RECORDINGS OF PRESSURE OBTAINED DURING CATHETERIZATION OF THE GREAT CARDIAC VEIN

BY

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The introduction of the cardiac catheter into the coronary sinus in man has been described by several authors (Bing et al., 1947; Bing et al., 1949; Culbertson et al., 1949; Soulié, Carlotti et al., 1949; Soulié, Servelle et al., 1949; McMichael and Mounsey, 1951; Smith et al., 1951; Miller et al., 1953). Usually, the pressure recorded in the coronary sinus has not been found to differ materially from that in the right atrium. However, in one of the cases reported by Bing et al. (1949), where the catheter had been pushed far into the sinus to obstruct the great cardiac vein, a pronounced pulse wave was found with a peak of 45–50 mm. Hg “which closely followed the left ventricular ejection period.” In one of the cases described by Culbertson et al. (1949) a pulse wave was also recorded deep in the coronary sinus which was at first mistaken for a right ventricular recording, and a similar instance is reported by Soulié et al. (1949a) when the pulse wave had some resemblance to a tracing from the pulmonary artery. Recently, Miller et al. (1953) have described several cases in which a prominent pulse wave was recorded when the catheter was inserted far into the coronary sinus. In our records there are nine manometric recordings taken when the catheter had been inadvertently introduced into this position. The following is a description and discussion of these tracings.

MATERIALS AND METHODS

The recordings were taken during the routine cardiac catheterization of patients, all of whom had abnormal hearts. The abnormalities were as follows.

Case A: Pulmonary valvular stenosis.
Case B: Atrial septal defect with possibly pulmonary valvular stenosis.
Case C: Atrial septal defect with severe pulmonary hypertension.
Case D: Pulmonary infundibular stenosis with patent ductus arteriosus.
Case E: Primary pulmonary hypertension with pulmonary incompetence.
Case F: Mitral stenosis.
Case G: Pulmonary valvular stenosis.
Case H: Mitral stenosis.

All the patients had hypertrophy of the right ventricle and a high right ventricular systolic pressure (Table I). They were all adults.

Courand catheters were introduced under local anaesthesia. A Size 7F catheter was used in all cases except for one occasion when an 8F catheter was employed. Recordings of pressure were taken by means of a capacitance manometer with simultaneous electrocardiogram, using the apparatus devised by Bareham (1954). Zero was taken at the level of the table on which the patient lay. The oxygen uptake of samples of blood was measured by means of the Haldane method (Haldane, 1920). The oxygen capacity of the blood was assessed from the haemoglobin estimation.

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TABLE I
PRESSURES RECORDED IN GREAT CARDIAC VEIN AND RIGHT VENTRICLE DURING CARDIAC CATHETERIZATION

<table>
<thead>
<tr>
<th></th>
<th>Case A</th>
<th>Case B</th>
<th>Case C, 1953</th>
<th>Case C, 1954</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>G.C.V.</td>
<td>R.V.</td>
<td>G.C.V.</td>
<td>R.V.</td>
</tr>
<tr>
<td>Interval between start of QRS and start of pulse wave (sec.)</td>
<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>Duration of pulse wave (sec.)</td>
<td>0.46</td>
<td>0.50</td>
<td>0.42</td>
<td>0.42</td>
</tr>
<tr>
<td>Height of pulse wave (mm. Hg)</td>
<td>50</td>
<td>115</td>
<td>68</td>
<td>100</td>
</tr>
<tr>
<td>Length of cardiac cycle (sec.)</td>
<td>1.02</td>
<td>0.92</td>
<td>0.78</td>
<td>0.76</td>
</tr>
</tbody>
</table>

G.C.V. = occluded great cardiac vein.  R.V. = right ventricle.

In every instance the catheter had been advanced along the coronary sinus as far as it would go, as judged by the resistance to the operator's hand and the kinking of the free length of catheter in the right atrium. In each case, the nearness of the tip of the catheter to the left border of the heart shadow in the antero-posterior view led us to believe that it actually lay in the great cardiac vein (Fig. 1). Often the catheter could be seen to pass for some distance anteriorly round the left border

TABLE II
OXYGEN CONTENT AND SATURATION OF BLOOD SAMPLES FROM GREAT CARDIAC VEIN AND CHAMBERS OF RIGHT HEART

<table>
<thead>
<tr>
<th>Case</th>
<th>Oxygen content (vols. per 100 ml.)</th>
<th>Oxygen saturation (percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Great cardiac vein</td>
<td>Right ventricle</td>
</tr>
<tr>
<td>A</td>
<td>5.6</td>
<td>14.0</td>
</tr>
<tr>
<td>B</td>
<td>6.2</td>
<td>17.9</td>
</tr>
<tr>
<td>C 1953</td>
<td>7.6</td>
<td>19.2</td>
</tr>
<tr>
<td>C 1954</td>
<td>4.1</td>
<td>14.8</td>
</tr>
<tr>
<td>D</td>
<td>6.7</td>
<td>13.1</td>
</tr>
<tr>
<td>E</td>
<td>9.2</td>
<td>19.9</td>
</tr>
<tr>
<td>F</td>
<td>5.8</td>
<td>13.7</td>
</tr>
<tr>
<td>G</td>
<td>7.9</td>
<td>14.0</td>
</tr>
<tr>
<td>H</td>
<td>5.8</td>
<td>—</td>
</tr>
</tbody>
</table>
of the heart. The catheter can not always be introduced as far into the coronary sinus system as occurred in these cases and presumably the valve of Vieussens will often prevent its passage into the great cardiac vein.

RESULTS

No discomfort or mishap occurred in any of the patients during or after the time that the catheter lay in the coronary sinus, nor was there any change in the electrocardiogram. Samples of blood could be withdrawn freely from the catheter when it was wedged in the great cardiac vein and all had a very low oxygen content (Table II).

