Compliance of the Left Ventricle

IT HAS OFTEN been assumed that elevated filling pressures of the ventricles are indicative of myocardial failure. As emphasized by Braunwald and Ross, factors other than the functional state of the myocardium may be responsible for substantial alterations in ventricular end-diastolic pressure. Of these factors, perhaps the most important is the diastolic compliance of the ventricle. In a general sense, the term ventricular compliance refers to the distensibility of the relaxed ventricle, defined in terms of its diastolic pressure-volume relationship. While it is true that most patients with myocardial failure exhibit elevated diastolic filling pressures, the subject with left ventricular hypertrophy, infiltrative disease of the myocardium, or constrictive endocardial or pericardial disease may have markedly elevated end-diastolic pressures and a normal end-diastolic volume as a consequence of a noncompliant ventricle, with little evidence of depressed myocardial function. A better understanding of these and other cardiac syndromes will require a close examination of diastolic as well as systolic properties of the heart.

A variety of physiologic expressions have been used to characterize the diastolic pressure-volume (P-V) relationships of the left ventricle. The expression dV/dP, representing the change in ventricular volume per unit change in diastolic filling pressure, may be considered an index of distensibility of the entire ventricle. The reciprocal of this expression (dP/dV) has been used as an index of stiffness or elasticity of the ventricle. When either of these indices is normalized for ventricular volume, the resultant figures can be expressed in terms of a coefficient or modulus. Thus, dV/VdP represents a coefficient of distensibility or compliance of the ventricular myocardium, while VdP/dV has been called the modulus of elasticity. Admittedly, some workers would disagree with the above description of these terms. However, it is of great importance to identify precisely the terms used in a particular assessment of ventricular compliance, and to define which methods were being employed and the assumptions made in each instance.

Of signal importance in any consideration of ventricular compliance is the fact that the diastolic P-V curve is not linear, and the compliance of every ventricle falls as the filling pressure rises. A structurally normal ventricle, then, may exhibit the same low compliance as a scarred or hypertrophied ventricle if its filling pressure is elevated sufficiently. Furthermore, variations in dV/dP from one heart to another will have a different implication than variations in dV/VdP. Consider, for example, two ventricles: one, the

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consequence of concentric hypertrophy with a normal or small end-diastolic volume; the other, the result of advanced myocardial failure with a greatly dilated ventricle. Let us assume that in both instances compliance (dV/VdP), calculated at end-diastole, was decreased to one fourth of normal. Thus, at that moment (end-diastole), the distensibility of a unit volume of the ventricle was the same in the two hearts. However, if we consider the ventricle as a whole, the distensibility (dV/dP) of the dilated ventricle will be substantially greater than the heart with concentric hypertrophy. In one instance (dV/VdP) it is the distensibility of the muscle which is under scrutiny, while in the other (dV/dP) the entire ventricle is examined.

The variable compliance of a given ventricle is based on a P-V curve which is exponential rather than linear. This exponential relationship has been demonstrated repeatedly in the freshly isolated dog heart except at the extremes of the stress-strain curve. Similarly, Noble et al. have confirmed this observation in the conscious dog. These workers further sought to examine whether mechanical properties other than compliance influenced the P-V relations of the dog left ventricle. Their experiments indicated that neither plastic properties of the ventricle nor series viscosity influenced these P-V relations. Viscous and inertial properties, however, were felt to affect the P-V curve, although other workers failed to demonstrate this effect.

It would appear, then, that in the normal dog left ventricle, as a consequence of an exponential P-V curve, the relationship between dP/dV and P is linear and, as shown by Diamond and co-workers, the slope of this linear function provides a constant which is affected primarily by changes in ventricular wall stiffness. The degree to which other variables affect this constant is not fully known. In this regard, Diamond and his associates observed that changes in ventricular geometry did affect the slope of the dP/dV to P relationship, although substantially less than alterations in wall stiffness. It should be noted, however, that the changes in geometry produced in these experiments were both acute and traumatic, and the extent to which geometric differences among diseased human hearts affect the slope of the dP/dV to P relationship remains unknown.

