Functional Characteristics of the Left Ventricular Inflow and Outflow Tracts

By Donald V. Priola, B.S., Charles E. Osadjan, M.D., and Walter C. Randall, Ph.D.

The heart chambers contract sequentially and any significant alteration in spread of the electrical activity may bring about a change in sequence. While recording contractile force from multiple sites on a single ventricle, it was noted that different portions of the ventricular myocardium showed consecutive onset of systole. Puff employed high-speed cinematography to study changes in ventricular configuration during systole. He reported that the inflow and outflow tracts contract consecutively, simulating a peristaltic wave along the main blood pathway through each ventricle. His observations suggest that, during ventricular systole, the oblique tracts of fibers twist and the papillary muscles become opposed in such a way as to produce physical separation of the inflow and outflow tracts.

While studying synchrony of ventricular contraction, we observed that the intervals between initial pressure elevations in different chambers were influenced by the position of the catheters within the chambers. We also noted that the wave form of force recordings from the ventricular muscle depended strongly upon the placing of the strain gauges relative to the orientation of the muscle layers. We examined therefore the inflow and outflow tracts of the left ventricle to learn whether these are simply convenient anatomical terms or represent functional cardiac chambers. This report describes the results of these investigations.

Methods

Mongrel dogs were anesthetized with phen-cyclidine HCl (2 mg/kg IM) and α-chloralose (60 to 80 mg/kg IV). Thoracotomy was performed in the left fifth interspace and the entire sixth rib was removed. The animals were maintained on positive-pressure respiration. All pressure and force data were recorded on an Offner type R ink-writing oscillograph at a paper speed of 250 mm/sec. Both the force and the pressure signals were capable of faithful recording (output > 90% of input) to 125 cycles/sec.

In order to record multiple intraventricular pressures, specially constructed intraventricular cannulas were inserted into the inflow and outflow tracts of the left ventricle (fig. 1). The inflow tract cannula was inserted through the lateral portion of the free wall about 1 cm below the A-V groove. Outflow pressure was obtained from a similar cannula in the anterior wall situated midway between the base and apex and about 1 cm lateral to the interventricular septum. The inflow tract extends from the mitral valve to the apex of the ventricle, and is bounded laterally by the free wall and medially by the anterior and posterior papillary muscles. Dimensions of the interpapillary space appear to vary considerably during the cardiac cycle. The outflow tract extends from the apical region to the aortic valve, and is bounded by the interventricular septum and the papillary muscles. Care was taken to avoid placing the cannula in intraventricular "pockets" at the base of the papillary muscles. Catheter placement was confirmed by postmortem observation and all data from experiments with questionable placement were discarded. Each of the cannulas was connected to a Statham P23Db transducer via identical lengths of PE50 polyethylene tubing, calculated to give a slightly underdamped recording.

From the Department of Physiology, Stritch School of Medicine and the Graduate School, Loyola University, Chicago, Illinois.

Supported by Grants HE-02705 and HE-08682 from the National Institutes of Health, U. S. Public Health Service.

Accepted for publication January 4, 1965.
Diagrammatic representation of interior of the left ventricle showing positions of cannulas in inflow and outflow tracts, hypothetical boundaries of the two tracts, and direction of flow (arrows). LA: left atrium, A and P: anterior and posterior papillary muscles, RA and RV: right atrium and ventricle.

Direct recordings of the instantaneous pressure differentials between the inflow and outflow tracts were obtained by connecting the inflow and outflow cannulas to a Pace P21D differential pressure transducer. Because this transducer measures the pressure differential across a single diaphragm, the possibility of error resulting from phase differences between two transducer diaphragms was eliminated. The Pace transducer has a frequency response that is flat to 600 cycles/sec.

Force recordings of inflow and outflow tract contractions were obtained by suturing Baldwin SR4 strain gauges (modified Walton gauges) directly to the ventricular surface. The stitches which fixed the gauges in position penetrated to a depth of about 5 mm.

