THE ACCELERATOR REFLEX IN NORMAL AND DEHYDRATED DOGS

By

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The thermal polypnea, and the superimposed further reflex acceleration of the respiration brought about by opening of the mouth (the 'accelaration reflex') have been studied in normal dogs and in the animals dehydrated by fluid deprivation for 48 hrs. Both these responses were suppressed in dehydrated dogs. After saline transfusion in the dehydrated dogs they appeared in an exaggerated manner. It is suggested that during hyperthermia, dogs conserve fluids in preference to the rise in body temperature. The temperature regulating mechanisms fail early in dehydrated than in normal dogs.

Dogs are known to respond by panting when their body temperature is raised. Mathur and Choudhury (1952) showed that superimposed on this panting is a further reflex acceleration of respiration rate brought about by opening of the mouth. This reflex acceleration of respiration rate on opening of the mouth was investigated by Kumar (1953a), and Sharma and Sharma (1958). Kumar and Sinha (1961b) have recently shown that in dogs deprived of water for 24 hrs the panting type of respiration did not appear on raising their body temperature, and that saline transfusion in such dogs resulted in panting. Since water deprivation affects the panting mechanism of heat loss in these animals, it was therefore, thought to investigate as to how dehydration affects the reflex mechanism of heat loss described by Mathur and Chaudhury and later designated as the 'accelerator reflex' by Kumar and Sinha (1961a).

METHODS

The study was undertaken on 34 dogs of both sexes weighing between 6 to 15 kg. Out of these 14 dogs were fed on dry bread and deprived of all fluids for 48 hrs before the start of the experiment. In the remaining 20 dogs no fluid restriction was observed before starting the experiment. They were anaesthetised with chloralose (80 mg per kg body weight) given intravenously. The methods of recording their respiration and rectal temperature, and of rendering them hyperthermic were the same as described by Kumar and Sinha (1961a).

RESULTS

Since the respiration rate of dogs both at their initial body temperatures and in response to increase in their body temperature showed very wide variations from animal to animal (Kumar, 1953b), the relative change in their respiration rate was calculated from the formula $R = \frac{F_2}{F_1}$ where R is the relative change in respiration rate, and F_1 and F_2 are the frequencies of respiration at their initial and the observed rectal temperatures respectively. Calculated in this way, it was observed that in normal dogs (Fig. 1) the respiration rate with their mouths closed increased with the rise in their body temprature. This increase was very much marked between 40°C-42°C rectal

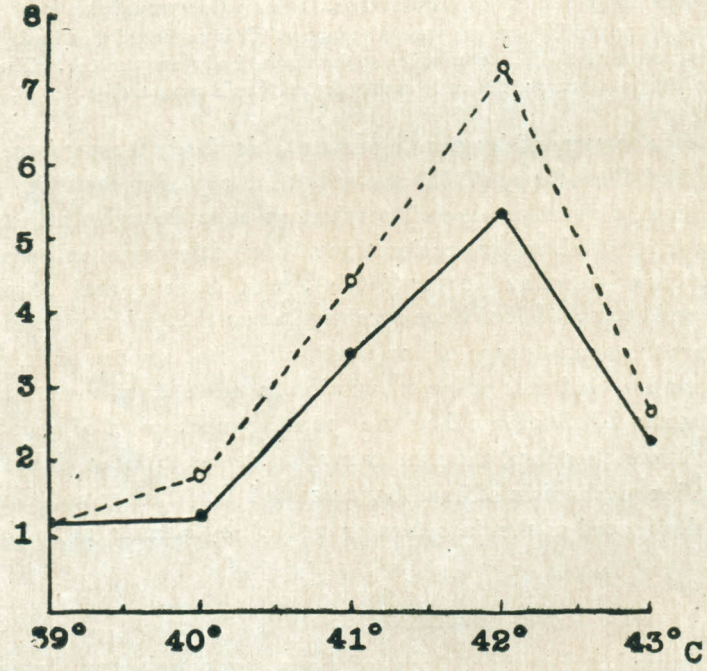

Fig. 1. The relationship of the respiratory rate with the rising body temperature in dogs. Continuous-line mouth closed; interrupted line-mouth open.

temperature. Above 42°C, the respiration rate declined and death occured at about 43°C. On opening the mouth as expected further acceleration of respiration occured. The magnitude of this acceleration also increased upto 42°C rectal temperature and then declined when their temperature was further raised.

