signals previously labeled as isovolumic relaxation flow, add further interest to these interesting signals. Certainly it is conceivable that the reversal of pressure difference between the left ventricular apex and left ventricular outflow tract that they have nicely shown on their pressure tracings may be the pressure correlate of flow from the base toward the apex that we have observed (1). Such careful studies will surely aid in understanding this interesting signal.

We also admire the work of Brutsaert et al. (2) and we believe the principles they have suggested may be most interestingly related to patients with hypertrophic cardiomyopathy with systolic gradient.

Although we encourage and admire the careful studies done by these authors, we are troubled by some features of the tracings in the figure they present. There is some question regarding the origin of the phonocardiographic signals and, more significantly, some question of correct registration in time between the Doppler and pressure recordings. Inspection of this figure shows the QRS complex of the electrocardiogram as much broader on the Doppler tracing than on the pressure tracing. This has apparently been used for primary registration, and the termination of the QRS complex on both records matches. However, if one assumes the onset of the QRS complex may be the correct registration, then the onset of the intraventricular flow velocity signal would occur beginning with S_2 registered on the phonocardiogram rather than 40 ms before it. Thus, the signal in question would be during the isovolumic relaxation period. The phonocardiogram itself is presumed from one of the catheters within the ventricle. However, there is no appropriate mitral component of the first sound registered because "S_1" occurs coincident with a left ventricular pressure >100 mm Hg. The recorded murmur stops before mid-systole, and the origin of the prolonged vibrations after the presumed S_2 are unclear. If we assume that the pressure and Doppler signals are properly registered, then mitral valve opening occurs approximately 40 ms after left ventricular pressure reaches its nadir and plateau. The ability of the mitral regurgitation Doppler flow velocity signal to predict the pressure difference between chambers allows certainty that mitral flow immediately follows the left ventricular-left atrial pressure crossover (see Fig. 2, ref. 1). Similarly, the left ventricular outflow Dopple: signal suggests that the flow under discussion begins as the pressure difference between the left ventricular body and the aorta or subaortic area disappears (see Fig. 2, ref. 1). Thus it is difficult to be sure that both tracings of Maier et al. are matched in time even though they were recorded simultaneously in the laboratory. Because they were not recorded on the same piece of paper, this possibility always exists. It would be optimal to use pulsed wave Doppler ultrasound because the beginning and end of such a velocity signal are better defined with this method (1).

Review of our patients shows a variety of patterns for this intraventricular signal, with some showing early termination of the signal as presented by Maire et al., some showing flow velocities sustained until mitral opening, and some showing reversal of this flow velocity before mitral opening (1). Review of our tracings suggests that these signals begin about the time of S_2. In our patients, the signals may begin very slightly before S_2 by external phonocardiography, but none of them seems to be as early as those in the three patients studied by Maier et al.

These signals have been noted in Japan (3), the United States and Switzerland. It is hoped that sophisticated studies such as those done in Switzerland will lend insight not only to the origin of these signals, but also to their implications for understanding relaxation of the myocardium.

ZION SASSON, MD
LIV HATLE, MD
RICHARD L. POPP, MD, FACC
School of Medicine
Stanford University
Stanford, California 94305

References

1. Sasson Z, Hatle L, Appleton CP, Jewett M, Alderman EL, Popp RL. Interventricular flow during isovolumic relaxation: description and characterization by Doppler echocardiography. J Am Coll Cardiol 1987;10:539–46.
2. Brutsaert DL, Rademakers FE, Sys SU. Triple control of relaxation: implications in cardiac disease. Circulation 1984;69:190–6.
3. Hiroyuki A, Kazuhiro T. Asynchronous relaxation of the ischemic left ventricle. Jpn Circ J 1982;46:102–12.

Problems in Nomenclature: Bulboventricular Foramen Versus Ventricular Septal Defect

The morphologic relation between hearts with tricuspid atresia and those with double inlet left ventricle continues to be a controversial topic. Clinical experience, however, has now shown that the problems presented by both lesions in the setting of discordant ventriculoarterial connections are very similar. This is further endorsed by a recent article in the journal (1). The problem is caused by restriction, in both entities, of a communication between the large and small ventricular chambers. The nomenclature of these ventricular chambers has been extensively debated and, as yet, there is no consensus concerning the most appropriate terminology. Surely the time has now come to move toward a unified terminology because to call the obstructed communication a "bulboventricular foramen" in one lesion yet a "ventricular septal defect" in the other (1) must suggest that the structures are morphologically dissimilar. They are not (2). In terms of anatomy, the interventricular communications in "single left ventricle" and tricuspid atresia are identical. Common sense should now dictate that hearts with an interventricular communication cannot logically be described either as "single ventricles" or "univentricular."

In the past, my colleagues and I (3) constructed formidable conventions to distinguish "ventricles" from "nonventricles" so as to preserve a contorted logic that justified calling hearts "univentricular" when they possessed two ventricles. We now recognize the futility of this approach (4). There is not need to describe hearts as "single left ventricle" when they can be described with greater accuracy and precision as "double inlet left ventricle." Acceptance of the latter term would also remove the ambiguity of "a univentricular heart of left ventricular type" with a "rudimentary right ventricle" (5). Nomenclature, nonetheless, is a matter of personal choice, and observers must retain their freedom to describe structures according to their preference. My reason in writing, therefore, is not so much to complain about use of the term "single left ventricle" (although double inlet left ventricular is, in my opinion, preferable) but to draw your reader's attention to the fact that the "bulboventricular foramen" in single left ventricle is no different

from the "ventricular septal defect" in tricuspid atresia. Both communications would be better described as ventricular septal defects, recognizing at the same time the inescapable fact that both groups of hearts possess two ventricles irrespective of how we choose to describe them.

