Analysis of in Situ Heart Mechanics

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Analysis of the *in situ* heart performance has been guided by the concepts of pump and muscle mechanics derived from studies on isolated heart and papillary muscle, respectively. It is occasionally beneficial to evaluate the cardiac performance separately on heart as a pump or as a muscle. Cardiac output is determined by the interaction among preload, afterload and contractility. Thus, it is sometimes difficult to estimate the contractile state of the diseased heart simply from hemodynamic measurements. Therefore, it is necessary to compare hemodynamics between rest and stress states or to apply concepts of "afterload mismatch and preload reserve" as well as end-systolic pressure volume relations in quantitative evaluation of the cardiac performance. Regional wall motion measurements are also useful to detect regional myocardial ischemia.

THE contractile state of the diseased heart has been estimated by hemodynamics or wall motion analysis obtained non-invasively at bed-side or invasively at the cardiac catheterization laboratory.¹ Recent advances in graphic recordings, such as echocardiography, radionuclide angiography and X-ray cineangiography, have provided us the instantaneous contour of the beating heart. From these tracings, we can derive information concerning both total and regional myocardial performance, guided by the traditional muscle and pump concepts. The objective of this article is to review these traditional concepts and to summarize the usefulness and the pitfalls of applying them to the analysis of the behavior patterns of myocardial wall motion.

Performance Characteristics of the Heart

Cardiac performance can be described from the standpoint of both pump and muscle me-

chanics. In clinical cardiology it is occasionally convenient for understanding cardiac performance to distinguish the heart as a pump from the heart as a muscle. We have seen patients with heart failure but without a significant decompensation of the cardiac muscle (for example, valvular heart disease) or those without pump failure but with obvious depression of their myocardial function.

Heart as a Pump

Behavior of the heart as a pump was first described by the relation of filling volume or venous inflow to cardiac output (Starling's mechanism), the so-called heterometric contraction, which indicates that the heart has an intrinsic ability to regulate its performance.² Starling² pointed out two important properties of the heart: 1) filling volume primarily determines the output of the isolated heart and 2) there is a breaking point over which cardiac output decreases in spite of the increased filling volume (Fig. 1). However, the existence of this descending limb has been one of the controversies.

Then, Sarnoff et al³ proposed the concept of a ventricular function curve based on the filling

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Fig.1. Schematic representation of pump function. Four important relations have been used to describe the cardiac performance. In each graph, a solid line represents a control state (basal state) and a dashed line represents a pump performance of enhanced contractility. Depressed contractility is shown by -.-.-. In pressure-volume curve (right bottom), when preload increases from A to D and F without altering both systolic pressure and contractility, end-systolic point C will never deviate from the control solid line and stroke volume increases from B-C to E-C and G-C.

pressure. They are 1) the relation between mean left atrial pressure and left ventricular stroke work, 2) the relation between left ventricular end-diastolic pressure and left ventricular stroke work and 3) the relation between changes in left ventricular fiber length and changes in left ventricular stroke work. These relations between filling pressure and work of the ejecting heart are easily modified by the contractile state (Fig. 1). Accordingly, the ventricular function curve is a practical concept based on the Frank-Starling mechanism.

Frank⁴ derived a concept on the frog heart that the diastolic volume and pressure developed in an isovolumetric contraction analogue to the initial stretch and tension of the isolated skeletal

muscle. On the similar framework, Suga⁵ advocated an end-systolic pressure volume relation that is independent of the initial end-diastolic volume (preload) and the afterload (Fig. 1).

Guyton⁶ noticed that in an *in vivo* heart an increase venous flow usually does not change cardiac output remarkably and they emphasized the relation of right atrial (RA) pressure to venous return as well as cardiac output. They derived two curves: one is the RA pressure and venous return and the other is the relation of RA pressure to cardiac output (Fig. 1). In an *in situ* heart, cardiac output is determined by the equilibrium state of these two relations.

Heart as a Muscle

Analytical studies of skeletal muscle mechanics have been extended to the papillary muscle and then to the intact heart! Muscle mechanics have been described on the framework of preload, afterload and contractility. As shown in Fig. 2, several basic characteristics have been determined: length-velocity curve, active length-tension curve, resting length-tension curve and force-velocity curve. These relations during active phase are sensitively moved upward or downward by a positive or negative inotropic effect.

In the whole heart preload analogues to the end-diastolic pressure, end-diastolic volume, end-diastolic fiber length (wall thickness), mean LA (RA) pressure, mean pulmonary arterial wedge pressure or end-diastolic tension. Afterload also analogues to the systolic pressure, systolic wall tension or input impedance of the arterial tree. However, the solid concept of the afterload in an *in situ* heart is still unsettled.

