valve when compared with that in patients receiving larger valve sizes.<sup>27</sup> The regression of left ventricular hypertrophy as a means of assessing the adequacy of the implanted aortic valve prosthesis could be useful, but is confounded by other factors, including postoperative medical therapy to promote regression and the presence of

concomitant fibrosis and extreme hypertrophy for which substantial regression is unlikely to occur.

*Patient–prosthesis mismatch* is a useful concept but, in practice, it is often difficult to confirm its presence in an individual patient. Rahimtoola defined patient–prosthesis mismatch as follows: “mismatch can be considered to be present when the effective prosthetic valve area, after insertion into the patient, is less than that of a normal human valve.”<sup>10</sup> Applying that definition, patient–prosthesis mismatch should be present after virtually every aortic valve replacement, but when a surgeon is choosing a prosthesis, especially when the size of the prosthesis to be inserted may be inadequate, it is worthwhile conceptualizing the problem as, for example, in aortic stenosis, trading severe native valve disease for mild or moderate aortic stenosis. Rahimtoola indicated that severe aortic stenosis is present with an aortic valve area index of  $0.75 \text{ cm}^2/\text{m}^2$ .<sup>10</sup> The aortic valve area of a 19 mm and a 21 mm St. Jude medical prosthesis is  $0.86 \text{ cm}^2/\text{m}^2$  and  $1.08 \text{ cm}^2/\text{m}^2$ , respectively.<sup>28</sup> Because of the curvilinear shape of the relationship between a prosthetic valve gradient and valve area, a relatively modest improvement in the aortic valve area produces a proportionately greater reduction in valve gradient. Symptomatic patient–prosthesis mismatch, implying an inadequate-sized prosthesis, is usually very difficult to confirm because postoperative heart failure caused by patient–prosthesis mismatch is difficult to distinguish from other mechanisms such as left ventricular systolic and diastolic dysfunction (although any combination may be present). Therefore, symptomatic patient–prosthesis mismatch is a very unreliable and insensitive indication of implantation of an inadequate-sized aortic prosthesis.

The measurement of *Doppler gradients* across prosthetic valves is a useful means both for determining prosthetic valve function and, potentially, for determining the adequacy of the prosthesis in the small aortic root. At the outset it should be recognized that although there is good relationship between catheter-derived gradients and Doppler-derived gradients across bioprosthetic and some mechanical valves, Doppler gradients across bileaflet valves may be overestimated. These potential inaccuracies occur from (1) early pressure recovery downstream (as a result of decreased flow velocity) and (2) nonuniform local velocities (with a higher velocity between the two leaflets as a result of the partitioning of the blood flow through the valve by the two leaflets).<sup>29,30</sup> Dobutamine-stress Doppler echocardiography has been used to determine flow velocity across the aortic valve prostheses under the circumstances of increased cardiac output to simulate an exercise gradient.<sup>26,31</sup> With dobutamine stress, mean gradients across 19 mm and 21 mm St. Jude medical and Medtronic-Hall prostheses have been reported as between 35 and 40 mm Hg.<sup>29,32</sup> Although these gradients are described as “normal” or “within a clinically acceptable range” (although dopamine stress

gradients appear to be higher than that of exercise gradients), the clinical significance of gradients of this magnitude (particularly in regard to the long-term structure and function of the left ventricle) is unknown. Gradients of this magnitude do reflect the compromise inherent in valve surgery, substituting native valve obstruction with devices that are, despite decades of progress, still obstructive, a feature that is particularly evident in the small aortic root.

### Prosthetic Valve Sizing and the Small Aortic Root

It has been pointed out on a number of occasions that the labeling of prosthetic valves (both mechanical and bioprosthetic) and valve sizers does not bear a consistent relationship to the internal diameter of the valve orifice, which is the dimension that is probably the most hemodynamically significant in terms of postoperative gradients.<sup>33,34</sup> For example, the internal diameter of the St. Jude standard mechanical aortic valve, which is labeled as 21 mm, is 16.7 mm, and the internal diameter of the Modified Orifice Hancock II porcine aortic valve, which is labeled as 21 mm, is 18.0 mm.<sup>33</sup> This discrepancy between the labeled size and the true internal diameter is particularly germane to the insertion of mechanical bioprosthetic valves in the small aortic root, where prostheses with a small internal orifice diameter may be unwittingly inserted in the small aortic root, resulting in excessive postoperative prosthetic valve gradients. Furthermore, comparison of the hemodynamic performance between different prosthetic valves when implanted in the small aortic root become difficult to judge.

