Pressure Events of the Cardiac Cycle in the Dog
Normal Right and Left Heart

By Howard L. Moscovitz, M.D. and Robert J. Wilder, M.D.

Simultaneous pressure curves from the cardiac cavities, aorta and pulmonary artery of dogs were recorded by manometers of equal sensitivity. It was found that left ventricular pressure during ejection is practically identical with that of the aorta. In the majority of dogs atrial and ventricular end-diastolic pressures were equal. The temporal relations of events in the right and left heart were similar to those reported in man, but the duration of the cardiac cycle is too dependent on cycle length to permit comparison.

The classic physiologic studies of Wiggers have defined the dynamic details of the phases of the cardiac cycle in the dog. Wiggers, however, commented upon the technical difficulty experienced in attempting to record simultaneously the three pressure pulses of each side of the heart, i.e., left atrium, left ventricle and aorta or right atrium, right ventricle and pulmonary artery. Pressures recorded separately must be reduced to a standard scale of ordinates and artificially superimposed and retraced in order to reproduce graphically and quantitatively the simultaneous pressure relationships of the cardiac cycle. Because of the uncertain reliability of such reconstructed curves, differences of opinion still exist as to the magnitude of the difference between the ventricular and aortic pressures during ejection and as to the agreement of ventricular and atrial end-diastolic pressures.

The use of multiple channel oscillographic instruments and strain gage manometers makes it possible to inscribe at one time and at identical sensitivities the pressure pulses of the entire cardiac cycle of either side of the heart. The use of this technic in studying the left heart in normal man and in patients with mitral stenosis has been reported. This report concerns a reinvestigation of cardiovascular hemodynamics in dogs utilizing these newer methods.

METHOD

Adult mongrel dogs averaging 17 Kg. in weight were anesthetized with intravenous Nembutal, using a dose of 30 mg./Kg. The trachea was intubated and respiration maintained with a pneumo-phyre demand valve supplying 100 per cent oxygen delivered under a cycled pressure of 10–15 cm. of water. Respiration was momentarily suspended during pressure measurements. The pericardium was opened widely and sutured to the chest wall providing a loose hammock support for the heart. Intracardiac pressures were obtained in 40 dogs by direct puncture of the cardiac chambers and great vessels.

The manometric system consisted of three Statham P23A strain gages set up in a horizontal bank, levelled at the height of the midthorax. Each gage was connected to a 122 cm. length of noncompressible vinyl tubing with an inside diameter of 1.9 mm., attached to 1/4 inch long 20 gage needles. The transmission time delay through this system was found to be 0.005 sec. and this figure was deducted from the measured value in determining the relationship of electric to mechanical events. A four-channel oscillographic recorder utilizing a single electronic gun with a vertical sweep and a paper speed of 50 mm./sec. was used. Time lines allowed measurements to the nearest 0.01 sec.

RESULTS

Representative tracings of the pressure events of the cardiac cycle of the left heart are shown in figure 1, and of the right heart in figure 2. Table 1 summarizes the data concerning the duration of the individual phases of the cardiac cycle, the time relationship of
electric to mechanical events, and the pressure levels on both sides of the heart.

The description of the phases of the cardiac cycle, designated by the alphabetic symbols in figures 1 and 2, largely follows that of Wiggers. Atrial contraction begins at A, producing the a wave of the atrial pulse. In the illustrated curves, the end-diastolic pressures in the atrium and ventricle appear to be practically equal, but in some records a brief reversal of the atrioventricular pressure gradient can be detected following the a wave. Ventricular systole begins at B as the pressure starts to rise, slowly at first then more rapidly. The c wave of the atrial pulse begins its ascent at the onset of ventricular contraction. Isometric contraction commences at the point of the crossover of the atrial and ventricular curves, and this point marks the closure of the atrioventricular valve. Isometric contraction terminates at C as the semilunar valve opens and rapid ejection into the aorta or pulmonary artery begins. The pressure of the ventricle barely exceeds that of the efferent artery as they rise together in a parallel manner to reach their common summit at D, the beginning of reduced ejection. Reduced ejection continues as the pressures decline together to E, the onset of the incisura of the aortic or pulmonary artery curve. This point, the termination of systole, may be difficult to locate precisely on the pressure tracing. The phase of protodiastole (E-F) ends at the deepest point of the incisural notch as the semilunar valve closes, and the curves of the ventricle and great vessel then diverge. Isometric relaxation begins at F and continues until the atrioventricular valve opens at G as the ventricular and atrial curves again cross. Rapid diastolic inflow into the ventricle begins at G and the atrial and ventricular curves decline together to the point H, with a barely detectable pressure gradient between them. This falling pressure curve during rapid ventricular filling marks the descending limb of the atrial v wave. With the slow heart rates, a phase of diastasis (H-A) is present as the pressure in the filling ventricle and communicating atrium gradually begin to rise. Diastasis terminates at A when atrial systole initiates a new cycle.

