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LETTERS TO THE EDITOR

Ode to the Electrophysiologist

I.

Electrically charged,
Mathematically inclined,
Logical physicians,
Sons and daughters
Of the His-Purkinje system.
Riding down the AV trail,
They like to climb retrograde.
They are a special breed;
They are electrophysiologists;
They are masters of their trade.

II.

They crowd the cardiac chambers
With long, electrical wires,
To tickle the whiskers of
The maestro of the electrical system
And record electrograms
Of the His and Purkinje system
And micro signals from the myocardium.
They are a special breed, indeed,
These ecstatic electrophysiologists!

III.

Their motto is to stimulate,
To induce and terminate,
To rock and roll
The cardiac system
Into a frenzied voodoo rhythm.
Then take a break
Within the sinus or AV junction.
They are a special breed, indeed,
These magical electrophysiologists!

IV.

Their job is to induce
VT and V fib;
To shock the poor patient
Into a flat line, then sinus rhythm.
With drugs, surgery
And devices, they challenge
God in His infinite wisdom.
They are a special breed, indeed,
These omnipotent electrophysiologists!

V.

Their world is measured in msec,
They map the heart in a few seconds.
To perform daredevil acts,
While ablating bypass tracts
Hidden around the AV junction.
Their goal is to seek the "focus"
And pathways of reentrant rhythms,
Then lash out with all the power,
With scalpel, radiofrequency and cryopower.

VI.

In my new-found wisdom
I would like to pay tribute
To those electrophysiologists
Who came out of the Public Health system.*
These creative and visionary men
Led the way for those
Who came after them.
They are today's electrophysiologists.
They are, indeed, very fine men.

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*Public Health System refers to the U. S. P. H. S. Cardio-pulmonary Laboratory in Staten Island where, under the direction of Dr. Anthony N. Damato and Dr. Sun A. Lau, the following electrophysiologists were trained: Masood Akhtar, William Batsford, Walter Berkowitz, David Cannan, Antonio Caracia, Stafford Cohen, Marcello Ellizzari, John Gallagher, Bruce Goldreyer, J. Anthony Gomes, Jacob Haft, Richard Helfant, Mark Josephson, Bernard Kosowsky, John Lister, M. Mirowski, J. Bimbola Ogunkelu, Robert Patten, Kasten Pauley, Andrew Przybyla, Michael Ricciutti, C. Pratap Reddy, Ken Rosen, Jeremy Ruskin, Benjamin Scherlag, Robert Schnitzler, Stuart Seides, Emanuel Stein, Charles Steiner, Andres Ticzon, Guillermo Vargas, P. Jacob Varghese, Gerald Weisfogel, Melvin Weiss, Andrew Wit, and Melvin Young.

Overestimation of Valve Area by the Gorlin Formula

In the January 1990 issue of the Journal, Gorlin and Gorlin (1) proposed a generalized formulation of the Gorlin formula. However, in this derivation they neglected the prestenotic velocity (V_1). This neglect may cause significant overestimation of calculated valve area.

The Gorlin formula including the prestenotic velocity V_1 can be written as:

$$A = \frac{Q}{c_c c_v \sqrt{2\Delta P/\rho}} \sqrt{1 - (V_1/V_2)^2}, \qquad [1]$$

where A = valve area, Q = flow rate, V_1 and V_2 = pre- and intrastenotic velocity, respectively, c_c and c_v = coefficients of contraction and velocity, ΔP = mean pressure difference and ρ = mass density.

On the basis of the data from 39 patients with aortic stenosis reported by Zoghbi et al. (2), the overestimation due to neglect of V_1 ranges from 15% in mild stenosis (gradient 23 mm Hg, valve area 1.5 cm^2, overestimated by 0.23 cm^2) to 1% in severe stenosis (gradient 109 mm Hg, valve area 0.51 cm^2). However, an overestimation >10% could be found in 6 (15%) of these patients. In patients

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with severe aortic insufficiency this overestimation may be even more marked because of the high prestenotic velocity V_1.

The same overestimation of valve area occurs when evaluating prosthetic valves with the Gorlin formula. With the continuity equation, the area determined by the tissue annulus diameter and the effective orifice area, respectively, can be substituted for the pre- and intrastenotic velocities in equation 1. On the basis of the data published by Horstkotte et al. (3) for the 29-mm St. Jude Medical prosthesis, an overestimation of valve area of 16% can be calculated. This may, at least in part, explain the overestimation of prosthetic valve area by the Gorlin formula (4).