## A Strategy for Dealing with the Small Aortic Root

The fact that surgery involving the small aortic root is still controversial implies incomplete information and the confounding of the issue by a number of imponderables (as previously outlined). However, because prosthetic valves are inherently obstructive and because there is at least some evidence that residual left ventricular outflow tract obstruction may have deleterious long-term consequences, placement of a valve replacement device with the lowest possible gradient seems prudent. Therefore, our strategy is to not use either a 19 mm mechanical or stented bioprosthesis or a 21 mm mechanical or stented bioprosthesis in patients with a body surface area of greater than  $1.9 \text{ m}^2$ . Because in the light of current information this strategy is discretionary, increasing the aortic root dimension to accommodate a larger prosthesis must be achieved with minimal or no increase in operative mortality. Consequently, this policy is not employed in patients for whom the surgeon determines that the risk of an annulus-enlarging procedure would increase the operative risk more than a minimal degree, and this particularly applies to frail, elderly patients in whom the putative

long-term advantages of a lower postoperative prosthetic valve gradient would not be seen.

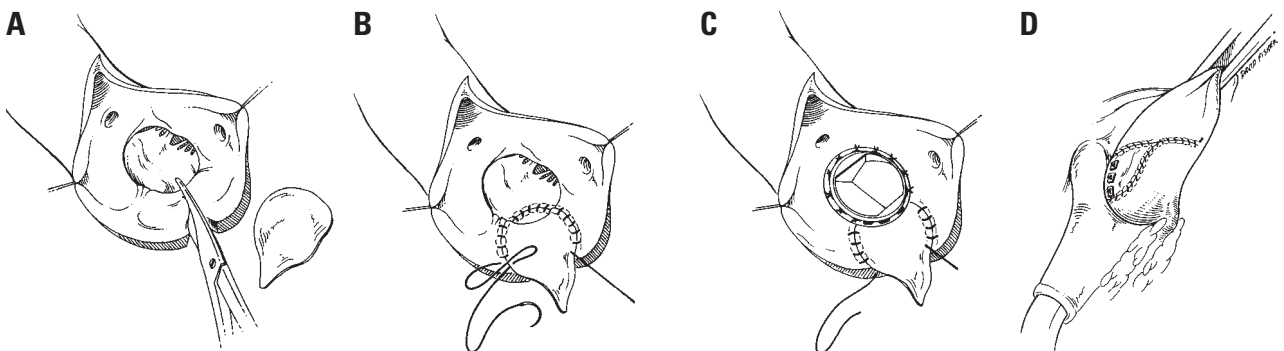
## Surgical Strategies for the Small Aortic Annulus

The fundamental goal of this surgical strategy is avoidance of implantation of a 19 mm or 20 mm mechanical or stented bioprosthesis. The surgical options are as follows: (1) *Mechanical dilatation of the small aortic root.* Although mechanical dilatation of the aortic root with Hegar metal dilators has been described in only one patient, its simplicity does make it appealing and worthy of future investigation.<sup>35</sup> (2) *Implantation of stentless tissue valves.* Allograft aortic valves inserted by either the subcoronary technique or inclusion technique are well known to have a postoperative transvalvar gradient that is lower than that of the mechanical or stented bioprosthetic valve. Similarly, the stented xenograft valves have the same excellent hemodynamics (a superior effective orifice area to that of a stented bioprosthesis) and, because of the flexibility of the valve, may allow for some oversizing and accommodation of an even larger valve in the small aortic root.<sup>36</sup> Furthermore, there is also the suggestion that the postoperative orifice area of the implanted stentless xenograft may increase over the first postoperative year.<sup>37</sup> Stentless xenograft valves do offer the solution to the problem of limited availability of allograft aortic valves. Allograft valves and stentless xenograft valves do appear to offer a very satisfactory option for the small aortic root. (3) *Supra-annular positioning of a valve replacement device.* Because the noncoronary sinus at its nadir may be lower than the nadir of the left and right aortic sinuses, a prosthesis can be implanted in the supra-annular position within the noncoronary sinus. The prosthesis is sutured along the aortic annulus in the left and right coronary sinuses but attached by pledgeted mattress sutures (the pledgets being outside the aorta in the noncoronary sinus). This technique usually allows implantation of a prosthesis that is one valve size larger than the size of the

