17295. Intraventricular Pressure Curves of the Human Heart Obtained by Direct Transthoracic Puncture.*

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In 1943, one of us (W.C.B.) studying blood pressures by the direct puncture technic had occasion to obtain a pressure curve from both the right and left ventricle by passing a needle directly into the cavity of these heart chambers. The recent development of right heart catheterization and the attempt to catheterize the left ventricle in man via the carotid artery, a procedure not without hazard, suggested to us the merits of publishing this experience with direct heart puncture. This is particularly timely because the experience with catheterization has shown not only how difficult but also how important for proper diagnosis it is to obtain adequate pressure curves. This difficulty obviously could be avoided by using the direct puncture technic as opposed to the long catheter method. Unfortunately, the hazards associated with direct puncture rule out this method for ordinary use.

The record shown in Fig. 1 was obtained on a 65 year old male who was moribund because of a cerebral thrombosis and terminal bronchopneumonia. The procedure had no significant effect upon the clinical course. A Hamilton needle manometer of adequate frequency similar to that usually employed for intra-arterial pressure recordings was used to record the intracardiac pressures. This was mounted on a portable electrocardiographic machine as previously described.[1] The right ventricular pressure curve (Fig. 1) was obtained by inserting the needle in the fourth intercostal space just to the left of the sternum. No electrocardiogram was recorded at this time. A respiratory fluctuation in the right ventricular pressure curve can be seen, the minimum end diastolic presure was 15 mm Hg and the maximum systolic pressure was 40 mm Hg. There was thus evidence of right heart congestion with little increase in systolic pressure. The contour of the curve is very satisfactory and shows none of the distorting artifacts so commonly seen with the catheter technic.

The top segment shows lead 2 of the electrocardiogram together with the left ventricular pressure curve. This was obtained by inserting the needle in the fifth intercostal space just beyond the left nipple line where the maximum apex beat was felt. In this instance there are frequent ventricular premature systoles occurring at times as pairs and triads. The third beat after the first pair of ventricular premature systoles is a fusion beat (labelled 3 on the left ventricular pressure curve). The beginning and end of the record present an acceptable undistorted left ventricular pressure curve. The pressure in this ventricle varied with respiration, showed a minimal end diastolic pressure of 15 mm Hg and a maximum systolic pressure of 100 mm Hg.

In both the left and right ventricular pressure curves, a noticeable auricular pressure wave is seen to precede the ventricular curve and is responsible to a large extent, especially in the left side, for the high diastolic end pressure. The slope of the ventricular pressure curves throughout systole and diastole is similar to that obtained by adequate pressure recordings from the cavities of the dog's heart.

The middle section of the top record offered the opportunity of studying the effect of premature systoles upon the pressure curve of the left ventricle. The beats of interest in the *middle section* have been indicated by numbers from 1 to 10. They will be referred to by these numbers in the subsequent discussion.

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† This department is supported, in part, by the Michael Reese Research Foundation.

* Aided by the A. D. Nast Fund.

1 Buchbinder, W. C., and Sugarman, H., *Arch. Int. Med.*, 1940, **66**, 625.

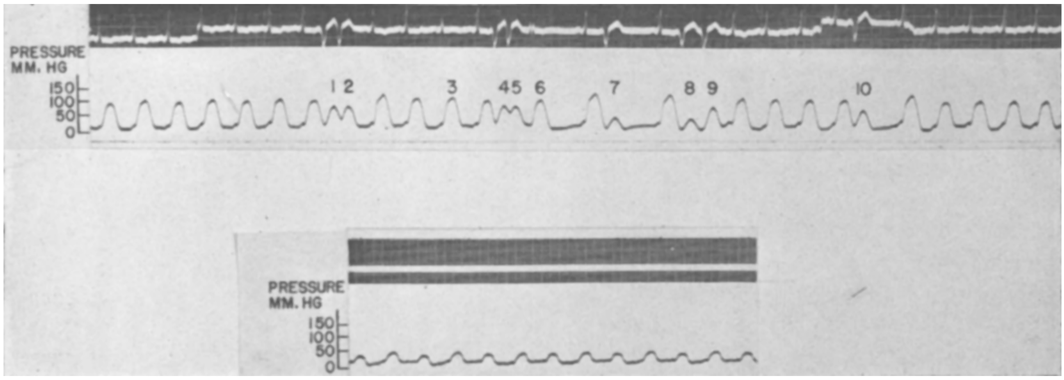

FIG. 1.
Pressure curves of left (above) and right (below) ventricle obtained in a 65-year-old moribund male. Time in each record is in the simultaneously recorded electrocardiographic record (each space between heavy lines equals 0.2 sec.). In the middle section of the upper strip, 1 to 10 indicate the successive premature systoles; 3 is a fusion beat. Discussed in text.