FIG. 1. Simultaneous pressure pulses from the left atrium, left ventricle and aorta of a normal dog. Electrocardiogram is lead 2. Heart rate is 130/min. (A-B) atrial systole; (B-C) isometric contraction; (C-D) rapid ejection; (D-E) reduced ejection; (E-F) protodiastole; (F-G) isometric relaxation; (G-H) rapid diastolic inflow. No phase of diastasis is present.

DISCUSSION

Pressure pulses from atrium, ventricle and efferent artery inscribed in superimposed fashion at equal sensitivities provide complete graphic representations of the pressure events of either side of the heart. The points of crossover of atrial and ventricular curves and the onset of ejection from the ventricle into its great vessel can be precisely designated on such composite curves. Records such as these support the view that ventricular pressure exceeds aortic by a barely measurable amount during ejection. The exact relationship of atrial and ventricular end-diastolic pressures has been the cause of speculation. Little has postulated that a brief reversal of the normal atrioven-
tricular pressure gradient follows atrial contraction and is responsible for presystolic closure of the A-V valves. In the present study, pressure pulses of the right side of the heart were recorded at a sufficiently high sensitivity to test this hypothesis. While not a constant finding, 13 of 28 dogs demonstrated such a phenomenon, indicating that at the atrial \( z \) point, right ventricular pressure may transiently exceed right atrial. This pressure differential may in some cases float the A-V valves into apposition before ventricular systole begins.

The data concerning the duration of the individual phases of the cardiac cycle of the left heart of dogs (table 1) are at variance with those of Wiggers\(^5\) by at most 0.01 sec., in those instances where average values are available for comparison. Accurate correlation with the reported data on the human heart\(^5,6,7\) is difficult because of considerable differences in cycle length in the various studies. However, an examination of the sequence of dynamic events of the two sides of the dog heart yields data essentially similar to the findings of Braunwald and associates\(^8\) in man. Atrial contraction begins sooner on the right, but left ventricular systole precedes that of the right ventricle. Because isometric contraction of the left ventricle is considerably longer than that of the right, ejection into the pulmonary artery precedes ejection into the aorta.

Comparison of other characteristics of the cardiac cycle of the right and left heart can be made from the data. The left ventricle requires 0.06 sec. to elevate its pressure 80 mm Hg in an isometric fashion in order to eject blood into the aorta. Right ventricular ejection occurs after an increment in pressure of only one-eighth to one-tenth this amount, but one third of this time is required (0.02 sec.). This is in part due to the fact that the initial phase of isometric contraction occurs at a slower rate than the final portion, and propor-

**Fig. 2.** Simultaneous pressure pulses from right atrium, right ventricle and pulmonary artery of a normal dog. (Ordinate, mm. Hg.) Lead 2. Heart rate 80/min. Phases of cardiac cycle are lettered in same manner as figure 1. A phase of distasis (\( H-A \)) is present. Time lines 0.04 sec. apart.
TABLE 1.—Summary of Duration of Phases of Cardiac Cycle, Relation of Electric to Mechanical Events and Average Pressure Levels.

