ROLE OF VAGUS DURING EXERCISE IN THERMAL PANTING

R. SAXENA, S. KUMAR, S. . TANDON AND P.C. JAIN

Department of Physiology,

King George's Medical College, Lucknow—226 003

(Received on May 5, 1978)

Summary: In dogs anaesthetised with pentobarbitone sodium raising the body temperature from 37°C during mild exercise increased the rate of respiration and pulmonary ventilation but decreased the tidal volume. Cold blocking the vagi during the exercise resulted in decrease in respiration rate and minute ventilation, but increase in tidal volume. At 40°C body temperature vagal block was not effective in decreasing the respiration rate and minute ventilation, which may be due to stimulation of lung irritant receptors through hyperthermia.

Key words: thermal panting; temperature regulation; lung irritant receptors; exercise

INTRODUCTION

Increase in both pulmonary ventilation and respiration rate occur in dogs under hyperthermic conditions as also by exercise. Sensory impulses from the exercising muscles are predominantly responsible for the respiratory change though humoral factors from the muscles also play a role (5). Resting body temperature of the dogs may be as high as 40.5°C during summer months in this country (4). Even at such high body temperature the dogs are known to run about. Cold blocking the vagi increased the respiration rate at 40°C, whereas at body temperatures below and above this it reduced the respiration rate (8). How will vagi affect the pulmonary ventilation and respiration rate when it is already under hyperthermic stimulation and is not stimulated by impulses from the exercising muscles is the object of study in the present investigation.

MATERIAL AND METHODS

The present study was conducted in a set of 25 healthy mongrel dogs of either sex. They were anaesthetized with sodium pentobarbital (30 *mg/kg*) given intraperitoneally. The temperature of the animal was brought to 37°C by cooling or warming of the animal as the case required. Respiration rate, tidal volume and minute ventilation were recorded by method described earlier (8). The hind limbs were exercised by square wave impulses of 25 m sec duration delivered every one second at 60 V from a Radart Electronic Stimulator. For maximum effectiveness stimuli were delivered through broad based brass electrodes tied on the thighs after shaving off hair from the skin. To rule out the possibility of any sensory nerve impulses from the exercising muscles affecting the respiration both the hind limbs of the animal were denervated by cutting, sciatic, obturator and femoral nerves (1). Vagal impulses were cold blocked by the method described earlier (8). The temperature of the animal was raised by applying radiant heat from two 40 watt carbon bulbs and by raising the temperature of the animal plate. Starting at 37°C, readings were taken at every 1°C rise of body temperature till the animal died or temperature of 42°C was attained. Exercise of hind limbs was continued for 5 minutes and record was made during the last one minute both before and during the vagal block.

Percentage change at each degree rise of body temperature in respiration rate, tidal volume and minute ventilation was calculated with respect to their values at 37°C, without and with vagi blocked respectively.

RESULTS

The initial respiration rate at 37°C body temperature varied between 12-30 min with a mean rate of 19±5, while during exercise at the same temperature the respiration rate varied between 14 to 36 with a mean of 23±6.3/min. With the rise in body temperature the respiration rate increased, and it further increased when the animal was subjected to exercise. The percentage increase of respiration rate during exercise at 38, 39, 40,41 and 42°C body temperature was 48,78,322 465 and 282% respectively. With the increase in respiration rate the tidal volume decreased and the minute ventilation increased (Table I). Since the magnitude of these parameters decreased at a body temperature of 42°C, it signified onset of respiratory failure.

Blocking the vagi while the dog was being exercised did not produce any change of mean rate of respiration at 37°C, but on raising the body temperature and continuing the exercise vagal block resulted in a decrease in respiration rate, except at 40°C, when it was found to increase (Table I). This increase was found to be statistically significant.

TABLE I : Percentage changes in the mean respiration rate, tidal volume and minute ventilation during exercise at rising body temperature.

		Vagi not blocked			*Vagi blocked*		
Rectal temp.	*No. of dogs*	*Resp. rate*	*Tidal volume*	*Minute ventilation*	*Resp. rate*	*Tidal volume*	*Minute ventilation*
38°C	20	+ 48	— 3	+ 20	+ 39	— 22	+ 26
39°C	20	+ 78	— 4	+ 30	+ 39	— 27	+ 37
40°C	17	+322	—16	+ 85	+334	—140	+116
41°C	13	+465	—23	+200	+439	— 65	+208
42°C	9	+282	—32	+ 55	+182	—280	+ 13

DISCUSSION

The exercise stimulus to the hind limbs at 1/sec was considered as a mild degree of exercise. Exercise increased the respiration rate. How does the exercise stimulus reach the respiratory centre is a matter of speculation. It obviously is not nervous because hind limbs had been denervated. It possibly is not a change in arterial pCO_2, pO_2 or pH as changes in these parameters in mild to moderate exercise were not observed by Sinnott (9).