In the first seven patients, a distinct systolic pulse wave was recorded. The dimensions of this wave and its position in the cardiac cycle are given in Table I where they are compared with similar figures for the systolic pulse wave in the right ventricle. The recordings from these two sites were not simultaneous and the heart rate was, therefore, different in each tracing. Nevertheless, in six of these cases, the timing of the systolic pulse wave in the great cardiac vein is seen to coincide approximately with that in the right ventricle (Fig. 2). In Case F, the wave in the great cardiac vein starts considerably later than that in the right ventricle, but they both end at approximately the same point (Fig. 2 and 3).

The shape of the pulse wave varied. In Cases A, B, and G it simulated the contour of a right ventricular pulse wave (Harris, 1955) although in all these instances it differed from the shape of
the right ventricular systolic wave itself (Fig. 4 and 5). In Case F, the appearance of the pulse wave in the occluded great cardiac vein mimicked the form of wave normally found in the pulmonary artery (Fig. 3). In Cases C, D, and E, the wave was less specific (Fig. 6). Patient C was catheterized a year later and the two recordings from the great cardiac vein are very similar (Fig. 6). Occasionally the main pressure wave was preceded by a small wave that seemed to coincide with auricular systole.

The height of the pulse wave ranged between 25 and 68 mm Hg. In every instance it was lower than the systolic pressure in the right ventricle. In all cases, however, the right ventricular systolic pressure was abnormally high.

![Fig. 3. Recordings from (A) occluded great cardiac vein and (B) main pulmonary artery in Case F.](http://heart.bmj.com/content/17/4/453)

![Fig. 4. Recordings from (A) occluded great cardiac vein, (B) right atrium, and (C) right ventricle in Case A.](http://heart.bmj.com/content/17/4/453)

![Fig. 5. (A) Recording from the occluded great cardiac vein in Case B. (B) Recording from the right ventricle of a patient with mitral stenosis. The tracings are compared in order to show how closely the recording from the great cardiac vein can simulate a right ventricular tracing.](http://heart.bmj.com/content/17/4/453)
FIG. 6.—Recordings from the occluded great cardiac vein taken on two different occasions from Case C.

DISCUSSION

In 6 out of 7 cases, the pulse wave recorded when the catheter was wedged in the great cardiac vein coincided approximately with the systolic wave in the right ventricle (Fig. 2). It seems most likely, therefore, that the rise in pressure in the great cardiac vein was in fact caused by the contraction of the ventricles. The outflow of blood from the coronary sinus of the dog has been shown to occur mainly or entirely during the period of ventricular systole (Anrep et al., 1927; Johnson and Wiggers, 1937). A systolic pulse wave would therefore be expected in the coronary sinus if the flow of blood were obstructed to any degree. The curves of velocity of flow in the coronary sinus described by Anrep et al. (1927), and Johnson and Wiggers (1937) reach a peak towards the end of systole, and in our records the peak of the pressure wave also occurred at this point.

Johnson and Wiggers (1937), Katz et al. (1938), and Gregg and Shipley (1947) have shown that, under experimental conditions, a rise in right ventricular systolic pressure causes an increase in outflow of blood from the coronary sinus. It so happened that in all the patients described here, there was in fact a high right ventricular systolic pressure. There was, however, no correlation between the height of the pressure wave in the great cardiac vein and the systolic pressure in the right ventricle. Of the ten cases described by Miller et al., the right ventricular pressure was found to be normal in one and was probably normal in one other; seven patients had a raised pressure in the right ventricle.

Johnson and Wiggers (1937) and Katz et al. (1938) suggest that a high right ventricular systolic pressure causes a retrograde flow of blood in the Thebesian veins from the cavity of the right ventricle into the coronary sinus. It seems unlikely that this occurred to any substantial extent in our patients, since the samples of blood withdrawn from the great cardiac vein all had the usual low content of oxygen (Table II) (Bing et al., 1949). Most probably, therefore, the pulse wave in the great cardiac vein was due simply to a squeezing of blood from the capillaries and venules of the myocardium during contraction of the ventricles.

The pulse waves deep in the coronary sinus described by Miller et al. (1953) occurred a little later in the cardiac cycle and these authors suggest that they were due to a transmission of the pulse wave in the coronary artery across the capillary bed. This explanation was also given by Bing et al. (1949). Such a mechanism can certainly occur across the pulmonary capillary bed (Weissel et al., 1952). As far as the coronary circulation is concerned, however, it cannot explain the appearance of a venous pulse wave as early in the cardiac cycle as is shown by most of our tracings.

The pulse wave in the great cardiac vein may resemble a right ventricular tracing not only in its timing but in its shape (Fig. 5) and in its height (Table I). It may, therefore, be impossible to say from the manometric tracing alone that the catheter is not in the right ventricle. This may be an added source of confusion since in the postero-anterior view the catheter will often appear to be in the right ventricle or the main pulmonary artery. The recognition of this fact could be important.
in view of the possible dangers attached to deep catheterization of the coronary sinus (McMichael and Mounsey, 1951; Smith et al., 1951).

**SUMMARY**

In eight patients, a cardiac catheter was passed far into the coronary sinus and was thought to have occluded the great cardiac vein. Manometric recordings at this site revealed a prominent systolic pulse wave in seven patients. This wave coincided approximately with the systolic pulse wave of the right ventricle in six cases and it seems to be caused by ventricular contraction. The pulse wave may simulate closely the appearance of a right ventricular recording and lead to confusion as to the position of the catheter. Samples of blood taken from this position all had an extremely low oxygen content.

We wish to thank Dr. Terence East for his advice and encouragement.

**REFERENCES**