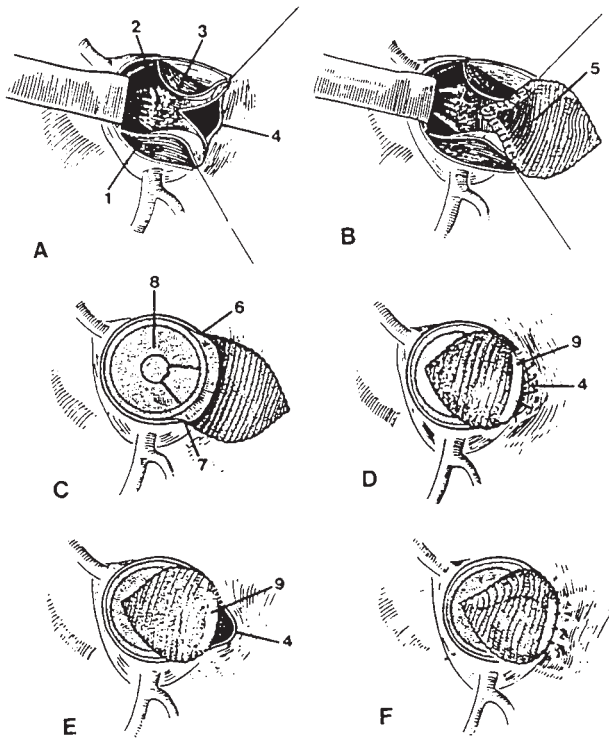
aortic annulus. (4) *Nicks procedure.* The Nicks procedure allows for implantation of a prosthesis that is usually two sizes larger than the measured size of the aortic annulus and is the simplest of the annulus-enlarging procedures to be performed. The Nicks procedure involves extension of the aortotomy into the noncoronary sinus at its nadir, through the aortic annulus and the aortic-mitral septum to the attachment of the left atrium (Figure 25-10A).<sup>38</sup> An autologous pericardial patch is fashioned in a teardrop shape, and this patch must be at least 4 cm in its transverse diameter. The pericardial patch is sutured along the incision through the aortic-mitral septum and aortotomy with continuous 4-0 polypropylene (Figure 25-10B). The prosthesis is sutured to the aortic annulus, and in the region of the patch, the prosthesis is secured with pledgeted mattress sutures passed from the prosthesis outward through the patch and tied over felt pledgets (Figure 25-10C). The remainder of the aortotomy is then closed (Figure 25-10D). The operative risk may be increased (in one series, 3.5% without annulus enlargement to 7.1% with annulus enlargement,  $p = .1$ ; but this may be explained by factors other than the procedure).<sup>39</sup>

## Manouguian Procedure

The Manouguian procedure, like the Nicks procedure, is a posterior annular enlargement procedure, but it produces on average a 5.4 mm increase in annular size.<sup>40,41</sup> The aortotomy is carried through the aortic annulus at the commissure between the left and noncoronary cusps (as opposed to the nadir of the noncoronary cusp with the Nicks procedure) (Figure 25-11). The incision is carried through the aortic-mitral septum and on to the anterior leaflet of the mitral valve for up to 2 cm (and this opens the roof of the left atrium). A pericardial patch (or Dacron patch as in Manouguian's original description) is sutured along the mitral valve incision, aortic-mitral septum, and the aortotomy, and, as with the Nicks procedure, the prosthesis is secured to the patch with horizontal mattress sutures. The roof of the left atrium is then repaired