TABLE II : Average changes in respiration rate, tidal volume and minute ventilation at rising body temperatures in dogs exposed to exercise, both without and with vagal block.

Body temperature		37°C	38°C	39°C	40°C	41°C	42°C
No. of dogs		18	20	20	14	11	8
	A	+ 2.89	+ 1.70	+ 3.50	+17.28	± 0	— 4.25
Respiration rate	B	+ 2.11	+ 2.60	+ 3.70	+ 1.57	+ 1.45	+ 6.25
	(A-B)	+ 0.77	— 0.90	— 0.20	+15.71	— 1.45	—10.50
	A	+ 8.55	—10.40	— 9.85	—10.43	— 9.18	— 5.0
Tidal volume	B	+ 2.89	— 4.25	— 3.45	— 6.0	+ 3.64	+ 1.25
	(A-B)	+ 5.66	— 6.15	— 6.40	— 4.43	—12.82	— 6.25
	A	+ 0.50	— 0.15	+ 0.19	+ 1.58	— 0.99	— 1.01
Minute ventilation	B	+ 0.44	+ 0.22	+ 0.86	— 0.44	+ 0.53	— 0.01
	(A-B)	+ 0.06	— 0.37	— 0.67	+ 2.02	— 1.52	— 1.00

Note A = Without vagal block.
B = With vagal block.

Bozler and Burch (2) observed that blocking the vagi increased the respiration rate in a few of their experiments. They suggested this to be due to chemical regulation of respiration. But recently Mills *et al.* (7), Luck (6) and Hillary and Widdicombe (3) revealed a distinct group of endings called the lung irritant receptors localized to the lungs by mechanical stimulation with an endobronchial catheter. Stimulation of these receptors resulted in increase in respiration rate. These irritant receptors, although mechano-sensitive, have properties quite different from other pulmonary stretch receptors. They usually have no spontaneous discharge in eupnoeic anaesthetized animals, although if the animal is breathing deeply (as after vagotomy) there may be a few impulses with each inspiration and/or expiration. Their response to a chemical or mechanical stimulus is rapidly adapting, with brief high frequency burst of irregular discharge. The authors have not come across any reference suggestive of body temperature affecting their rate of discharge. It may be that rise in body temperature stimulates either certain yet unknown pulmonary receptors or the lung irritant receptors themselves resulting in polypnoea. The significance of 40°C body temperature in the physiology of these receptors became obvious when during exercise the difference in respiration rate on blocking the vagi was calculated (Table II) and plotted against temperature (Fig 1). Exercise alone produced only a moderate increase in respiration rate at all body temperatures. This was however, maximum at 40°C. On blocking the vagi this difference due to exercise became minimum at 40°C. It was therefore, argued that the vagi at 40°C body temperature play a very

