CT-ZONE AND THERMAL PANTING

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Summary: Body temperature of 35 anaesthetised dogs was raised gradually and their respiratory rate recorded. The extent of respiratory response at a given temperature varied from animal to animal. However 28 out of 35 dogs showed panting when the temperature became 42.5°C.

Lesions in the chemoreceptor trigger zone (CT-zone) of the medulla oblongata decreased the respiratory response to hyperthermia at all temperatures. At 42.5°C only one out of nine such dogs showed panting. It is possible therefore that the CT-zone is also involved in the control of panting produced by increase in body temperature.

Key words: thermal panting chemoreceptor trigger zone body temperature regulation

INTRODUCTION

The relative significance of peripheral and central mechanisms of thermal panting has not yet been adequately understood. In unanaesthetised conscious animals panting sets in on mere exposure to hot environment much before the body temperature shows any rise (14, 3 and 4). In anaesthetised animals on the other hand, both peripheral and central mechanisms are said to be responsible for panting (14, 9, 10, 12). Richet (14) thought that the centre for thermal panting was situated in the medulla, and Hammouda (9) found that dogs could pant to thermal stimulus only when the optic thalamus was intact.

The chemoreceptor-trigger (CT) zone in the area prostrema is the site of action for vomiting induced by cardiac glycosides (6), morphine and hydergine (17), nitrogen mustard (5), nicotine and lobeline (11), and sodium salicylate (1). It is also the site of mediation of vomiting due to nonchemical agents like irradiation (8) and swinging (15). In this communication we report the experiments which lead to the inference that CT-zone of the medulla oblongata has a role to play in the control of respiratory responses to increased body temperature in the dog.

MATERIALS AND METHODS

In healthy mongrel dogs of both sexes weighing between 9 and 15 *kg* and anaesthetised with 35 *mg/kg* i.v. pentobarbitone sodium, body temperature was recorded by a thermometer placed high inside the rectum, and respiration was recorded through a balloon placed in between the liver and diaphragm and connected to a tambour. The temperature of the animals was raised by keeping it on a metal plate warmed by means of electric bulbs.

Panting was arbitrarily assumed to appear if the respiration rate increased above 100 per minute (12). Some of the dogs could not bear the thermal stress and died during the course of the experiment. Only 35 dogs that survived upto 42.5°C body temperature were taken into considaration.

In another 9 dogs the fourth ventricle was opened and a lesion made in the area postrema by the method described by Wang and Borison (16). Antibiotic coverage was given upto seven days after the operation. Ablation of the CT-zone was thought complete when 25 *μg/kg* apomorphine given intravenously on two consecutive days did not produce vomiting after the operation (2). Presence of vomiting to a total dose of 300 *mg* copper sulphate given orally through a stomach tube confirmed not only the intactness of the vomiting centre, but also showed that the lesion was not very deep. All the operated dogs started eating normally by the 4th day after the operation, and 7-12 days afterwords these animals were anaesthetised again to observe the effect of induced hyperthermia on respiration.

RESULTS

Results are tabulated in Table I. The mean respiration rate in normal dogs increased gradually upto a temperature of 40.5°C and thereafter showed a sudden and steep rise. Some of the dogs started panting at 41°C, and at 41.5°C the mean respiration rate reached panting level even though only 9 dogs had a respiration rate of over 100 per min. At 42°C and 42.5°C, 25 and 28 dogs respectively were panting out of a total of 35.

The CT-zone ablated dogs also showed a similar behaviour of respiration rate on raising the body temperature, but the response was very feeble. The mean respiration rate never reached panting levels. None of the CT-zone ablated dogs panted throughout the experiment except one out of 9 dogs that panted only at 42.5°C. The number of CT-zone ablated dogs panting at 42°C and 42.5°C differed very significantly from the normal dogs ($P<0.001$).

DISCUSSION

In CT-zone ablated dogs the respiratory response to hyperthermia was feeble and the incidence of panting was low ($P<0.001$). These observations suggest that CT-zone has a role to play in the thermal panting of anaesthetised dogs. The lowered respiratory rates and absence of panting in CT-zone ablated dogs could have been due to the elimination of vagal pulmonary afferents to the nucleus solitarius situated just anterior to the CT-zone. However vagotomy in dogs when panting has set in does not change the respiratory rate (9). Nor, does the inflation or deflation of lungs influence the thermal panting of dogs (9).

It has been suggested that the centre responsible for panting is situated in the hypothalamus (9, 13). Hammouda (9) observed respiratory rate of about 70 per minute in hyperthermic dogs where hypothalamic centre had been ablated. In the light of the present work it would appear that the high respiratory rate in their hyperthermic dogs could be due to the intact CT-zone. Since similar high rate of respiration was also observed in the CT-zone

TABLE I: Showing the effect of CT-Zone ablation on respiration rate and frequency of panting in dogs.