**FIGURE 25-10.** The Nicks procedure. *A*, Extension of the aortotomy through the noncoronary sinus and nadir of the annulus and aortic and mitral septums. A teardrop-shaped autologous pericardial patch is fashioned. *B*, The pericardial patch is sutured to the aortic-mitral septum and aortotomy with continuous 4-0 polypropylene. *C*, The prosthesis is sutured along the annulus and to the pericardial patch with mattress sutures through the prosthesis and pericardial patch with pledgets on the outside of the pericardial patch. *D*, The remainder of the aortotomy is closed.



**FIGURE 25-11.** Manouguian procedure: (1) left coronary cusp; (2) anterior leaflet of the mitral valve; (3) noncoronary cusp; (4) left atrial wall; (5) patch; (6 and 7) enlargement of the aortic valve ring; (8) aortic valve prosthesis; (9) sewing ring of the prosthesis. *A*, The incision; *B*, initial suture of patch; *C*, implantation of prosthesis; *D*, suture of the left atrium and patch to the sewing ring with the same suture in small atriotomies; *E* and *F*, separate suture of larger left atriotomy following larger posterior enlargements. Reproduced with permission from Manouguian S et al.<sup>40</sup>

by suturing the cut edge to the patch or, as is often necessary, with a pericardial patch. The remainder of the aortotomy is then closed. Both the Nicks and the Manouguian procedures can be used to facilitate implantation of a larger-sized allograft aortic valve. The use of the Manouguian procedure should be rare as the other options, such as the Nicks procedure, on insertion of an allograft or stentless xenograft valve, are usually perfectly adequate solutions.

### Radical Solutions to the Small Aortic Root

The Konno procedure (aortoventriculoplasty) is mentioned for completeness, but its use in the small aortic root associated with aortic valve disease is exceedingly rare.<sup>42</sup> Its role is really for complex left ventricular tract obstruction. It is an anterior enlargement procedure that involves a longitudinal aortotomy that is carried through the anterior wall of the aortic root and into the ventricular septum and free wall of the right ventricle. The procedure involves patch repair of the interventricular septum

and patch closure of the defect created in the right ventricle. Similarly, left ventricular apicoabdominal aortic conduits have been used but should be a procedure of the last resort, particularly in patients with no other identifiable option in the setting of multiple previous procedures on the aortic root.

### Conclusion

There is abundant evidence that the structure and function of the left ventricle after aortic valve replacement is an important determinant of long-term survival and symptoms of heart failure, and, although unproven, the goal of facilitating the regression of left ventricular hypertrophy after aortic valve replacement seems important. The definition of the small aortic root (and the aortic prostheses) and its management seems less important than an overall strategy for aortic valve replacement that embodies the concept that the lowest possible transvalvar prosthetic gradient possible is the most desirable. However, except in unusual situations in which the prosthesis selected (without root enlargement) is likely to reduce late survival or impair desired activity level, the surgical methods used to minimize the transvalvar prosthetic gradient should not significantly increase the operative risk above that of standard aortic valve replacement. There are now available several surgical options that should facilitate this strategy and, it is hoped, make the small aortic root a less controversial issue.