In the dehydrated dogs (Fig. 2), on the other hand, it was observed that the rate of the change of respiration was abrupt and more than in the normal dogs upto 40°C, and then upto 41°C it was gradual but less than in normal dogs. On opening the mouth the further acceleration of respiration did occur

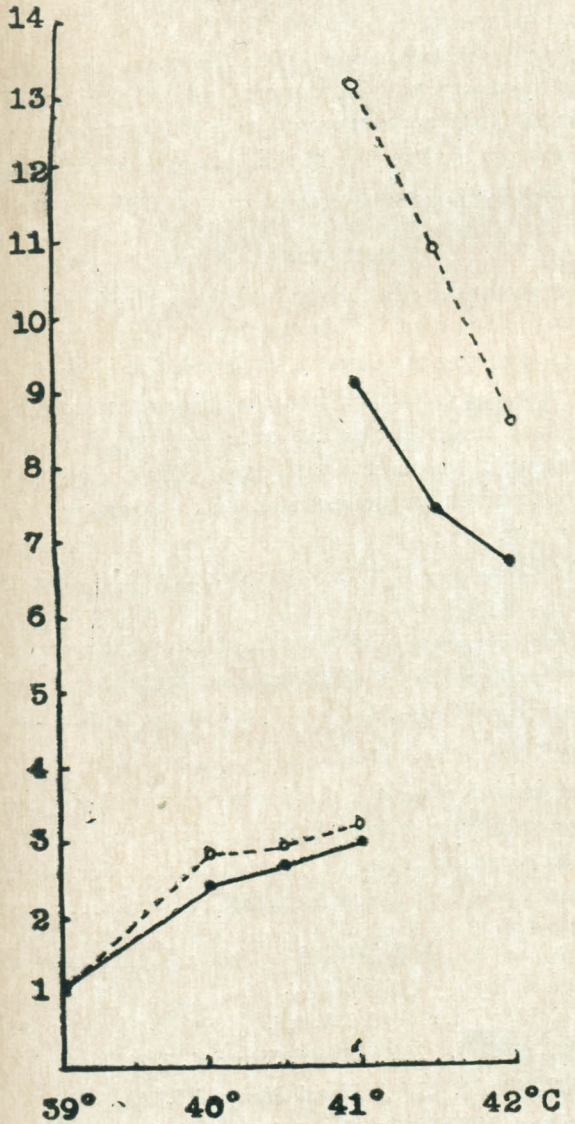

Fig. 2. The relationship of the respiratory rate with the rising body temperature in dogs. Continuous line-mouth closed; interrupted line, mouth open. The top right bold curves are after intravenous saline tronsfusion,

but the magnitude of this acceleration was much less than observed in normal dogs. At about 40.5°C-41°C body temperature these dogs were given 250 to 300 ml normal saline intravenously. The transfusion was complete in about five mins. Dogs not given any normal saline transfusion invariably died at about this temperature. As observed by Kumar and Sinha (1961b) earlier, the respiration rate suddenly and remarkably increased on saline transfusion. On further raising the temperature above 41°C the respiration rate, however, declined. On opening the mouth, after saline infusion, the acceleration in respiration rate was pronounced. The magnitude of the acceleration was maintained when the temperature was raised to 42°C even though the rate was declining. These dogs died inspite of the saline transfusion when their temperature was raised above 42°C.

DISCUSSION

Since dogs do not sweat they lose their body heat predominantly through alterations in their respiration rate. This respiratory temperature regulating mechanism in dogs has two components. The first is the simple acceleration of respiration rate in response to rise in body temperature, and the second, superimposed on the first a further reflex acceleration of respiration rate brought about by opening of the mouth. In dogs dehydrated by fluid deprivation both these responses remain suppressed. But when normal saline is injected intravenously both the respiration rate and the magnitude of acceleration rate of opening the mouth, rise remarkably. It is probable that dehydration in dogs keeps the temperature regulating mechanisms suppressed, and that they are thrown into action with greater emphasis when water is made available due to saline transfusion. It can therefore, be said that dogs, at least during hyperthermia, conserve body fluids in preference to the rise of body temperature.

The decline in respiration rate with the further rise in body temperature in the dehydrated dogs even after saline transfusion is suggestive of the gradual failure of the temperature regulating mechanism. Persistence of the 'accelerator reflex' is the last effort to lose heat before death occurs.

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