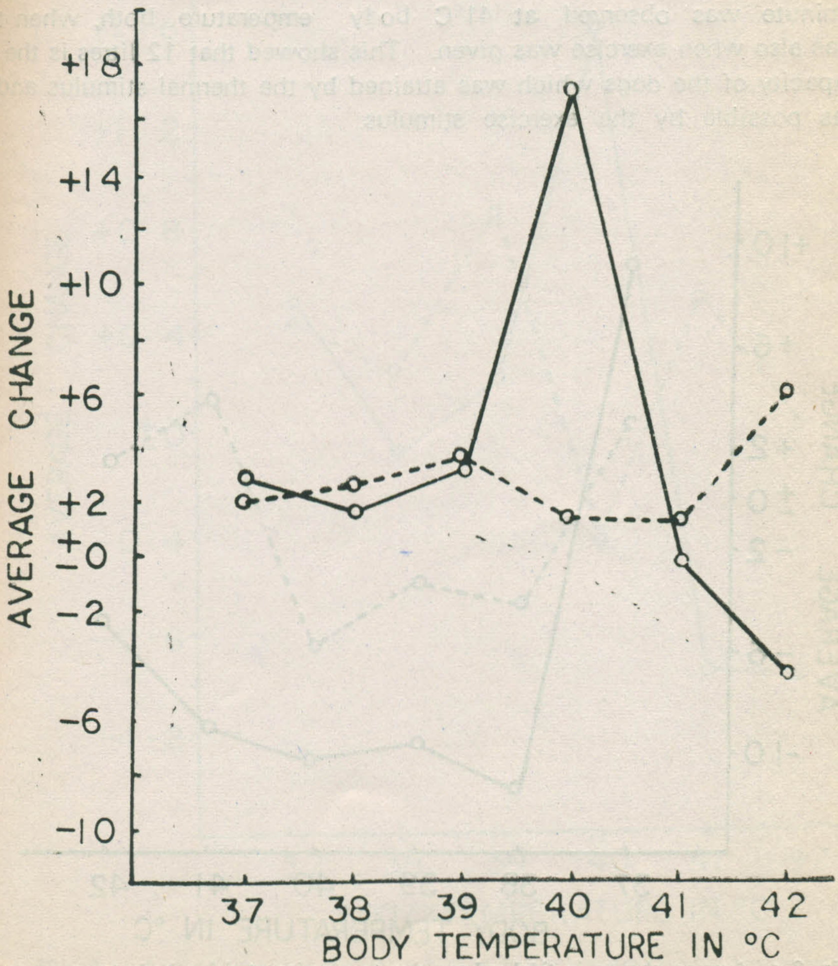

Fig. 1 Average change in respiration rate due to exercise at rising body temperatures both without (continuous line) and with (interrupted line) vagal block.

dominant role in the regulation of respiration rate. Vagi also exerted a regulatory effect on minute ventilation (Fig. 3) but the effect on tidal volume was not very remarkable (Fig. 2).

The pulmonary ventilation under exercise at 37°C body temperature increased 4±0.6 litre from the resting level of 3.5±0.4 litre. When the body temperature was raised to 38, 39, 40, 41 and 42°C and exercise was continued the minute ventilation increased by 20, 30, 85, 200 and 55% respectively (Table I). On blocking the vagi the magnitude of this increase was 26, 37, 116, 208 and 13% respectively (Table I). Statistically the finding was significant at 40°C temperature ($P<0.001$). Maximum ventilation of 12 litres per minute was observed at 41°C body temperature both, when there was no exercise as also when exercise was given. This showed that 12 litres is the maximum breathing capacity of the dogs which was attained by the thermal stimulus and no further increase was possible by the exercise stimulus.

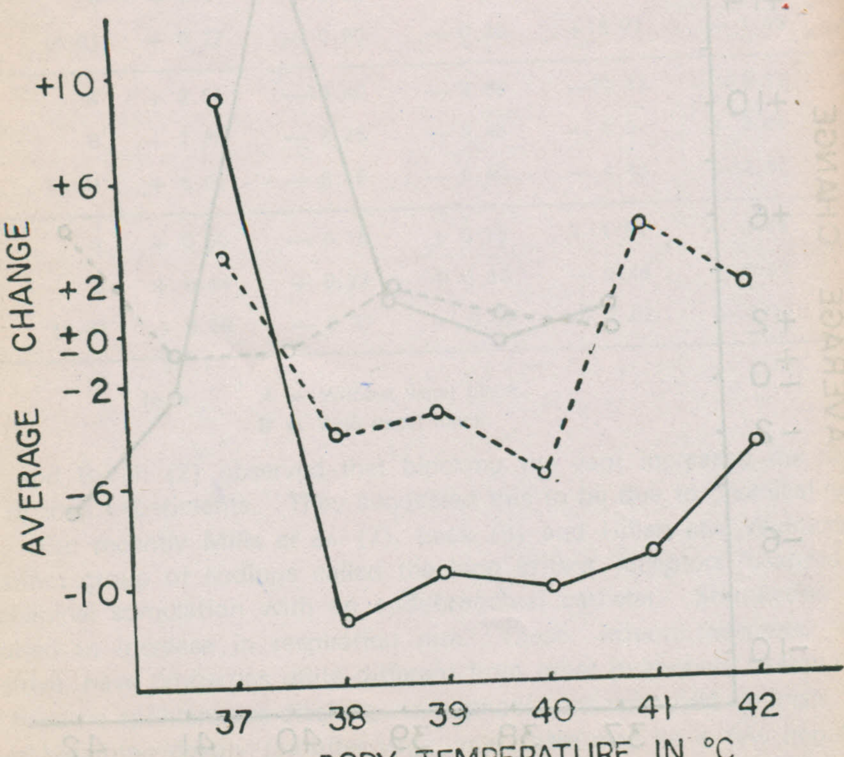

Fig. 2 : Average change in tidal volume due to exercise at rising body temperature both without (continuous line) and with (interrupted line) vagal block.