### References

1. Anderson RH, Devine WA, et al. The myth of the aortic annulus: the anatomy of the subaortic outflow tract. *Ann Thorac Surg* 1991;52:640-6.
2. Nidorf SM, Picard MH, Triulzi MO, et al. New perspectives in the assessment of cardiac chamber dimensions during development and adulthood. *J Am Coll Cardiol* 1992; 19:983-8.
3. Westaby S, Karp RB, Blackstone EH, Bishop SP. Adult human valve dimensions and their surgical significance. *Am J Cardiol* 1984;53:552-6.
4. Hutchins AM, Araya OA. Measurement of cardiac size, chamber volumes, and valve orifices at autopsy. *Johns Hopkins Med J* 1973;133:96-106.
5. Chambers J. Echocardiography and the small aortic root. *J Heart Valve Dis* 1996;5 Suppl III:S264-8.
6. Schaff HV, Borkon AM, Hughes C, et al. Clinical and hemodynamic evaluation of the 19-mm Bjork-Shiley aortic valve prosthesis. *Ann Thorac Surg* 1981;32:50-7.
7. Davidson WR, Pasquale MJ, Fanelli A. Doppler echocardiographic examination of the normal aortic valve and left ventricular outflow tract. *Am J Cardiol* 1991;67:547-9.
8. Singh B, Mohan JG. Doppler echocardiographic determination of aortic and pulmonary valve orifice areas in normal adult subjects. *Int J Cardiol* 1992;37:73-8.
9. Dumesnil JG, Honos GN, Lemieux M, Beauchemin J. Validation and applications of indexed aortic prosthetic