Temperature in degrees C	38.0	38.5	39.0	39.5	40.00	40.5	41.0	41.5	42.0	42.5
Respiration rate Mean ± S.D.										
Control (35)	17 ± 5	23 ± 13	21 ± 8	26 ± 11	30 ± 22	33 ± 18	71 ± 61	101 ± 72	135 ± 76	133 ± 47
CT-zone ablated (9)	8 ± 3	10 ± 9	13 ± 10	14 ± 10	16 ± 11	31 ± 9	22 ± 20	30 ± 24	42 ± 13	71 ± 64
Number of dogs showing panting										
Control (35)	0	0	0	0	1	1	6	9	25	28
CT-zone ablated (9)	0	0	0	0	0	0	0	0	0	1

ablated but with intact hypothalamus dogs of this study it appears that the two regions are independent of each other.

It has been observed that thermal panting disappears whenever the central temperature is lowered to 37°C by cooling the carotid blood even though the body temperature remains elevated (12). Heating the carotid blood did not produce panting in all the dogs. Instead 1/3 of their animals did not pant even though the carotid blood temperature was as high as 46°C. This could be due to the lack of sufficient perfusion of CT-zone with the hot blood which would have occurred in their preparations as a result of the ligation of vertebral arteries.

Brodie and Borison (7) observed an increase in the rate of gasping by electrically stimulating a circumscribed area about 6 mm rostral to the obex and 3 mm outside the midline in the floor of the 4th ventricle in unanaesthetised decerebellate cats. By stimulating areas lateral to this region they observed hyperpnoeic response. Although they have not specifically demarcated these latter regions of the bulb it is likely that they were infact stimulating the CT-zone lying in the lateral aspects of the floor of the 4th ventricle.

REFERENCES

1. Bhargava, K.P., O. Chandra and D.R. Verma. The mechanism of emetic action of sodium salicylate. *Brit. J. Pharmacol.*, **21**:45-50, 1963.
2. Bhargava, K.P., P.C. Gupta and O. Chandra. Effect of ablation of the chemoreceptor trigger zone on the emetic response to intraventricular injection of apomorphine and emetine in the dog. *J. Pharmacol. Exp. Therap.*, **134** : 329-331, 1961.
3. Bligh, J. The initiation of thermal polypnoea in the calf. *J. Physiol*, **136** : 413-419, 1957.
4. Bligh, J. The receptors concerned in the thermal stimulus to panting in sheep. *J. Physiol.*, **146** : 142-151, 1959.
5. Borison, H.L., E. B. Brand, and R.K. Orkand. Emetic action of nitrogen mustard (Mechlorethamine hydrochloride) in dogs and cats. *Am. J. Physiol.*, 192 : 142-151, 1959.
6. Borison, H.L. and S.C. Wang. Locus of the central emetic action of cardiac glycosides. *Proc.Soc. Exp.Biol. and Med.*, **76** : 335-378, 1951.
7. Brodie, D.A. and H.L. Borison. Evidence for an inspiratory pacemaker. Functional concept of central regulation of respiration. *Am. J. Physiol.*, **188** : 347-354, 1957.
8. Chinn, H.I. and S.C. Wang. Locus of emetic action following irradiation. *Proc.Soc.Exp. Biol. and Med.*, **85** : 472-474, 1954.
9. Hammouda, M. The central and reflex mechanism of panting. *J. Physiol.*, **77**: 319-336, 1933.
10. Hemingway, A. The effect of rate of heating and environmental temperature on panting threshold temperatures of normal dogs heated by diathermy. *Am. J. Physiol.*, **122** : 511-519, 1938.
11. Laffan, R.J. and H.L. Borison. Emetic action of nicotine and lobeline. *J. Pharmacol. and Exper. Therap.*, **121** : 468-476, 1957.
12. Lim, P.K. and F.S. Grodings. Control of thermal panting. *Am. J. Physiol.*, 180 : 445-449, 1955.
13. Magoun, H.W., F. Harrison, J.R. Brobeck and S.W. Ranson. Activation of heat loss mechanism by local heating of brain. *J. Neurophysiol.*, **1** : 101-114, 1938.
14. Richet, C. *Dictionnaire de physiol.*, **3** : 175, 1898.
15. Wang, S.C. and H.I. Chinn. Experimental motion sickness in dogs. Functional importance of chemoreceptive emetic trigger zone. *Am. J. Physiol.*, **178** : 111-116, 1954.
16. Wang, S.C. and H.L. Borison. Vomiting centre: Critical experimental analysis. *Arch. Neurol. and Psychiat.*, **63**: 928-941. 1950.
17. Wang, S.C. and V.V. Glaviano. Locus of emetic action of morphine and hydergine in dogs. *J. Pharmacol. and Exper. Therap.*, **111** : 329-334, 1954.