Fig. 1
Peptone Shock with a normal circulation.

Fig. 2
Peptone Shock with a "Short circuit" circulation which has eliminated all abdominal viscera and the posterior extremities from the circulation.

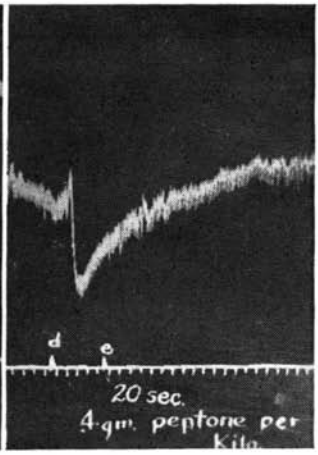

Fig. 3

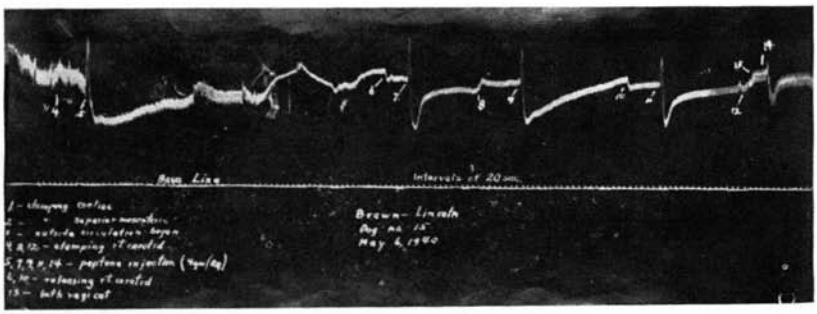

Fig. 4
Peptone Shock showing slow recovery with the cranial circulation obstructed at 5 and 9 as compared with unimpaired cranial circulation at 7 and 11.

PRELIMINARY STUDIES ON THE SITE OF CANINE PEPTONE SHOCK

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In 1880 Schmidt-Mulheim observed that on rapid injection of "peptone" into a dog a marked fall in blood pressure immediately results, accompanied by a delay or failure of the blood to clot.

Four possible causes for the fall are recognized: (a) peripheral vasodilatation, (b) diminished heart action, (c) reduced blood volume, and (d) obstruction at some strategic point in the circulation.

Schmidt-Mulheim noticed an effect on the tone of the blood vessels during the arterial fall and Pollitzer (1886) thought the vasodilatation was manifested chiefly in the splanchnic region, the mesenteric vessels being always very congested. He believed the fall in pressure to be due to vasomotor paralysis.

Thompson (1896) showed that the vasodilatation is shared by vessels other than those in the splanchnic area, and that the vasomotor depression is peripheral, apparently at the myoneural junction. Geiling and Kolls (1924) have confirmed the peripheral origin of the dilatation as well as its general effect on venules and capillaries of both splanchnic and skin areas in the unanesthetized dog. These investigators also found the cardiac output to be greatly reduced during the low pressure period. This, they believe, is evidence of reduced venous return and they regard it as the natural result in increased peripheral capacity, and as the prime cause of the fall in blood pressure.

Arey and Simonds (1920) found a relatively large amount of smooth muscle in the hepatic veins of the dog and feel that this lends support to the theory of decreased venous return owing to hepatic obstruction.

Manwaring, Hosepian and Beattie (1925) found a marked increase in the weight of the liver in peptone shock, which represented a considerable withdrawal of plasma from the blood. Peterson and coworkers (1923) substantiated this point by noting an increase in the permeability of the liver endothelium.

Dragstedt and Mead (1937) have brought forth considerable evidence to show the drop in blood pressure to be due to the explosive liberation of a vaso-depressor substance which they have identified tentatively as histamine.

Such a diversity of opinion as to how the "shocking" agent becomes effective challenged us to undertake some experiments to throw some light upon the point of attack.

EXPERIMENTAL

Nineteen full grown dogs selected at random without regard for breed or sex were used. Ether anesthesia was applied by direct tracheotomy and carotid blood pressure was recorded with a mercury manometer.

In consideration of the general opinion that the liver and splanchnic area form the strategic points for the stagnation of a great volume of the circulating blood, we eliminated both from the circulation. This was done as follows:

The coeliac axis was ligated as was also the superior mesenteric artery. A straight cannula was then tied into the abdominal aorta just anterior to the renal arteries. From this cannula rubber tubing was used to set up a collateral circulation to the right jugular vein. The tubing was filled with 2% sodium citrate solution to prevent coagulation when the blood was turned through it. A "T" near the anterior end served to drain the sodium citrate as the blood came through, thus preventing excess sodium citrate from entering the circulation.

By this scheme, all arterial blood was "short circuited" away from the viscera and extremities posterior to the diaphragm. The dosage of peptone used was 0.4 gram of Difco Peptone per kilogram of body weight. The peptone, made up as a 10 percent aqueous solution, was administered by cannula into the jugular vein or into the rubber tubing used to establish the collateral circulation. Dogs were kept alive and given duplicate doses of peptone during periods ranging from 30 minutes to 2½ hours.

When peptone was introduced into the unaltered circulation, a typical reaction, showing a very abrupt drop in blood pressure, was produced as is shown by the carotid tracing of Fig. 1.

Figures 2 and 3 show that a very similar reaction occurred when the blood was shunted past the entire visceral capillary bed. This is excellent evidence that, whatever the cause for the drop in blood pressure, it does not necessarily reside in the liver and splanchnic area alone.

Our next interesting phenomenon came from an altered blood supply to the brain. With the dog fully prepared, and following a typical reaction, the right carotid artery was clamped. This eliminated the blood supply to the cranium except for the vertebral arteries. An injection of peptone produced the typical drop in blood pressure but the recovery period was 7 or 8 times longer than it was in case of a general cranial circulation, as shown in Fig. 4. This was repeated several times alternately permitting and inhibiting cranial flow, always with the same result. This indicates that probably some cranial nerve center plays a very important role in the reestablishment of circulatory equilibrium.

SUMMARY

1. A new method for investigating the mechanism of shock production has been developed.
2. The visceral region does not contain the whole structure involved in the production of peptone shock.
3. There is a great probability that the blood supply to the brain has some effect on recovery.

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