The tidal volume during exercise at body temperature of 37°C insignificantly increased to a mean value of 155±22 *ml* from the resting level of 149±18 ml. On continuing the exercise at the rising body temperatures of 38,39,40,41 and 42°C the tidal volume decreased by 3,4,16,23 and 32% respectively. On blocking the vagi under similar condition the decrease in tidal volume was 22,27,140,65 and 280% respectively (Table I). The consistant decrease in tidal volume at rising body temperatures whether vagal afferents were blocked or not resulted in better and more effective heat loss. Such a respiration is not meant as much to meet out the gaseous requirement of exercise as

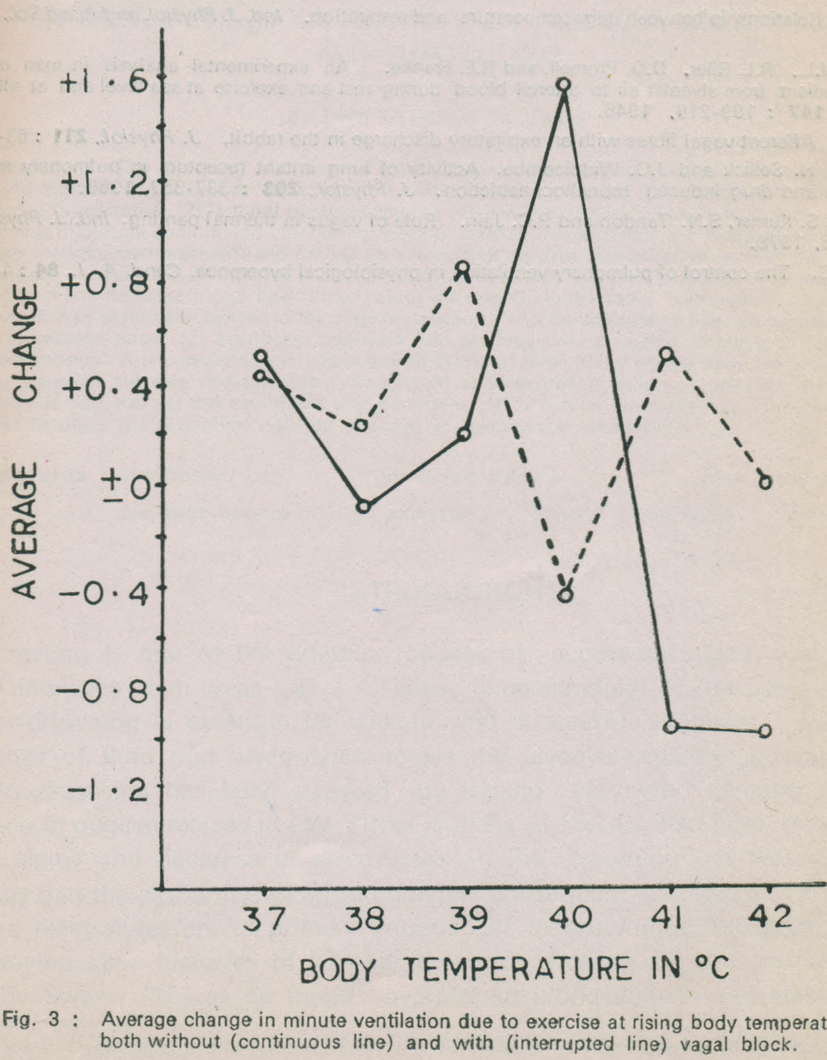

Fig. 3 : Average change in minute ventilation due to exercise at rising body temperature both without (continuous line) and with (interrupted line) vagal block.

much as it appears to be playing temperature regulatory function of respiration unde hyperthermic state of the body.

REFERENCES

1. Biscoe, T.J. and H.J. Purves. Factors affecting the cat carotid and cervical sympathetic activity with spe reference to passive hind limb movements. *J. Physiol.*, **190** : 425-441, 1967.
2. Bozler, E. and B.H. Burch. Role of the vagus in the control of respiration. *Amer. J. Physiol.*, **166** : 255-2 1951.
3. Hillary, S. and J.G. Widdicombe. The activity of lung irritant receptors during pneumothorax, hyperpnoea an pulmonary vascular congestion. *J. Physiol.*, **203** : 359-381, 1969.
4. Kumar, S. Relationship between dogs temperature and respiration. *Ind. J. Physiol. and Allied Sci.*, **7** : 203-2 1953.
5. Lilienthal, J.L., R.L. Riler, D.D. Promell, and R.E. Franke. An experimental analysis in man of the oxyg pressure gradient from alveolar air to arterial blood during rest and exercise at sea level and at altitude. *Ame J. Physiol.*, **147** : 199-216, 1946.
6. Luck, J.C. Afferent vagal fibres with an expiratory discharge in the rabbit. *J. Physiol.*, **211** : 63-71, 1970.
7. Mills, J.E., H. Sellick and J.G. Widdicombe. Activity of lung irritant receptors in pulmonary microemboli anaphylaxis and drug induced bronchoconstriction. *J. Physiol.*, **203** : 337-357, 1969.
8. Saxena, R., S. Kumar, S.N. Tandon and P.C. Jain. Role of vagus in thermal panting. *Ind. J. Physiol. Pharmac.* **22** : 39-45, 1978.
9. Sinnott, J.G. The control of pulmonary ventilation in physiological hyperpnea. *Cand. A. J.*, **84** : 471-483, 196