

third week. At this time embryonic trichinæ are in process of transmission from the intestinal mucosa, by way of the lymphatic vessels and the blood through the lungs, to the vascular system.

Eosinophile cells accumulate in the mesenteric lymph glands and in the lungs, and form foci resembling small abscesses, in which polynuclear leukocytes are replaced by eosinophile cells. These cells are provided with polymorphous nuclei and do not differ from the eosinophile leukocytes of the circulating blood. Accumulation of the eosinophile cells in the mesenteric lymph glands and in the lungs is explained by the transmission of the embryonic parasites through these organs.

Increase of eosinophile cells in the blood and in other organs is accompanied by characteristic changes in the bone marrow. The fat is diminished in amount and cellular elements replace it. Cells with eosinophile granulation are present in immense number and particularly numerous are the eosinophile myelocytes, cells peculiar to the bone marrow. Eosinophile cells undergoing mitotic division are more numerous than usual.

The number of eosinophile leukocytes in the blood always diminishes before death, so that the proportion is usually less than 1 per cent. Infection with a very large number of trichinæ causes a rapid diminution of the number of eosinophile leukocytes, and is quickly fatal. The eosinophile cells of the bone marrow exhibit degenerative changes, of which nuclear fragmentation is most characteristic. Similar changes may affect the eosinophile cells of the intestinal mucosa and of the mesenteric lymph glands. Mild infection stimulates the eosinophile cells to multiplication, but severe infection causes their destruction.

30. "Subcortical expressive reflexes and their spinal pathways": ROBERT S. WOODWORTH.

The author reported on some experiments done in collaboration with Professor Sherrington in the latter's laboratory. It was shown that in a recently decerebrated cat, powerful sensory stimuli evoked reactions such as in a normal animal would be expressive of pain, anger, and other similar emotions. Such reactions are therefore primarily subcortical reflexes and not dependent on the organ of consciousness. The "ether cry" also appeared in decerebrate animals. The sensory spinal pathway, by which

these signs of pain were aroused, was found by experiments in which partial cross-sections of the cord were made, to run not in the posterior, but in the lateral columns. The pain pathway from either side of the body runs up both halves of the cord, but more largely up the opposite half.

31. "An experimental study of the cause of shock": WILLIAM H. HOWELL. [Presented by S. J. MELTZER.]

Professor Howell's experiments were made upon dogs anaesthetized with morphin and ether, and brought into a condition of shock by operations of various kinds. Blood-pressure records were obtained in the usual way during the experiment. The following general conclusions were reached:

1. The most important and dangerous feature of severe shock is a long continued, practically permanent fall in blood-pressure to about 20 mm. to 40 mm. of Hg. This condition is designated as vascular shock and is due to a long lasting loss of activity of the vasoconstrictor center.

2. A second important result of shock is a very rapid and feeble heart-beat. This condition is designated as cardiac shock; since, although it may result secondarily from the permanent fall in blood-pressure, it may also occur quite independently of the vascular shock as a primary result of the operations. Cardiac shock, so far at least as the rate of beat is concerned, is due to a more or less permanent loss of activity of the cardioinhibitory center.

3. Intravenous infusions of alkaline salt solutions (NaCl, 0.6% — Na₂CO₃, 0.5%) cause a rise of pressure by increasing the force of the heart-beat. The effect is more durable than with salt solution alone and may be renewed by repeating the injection.

4. The fundamental cause of vascular and cardiac shock is not exhaustion of the vasomotor and cardioinhibitory centers from over activity, but a more or less permanent inhibition of these centers from excessive stimulation of the inhibitory paths.

Sixth meeting.¹

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