that, even if distensibility varies, pressure cannot fall as volume increases. Even if distensibility could become infinitely large as the ventricle continues to relax, the pressure would be required to increase as volume increases. Using this equation, our data showed a calculated negative value of distensibility. In our judgment, the very measurement of a negative distensibility means that the ventricle was undergoing a self-enlarging action.

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Additional Comments (and Response to Drs. Sabbah and Stein)

The usual definitions of ventricular distensibility should not yield negative values from data in early diastole. When the definition first used by Sabbah and Stein ($D = V/P$) is applied with the correction which I suggested (see above), negative values of distensibility are not obtained. The definition used by Mirsky ($D = dV/dP$) was proposed for a passive ventricle; one in which distensibility does not change with time. This latter definition could be modified to include the possibility of a varying ventricular distensibility during diastole by using partial derivatives, i.e., $D = \delta V/\delta P$, in which the increments of volume and pressure are obtained with time held constant. This may be approached experimentally by rapidly (rapid in comparison to the time course of change in distensibility) introducing increments of volume ($\delta V$) and observing the effect or pressure ($\delta P$) at various time intervals in diastole. Measured in this way, $D$ always will be positive and finite throughout the cardiac cycle, but will vary significantly at various points in the cycle.

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Comments on
"Pressure-Diameter Relations during Early Diastole in Dogs: Incompatibility with the Concept of Passive Left Ventricular Filling"
and
"Negative Diastolic Pressure in the Intact Canine Right Ventricle: Evidence of Diastolic Suction"
which appeared in
and

In two recent elegant studies, Sabbah and Stein (1981a, 1981b) described that the early rapid filling of the heart is due to forces within the ventricular wall that act to restore the ventricle to its diastolic dimensions. Pressure in the ventricles will be lower than that in the atria, and blood will be drawn from atria to ventricles. These authors reemphasized thereby the familiar concept of diastolic suction, as resulting from the contribution of elastic recoil ("passive" restoring forces) and "active" restoring forces thought to result from compression of sarcomeres (Farsons and Potter, 1966).

The concept of diastolic suction and the proposed mechanisms that determine relaxation can be more fully appreciated by taking into consideration the dual control of relaxation in the mammalian heart (Brutsaert et al., 1978a; Brutsaert et al., 1980; Goethals et al.). As summarized in a recent Brief Review in Circulation Research (Brutsaert et al., 1980), relaxation is controlled by the continuous interplay of the high sensitivity of the myocardium to the loading conditions (load dependence), requiring well-functioning calcium-sequestering membranes (Brutsaert et al., 1978b; Lecarpentier et al., 1979), and the rate of inactivation (inactivation dependence).

Diastolic suction requires the cooperation of load-dependent relaxation and the prevailing loading conditions, of which (i) elastic recoil (due to release of potential energy stored during systole), (ii) arterial impedance, (iii) filling of the coronary reservoir and (iv) implications of the Laplace relationship constitute the four major components in the intact heart. Among these four components, elastic recoil (as extensively discussed by Sabbah and Stein) and the Laplace load will become predominant after mitral valve opening. Provided muscle relaxation is load-sensitive, these two loadings will result in the almost explosive character of early diastolic filling in physiological conditions. In this sense, the phenomenon of diastolic suction is merely a manifestation of load dependence of relaxation.

In the absence of load sensitivity of relaxation, such as in frog heart or in mammalian heart after hypoxia or caffeine (Chuck et al., 1981; Paulus et al., 1982) diastolic suction cannot easily be demonstrated. By contrast, in high inotropic states and some clinical conditions, such as mitral stenosis, constrictive pericarditis, and cardiac tamponade where end-systolic volumes are smaller, cardiac muscle fibers may indeed
benefit from additional restoring forces accumulated at lengths shorter than slack length (Rademakers et al., 1981). This adds further support to the concept of diastolic suction proposed by Sabbah and Stein, as manifested by negative early diastolic pressures, particularly under the clinical conditions cited above.

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