ROBERT H. ANDERSON, MD
Visiting Professor
Children's Hospital of Pittsburgh
Pittsburgh, Pennsylvania 15213

References

1. Rothman A, Lang P, Lock JE, Jonas RA, Mayer JE, Castaneda AR. Surgical management of subaortic obstruction in a single left ventricle and tricuspid atresia. J Am Coll Cardiol 1987;10:421–6.
2. Desanfield JE, Tommasin G, Anderson RH, Macartney FJ. Tricuspid atresia: analysis of coronary artery distribution and ventricular morphology. Br Heart J 1982;48:485–92.
3. Anderson RH, Becker AE, Freedom RM, et al. Problems in the nomenclature of the univentricular heart. Herz 1979;4:97–106.
4. Anderson RH, Becker AE, Tynan M, Macartney FJ, Rigby ML, Wilkinson JL. The univentricular atrioventricular connection: getting to the root of a thorny problem. Am J Cardiol 1984;54:822–48.
5. Freedom RM. The dinosaur and banding of the main pulmonary trunk in the heart with functionally one ventricle and transposition of the great arteries: a sage of evolution and caution. J Am Coll Cardiol 1987;10:427–9.

Standardized ECG Examination

The article by Hancock et al. (1) was interesting, somewhat provocative with respect to future possibilities for both testing and standardization of interpretation of electrocardiograms (ECGs) and provided a baseline for supplying ECG interpretation, looking toward possible computerization of the checklist on the interpretation sheet. I think at least two items have been overlooked. 1) I suggest a line on the checklist for multiform premature ventricular complex(es) as well as one for interpolated ventricular ectopic beats. 2) A shortcoming of the list is in the pacemaker function and rhythm section as there is no good way to include interpretation of an ECG in a patient who has a DDD pacemaker. I propose the addition of a line suggesting atrial sensing with ventricular pacing for ventricular activation) as well as a line indicating intermittent pacing.

This is a good baseline evaluation, but it may also benefit from an additional suggested or probable clinical disorder that would include elevated intracranial pressure.

STEVEN M. KOROTKIN, MD, FACC
Cardiology Associates of Birmingham, P.C.
32270 Telegraph Road
Birmingham, Michigan 48010

Reference

1. Hancock EW, Norcini JJ, Webster GD. A standardized examination in the interpretation of electrocardiograms. J Am Coll Cardiol 1987;10:382–6.

Comment

The items mentioned as overlooked were omitted for the sake of keeping the list to a practical length for the examination. Other such items could have been added for the sake of completeness. DDD pacing is partially covered by the term AV sequential pacing, although I agree that modes such as atrial-sensed ventricular-paced rhythm are not covered. A list used for diagnostic purposes should certainly be expanded beyond that needed for the purpose of the examination.

E. WILLIAM HANCOCK, MD, FACC
Cardiology Division
Stanford University School of Medicine
Stanford, California 94305

Doppler Echocardiographic Appearance of Cusp Tears in Tissue Valve Prostheses

Chambers et al. (1) report a Doppler sign of rapidly oscillating intracardiac structures without seeming to realize that this sign was reported and the underlying physics elegantly worked out recently by my colleagues Holen et al. (2) at the University of Rochester.

RICHARD MELTZER, MD, PhD, FACC
University of Rochester Medical Center
Rochester, New York 14642

References

1. Chambers JB, Monaghan MJ, Jackson G, Jewitt DE. Doppler echocardiographic appearance of cusp tears in tissue valve prostheses. J Am Coll Cardiol 1987;10:462–6.
2. Holen J, Wiag RC, Gramiak R. Representations of rapidly oscillating structures on the Doppler display. Ultrasound Med Biol 1985;11:267–72.

Reply

The main thrust of our study was the association in failing bioprostheses of a common pathologic finding (one torn and two normal cusps) and the same Doppler sign (striation of the regurgitant jet). We were careful to point out that the sign had also been described in other situations (1) and that diastolic flutter in mitral prostheses had also been shown with paraprosthetic leaks (2, 3). The paper referred to by Meltzer, in which the emphasis is on the physical basis of the phenomenon, is complementary to ours.

J. B. CHAMBERS, MA, MRCP
M. J. MONAGHAN, MSc
G. JACKSON, MB, FRCP
D. E. JEWITT, BSc, FRCP
King's College Hospital
Denmark Hill
London SE5 #9RS, England

References

1. Hatle L, Angelsen B. Doppler Ultrasound in Cardiology. Philadelphia: Lea & Febiger, 1985:278.
2. Harrison EE, Starr S, Spotto E, Clark P. Echocardiogram in porcine mitral valve dysfunction. Am J Cardiol 1980;45:968.
3. Iacovella G, Narducci C, Pino PG, Saloib A. Echocardiographic features of porcine valve dysfunction. Am J Cardiol 1980;46:526.