In order to determine the interior configuration of the ventricle during systole and diastole, plaster of Paris was injected into the ventricle via the left atrium: (a) in the heart of a freshly-sacrificed animal, (b) immediately before the heart was stopped in diastole following the infusion of KCl, or (c) before the heart was stopped in systole following the infusion of CaCl2. The entire heart was then excised and immediately immersed in liquid nitrogen. After the heart was completely frozen, it was cut transversely and each section photographed.

Results

Simultaneous pressures from the inflow and outflow tracts of the left ventricle were successfully recorded in 14 animals. Faithfully recorded pressure tracings from both inflow and outflow tracts showed the characteristic components of a ventricular pressure curve. The presystolic or atrial filling wave was frequently prominent in both traces. In 10 of the 14 experiments, the first elevation of intraventricular pressure, measured from the onset of the entrant phase occurred in the inflow tract. This was followed 4 to 18 msec later by the beginning of pressure rise in the outflow tract (fig. 2). In the upper panel are the intraventricular pressure recordings from the inflow (top) and outflow (bottom) tracts of the left ventricle. The interval between the beginning of the entrant phases of systole in the two tracts is indicated by the two vertical lines and measures 12 msec. The lower panel shows a record taken from the same animal a few minutes later with amplifier gain increased by a factor of ten and with the pen excursions electronically limited. This amplified the diastolic and entrant periods but truncated the pressure curve be-
between early and late systole. The entrant phases were more sharply defined and measurement of the inflow-outflow interval facilitated. This interval measured 17 msec as denoted by the two vertical ordinates. In the remaining four animals of this series, the pressure elevations in the inflow and outflow tracts were simultaneous or only slightly asynchronous. On rare occasions, and particularly during arrhythmias, outflow tract pressure rose slightly before inflow tract pressure.

Direct recordings of the instantaneous inflow-outflow pressure gradients in the left ventricle were obtained in 12 additional animals. In 10, the differential pressure trace indicated earliest pressure development in the inflow tract (fig. 3). The initial small downward deflection is presumably related to left atrial contraction. The differential pressure approaches zero during the development of the T wave of the ECG, and in late systole the tracing indicates a higher outflow tract pressure. This is the type of curve which would be expected if the two intraventricular pressures are similar in form, amplitude and duration but displaced in time.

Thus, it appeared that the mechanical contraction of the inflow tract preceded that of the outflow tract. This was explored in four animals with strain gauges applied to the left ventricular myocardium in the same areas in which the inflow and outflow tract cannulas were inserted routinely (fig. 4). The inflow tract force recording showed a definite rise 20 msec before the initial increase of force in the outflow tract. When the strain gauges were moved closer together, this asynchrony became less apparent and it was exaggerated if the gauges were moved farther apart. This confirmed the suggestion2,3 that mechanical contraction proceeds in a wave-like fashion across the ventricle.

The apparent asynchrony of pressure changes in the inflow and outflow tracts is difficult to explain if the ventricle is truly a single, openly communicating chamber throughout systole. If this were the case, pressure in such a system should be transmitted rapidly to all parts of the chamber, and a differential could not exist except for the brief period required for the transmission of pressure. This required a measurement of conduction velocity of a pressure wave across the ventricle.

Catheters were inserted into the inflow and outflow tracts of the left ventricle as described above. An impact of essentially constant force and rate of application was delivered to a small area on the external surface of the heart as close to the inflow tract cannula

![Figure 3](http://circres.ahajournals.org/)

**FIGURE 3**

Simultaneous recordings of pressure pulses in the left atrium (LA) and mammary artery (BP), lead II of the ECG, and differential pressure trace from inflow and outflow tract cannulas in the left ventricle (LV<sub>0</sub>-LV<sub>i</sub>). Note simultaneous ordinates at right of figure.

*Circulation Research, Vol. XVII, August 1965*
as possible. The resulting pressure wave was easily detectable in the ventricular recordings. Assuming the distance between the two catheters to be constant, the apparent conduction velocity of the superimposed pressure wave was calculated during successive portions of the cardiac cycle (fig. 5). The straight line distance between the cath-

**FIGURE 4**

Strain gauge force recordings from muscle overlying the inflow (top) and outflow (middle) tracts of the left ventricle. Bottom trace shows left ventricular pressure pulse recorded from the outflow tract.

