6983

Effects of Primary Shock on Cardiac Output and Blood Pressure.*

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In a previous study,[1] the effects of uncomplicated hemorrhage on cardiac output and blood pressure were determined. Repeated removal of blood was usually associated with a decline in the cardiac output from 30 to 50% below the normal level before a marked diminution in the blood pressure occurs. Johnson and Blalock[2] found that secondary shock as a result of trauma to muscles, trauma to the intestines or burns is associated with a definite decrease in the output of the heart before the blood pressure is altered appreciably. On the contrary, the introduction of histamine[2] causes an initial decline in the blood pressure followed by a decrease in the cardiac output. Following the removal of the adrenal glands,[3] the blood pressure usually declines before the cardiac output is altered. Trauma to the central nervous system[4] is usually associated with a simultaneous decline in both the cardiac output and blood pressure.

The alterations in the cardiac output and blood pressure in primary shock were determined in the present experiments in order to compare these changes with those observed in secondary shock.

Dogs were profoundly anesthetized by sodium barbital administered intravenously. The dosage was 0.3 gm. per kilo of body weight. The control determinations of the cardiac output and blood pressure were performed approximately 2 hours after injection of barbital. The anterior abdominal wall of the animal was then struck many blows in rapid succession with flat surface of a board. This procedure occupied approximately 2 minutes. Thirty to sixty seconds later, the cardiac output and blood pressure were again determined. If free hemorrhage into the peritoneal cavity resulted from the traumatization, the experiment was discarded.

A cannula that was placed in the femoral artery was used for the determination of the blood pressure. The cardiac output was computed by the Fick principle:

$$\frac{\text{Oxygen consumption per minute}}{\text{Arterial oxygen content minus venous oxygen content}} = \text{No. cc. of blood flowing through lungs per min.}$$

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1 Blalock, Alfred, *Arch. Surg.*, 1927, **15**, 762.

2 Johnson, G. S., and Blalock, Alfred, *Arch. Surg.*, 1931, **23**, 855.

3 Blalock, Alfred, and Beard, J. W., *J. Lab. and Clin. Med.*, 1933, **18**, 941.

4 Blalock, Alfred, and Bradburn, H. B., *Arch. Surg.*, 1929, **19**, 725.

A Benedict spirometer was employed in determining the oxygen consumption. Arterial blood was obtained from the femoral artery and mixed venous blood from the right side of the heart. The oxygen content of the blood was determined with the Van Slyke-Neill manometric apparatus.

Ten experiments were performed. In all, the striking of the abdomen was followed by a decline in the blood pressure. The drop in the mean arterial blood pressure in the different experiments varied from 38 to 75 mm. Hg. The decline in blood pressure was usually of short duration. Associated with the drop in blood pressure, the output of the heart increased slightly in 7 experiments and decreased in 3. The consumption of oxygen increased during and immediately following the traumatization in 9 of the 10 experiments. The arterio-venous oxygen difference increased in some instances and decreased in others. These alterations are quite different from those that are found in secondary shock.

6984 P

Specific Carbohydrate of Type I Pneumococcus.

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(Introduced by Hans Zinsser.)

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When a Type I pneumococcus antiserum is absorbed with the homologous soluble specific carbohydrate until no further precipitin reaction occurs with the latter, the absorbed serum still reacts type-specifically with a substance in pneumococcus autolysates in high dilution, as shown by Enders[1] and confirmed by Wadsworth and Brown[2, 3] and by Sabin.[4] We have now succeeded in isolating this substance, called the "A substance" by Enders, from Type I pneumococcus broth cultures.

The initial steps in the preparation of the A substance are essentially those given by Heidelberger and Kendall[5] in their preparation

[1] Enders, J. F., *J. Exp. Med.*, 1930, **52**, 235.

[2] Wadsworth, A., and Brown, R., *J. Immunol.*, 1931, **21**, 245.

[3] Wadsworth, A., and Brown, R., *J. Immunol.*, 1933, **24**, 349.

[4] Sabin, A. B., *J. Exp. Med.*, 1931, **53**, 93.

[5] Heidelberger, N., and Kendall, M., *J. Exp. Med.*, 1930, **52**, 477.