The Mechanism of Protracted Collapse in Young Animals Due to the Action of Bacterial Toxins

I. A. Kornienko

From the Laboratory of Physiology and Pathology of Aging (Head - Prof. I. A. Arshavskii) of the Institute of Normal and Pathological Physiology (Director - Active Member AMN SSSR Prof. V. N. Chernigovskii) of the AMN SSSR, Moscow.

(Received December 20, 1957. Presented by Active Member AMN SSSR V. N. Chernigovskii)

Research in our laboratory has shown that different pathogenic agents (bacterial toxins, pharmacological substances in suitable doses) cause a state of protracted collapse in young animals [5, 7]. Several workers have shown that this type of reaction in young animals is associated with the special features of tissue metabolism which permit the utilization of anaerobic energy in a state of collapse [6, 9]. This usually accounts for the ability of young animals to survive for a long time in conditions of severe anoxia. At the same time it has been shown in our laboratory that during anoxia, protracted collapse can develop in young rabbits and kittens only when the body temperature is constantly falling; prevention of cooling very rapidly leads to death with signs of rapid collapse of the type characteristic of adult animals [1, 3].

The aim of the present investigation was to assess the importance of a falling body temperature as a factor permitting the prolonged survival of young puppies subjected to the action of bacterial toxins, and to study the changes in the condition of certain nerve centers.

Experimental Method

Experiments were performed on puppies ages from 1 day to 3 months. The animals were given an intravenous injection of a lethal dose of staphylococcal or dysentery toxin causing death after 4-12 hours. For this purpose we used liquid staphylococcal toxin in a dose of 0.2-0.5 ml/kg body weight and complete Hiss-Flexner antigen in a dose of 5-7 mg/kg body weight. In order to determine the state of the cerebral cortex, the electroencephalogram (EEG) was recorded. Bipolar leads were taken, using needle electrodes, which were inserted into the bones of the skull at the site of projection of the frontal or parietal lobe. In the majority of experiments the animal's head was firmly fixed in order to prevent artefacts. In addition, throughout the experiment, recordings were made of the electrocardiogram, the respiration and the body temperature. In a special series of experiments the animals were placed in an incubator with an air temperature of 30-31°C. Altogether 78 experiments were performed.

Experimental Results

Injection of lethal doses of staphylococcal or dysentery toxin to puppies ages 1½ -3 months caused the reaction already described above, with a four-phase change in the rhythm of the heart's contractions. Workers in our laboratory [2, 5] have shown that the first phase, appearing soon after injection of the toxin, when the cardiac rhythm became slower on account of the increased tone of the center of vagus innervation of the heart. The second phase—a quickening of the activity of the heart—was due to a fall in the tone of the vagus. Later, during the development of a terminal state, a second increase arose in the tone of the center of vagus innervation of the heart; this was expressed by the phenomenon of syncope with subsequent change of the activity of the heart to an
Fig. 1. The course of staphylococcal toxemia. a) In a puppy age 1 month 20 days; b) in a puppy age 7 days. Along the vertical axis — rectal temperature; P) number of contractions per minute of the heart; D) respiration rate. Along the horizontal axis — time in hours. 6) Time of injection of toxin.

Fig. 2. Changes in the EEG (frontal lead) in a puppy age 2 months injected with staphylococcal toxin. a) Recording before injection of toxin; b) in the phase of the first slowing of the heart's contractions; c) in the phase of quickening of the heart's contractions; d) 5 minutes before cardiac syncope; e) during cardiac and respiratory syncope.

Aschoff-Tawara rhythm. This phase coincided with phenomena of syncope in the respiratory center also. Next followed the fourth phase — the change of the heart to an automatic rhythm and, possibly, to fibrillation. In this last phase terminal inspirations were observed at the same time. The last two phases, beginning with cardiac and respiratory syncope, lasted on the average 5-8 minutes, after which death ensued. These two phases were also characterized by what in adult dogs would in similar conditions be called collapse. Under these circumstances in adult dogs the body temperature, if lowered at all, does not fall by more than 1-3°. In puppies ages 1½ - 2 months it may fall by 3-5° (Fig. 1).