Effect of Heart Irregularity on Left Ventricular and Arterial Peak Systolic Pressures in Aortic Stenosis

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In aortic stenosis, systemic arterial peak systolic pressure may remain relatively steady in the face of marked, arrhythmia-induced, variations in left ventricular peak systolic pressure. A possible basis for this phenomenon is presented. It is suggested that beat-to-beat variations in the systolic pressure gradient across the aortic valve region may not always reflect variations in stroke volume.

Under most circumstances when the pulse is irregular, systemic arterial systolic pressure closely mirrors the variations in left ventricular systolic pressure although the 2 are not necessarily identical. By contrast, left heart catheterization of patients with aortic stenosis, in whom the pulse was irregular, revealed exaggerated beat-to-beat variation in left ventricular peak systolic pressure, with little variation in simultaneously recorded systemic arterial peak systolic pressure. The purpose of this communication is to call attention to this phenomenon, and to provide a possible explanation.

Material

All left heart catheterization data (posterior percutaneous approach1 and data obtained at the time of cardiac surgery were reviewed. Cases demonstrating beat-to-beat variations in left ventricular systolic pressure with simultaneously recorded radial, brachial, or aortic pressure were selected for analysis. Variations were due to arrhythmias or were respiratory. Arrhythmias included premature beats, atrial fibrillation, and sinus arrhythmia. Aortic stenosis was present in some cases, absent in others. The cases selected for presentation are representative.

Results

Figures 1 and 2 depict simultaneously recorded left ventricular and systemic arterial pressure pulses in 2 cases of aortic stenosis. Noteworthy, particularly in figure 1, are the markedly variable left ventricular peak systolic pressures and the much less variable arterial peak systolic pressures. The systolic pressure gradient across the aortic valve region is correspondingly variable. In contrast, in the absence of aortic stenosis (fig. 3), systemic pressure mirrors left ventricular pressure with great fidelity.

The relationship of ventricular and arterial peak systolic pressure level to previous cycle length has been plotted as a pressure-time curve for each of cases 1 and 3 in figures 4 and 5 respectively. The expected dependence of left ventricular peak systolic pressure level on previous cycle length is clearly indicated in both figures. Noteworthy is the flat arterial curve in figure 4 in the presence of relatively severe aortic stenosis, contrasting with figure 5 in which, stenosis being absent, the arterial curve parallels the ventricular curve.

Discussion

The left ventricular pressure-time curves in figures 4 and 5 are of a contour one would expect from the classic observation of dependence of ventricular systolic pressure on end-diastolic volume.2 In the arrhythmias under discussion, a greater diastolic filling time presumably acting through increased end-diastolic volume, results in a more forceful ventricular contraction. The force of ventricular contraction is a major determinant of left ventricular systolic pressure. In the normal heart, the left ventricle and major arteries act as a common chamber during systole, pressures being approximately equal al-

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though in the peripheral vessels some variation occurs due to deformation in transmission of the pulse. When aortic stenosis is present, the left ventricle and systemic arterial tree can no longer, from the physiologic point of view, be considered as a single chamber in systole. The severity of the stenosis is an important determinant of the systolic pressure gradient across the aortic valve region. This pressure gradient, however, varies not only with the degree of stenosis but apparently also with the square of the stroke volume. While it is evident from the foregoing that relatively small changes in stroke volume may markedly affect the gradient, consideration of the hemodynamics in individuals without aortic stenosis would lead to the conclusion that arterial systolic pressure in aortic stenosis should still mirror left ventricular systolic pressure but with less marked variations as the gradient widens or narrows from beat to beat in relation to changes in stroke volume. Case 1 (figs. 1 and 4) illustrates how variations in left ventricular peak systolic pressure as great as 120 mm. Hg may be almost totally unreflected in the systemic arterial pulse, and suggests that other factors are active.

