Pressure-Volume Relationships in Right Ventricle

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Recently, we have reported on the pressure developed in the left ventricle when that chamber was made to contract under conditions closely approaching that of being isovolumic. More recently, the use of a latex balloon to change the amount of effort of the right ventricle yielded data on the pressure developed in the right ventricle at different volumes under nearly isovolumic conditions. The method is described in detail in a report published in this issue. It is deemed appropriate to present the data obtained and to compare it with that found in the left ventricle.

The curves (fig. 1) are similar to that seen in the isovolumic left ventricle, except for scale values. The pressure curve increased slightly in duration, the steepness of the pressure rise and fall was augmented, and the early diastolic dip was exaggerated as the volume of the ventricle increased.

The occurrence of spontaneous tachycardia with a concomitant rise of right intracardiac pressure is also illustrated in figure 1. A rise in aortic pressure occurs with the tachycardia, accompanied by a marked change in aortic pressure contour. The marked augmentation in the pressure pulse of the right ventricle is also evident, as is the augmentation of the gradient of pressure rise and fall. An early diastolic dip develops which brings the ventricular pressure below the zero level. This may represent suction (cf. reference 4), or it may be an artifact dependent on underdamping of the right ventricle-latex balloon system. It could be caused by a drop in intraballoon pressure below the ambient pressure because of elasticity of shape of the isovolumic ventricle and its contained balloon, or it could be a drop in this chamber pressure below ambient pressure caused by a movement of the interventricular septum toward the left during the early relaxation of the left ventricle. These possibilities merit further exploration.

L-epinephrine, like spontaneous tachycardia, caused an augmentation of the peak right ventricular pressure at any given volume, augmented the steepness of the pressure rise, abbreviated the duration of the pressure curve, and led to the appearance of an early diastolic dip.

In figure 2, the pressure-volume (P/V) relationship of two dogs is shown. The graph on the left shows the change following the appearance of spontaneous tachycardia; the one on the right shows the influence of L-epinephrine. In these experiments, which are typical, the lower curves, concave upward, show the relation of balloon volume to end-diastolic pressure, and the upper ones, convex upward, are the relation of volume to peak (end) systolic pressure. With the balloon in its cavity, the ventricle was doing practically no external work; it is, therefore, considered to be an isovolumic chamber. The conditions, when no balloon was in place, are assumed to be zero ventricle volume and zero cavity pressure during the cycle (because of the siphonage).

As in the case of the left ventricle, the diastolic P/V curve shows little rise, at first, in end-diastolic pressure as the cavity volume increases and then begins to rise with ever-increasing steepness. No deviation in the
The upper part of this figure shows the change in contour, amplitude, and duration of the right ventricular pressure pulse when the ventricular volume was altered by means of a balloon in its cavity. Time in 0.04 and 0.2 second. The curves represent, from left to right, the pressure pulse when the intraventricular volume was, respectively, 20, 30, and 40 cc. Discussed in text.

The lower part of this figure shows the effect of spontaneous tachycardia. The slow speed record on the left represents the development of the spontaneous tachycardia with its associated rise in aortic blood pressure (lower curve) and augmentation of right ventricular pressure pulse (upper curve). The two fast speed records on the right represent the contour of the aortic and right ventricular pressure pulses during the appearance and after the disappearance of the spontaneous tachycardia. Discussed in text.

Character of the curve was demonstrable when spontaneous tachycardia occurred or when the catecholamine was exhibited.

In the case of the systolic P/V curve, the curve rose rapidly at first as the ventricular volume was increased, and then at a decreasing rate with larger volumes. In one of the four experiments, the systolic P/V curve appeared to reach a plateau and actually became horizontal at 40 cc. volume. In none of the four cases was a real decline obtained in the systolic P/V curve at higher volumes. The systolic P/V curve was similar, except for magnitude, to that of the left ventricle.

Both spontaneous tachycardia and the exhibition of L-epinephrine, caused a shift in the systolic P/V curve such that the pressure for a given volume was increased. No apparent effect on the end-diastolic P/V curve accompanied this change in the systolic curve.

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can mean that no change occurred or that any change was within the experimental error of measurement.

The changes in the right ventricular pressure curve contour following L-epinephrine and spontaneous tachycardia in our experiments as regards the rate of relaxation lends some support to the interpretation proposed by Buckley et al.\textsuperscript{5,6} who suggested that changes in the viscous-elastic properties of the wall can alter the impedance to filling. The changes produced by these procedures on active relaxation contrast sharply with the apparent lack of effect upon the relaxed ventricle which fills passively toward the end of diastole.

**Summary**

Contour changes of the pressure curve of the isovolumic right ventricle were analyzed as its volume increased and during spontaneous tachycardia and L-epinephrine exhibition. Evidence of an early diastolic dip going below ambient pressure was observed when spontaneous tachycardia developed. Evidence was clear that the impedance to filling during the phase of active relaxation lessened in the right ventricle when spontaneous tachycardia occurred and L-epinephrine was exhibited, and to a lesser extent when the ventricle became more dynamic. However, there was no evidence of any shift in the right ventricular pressure/volume relationship at the end of diastole during spontaneous tachycardia or L-epinephrine action. On the other hand, the pressure/volume relationship at the end of systole was altered under these two conditions, in that the pressure for any volume was increased. The curves of the pressure/volume relationship of the right ventricle at the end of systole and at the end of diastole were found to be similar in contour to those of the left ventricle except for the magnitude of change.

**References**

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