tive prosthetic orifice size may have to be performed at least a few weeks after valve insertion. Second, in some patients, the problem is compounded by the small size of the annulus compared with the size of the patient. My article was not written just to highlight the difficulties of a small aortic annulus.

Dr. Kinsley’s ability to insert a prosthetic valve size commensurate with the patient’s body size is to be applauded. However, Dr. Kinsley’s letter and previous papers leave many questions unanswered. The technique of anticipating stroke volume and tailoring the prosthetic valve to that stroke volume is not described. The belief that favorable hemodynamic characteristics are actually enhanced is not based on data that have been presented. Although gradients obtained at surgery are cited, calculated prosthetic valve areas and valve area indices of patients studied some weeks or months after surgery have not been presented. It would also be of interest to know how many patients had aortic valve replacement without use of this technique during the same period of time when the 52 patients were operated on with use of this technique. There have been two deaths with the use of this technique, and some patients already have aortic incompetence; moreover, the device is inserted in an abnormal position. Before one is convinced that the technique is safe and effective, one should know: 1) the long-term mortality and morbidity of patients so treated and the data analyzed with the use of actuarial techniques; and 2) the results of detailed hemodynamic evaluation which provide information about prosthetic valve areas, frequency and severity of valvular regurgitation and about ventricular performance.

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End-Systolic Pressure-Volume Relations

To the Editor:

Referring to a recent article by Grossman et al., I Dr. Iizuka commented that the end-systole identified as the moment for the smallest volume may not necessarily be the true end-systole contributing to the end-systolic pressure-volume relationship. Both Dr. Iizuka’s comment and Dr. Grossman’s response to it emphasize the problems in identifying the end-systole in the clinical setting.

As one of the researchers who originally proposed the end-systolic pressure-volume ratio as an index of ventricular contractility from animal experiments, I would like to reemphasize our definition of end-systole to prevent misunderstanding of the end-systolic pressure-volume relationship.

There is no unanimous definition of systole and end-systole. In our definition, the end of mechanical systole is the moment at which the contraction becomes maximal and the relaxation starts. This end-systole may not coincide with the end-systole identified as the end of ejection or the moment of the dicrotic notch of aortic pressure. In an isovolumic contraction, the peak pressure corresponds to our end-systole, as seen in figure 1, loops a and b. In a natural in-vivo type of contraction, the left upper corner of the pressure-volume loop corresponds to our end-systole. All these end-systoles come on or very near a line representing the end-systolic pressure-volume relationship. When the pressure-volume loop of an abnormal contraction has a sharp left upper corner as loop c in figure 1, this corner can be identified as our end-systole because it is situated on the end-systolic pressure-volume line.

However, the identification of our end-systole is very difficult if the pressure-volume loop of an abnormal contraction has a rounded shoulder at its left upper part, as loop d in figure 1. The point on the loop nearest the end-systolic pressure-volume line should be identified as our end-systole (solid circle). However, if loop d were alone in the diagram, it would be very difficult to pinpoint the end-systole on the loop. Obviously, the point with the smallest volume (open circle) should not be identified as the end-systole of loop d because it occurs late in the relaxation phase.

From our animal experiments, I have been impressed that the close coincidence of the end of ejection with end-systole in natural in vivo contractions is circumstantial. This coincidence is probably due to an appropriate interaction among the heart, the valve and the artery. If this interaction becomes abnormal, the end of ejection may no longer coincide with the end-systole, and the identification of the end of ejection as the end of systole may no longer be correct. Further studies are needed to discover the relationship between the ends of systole and ejection and the factors influencing this relationship.

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“Trifascicular Block”

To the Editor:

In a recent comment relating to my paper, “Prognosis for Patients with Congenital Heart Disease and Postoperative Intraventricular Conduction Defects,” (Circulation 57: 867, 1978), Dr. Gillette states that an error in terminology should be brought to the attention of the readers. There is no error in this paper.

Dr. Gillette’s argument is that the term “trifascicular block” is an electrocardiographic pattern which may result from various etiologies, such as a true trifascicular disease, or “bifascicular disease” and His bundle and/or atrioventricular (AV) nodal disease or no fascicular disease and P-R prolongation due to atrial conduction delay due to atrial enlargement. This last pattern is commonly seen in patients with various types of AV canal defects and other congenital heart defects.

I wholeheartedly agree with Dr. Gillette’s comment that “trifascicular block” is a misnomer referring to a descriptive electrocardiographic pattern rather than to an implication of an underlying pathophysiologic mechanism or process. Indeed, our group has commented extensively in the past on this problem and the risks involved by confusing descriptive electrocardiographic terminology with possible underlying pathophysiologic mechanisms.

Because of the brevity of descriptive terms and the day-to-day convenience inherent in the use of these terms, rather than a long explanation of a possible mechanism, their use is extremely widespread. For example, “bifascicular block,” “AV block,” “intraventricular conduction defects” — all may be due to mechanisms other than “block” as implied in these terms.

We have previously stated that “attempts to indicate a precise electrophysiologic mechanism from the scalar electrocardiogram may lead to oversimplification and misinterpretation of the true underlying cardiac abnormality.”

The section referred to by Dr. Gillette is entitled, Right Bundle Branch Block and Left Anterior Hemiblock Pattern and P-R Prolongation (“trifascicular block pattern”). Use of quotation marks for identification of a mistaken idea is common and proper in the English language (Professor Michael Hayes, Department of English, Columbia University; personal communication), and I used it extensively throughout the article when I felt that the term used did not properly reflect the underlying pathophysiologic mechanism. When Dr. Gillette uses the term “trifascicular disease” in an identical way to mine, he identifies the underlying impropriety of this term by placing it within quotation marks.

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