**FIGURE 5**

Impact pressure waves are shown superimposed upon pressure pulses recorded from the left ventricular inflow (top) and outflow (bottom) tracts at successive periods of the ventricular cycle. Each panel shows impact waves delivered in the inflow tract and recorded simultaneously in both traces. Apparent conduction velocity of the impact wave between the tracts is shown.
Ventricular inflow and outflow tracts

Parameters were measured postmortem and found to be 5.5 cm. The upper record in each panel represents inflow tract pressure and the lower panel outflow tract pressure. In mid-diastole, the transmission time was 4 msec and the apparent conduction velocity was calculated to be 14 m/sec. In late diastole, the transmission time was unchanged and the apparent velocity again was 14 m/sec. However, in early systole, transmission time lengthened abruptly to 8 msec and the apparent conduction velocity decreased to 6.9 m/sec. During maximum systole, the impact wave was markedly attenuated and could not be clearly detected in the outflow tract pressure tracing. When the impact was delivered in early diastole, apparent conduction velocity was reduced to 5.5 m/sec. Delivery of the impact to the ventricular wall over the outflow tract revealed comparable alterations in velocity of transmission.

In order to visualize the internal configuration of the ventricle in the various stages of the cardiac cycle, three techniques were used: 1) Hearts were excised from freshly-sacrificed animals, filled with plaster of Paris which was allowed to set, and then sectioned and photographed. 2) Hearts were arrested in diastole by intracoronary or intravenous infusion of KCl, filled with plaster of Paris, frozen in liquid N₂, sectioned and photographed. 3) Hearts were arrested in systole by CaCl₂ infusion via the left atrium or femoral vein and treated in a manner identical to the second group. A photograph of a macrosection of a heart arrested in diastole by KCl is illustrated in figure 6A. It is clear that the left ventricle consisted of a single chamber with the papillary muscles widely separated. Hearts from freshly-sacrificed animals showed similar internal configuration.

Hearts which were sectioned and examined after arrest in systole by CaCl₂ exhibited a completely different appearance (fig. 6B). The left ventricular cavity was discontinuous, the inflow and outflow tracts being separated by the apposition of the anterior and posterior papillary muscles. Basilar sections of hearts in the CaCl₂ series showed partitioning of the ventricle by the dependent mitral valve leaflet.

Discussion

The terms “inflow tract” and “outflow tract” are not new. They have been used clinically for many years to relate ventricular anatomy to the functional pathway of blood through the working heart. However, the description of the sequential and reciprocal contractions of the two tracts offered by Puff emphasizes this functional relationship. He describes an initial contraction of the inflow tract which causes expansion of the outflow tract, and is followed by contraction of the outflow tract beginning with the subaortic recessus. During contraction the oblique tracts of fibers in the inflow tract twist and

Circulation Research, Vol. XVII, August 1965

FIGURE 6

Photographs of macrosections through the apical portions of two dog hearts injected with plaster of Paris and frozen instantaneously in liquid nitrogen. A: heart stopped in diastole; arrows indicate the widely separated papillary muscles in the left ventricle. B: heart stopped in systole. Shows close apposition of the anterior and posterior papillary muscles in the left ventricle completely separating the inflow (right) from the outflow (left) tract.
the papillary muscles become intertwined, closing the interpapillary space. Thus a new wall, nonexistent in diastole, is formed by the papillary muscles and the contracted recessus. If this hypothesis is correct, a distinct intraventricular pressure gradient may be predicted during systole. Examination of differential pressure recordings from inflow and outflow tracts (fig. 3) revealed precedence in the inflow side, giving functional verification of the motion picture analysis of Puff.