<table>
<thead>
<tr>
<th>Event</th>
<th>Avg. Duration (sec)</th>
<th>Left heart (34 dogs)</th>
<th>Right heart (28 dogs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average heart rate/min.</td>
<td>130</td>
<td>125</td>
<td></td>
</tr>
<tr>
<td>Atrial systole (A-B)</td>
<td>0.076</td>
<td>0.063</td>
<td></td>
</tr>
<tr>
<td>Isometric contraction (B-C)</td>
<td>0.050</td>
<td>0.025</td>
<td></td>
</tr>
<tr>
<td>Rapid ejection (C-D)</td>
<td>0.107</td>
<td>0.097</td>
<td></td>
</tr>
<tr>
<td>Reduced ejection (D-E)</td>
<td>0.052</td>
<td>0.085</td>
<td></td>
</tr>
<tr>
<td>Protodiastole (E-F)</td>
<td>0.017</td>
<td>0.020</td>
<td></td>
</tr>
<tr>
<td>Isometric relaxation (F-G)</td>
<td>0.064</td>
<td>0.032</td>
<td></td>
</tr>
<tr>
<td>Rapid diastolic inflow (G-H)</td>
<td>0.073</td>
<td>0.084</td>
<td></td>
</tr>
<tr>
<td>Diastasis (H-A)</td>
<td>0.030</td>
<td>0.035</td>
<td></td>
</tr>
<tr>
<td>Onset P to a wave</td>
<td>0.058</td>
<td>0.045</td>
<td></td>
</tr>
<tr>
<td>Onset Q to ventricular systo-</td>
<td>0.037</td>
<td>0.044</td>
<td></td>
</tr>
<tr>
<td>le</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset Q to ventricular ejection</td>
<td>0.095</td>
<td>0.064</td>
<td></td>
</tr>
<tr>
<td>Pulmonary artery pressure</td>
<td>108/53</td>
<td>21/10</td>
<td></td>
</tr>
<tr>
<td>Aortic or pulmonary artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pressure</td>
<td>%</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Pulse pressure average</td>
<td>25%</td>
<td>12%</td>
<td></td>
</tr>
</tbody>
</table>

Simultaneously this slow phase occupies more of the total isometric contraction period of the right ventricle than the left. The rate of pressure ascent in the left ventricle during isometric contraction is approximately 1300 mm./sec. while in the right ventricle it is closer to 500 mm./sec.

The relative width of the pulse pressure of the pulmonary artery is considerably greater than that of the aorta. The aortic pulse pressure varies from 20-30 per cent of the left ventricular systolic height while the pulse pressure of the pulmonary artery is approximately 60 per cent of the peak right ventricular pressure. This explains in part the fact that ejection of blood into the pulmonary artery usually precedes aortic ejection and persists for a longer period. The shorter phases of isometric contraction and relaxation of the right ventricle, as compared to the left, and the relatively wider pulse pressure of the pulmonary artery than the aorta appear to be dependent upon differences in peripheral resistance and distensibility of the pulmonary and systemic arterial circuits.

SUMMARY

Simultaneous pressure curves of equal sensitivity were obtained from the left atrium, left ventricle and aorta of 34 normal dogs and from the right atrium, right ventricle and pulmonary artery of 28 normal dogs.

It was found that left ventricular pressure during ejection is practically identical with that at the root of the aorta. In the majority of the dogs, atrial and ventricular end-diastolic pressure were equal, but in 13 of 28 dogs a transient reversal of the normal pressure gradient appeared just prior to ventricular systole.

The temporal sequence of events on the two sides of the human and dog heart is similar, but the duration of the phases of the cardiac cycle is too dependent upon cycle length for adequate comparison.

SUMMARY IN INTERLINGUA

Simultanee curvas de pression de equal sensibilitate esseva obtenite ab le atrio sinistre, le ventriculo sinistre, e le aorta de 34 canes normal e ab le atrio dextere, le ventriculo dextere, e le arteria pulmonar de 28 canes normal.

Esseva constatare que le pression sinistro-ventricular durante le ejection es practicamente identic con le pression al radice del aorta. In le majoritate del canes, le pressiones atrial e ventricular al fin del diastole esseva equal, sed in 13 ex 28 canes un transiente reversio del normal gradiente de pression appa-reva immediatemente ante le systole ventricular.

Le successione temporal del evenimentos al duo lateres del corde human e canin es simile. Sed le duration del phases del cyclo cardioes nimir dependent del longor del cyclo pro permetter un comparation adequate.

REFERENCES


2 —: Studies on the consecutive phases of the cardiac cycle. I. The duration of the consecutive phases of the cardiac cycle and the criteria for
their precise determination. Am. J. Physiol. 56: 415, 1921.


The Pulse Form in Sclerotic Arteries

In patients with suspected sclerosis of the large arterial trunks, the characteristics of the central pulse—large amplitude, pronounced incisura followed by rapid decline of the curve—are transmitted to the radial artery. (Wiggers, Ann. Int. Med. 6: 12, 1932). However, this valuable criterion of stiffening of the large arteries has not received much clinical recognition.

The demonstration that femoral pulses taken from patients with sclerosis of the leg vessels also show the same changes, is therefore of timely interest.

Animal experiments confirmed the conclusion that the characteristic femoral pulses are not caused by the demonstrable sclerosis of leg vessels but by reduced distensibility of the aorta.

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