LEFT VENTRICULAR PRESSURES IN PATIENTS WITH AORTIC INSUFFICIENCY STUDIED BY INTRACARDIAC CATHETERIZATION

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(Submitted for publication June 22, 1950; accepted, September 21, 1950)

The purpose of this communication is to report on the pressure levels recorded within the cavity of the left ventricle in ten human subjects with aortic insufficiency. The technique of catheterization has been described in detail (1); it should be again emphasized that this may be a hazardous procedure. However, the Mexico workers have carried out 56 left ventricle catheterizations without incident. Pressure tracings were inscribed by means of the Brush Six Channel Oscillograph (2).

The recording reference point was the antecubital fossa of the outstretched and level arm, approximately 5 cm. below the level of the sternum. Figure 1 shows simultaneous pressure tracings from the left and right ventricles and femoral artery.

Obvious congestive failure was present in seven patients, and absent in three at the time of study. Numerous investigators (3–6) have shown by catheterization of the right side of the heart, that

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**Fig. 1.** A Representative Tracing Recorded by the Brush Six Channel Oscillograph Showing Phase of Respiration, Right Ventricular Pressure, Left Ventricular Pressure, Femoral Arterial Pressure and Lead 2 of the Electrocardiogram

The chart speed was 25 mm./sec. Note the mechanical alterations present in the pressure tracings.
the right ventricular diastolic pressure is elevated beyond the normal range of 0 to 5 mm. of mercury in congestive failure. Our data indicate that in left ventricular failure, the left ventricular diastolic pressure is likewise elevated (Table I).

In the three patients without evidence of failure, diastolic pressure levels of 0, 0, and 4 mm. of mercury were recorded, an average of 1.3 mm. of mercury; in the seven patients with congestive failure the left ventricular diastolic pressure ranged between 15 and 39 mm.; with an average pressure of 25 mm. of mercury.

These observations support the conclusions of Wiggers (7). From his studies of the hemodynamic changes created by inducing acutely aortic insufficiency in dogs, he concluded that the amount of blood regurgitated into the left ventricle in human aortic insufficiency may be quite small. When aortic incompetency is thus artificially induced, left ventricular diastolic pressure rises immediately. The low diastolic pressure levels obtained in the three subjects without failure may reasonably be explained by the assumption that during the slow development of rheumatic or syphilitic aortic insufficiency, the left ventricle can compensate for the relatively small and slowly incremental reflux of blood by an alteration in its distensibility. In this fashion diastolic filling pressure would be maintained within normal limits compatible with levels most efficient for cardiac function.

The consistent rise in left ventricular diastolic pressure noted in the seven patients in congestive failure is in keeping with Starling's concept of the failure mechanism: a progressive accumulation of blood within the left ventricular cavity, accompanied by a progressive stretching of the myocardium until a critical point is reached where the muscle fibers offer an increasing resistance to the incoming blood, with a resultant rise in the intracavitary diastolic pressure. The elevated diastolic pressure levels we have recorded are likewise in agreement with the demonstration by Nylin and Celander (8), that the human ventricles in congestive failure do not empty completely, permitting a large amount of residual blood to remain within the ventricular cavity following systole.

There are certain limitations in securing accurate pressure curves through the 100 cm. plastic catheter in use at the present time. This makes mandatory a repetition of this work with the use of more precise recording equipment, such as the catheter tip strain gauge, now under development in several laboratories.

**SUMMARY**

1. The pressure levels within the left ventricular cavity have been studied in ten human subjects with aortic insufficiency by catheterization of the left ventricle via the radial artery.
2. The three patients who exhibited no clinical evidence of congestive failure had normal left ventricular diastolic pressures.
3. The seven patients in obvious congestive failure demonstrated significant increases in diastolic pressure, averaging 25 mm. or mercury.

**ACKNOWLEDGMENTS**

The author is indebted to Miss Gladys Heckman, R.N., and Miss Hanna Janouskevec, R.N., for their technical assistance.
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