In aortic stenosis, left ventricular peak systolic pressure is determined primarily by the force of ventricular contraction and the resistive force of the stenotic valve. The assumption appears reasonable that the stenotic resistive force is dependent upon the force of left ventricular contraction; the greater the contracting force, the greater being the resistive force. Systemic arterial peak systolic pressure would be determined primarily by the force of ventricular contraction minus the stenotic resistive force. Should the resistive force vary with the contracting force, a possible result might be partial or almost complete failure of arterial peak systolic pressure to reflect variations in left ventricular systolic pressure, as best seen in figure 1. Related to this is the effect of graded severity of stenosis on stroke volume. The latter may be expressed as mean velocity of ejection × cross sectional area of the valve orifice in systole.
If aortic stenosis exceeds a certain critical severity, variations in force of ventricular contraction may no longer be able to influence velocity of outflow. It then follows that stroke volume will vary only with the duration of ejection. While variations in duration of ejection do occur, they are small and could not be expected greatly to influence stroke output from beat to beat in these circumstances. Thus, the almost complete lack of variation in arterial systolic pressure levels could be attributed in part to a relatively fixed stroke output, and the pressure gradient might then vary widely while the stroke output remains relatively fixed. A lack of correlation between pressure gradient and stroke volume would result when the circumstances illustrated in figure 1 exist.* Other factors contributing to systemic arterial systolic pressure level, such as peripheral vascular resistance, volume-elasticity characteristics of the systemic arterial tree, etc., are well known, and will not be further detailed here. There appears to be little reason for assuming that their quantitative contributions to beat-to-beat variations in arterial systolic pressure in the presence of aortic stenosis differ in any important respect from the normal.

When a more variable arterial peak systolic pressure is noted (fig. 2), one might assume either a less severe degree of stenosis or alternatively, a less rigid form of stenosis, in which the stenotic opening alters in size with changes in the force of ventricular contraction.

While the practical importance, in the assessment of aortic stenosis, of a systolic pressure gradient across the aortic valve region cannot be denied, particularly when flow past the aortic valve can be taken into account, the above observations cast some doubt on the assumed simple relationship be-

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*Expressed in terms of external work of the ventricle \( EW = PQ + \frac{Mv^2}{2g} \), where \( EW \) = external work, \( P \) = ventricular mean systolic pressure, \( Q \) = stroke volume, \( M \) = mass of blood moved, \( v \) = velocity and \( g \) = gravitational constant), if \( v \) and therefore \( Q \) and \( M \) are maximum and fixed, a rise in \( P \) will determine increase in \( EW \) which is entirely isovolumic and therefore unrelated to flow.

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**Fig. 4** Top. Systolic pressure-time curves for the left ventricle (solid line) and radial artery (dashed line) in aortic stenosis (case 1).

**Fig. 5** Bottom. Systolic pressure-time curves for the left ventricle (solid line) and aorta (dashed line) with no aortic stenosis (case 3).
terial pulse contour has been related in a general way, both experimentally and clinically, to the severity of stenosis. Therefore, the present observation suggests that the degree of stenosis is relatively exaggerated during certain beats. While it is possible that variations in stroke output might be, in part, the basis for this variation, the idea that the resistive force opposing outflow is in a sense variable, depending on the force of ventricular contraction, is a more attractive hypothesis.

The possibility exists that a relatively fixed systemic arterial systolic pressure level in the presence of an irregular pulse may be an additional clinical reflection of severe aortic stenosis.

**Summary**

In aortic stenosis with an irregular pulse, a relatively less variable or almost constant peripheral arterial peak systolic pressure is described in association with marked variation in left ventricular systolic pressure. Possible mechanisms for this phenomenon are discussed.

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**Summario in Interlingua**

In stenosis aortica con irregulare ateriae pulmonary quase constantem maximum tensionis peripherico-arterial es describite in association con marcata variationes del tension systolic sinistro-ventricular. Mechanismos possibile de iste phenomeno es discutite.

**References**


I think that knowledge of every kind is useful in proportion as it tends to give people right ideas, which are essential to the foundation of right practice, and to remove wrong ideas, which are the no less essential foundations and fertile mothers of every description of error in practice. And inasmuch as, whatever practical people may say, this world is, after all, absolutely governed by ideas, and very often by the wildest and most hypothetical ideas, it is a matter of the very greatest importance that our theories of things, and even of things that seem a long way apart from our daily lives, should be as far as possible true, and as far as possible removed from error.—THOMAS H. HUXLEY. *American Address with a Lecture on the Study of Biology*. London, MacMillan and Co., 1877, p. 142.
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