Kumar (1953) observed that the normal respiratory rate of the dogs varied between 10 to 30 per minute or even less at their resting body temperatures, and rose to 300 per minute or even more when the body temperature was raised to 42°C or slightly above it. The dogs pant to a remarkable degree but seldom suffer bad consequences. Haldane and Priestley (1935) found that such a degree of panting, if artificially induced in man, produced severe condition of acapnia incompatible with life. Collip and Backus (1920) found that prolonged voluntary hyperventilation in normal adults for 15 to 20 minutes caused a fall in carbon dioxide tension of the alveolar air. It was therefore considered necessary to investigate if a similar fall occurred in carbon dioxide content of arterial blood during thermal panting in dogs.

METHODS

Experiments were done on 10 healthy dogs of both sexes weighing from 4.5 to 9 kg. After preliminary ether anesthesia, chloralose (75 - 80 mg. per kg. body weight) was given intravenously. Respiratory rate was recorded through a toy balloon by introducing and inflating it in between the liver and the diaphragm and connecting it to a recording tambour. Rectal temperature was recorded by a Centigrade thermometer. The temperature of the dog was raised by applying carbon bulbs all round the animal and by warming the plate, on which the animal rested, from below. Arterial blood was collected from the femoral artery through a paraffinised cannula, in a paraffinised tube under previously boiled and cooled liquid paraffin. Arterial carbon dioxide content was determined by the method of Haldane.

RESULTS

The initial carbon dioxide content of the arterial blood varied between 41 - 66 volumes per cent, with a mean value of 51.6 ± 6.09 volumes per cent. On raising the temperature it steadily decreased with the rise in respiratory rate till it went down to 27.3 ± 4.75 volumes per cent. In one dog it fell to 14.5 volumes per cent. The fall in carbon dioxide content was generally found to be in proportion to the rise in respiratory rate as shown in the graph. There was no change in the rhythm of respiration.
Fig. 1 Showing the effect of temperature on arterial CO₂ content (solid line) and respiratory rate (broken line) in dogs.

Further, by giving artificial respiration for about 10 minutes at rates above their normal respiratory rate at resting body temperature it was observed that the arterial carbon dioxide content fell to a mean value of 30.0 ± 4.62 volumes per cent, and their respirations ceased on stopping the artificial respiration.

DISCUSSION

In a non-sweating animal like dog, heat loss is by evaporation of the saliva. This is brought about by rapid increase in the respiratory rate. With the rise in respiratory rate the arterial carbon dioxide has been found to decrease. The heat polypnoea in dogs is therefore, a form of hyperventilation. At higher body temperatures it appears therefore that for the sake of temperature regulation there is no attempt on the dog's part to conserve the carbon dioxide even though its content may be severely reduced. Since artificial hyperpnoea produced cessation of normal breathing and fall in
arterial carbon dioxide, it is suggested that at high body temperature the effect of the loss of carbon dioxide on the respiratory centre is made up by rise in temperature and thus the activity of the respiratory centre is maintained.

SUMMARY

1. Investigations have been carried out on dogs under chloralose anaesthesia.

2. Arterial carbon dioxide content has been determined at different degrees of rise of body temperature.

3. A steady fall in carbon dioxide content of the arterial blood has been found with a rise in respiratory rate.

4. It has been shown that the effect of fall in carbon dioxide content of the arterial blood on the respiratory centre is made up by rise in temperature.

5. Further, it has been shown that temperature regulation takes precedence over conservation of carbon dioxide.

REFERENCES

