Some Observations on the Hepatic Venous Pulsation in the Anesthetized Dog

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Since direct evidence of transmission of propagated waves into the hepatic veins (liver pulse) has been lacking, pressure pulses were recorded in the hepatic vein of the anesthetized dog. A constant presystolic positive wave, amplified by venous congestion and pulmonic compression, is demonstrated and shown to be transmitted from the atrium. During tricuspid insufficiency, a second transmitted positive wave is inscribed late in ventricular systole.

WHILE MacKenzie¹ and many cardiologists after him² have recorded pulsations from the right hypochondrial region (liver pulse) and attributed them to transference of pressure from the right atrium, it has never been demonstrated experimentally that such pulsations can be propagated backward through the inferior cava to the liver. The possibility that the oscillations recorded over the hepatic region may be distorted by impacts from abdominal vessels could not be entirely excluded.

This investigation was undertaken to determine whether such pulsations can be transmitted from the right atrium to the hepatic vein with sufficient reliability to be useful and to study the alterations that take place during venous congestion and tricuspid insufficiency.

METHODS

Adult mongrel dogs weighing between 11 and 18 Kg. were premedicated with morphine and anesthetized with sodium barbital (180 mg. per kilogram). Hepatic pulsations were recorded through a brass cannula, 46 cm. long, with an internal diameter of 2.7 mm., slightly curved at the tip, and introduced through the right jugular vein. In some instances it was possible to introduce the tip of the cannula blindly into the hepatic vein, its position being verified at postmortem examination. In about 70 per cent of the experiments it was necessary to guide the cannula into position bi-manually via a small transabdominal incision below the xiphoid. Aortic pressures were recorded through an appropriate cannula introduced into the left carotid artery. All pressure pulses were recorded by Gregg manometers of suitable sensitivity and proper frequency responses. In four animals the hepatocaval junction was occluded during recording of the hepatic venous pulsations. For this purpose a small collapsible rubber balloon cemented circumferentially near the open distal tip of the cannula was inflated through an indwelling polyethylene tube. Tricuspid insufficiency was produced by introducing a brass trocar through the right ventricular apex and thence into the atrium, according to the method of Wiggers.³ Acute right heart congestion was simulated by sudden digital compression of the pulmonary conus.

RESULTS AND DISCUSSION

Present in all recordings with the tip of the sound properly placed is a presystolic positive wave, a (fig. 1A). This positive wave may be followed by a negative "dip," x, in about half the records (more apparent in fig. 1B). The negative "dip," x, cannot be attributed to a decrease in intrathoracic pressure occurring during ventricular systole as some would imply² from a study of extra-abdominal volumetric curves since, when definitely present, it persists unchanged following opening of the thoracic cage. The deep negative wave n ted during ventricular systole and the secondary positive wave inscribed early in ventricular diastole in external volumetric curves² could not be identified in these pressure wave recordings.

During ventricular systole there is usually a
gentle decline in pressure which returns to the original base line by the time that the next presystolic positive wave is inscribed. Small, inconstant wavelets may be recorded during this period. The presystolic wave generally occurs between 0.08 and 0.12 second before the onset of ventricular systole. Its amplitude may be increased by elevating venous pressure by a rapid intravenous infusion of saline (500 positive presystolic wave is dynamically comparable to the hepatic pulsations sometimes noted clinically in the presence of tricuspid atresia.

Pulsations could not be recorded if the cannula was introduced far into the hepatic vein or when it fitted so tightly as to interrupt blood flow into the inferior vena cava. If, after recording the hepatic pulse through the in-

cc. in 25 seconds); the increase in central venous pressure is reflected in the hepatic vein, and the accentuated negative "dip," \( x \), is then followed by a steady gradual rise of pressure to the original level before the onset of the succeeding presystolic wave (fig. 1B). Similar effects were produced when venous congestion was produced by compression of the pulmonary artery. Such amplification of the dwelling brass cannula (as in fig. 1B) the hepatic vein was occluded by the rubber balloon, the recorded pulsations were reduced to a mere trace or disappeared entirely, as in figure 1C. This demonstrates that the presystolic wave is not transmitted through hepatic vessels but represents a propagated presystolic wave.

To support this concept, serial recordings of
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Fig. 2. Progressive increase in a-c interval. Tip of cannula in following positions: A. Two cm. within hepatic vein. B. One cm. within hepatic vein. C. Hepatocaval junction. D. IVC, 2 cm. above C. E. IVC-RA junction, 2 cm. above D. F. Right atrium, 2 cm. above E. See text.

Fig. 3. A. Hepatic pulse, open chest. B. Hepatic pulse, tricuspid insufficiency. A secondary positive wave is noted during ventricular systole. (Venous membrane with lesser frequency response than that employed in recordings in figs. 1 and 2.)
isometric ventricular contraction, this time interval being virtually identical to that noted between the ascent of the positive presystolic wave a and ventricular systole. A ventricular impact could not account for this wave, since the time relationship favors genesis from a transmitted atrial wave. Its clinical counterpart is represented by the “expansile liver” of tricuspid insufficiency.

**Conclusions**

1. Pressure pulses in the hepatic vein of the anesthetized dog were recorded via a brass cannula by Gregg manometers.

2. A positive presystolic pressure wave usually followed by a negative “dip” is the one constant feature noted. Since these waves are abolished by occlusion of the hepatic vein proximal to cannulation, and since they can be followed with progressively decreasing a-c intervals from the right atrium to the hepatic vein, they represent propagated waves from the right atrium.

3. In the presence of tricuspid insufficiency a second positive wave is similarly transmitted during ventricular systole, whereas the only change noted during acute venous congestion is a marked amplification of the positive presystolic wave.

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**References**


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