- valve areas calculated by Doppler echocardiography. *J Am Coll Cardiol* 1990;16:637–43.
10. Rahimtoola SH. The problem of valve prosthesis-patient mismatch. *Circulation* 1978;58:20–4.
  11. Dumesnil JG, Yoganathan AP. Valve prosthesis hemodynamics and the problem of high transprosthetic pressure gradients. *Eur J Cardiothorac Surg* 1992;6 Suppl 1:S34–8.
  12. McGiffin DC, O'Brien MF, Galbraith AJ, et al. An analysis of risk factors for death and mode-specific death after aortic valve replacement with allograft, xenograft, and mechanical valves. *J Thorac Cardiovasc Surg* 1993;106:895–911.
  13. Fiore AC, Swartz M, Grunkemeier G, et al. Valve replacement in the small aortic annulus: prospective randomized trial of St. Jude with Medtronic Hall. *Eur J Cardiothorac Surg* 1997;11:485–92.
  14. Sawant D, Singh AK, Feng WC, et al. Nineteen-millimeter aortic St. Jude medical heart valve prosthesis: up to sixteen years' follow-up. *Ann Thorac Surg* 1997;63:964–70.
  15. Medalion B, Lytle BW, McCarthy PM, et al. Aortic valve replacement for octogenarians: are small valves bad? *Ann Thorac Surg* 1998;66:699–706.
  16. Medalion B, Blackstone EH, Lytle BW, et al. Aortic valve replacement: is valve size important? *J Thorac Cardiovasc Surg* 2000;119(5):963–74
  17. Kratz JM, Sade RM, Crawford FA Jr, et al. The risk of small St. Jude aortic valve prostheses. *Ann Thorac Surg* 1994;57:1114–9.
  18. He GW, Grunkemeier GL, Gately HL, et al. Up to thirty-year survival after aortic valve replacement in the small aortic root. *Ann Thorac Surg* 1995;59:1056–62.
  19. Adams DH, Chen RH, Kadner A, et al. Impact of small prosthetic valve size on operative mortality in elderly patients after aortic valve replacement for aortic stenosis: does gender matter? *J Thorac Cardiovasc Surg* 1999;118:815–22.
  20. Morris JJ, Schaff HV, Mullany CJ, et al. Determinants of survival and recovery of left ventricular function after aortic valve replacement. *Ann Thorac Surg* 1993;56:22–30.
  21. St. John Sutton M, Plappert T, Spiegel A, et al. Early post-operative changes in left ventricular chamber size, architecture, and function in aortic stenosis and aortic regurgitation and their relation to intraoperative changes in afterload: a prospective two-dimensional echocardiographic study. *Circulation* 1987;76:77–89.
  22. Henry WL, Bonow RO, Borer JS, et al. Evaluation of aortic valve replacement in patients with valvular aortic stenosis. *Circulation* 1980;61:814–25.
  23. Christakis GT, Joyner CD, Morgan CD, et al. Left ventricular mass regression early after aortic valve replacement. *Ann Thorac Surg* 1996;62:1084–9.
  24. De Paulis R, Sommariva L, De Matteis GM, et al. Extent and pattern of regression of left ventricular hypertrophy in patients with small size CarboMedics aortic valves. *J Thorac Cardiovasc Surg* 1997;113:901–9.
  25. Kahn SS, Siegel RJ, DeRobertis MA, et al. Regression of hypertrophy after Carpentier-Edwards pericardial aortic valve replacement. *Ann Thorac Surg* 2000;69:531–5.
  26. Anderson WA, Ilkowsky DA, Eldredge J, et al. The small aortic root and the Medtronic Hall valve: ultrafast computed tomography assessment of left ventricular mass following aortic valve replacement. *J Heart Valve Dis* 1996;5 Suppl III:S329–35.
  27. Sim FKW, Orszulak TA, Schaff HV, Shub C. Influence of prosthesis size on change in left ventricular mass following aortic valve replacement. *Eur J Cardiothorac Surg* 1994;8:293–7.
  28. Sawant D, Singh AK, Feng WC, et al. St. Jude medical cardiac valves in small aortic roots: follow-up to sixteen years. *J Thorac Cardiovasc Surg* 1997;113:499–509.
  29. Kadir I, Izzat MB, Birdi I, et al. Hemodynamics of St. Jude medical prostheses in the small aortic root: in vivo studies using dobutamine Doppler echocardiography. *J Heart Valve Dis* 1997;6:123–9.
  30. Baumgartner H, Schima H, Tulzer G, Kühn P. Effect of stenosis geometry on the Doppler-catheter gradient relation in vitro: a manifestation of pressure recovery. *J Am Coll Cardiol* 1993;21:1018–25.
  31. Izzat MB, Birdi I, Wilde P, et al. Evaluation of the hemodynamic performance of small CarboMedics aortic prostheses using dobutamine-stress Doppler echocardiography. *Ann Thorac Surg* 1995;60:1048–1052.
  32. Fiore AC, Swartz M, Grunkemeier G, et al. Valve replacement in the small aortic annulus: prospective randomized trial of St. Jude with Medtronic Hall. *Euro J Cardiothorac Surg* 1997;11:485–92.
  33. Christakis GT, Buth KJ, Goldman BS, et al. Inaccurate and misleading valve sizing: a proposed standard for valve size nomenclature. *Ann Thorac Surg* 1998;66:1198–203.
  34. Bartels C, Leyh RG, Bechtel JFM, et al. Discrepancies between sizer and valve dimensions: implications for small aortic root. *Ann Thorac Surg* 1998;65:1631–3.
  35. Bartels C, Sievers HH. Successful dilatation of the small aortic root for implantation of a larger valve prosthesis. *J Heart Valve Dis* 1999;8:507–8.
  36. Walther T, Falk V, Diegeler A, et al. Stentless bioprostheses for the small aortic root. *J Heart Valve Dis* 1996;5 Suppl III:S302–7.
  37. Sintek CF, Fletcher AD, Khonsari S. Small aortic root in the elderly: use of stentless bioprosthesis. *J Heart Valve Dis* 1996;5 Suppl III:S308–13.
  38. Nicks R, Cartmill T, Bernstein L. Hypoplasia of the aortic root—the problem of aortic valve replacement. *Thorax* 1970;25:339–46.
  39. Sommers KE, David TE. Aortic valve replacement with patch enlargement of the aortic annulus. *Ann Thorac Surg* 1997;63:1608–12.
  40. Manogueanu S, Seybold-Epting W. Patch enlargement of the aortic valve ring by extending the aortic incision into the anterior mitral leaflet—new operative technique. *J Thorac Cardiovasc Surg* 1979;78:402–12.
  41. Demmy TL, Magovern GJ. Aortic root enlargement procedures: In: Emery RW, Arom KV, editors. *The aortic valve*. Philadelphia: Hanley & Belfus, 1991.
  42. Konno S, Imai Y, Iida T, et al. A new method for prosthetic valve replacement in congenital aortic stenosis associated with hypoplasia of the aortic valve ring. *J Thorac Cardiovasc Surg* 1975;70:909–17.