Comprehensive Analysis of Pump and Myocardial Function

Determinants of the cardiac performance, such as preload, afterload and contractility cannot be rigidly controlled in the intact heart (Fig. 3), because each factor is easily influenced by others. In practice, hemodynamics and chamber size at the basal condition provide the essential information in describing the pump function of the heart. Then assessment of both the basal level of cardiac performance and its directional changes during the stress is helpful not only in detecting the impairment of contractile state, but also in describing the depressed pump performance quantitatively?

Ejection phase indices, such as systolic pressure, stroke volume, stroke work, ejection frac-

Fig.2. Muscle properties are shown. Basal control state is designated by normal as a solid thick line. Positive and negative inotropic effects are shown by upward and downward shifts of lines, respectively. P_o is a maximum tension at the isometric contraction.

Fig.3. Popular two-dimensional analysis of the *in situ* heart performance. As an example, changes in cardiac performance are shown when the preload is increased from A to B. Solid line is a basal control state. Positive and negative inotropic effects are shown by upward and downward thin lines, respectively.

tion and V_{cf} represent the pump performance, but these indices are easily influenced by preload or afterload (Fig. 3) and cannot express the myocardial function directly. Isovolumic phase indices, such as peak dP/dt , $(dP/dt)/P$, $(dP/dt)/DP_{40}$, V_{pm} , V_{max} and $(dP/dt)/IIT$ are sensitive to changes in contractility, however, theoretical problems, extrapolation technique and a necessity for high fidelity pressure recordings along with some preload dependency limit their clinical usefulness.¹ Accordingly, there is no sole index of the contractile state at the present time and it has long been desired to obtain a practical

concept or an index which expresses the contractility and/or contractile state not influenced by changes in preload and/or afterload.

Recently, two concepts have been advocated: 1) afterload mismatch and preload reserve⁷ and 2) end-systolic pressure-volume relations⁵ (Fig. 3). The former concept attempted to describe the failing heart in the framework of preload reserve: when the compensating ability induced by the preload increment is exhausted, the extent or the velocity of systolic shortening is depressed progressively along with increases of afterload, thus signifying an impairment of the

contractile state. As for the latter, Suga⁵ examined the left ventricular pressure-volume relations with a time-varying compliance, $E(t)$ (Fig. 3). This concept has been applied to clinical cardiology, because the end-systolic pressure-volume relation, so-called E_{max} , is insensitive to any change in preload and/or afterload and it represents the contractile state.

Therefore, above-mentioned two concepts may be helpful in evaluating regional as well as total cardiac performance (Fig. 3).

Analysis of Regional Wall Motion

Wall motion can be recorded non-invasively by echocardiography and radionuclide angiography. In the experimental study, regional wall motion has been measured in acute and chronic preparations using miniature piezoelectric crystals with high precision and excellent reproducibility⁸. In both clinical and experimental studies, regional myocardial function has been analyzed by applying the concepts derived from studies in papillary muscle or the isolated heart as described above: 1) chamber size information at end-diastole, 2) ejection phase indices such as extent or velocity of shortening, % shortening, and indices of regional power and regional work, 3) diastolic property such as $\Delta P/\Delta L$ and 4) pressure-length (wall thickness) loops.

Although the regional wall motion might behave similarly to an isotonic contraction of the isolated papillary muscle or an ejection of the isolated heart, segmental shortening sampled from the whole heart is invariably influenced by the muscle shortening in the segment next to the measuring site. We conveniently use the left ventricular end-diastolic pressure and systolic pressure as an index of preload and afterload on that segment, respectively, however, the rationale for this kind of analogy has not yet been proved. It is also unclear how well the traditional muscle and pump concepts are suited in determining the contractile changes of the regional myocardium of the *in situ* heart.

Despite these unsolved limitations, measurements of regional wall motion provide useful information concerning regional ischemia. Namely, regional wall motion is markedly suppressed by coronary occlusion, and direct linear relation between coronary flow and extent of shortening has been repeatedly observed.

Future Goals of Cardiovascular System Analysis

In this article I focused the analysis of cardiac performance on the concept of the heart as a muscle or as a pump. However, it is also essential to understand the interaction of the heart with the venous and arterial trees, both of which are affected by neural, humoral and metabolic conditions. Accordingly, as a future goal, the subsequent problems should be investigated: 1) What are the best practical indices in evaluating the contractile state? 2) What is the limitations of the traditional concepts in analyzing regional wall motion of the *in situ* heart? 3) Is there a practical model of the cardiovascular system for a quantitative understanding of the pump function and its interaction with the vascular system? 4) Is it possible to measure left ventricular pressure non-invasively?

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