In accordance with Wigger's rule[2] the amplitude of the pressure curve of the post-extrasystolic beat is an inverse function of the duration of the immediately preceding diastole. Thus the magnitude of the pressure pulse occurring after the first pair of premature systoles (beats 1 and 2) is greater than the pressure pulse occurring before the premature systoles. This increase in size of the pressure pulse is more marked after the next two pauses (after beats 6 and 7) and after the last pause (after beat 10). Because there is no pause after the last pair of premature systoles (beats 8 and 9), the size of the post-extrasystolic beat is not significantly larger than the beats that follow. Following the last pause (after beat 10) there is a temporary mechanical alternans in that the second beat after this pause is smaller than the third. The beat registered at the time of the fusion complex (beat 3) is no different than the others. This evidence tends to confirm for man what has been established in the experimental animal that the magnitude of the pressure pulse varies with the duration of the preceding diastole.

The fact that the premature systoles occur at varying time intervals from the preceding beat accounts for the varying size and summation of their pressure curves. The shorter the R-R interval preceding the premature beat the higher up on the pressure curve of the preceding beat does the premature beat begin. The height of the systolic pressure obtained however, is not determined exclusively by the preceding R-R interval. Thus the second and third beats in a run of premature systoles (beats 6 and 9) unexpectedly may have a higher systolic pressure than other immediately preceding premature beats in the same runs (beats 4 and 8). This might be explained by the fact that the first premature systoles are less effective in emptying the ventricular content so that the systolic residue added to the next filling makes the subsequent beat more effective. Furthermore, the duration of the contraction curve of the premature systole is shorter than the normal beat permitting more time for filling before the next premature beat. It may be, however, that this phenomenon is due to the presence of a supernormal phase expressing itself in an increased contractility of the second premature beat.

Another interesting phenomenon is revealed in the pressure curves obtained in the first (beats 1 and 2) and second (beats 4 and 5) runs of the premature systoles. These pressure curves may be said to resemble a summation curve such as is obtained with skeletal muscle. As a matter of fact the pressure during the first two premature beats of the second run (beats 4 and 5) decreases very little; in this instance, therefore, the heart has for practical purposes changed its activity

[2] Wiggers, C. J., *Am. J. Physiol.*, 1925, **72**, 188.

from its normal creation of pressure fluctuation intermittently to maintaining a sustained pressure rise. This suggests the possibility that a rapid run of ventricular premature systoles could leave the heart more or less in sustained systole.

Summary. The recording of direct pressure curves, in which technical errors are reduced to a minimum, from the ventricular cavities of the heart in man emphasizes the great need of interpreting with care pressure curves obtained by other means in this newly expanded field of human physiology.

Received July 18, 1949. P.S.E.B.M., 1949, **71**.

17296. Spiral Arterial Structures in the Fetal Placenta.

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Spiraled arterial vessels are known to exist in the uterus[1,2] and ovary.[3,4] In the course of studying the fetal vascular components of the human placenta, the spiral nature of the primary branches of the allanto chorionic vessels has been noted for the first time. These appear to penetrate the substance of the placenta in much the same way as the radiate arteries do the myometrium of the uterus. The fetal spiral arteries arise from the major subchorionic vessels and represent the main vascular channels for the primary villus stem.

One hundred and twenty-five born placentae have been injected with several different masses and the vascular ramifications of the placental fetal vessels are being studied with a variety of technics. Each injection mass has been introduced via a cannulated umbilical artery shortly after the expulsion of the placenta. Fifty of these have been injected either with a 28% or 12% solution of vinyl acetate. The more concentrated solution was injected at 200-250 mm Hg while the more dilute monomer was injected at 120 mm Hg. The placentae were corroded in commercial concentrated hydrochloric acid for 24-48 hours and in turn washed with jets of tap water. The plastic cast of the fetal vessels which was subsequently recovered represented an accurate 3 dimensional model of the vasculature. Serial reconstruction by comparison is a time consuming, laborious task which does not yield as good a result.

The spiral features can be demonstrated more easily in preparations made with the more dilute vinyl acetate injected at physiological pressures. It is our impression that the greater pressure head which is required to completely inject the more concentrated solution straightens out some of the arterial coils. The remainder of the placental preparations, comprising 3 groups of 25 each, were injected at physiological pressures with radio opaque gelatin mass, liquid latex, and India ink, respectively. Each of these masses has advantages as well as distinct limitations. In the overall study, the desirable features of each has been utilized to reconstruct the details of the vascular pattern. The gelatin and India ink injected placentae have been cleared with a modified Spalteholtz technic and then selected areas have been serially sectioned for histologic detail. Liquid latex can be made to set within the vessel wall without distortion of the lumen, diameter, or calibre of the vessel. Such preparations permit a geometric

* This investigation was supported, in part, by the Boston Lying-in Hospital Research Fund, Department of Obstetrics, Harvard Medical School, and in part by a research grant from the Division of Research Grants and Fellowships of the National Institute of Health, U. S. Public Health Service.

1 Hunter, Williams, Anatoma uteri humani gravidi tabulis illustrata, 1774.

2 Daron, G. H., *Am. J. Anat.*, 1936, **58**, 349.

3 Reynolds, S. R. M., *Am. J. Obst. and Gynecol.*, 1947, **53**, 221.

4 Delson, B., Lubin, S., and Reynolds, S. R. M., *Endocrinology*, 1948, **42**, 124.