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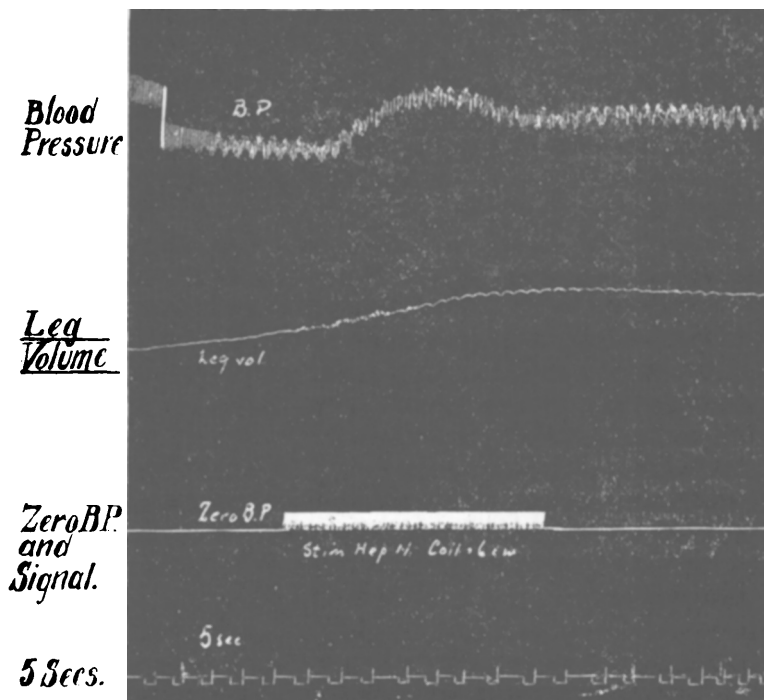
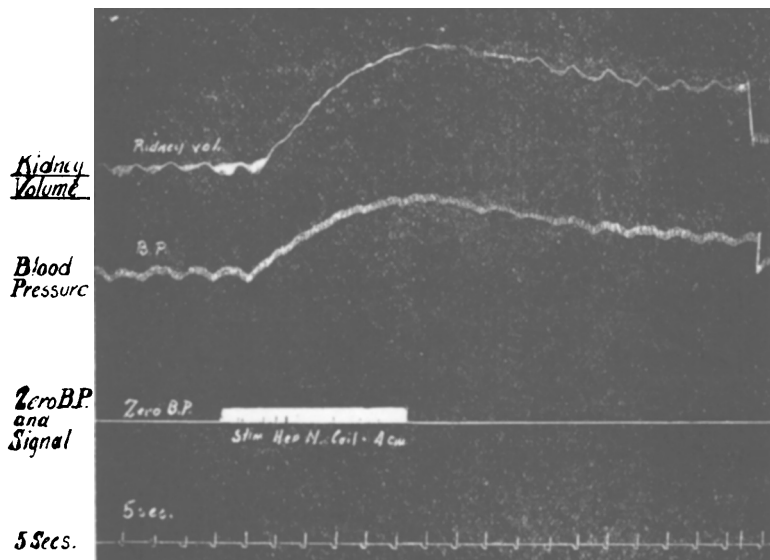
Vasoconstriction in the Liver.

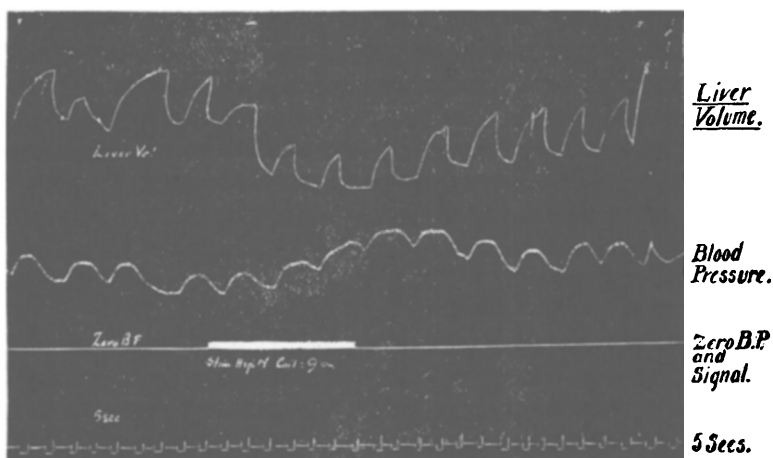
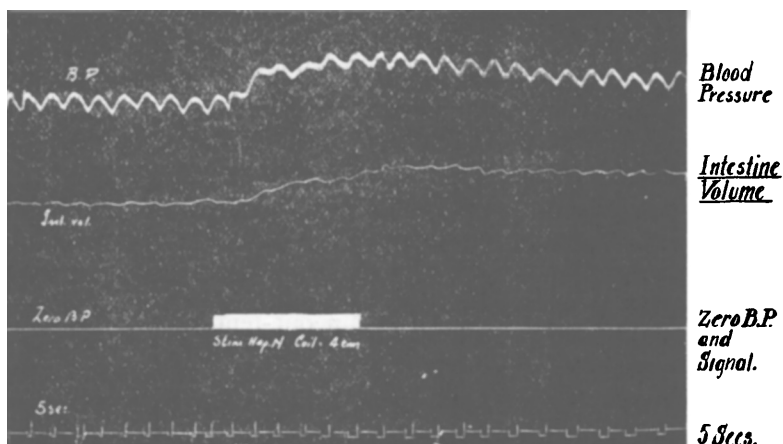
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Stimulation of the nerves going to the liver causes a rise in the arterial blood pressure in the cat.¹ It has been attempted to show that this is due to the release of a vaso-constrictor substance from the liver into the general circulation; but the effort to isolate such a substance from extracts of the liver has been unsuccessful.²

We have recently taken records of the volume of a hind leg, a kidney, an intestinal loop and the liver itself during stimulation of its nerves. The experiments were done on cats under urethane or chloralose anesthesia. Shielded electrodes were applied to the hepatic nerves after they had been dissected free from the artery proximal to its gastro-duodenal branch; the nerves were ligated and cut centrally to the electrodes and were stimulated with a tetanizing current from a Harvard inductorium. The leg, kidney and intestinal volumes were recorded in the usual way. A special plethysmograph was devised for use with the liver. This was, essentially, a flattened, bell-shaped device that could be lowered over the entire liver—the animal being on its back and the liver exposed by a ventral, mid-line incision. A notch on each side of the plethysmograph prevented occlusion of the arterial or venous (portal and hepatic) blood vessels; and the lower edges were conformed to the shape of the dorsal body wall so that when snugly in place the junction was practically air tight. Any possible leakage was entirely prevented by





pouring enough paraffine oil into the abdominal cavity to cover the lower edges.

The records, of which examples are reproduced in the accompanying figures, show that the rise of arterial blood pressure which results from stimulation of the hepatic nerves is accompanied by, or results in a passive dilatation of the leg, kidney and intestines; the liver alone decreases in volume. Such evidence would seem to indicate that the rise in blood pressure is due merely to the forcing of blood from the extensive vascular bed in the liver into the general circulation rather than to the release of a vasoconstrictor substance from the liver into the blood stream.

¹ Cannon, W. B., and Uridil, J. E., *Am. J. Physiol.*, 1921, lviii, 353.

² Cannon, W. B., and Griffith, F. R., *ibid.*, 1922, lx, 544.