Thus, in mild stenosis neglect of the prestenotic velocity may cause significant overestimation of valve area whereas in severe stenosis it is negligible. Therefore, in our opinion, the prestenotic velocity V_1 should not be neglected in a "generalized" formula, which can be written as shown above.

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2. Zoghbi WA, Farmer KL, Soto JG, Nelson JG, Quinones MA. Accurate noninvasive quantification of stenotic aortic valve area by Doppler echocardiography. Circulation 1986;73:452–9.
3. Horstkotte D, Haerten K, Herzer JA, Seipel L, Bircks W, Loogen F. Preliminary results in mitral valve replacement with the St. Jude Medical prosthesis: comparison with the Björk-Shiley valve. Circulation 1981;64 (suppl II):II-203–9.
4. Cannon SR, Richards KL, Crawford MH, et al. Inadequacy of the Gorlin formula for predicting prosthetic valve area. Am J Cardiol 1988;62:113–6.

Reply

The notation was meant to present the generalized equation, devoid of considerations of mass and density and, thereby, of gravitational acceleration. The latter is a by-product of the traditional method of measurement of pressure in terms of height of a given fluid density.

The issue of approach velocity is a different consideration and relates more to steady state flow conditions, as in a blood vessel. For the aortic and the mitral valve, respectively, blood velocity is zero in the ventricle before valve opening by the systolic force generation against aortic pressure, and likewise, in the atrium, velocity and flow at the closed mitral valve is zero and only begins as the valve opens to a low pressure diastolic ventricular chamber.

We commend Wipperman for using Bernoulli's equation in a more generalized form than was intended in original valve orifice formulas. These latter equations relied on the fact that initial pressure measurements were carried out at the site where initial velocity was either zero or relatively low, i.e., ventricular, atrial or aortic sites. Wipperman measures V_1 at the prestenotic aortic valve area site rather than in the preceding chamber, and is thereby measuring a velocity whose energy is derived from the ventricular systolic pressure. Consequently, pressure at the prestenotic aortic valve site would be proportionately less than the pressure in the preceding chamber. One could derive the most accurate calculation, and therefore application, of the Bernoulli equation if one could measure simultaneously and at the same site prestenotic velocity and pressure and jet velocity and pressure.

In practice, numerous authors have described other critical variables causing energy loss, such as orifice shape and size, and those losses of energy owing to paravalvular impediments to flow, such as inertia of opening a diseased or prosthetic valve, subvalvular tissue protrusions, etc. As we have noted (1), the pressure-flow derivation of area is generally smaller than that measured via imaging or by intrinsic prosthetic valve orifice design, but is the *functionally important* value in assessing the load placed upon the cardiac pump. The point is particularly evidenced in the different pressure-flow characteristics of the St. Jude and Björk-Shiley valves of similar lumen dimensions as shown by Horstkotte et al. (2). Thus, the orifice "constants" are often empirically derived to take account of intrinsic orifice differences (contraction, frictional losses, etc.) and techniques and precision of hydraulic measurements. One must also keep in mind that the site of measurement of pressure or velocity will slightly affect the correction factor owing to losses due to friction at sites other than the orifice itself.

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Role of the Cardiologist in Peripheral Vascular Disease

ACC President Robert L. Frye should be congratulated for his excellent review of the role of the cardiologist in peripheral vascular disease (1). The time is long overdue to recognize the fact that vascular medicine is more than just interventional therapy, imaging or the medical care of the vascular surgical patient. Without a thorough understanding of the etiology, pathophysiology and natural history of the disease, as well as a knowledge of medical, surgical and interventional technologies, care for the patient with peripheral vascular disease will be less than optimal. The notion that a cardiologist who is trained in invasive cardiac technology can apply these same procedures to the peripheral vasculature is a misconception.

As noted by Frye, formal or informal training in peripheral vascular disease by the cardiologist in most programs is seriously lacking. A number of training programs are emerging around the country to fill the need for the important subspecialty of vascular medicine. Unfortunately, there are still too few institutions to supply the need, both at academic medical centers and in private practice. Programs with a multidisciplinary approach to peripheral vascular disease emphasize the need to develop excellent bedside clinical techniques, as well as expertise, in not only arterial disease