LETTERS TO THE EDITOR

Note on the Way to Quantify the Pump Function of the Heart

In an interesting article Elzinga and Westerhof (1979) have stressed the importance of assessing the performance of the heart as a pump. They suggested for this purpose an empirical approach which consists of studying the mean pressure-mean flow curve for the left ventricle, in analogy to the case of the roller pump. A theoretical point of view would suggest that the quantification of the pump function of the heart can be achieved primarily by a study of its dynamics.

The study of the properties of the left ventricle has until now been confined mainly to static models. A study of the effect of inertial forces by Tallarida et al. (1970) has shown that these forces amount to less than 1% of the static forces and as such can be neglected. However as far back as 1966, Noble et al. published a study in which the potential clinical usefulness of measurement of the acceleration of the blood flow from the left ventricle was stressed. They noted that in cases of induced mild coronary occlusion "... the magnitude of the fall in maximum acceleration and the fact that this decrease occurred before any other changes made maximum acceleration by far the most sensitive index of the change in the myocardium."

In a study by Shoucri and Dumesnil (1978) of the dynamics of the left ventricle, it was suggested that the change in blood flow velocity and acceleration observed by Nobel et al. (1966) for cases of mild coronary occlusion was in fact directly induced by similar changes in the velocity and acceleration of the left ventricular wall. By using a two-dimensional cylindrical model, a relation was derived of the form (Equation B7) of Shoucri and Dumesnil, 1978

\[ Dh - p = \alpha \left( \frac{1}{2}a^2 - \frac{1}{2}b^2 \right) - \beta \ln \frac{b}{a}, \quad (1) \]

In Equation 1, h is the thickness of the wall of the left ventricle and is equal to b - a; a is the inner radius and b the outer radius of the left ventricle. D is the body force per unit volume directed radially and representing the contracting force during systolic motion, and Dh is consequently the resulting pressure on the inner wall of the left ventricle due to the contracting forces and p the intraventricular pressure,

\[ \alpha = \rho \left( \frac{da}{dt} \right)^2 = \left( \frac{\rho}{4\pi^2} \right) Q^2, \] \[ \beta = \frac{d}{dt} \left( \frac{da}{dt} \right) = \left( \frac{\rho}{2\pi l} \right) \frac{dQ}{dt}, \] where \( \rho \) is the density of the myocardium, l the length of the cylinder (assumed constant), Q the flow velocity, and \( \frac{dQ}{dt} \) the flow acceleration.\[ \frac{da}{dt} = R \frac{dR}{dt}, \]

where R is the mean radius \( \frac{a + b}{2} \). It is clear that the radial force D can be resolved into forces F acting along the direction of the fibers by using the rule of the polygon as shown in Figure 1. Equation 1 was derived by using the methods of the theory of elasticity. The concept of body forces, i.e., forces proportional to the volume or mass, is well known in the theory of elasticity, although usually neglected because inert masses do not exhibit body forces (apart from gravity forces which are neglected, because they are relatively small). Biomatter is a different case, because forces can be generated chemically.

Equation 1 is similar to an equation suggested by Abbott and Gordon (1975) from hemodynamic considerations

\[ F = F_m + C a_{cf} + D v_{cf}^2, \quad (2) \]

where \( F - F_m \) = force excess over the force necessary to match aortic pressure, \( a_{cf} \) = acceleration of the equatorial circumferential wall fiber, \( v_{cf} \) = velocity of the circumferential fiber, C and D = geometrical parameters related to the size and shape of the ventricle.

**FIGURE 1** Cross-section of a hollow cylinder with inner radius, a, and outer radius, b. The polygon method to decompose the radial force per unit volume D into tangential forces along the circumferential direction is shown.
It is interesting to mention here this comment of Abbott and Gordon (1975), "... We feel that it would be fruitful to extend the correspondence between isolated muscle experiments and intact heart investigations so that the meeting point of the two fields will not focus so completely on the issue of the validity of \( V_{\text{max}} \) (the maximum velocity of the contractile element). The intense preoccupation with this interesting question has diverted attention from the biophysics of the ejection phase and the possible implications of hemodynamics for muscle mechanics. After all, it is not the left ventricular pressure per se that determines directly the ejection of blood from the ventricle into the aorta but rather the small differential pressure between the ventricle and the aorta." We note that, in Equation 1, it is not the pressure difference between the left ventricle and the aorta that enters into consideration, but the pressure difference across the inner wall of the left ventricle. We note also from Equation 1 that a change in the pressure \( p \) reflects in a complicated way a change in the inotropic state (represented by the body force pressure \( D_h \)), a change in the geometry of the left ventricle represented by the factors \( \frac{a^2 b}{b^2} \) and \( \ln \frac{a}{b} \), and a change in the blood flow velocity and acceleration.

Equation 1 applies to instantaneous values and can be averaged over a given period, and it is unfortunate that all the data required for its application have never been measured simultaneously. One can however apply it approximately at the instant when \( Q \) is maximum (\( Q = 0 \)) to the results given by Tallarida et al. (1970) or Noble et al. (1966). During the ejection phase, when \( Q \approx 0 \), Equation 1 reduces to

\[
p \approx -K Q^2 + D_h,
\]

where \( K \) is a positive slowly varying factor. Equation 3 indicates a relationship between the instantaneous values of pressure and volume similar to the mean pressure-mean volume curve reported by Elzinga and Westerhof (1979).

It is hoped that the preceding discussion will help orient theoretical research towards the study of the dynamics of ventricular wall motion as a means to quantify the pump function of the heart, and also suggest new experiments to make up for the actual lack of experimental data required for the application of this kind of theoretical work.

Rachad M. Shoucri, Ph.D.
Institut de Cardiologie de Québec
2725, chemin Sainte-Foy
Sainte-Foy, Québec, G1V 4G5
Canada

References

Reply to the Preceding Letter

In his comment Dr. Shoucri stresses that the study of the dynamics of ventricular wall motion to quantify pump function of the heart is a neglected but interesting subject. He points towards a supposed similarity between his Equation 1 and Equation 4 of Abbott and Gordon (1975) and claims that his Equation 2 describes a relationship between the instantaneous values of pressure and flow similar to the mean pressure-mean output graph described by us.

Although of interest theoretically we do not think that Equation 2 is based on the same fundamental myocardial properties as our pump function graph. As far as we can see the term \(-KQ^2\) remains under all circumstances too small to be of any interest in determining cardiac pump function. We estimated its contribution for a thick-walled cylinder representing the isolated cat heart to be about 0.001 mm Hg at maximum peak flow.

The small pressure gradient between ventricle and aorta (Abbott and Gordon, 1975) and the difference between the pressure exerted on the inner wall of the left ventricle and that present in the cavity, as emphasized by Dr. Shoucri, are not determined only by the contractile force of the myocardial fibers. Other factors, like for instance the geometry of the outflow tract and the mass of the myocardial wall, respectively, determine the magnitude of each of the two pressure gradients as well. This seems to us to limit the usefulness of such small quantities as tools to measure pump function of the heart.

Gjis Elzinga, M.D., Ph.D.
Nicolaas Westerhof, Ph.D.
Physiological Laboratory
Free University
Van der Boechorststraat 7
Amsterdam, The Netherlands

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R M Shoucri

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