The technics used for estimating the compliance of the human left ventricle have varied greatly. Bristow et al. measured the volume change occurring during the last 0.2 sec of diastole and related this to the pressure difference produced by atrial systole. Diamond and Forrester determined total diastolic ΔP/ΔV by dividing the difference between end-diastolic and initial diastolic pressure by the stroke volume. By pooling the data of similar patients, the slope of a plot of ΔP/ΔV vs mean diastolic pressure provided a "passive elastic modulus" of the left ventricle. Analysis of these relationships proved of some prognostic value in patients with acute myocardial infarction. A different technic has recently been used by Gaasch et al. Assuming a linear relationship between log P and V and a constant pressure intercept at zero V, compliance (dV/VdP) was calculated at end-diastole from the slope of the log P-V relationship. In this fashion, decreased compliance at end-diastole was observed in patients with hypertrophy and small end-diastolic volumes and in those with markedly dilated ventricles due to congestive cardiomyopathy. While all of these methods have serious shortcomings, it is likely that each provides useful estimates of ventricular distensibility or compliance.

The information provided by these estimates of ventricular compliance has considerable theoretic and practical value. The experimental studies of Hood et al. indicate that elevated filling pressures of the ventricle following acute myocardial infarction may not signify ventricular failure, and that the observed fall in compliance 3–5 days following coronary ligation may represent a mechanism for improving ventricular function during recovery from acute myocardial infarction. Indeed, the role played by altered compliance of the left ventricle is essential to the clinical distinction between myocardial failure and
Thus, the symptom. Thus, the setting for congestive failure without myocardial failure is provided by the noncompliant ventricle. Other examples of congestive failure without myocardial failure include acute glomerulonephritis, volume overload, systemic arteriovenous fistula, beri, cirrhosis, etc. In each case, the myocardium may be functioning normally but near the peak of the ventricular function curve where filling pressures are high and ventricular compliance low. On the other hand, cardiologists are quite familiar with the occasional patient with mitral regurgitation or with congestive cardiomyopathy who has a dilated, hypokinetic ventricle and near-normal pulmonary venous and ventricular filling pressures. In this instance myocardial failure exists without congestive failure by virtue of a highly compliant ventricle. The result is a low-output state without pulmonary congestion.

Many of the clinical signs of congestive failure, therefore, are less indicative of myocardial failure than they are of a reduced myocardial compliance. Pulmonary rales, dyspnea, ventricular gallop rhythm, and even the characteristic “square wave” Valsalva response need not imply depressed myocardial contractility. In the case of the Valsalva test, a given reduction in effective filling pressure during the strain phase of the test will cause a greater reduction in ventricular dimensions (and therefore sarcomere length) at a low initial end-diastolic pressure than at a high pressure. Thus, the role of the Frank-Starling mechanism induced by a standard Valsalva maneuver will be less in the less compliant ventricle, irrespective of whether the noncompliant ventricle is the result of chronic myocardial disease or acute hypervolemia. It is clear from the foregoing that an evaluation of myocardial performance cannot be made in diseased hearts if one employs end-diastolic pressure, end-diastolic volume, or even end-diastolic stress as the index of muscle stretch. The extent to which a muscle fiber is stretched by a given preload will depend upon the compliance of that muscle. Thus, while indices of systolic performance can be easily measured in diseased heart, normalization of the horizontal axis of a ventricular function curve (fiber stretch) has been difficult. In recent years there has been growing enthusiasm for using isovolumic contractile element velocity measurements to assess the contractile state of the heart. Part of this enthusiasm was due to the fact that normalized values for $V_{\text{max}}$ were obtained without measuring ventricular dimensions or compliance. However, serious practical as well as theoretic objections to these isovolumic velocity measurements have been emphasized, not the least of which is how the diastolic load and extension of the contractile element is measured, and how it is then dealt with in the muscle model. These frustrations have led some workers to turn again to length-tension relationships with the hope of providing normalized values for muscle stretch in diseased hearts. Gaasch and associates have used compliance values as a means of estimating relative sarcomere lengths in diseased human hearts. Employing the product of end-diastolic stress and end-diastolic $dV/dP$ as a measure of “muscle fiber stretch,” subjects with hypertrophy, particularly those with IHSS, were found to have low values for muscle fiber stretch indicating short sarcomeres, while those with advanced congestive cardiomyopathy surprisingly had values for muscle fiber stretch which were within the normal range. These values of muscle fiber stretch, in turn, were related to systolic indices of ventricular performance to provide normalized ventricular function curves or length-tension diagrams.

Implicit in these observations is the realization that pump function of the heart cannot be studied adequately without a consideration of muscle function. It would also seem clear that if either analysis of cardiac performance is to enjoy a renaissance, characterization of the
diastolic properties of the diseased ventricle will be requisite.

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References