If the straight-line distance between the recording catheters is assumed to be constant throughout the cardiac cycle, apparent conduction velocity can be computed, as in figure 5. The apparent decrease in conduction velocity during systole might be explained: 1) by differences in the physical properties of the myocardium during systole and diastole, 2) by alterations of the intercatheter distance as a result of the change in systolic intraventricular dimensions, 3) by interposition of some intraventricular structure between the tracts. A decrease in pressure wave velocity would be expected if the distensibility of the systolic myocardium increased. We have no specific information concerning this. However, during the isovolumetric phase of systole, the increase of myocardial tension is great for a negligible change of fiber length and therefore, a decrease of distensibility should be expected. If data derived from striated muscle can be applied to cardiac muscle, the experiments of Buchthal and Rosenfalck should be relevant. These workers found that the elastic modulus (reciprocal of distensibility) of striated muscle was higher in contracting muscle than in relaxed muscle. Distensibility of the myocardium would thus decrease during systole, resulting in increased conduction velocity rather than reduced velocity. In this context the decrease in apparent conduction velocity during systole results probably from something other than a change in the physical characteristics of the myocardium. Increased intercatheter distance would also cause the apparent conduction velocity to decrease. However, Hawthorne has shown that the internal diameter of the ventricle decreases during systole. It must be concluded, therefore, that the effective path length of the pressure wave increases, or transmission of the wave is impeded by the interposition of some intraventricular structure between the inflow and outflow tract catheters. These conclusions are made tenable by the anatomical studies that compare the heart arrested in diastole with the heart arrested in systole.

It is believed generally that the papillary muscles and the trabeculae carneae are the first areas of the ventricles to enter mechanical contraction. By their contraction, the papillary muscles tend to increase their diameter, pull the edges of the mitral valve cusps downward and cause a decrease in the longitudinal dimension of the ventricle. All these changes tend to separate the ventricular cavity into an inflow and outflow tract. If this hypothesis is correct, the separation of the ventricle into inflow and outflow tracts should not occur when the papillary muscles are not allowed to come into apposition with each other during systole. Two preliminary experiments have demonstrated that, when the ventricular volume is increased by partial occlusion of the aorta, the inflow-outflow asynchrony disappears.

Severe subendocardial hemorrhages have been reported on the papillary muscles after prolonged stimulation of the stellate ganglion or after catecholamine infusion. It is believed that this results from increased cardiac contractile force and greater systolic ejection with direct mechanical impact between the anterior and posterior papillary muscles. The incidence of these lesions decreases during partial aortic occlusion or infusion of large amounts of fluid which maintain systolic volume at a high level.

**Summary**

Simultaneous pressures were recorded from the left ventricular inflow and outflow tracts in 14 dogs. In 10 the inflow tract entered systole 4 to 18 msec before the corresponding outflow tract. In 12 additional animals, differential pressure recordings from the two tracts confirmed precedence in the inflow tract. In four animals, strain gauge recordings...
from the epicardial surfaces of the inflow and outflow tracts exhibited asynchrony with precedence in the inflow tract.

Hearts excised from freshly-sacrificed animals or hearts stopped in diastole by KCl infusion showed a single, openly-communicating ventricular chamber. In contrast, hearts arrested in systole by infusion of CaCl$_2$ revealed a separation of the left ventricle by the apposition of the anterior and posterior papillary muscles and the dependent mitral valve leaflet.

The apparent conduction velocity of an impact pressure wave from inflow to outflow tract depended upon the portion of the cardiac cycle during which it was delivered. From a maximum of 14 m/sec in mid and late diastole, it decreased to 6.9 m/sec in early systole and 5.5 m/sec in early diastole. Complete attenuation of pressure wave transmission often occurred during maximum systole.

These data support the concept that the left ventricle may become separated, during systole, into an inflow and outflow tract. It appears that the division is accomplished by a change in the systolic ventricular architecture, with apposition of the anterior and posterior papillary muscles and the downward movement of the mitral valve leaflets. It is probable that this phenomenon is functional in hearts working near the lower limits of their systolic reserve.

References

Functional Characteristics of the Left Ventricular Inflow and Outflow Tracts
DONALD V. PRIOLA, CHARLES E. OSADJAN and WALTER C. RANDALL

Circ Res. 1965;17:123-129
doi: 10.1161/01.RES.17.2.123

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1965 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/17/2/123