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Effects of High Environmental Temperature and Exercise on Cardiorespiratory Function and Metabolic Responses in Steers

Takashi UENO[1]*, Yuji TAKEMURA[2], Kichiro SHIJIMAYA[1], Fumihiro OHTANI[3] and Riohei FURUKAWA[4]

Department of Animal Physiology, National Institute of Animal Industry
(Tsukuba, Ibaraki, 305–0901 Japan)

Abstract

Changes in the cardiorespiratory function and the metabolic responses induced by walking exercise (40 m/min, 80 m/min) in Holstein steers in thermoneutral (18ºC) and hot (33ºC) environments were investigated. Cardiac output and stroke volume at rest, during and after exercise in a hot environment, except during exercise at 80 m/min, were larger than in a thermoneutral environment. Stroke volume was larger in a hot environment and during exercise, whereas the mean arterial blood pressure was not different between thermal environments, or exercise levels. Total peripheral resistance was lower in a hot environment than in a thermoneutral one, and it also decreased in both environments when the exercise was imposed on the steers. Respiratory rate and respiratory minute volume during the resting period were larger in a hot environment than in a thermoneutral one. However, the extent of the increase in both parameters induced by the exercise was not affected by the environmental temperature. After exercise in a hot environment, alveolar ventilation reverted to the initial levels within 5 min, while both respiratory minute volume and dead space ventilation still remained at higher levels. These results indicate that the change in the respiratory minute volume after exercise in a hot environment was due mainly to the change of the respiratory dead space. The higher the walking speed was, the more the heat production increased during exercise. Also, heat production during exercise was somewhat higher in a hot environment than in a thermoneutral one.

Discipline: Animal industry
Additional key words: hot environment, walking, cattle, physiological response

Introduction

A hot environment induces a remarkable decrease in the productivity of domestic animals. Physiological aspects, however, have not been fully elucidated. The cardiorespiratory system is related to the energy metabolism and the body temperature regulation through gas exchange and heat transport, and its function is influenced by thermal environments[7].

In our previous report[10], the effects of a high environmental temperature on the cardiorespiratory function and energy metabolism were investigated in sheep. We reported that the dead space ventilation, which is associated with evaporative heat loss, increased more than the alveolar ventilation in a hot environment. Above thermal neutrality, cutaneous evaporation plays a secondary role in evaporative cooling in sheep, in contrast to its important role in cattle[2,6]. Therefore, the cardiorespiratory responses observed in a hot environment might be different between cattle and sheep.

These experiments were conducted to investigate the effects of high environmental temperature and exercise on the cardiorespiratory function and metabolic responses in Holstein steers. To analyze the range of adaptability of the cardiorespiratory function in each

Present address:
[1] Department of Research Planning and Coordination, National Institute of Animal Industry (Tsukuba, Ibaraki, 305–0901 Japan)
[2] Faculty of Agriculture, Tokyo University of Agriculture and Technology (Fuchu, Tokyo, 183–8509 Japan)
[3] Department of Animal Production, Hokkaido National Agricultural Experiment Station (Toyohira-ku, Sapporo, 062–8555 Japan)
[4] Department of Grazing, National Grassland Research Institute (Nishinasuno, Tochigi, 329–2793 Japan)
*Corresponding author: fax +81–298–38–8573, e-mail oneu@niai.affrc.go.jp
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thermal environment, exercise load tests were conducted.

Materials and methods

1) Animal preparation

Two Holstein steers, weighing about 317 and 334 kg, respectively, were used. Preliminarily, they were surgically prepared under anesthesia one month before the onset of the experiment, so that the left carotid artery was pulled out externally and was covered with the skin by a suture.

The steers were kept on their respective stanchion stool in a controlled-environment room (room-A) of the zootron facilities described by Furukawa et al.[4], and exposed for 2 weeks to each of the 2 climatic conditions, i.e. 18 and 33ºC. Relative humidity was 50% in both environments. Each steer was fed approximately 10 kg Italian ryegrass hay wafer and 1 kg concentrate daily. During the preliminary training period, the animals had to walk on the treadmill used in the walking trials, and to allow the attachment of a face mask to collect the expired air.

Approximately 2 h before the initiation of each trial, the steers were transferred to an another room (room-D) equipped with a treadmill apparatus, the climatic conditions of which were controlled in the same way as in room-A. Soon after entering the room, a silicon catheter with a bore of 1.0 mm and an external diameter of 2.0 mm was inserted into the right jugular vein and the left carotid artery. To prevent coagulation of blood, the catheters were filled with heparinized physiological saline (80 IU sodium heparin in 1 mL saline).

2) Measurements

The steers were subjected to each of the following experimental conditions on a separate day: (1) 40 m/min walking at 18ºC; (2) 80 m/min walking at 18ºC; (3) 40 m/min walking at 33ºC; (4) 80 m/min walking at 33ºC. Each trial was repeated twice a week.

Experimental schedules in each trial are shown in Fig. 1. The steers were exercised on a treadmill at the walking speed of 40 or 80 m/min for 20 min and then were allowed to rest for recovery for 40 min. Measurements of respiratory changes were performed using the open circuit method. Face mask was attached at each sampling time (Fig. 1). The expired air through a face mask was collected into a Douglas bag for 2–3 min and the respiration volume was measured with a wet-type gas meter (WT5A, Shinagawa, Japan).

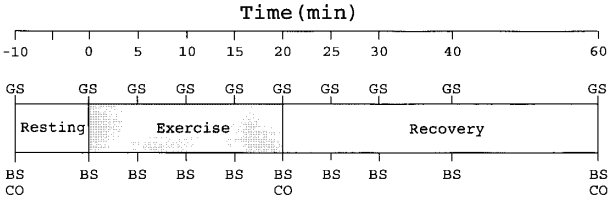

Fig. 1. Sequence of events during the experimental period
Expired gas samples (GS) and blood samples (BS) were obtained and cardiac output (CO) was measured at the indicated times.

The concentration of O_2 in the expired air was measured with a paramagnetic oxygen analyzer (Model-755, Beckman, USA) and those of CO_2 and CH_3 were measured with an infrared gas analyzer (URA-3B, Shimadzu, Japan). Heat production was calculated according to the Brouwer's formula[3].

Electrocardiogram (ECG) was continuously recorded by A-B lead, bipolar lead along the longitudinal axis of the heart, using bio-potential skin electrodes (Type-NS, Nihon Kohden, Japan) and a bioelectric amplifier (AB-621G, Nihon Kohden, Japan). Heart rates were counted from the ECG records. Cardiac output was measured by a dye dilution technique using indo-cyanine green dye (Diagnogreen, Daiichi Pharmacy Co., Japan) as an indicator. One mL of a 5 mg/mL solution of the dye in distilled water was injected into the right jugular vein through a catheter, and arterial blood samples from the left carotid artery were drawn through a catheter into a cuvette-densitometer at a rate of 16.5 mL/min with a withdrawal-infusion pump (Erma Optical Workes, Ltd., Japan). Cardiac output was calculated using a cardiac output computer (EQ-611V, Nihon Kohden, Japan). Arterial blood pressure was measured by using a pressure transducer (Model P23-1D, Gould Stathan Instrument, USA) in conjunction with a carrier amplifier (AP-601G, Nihon Kohden, Japan) and blood pressure curves were drawn on a thermal recorder (WT-647G, Nihon Kohden, Japan). Total peripheral resistance (W) was calculated by the Whittow's formula[12] , W=1332xP/Q [P: mean blood pressure (mmHg), Q:cardiac output (mL/min)].

Arterial and venous blood samples were drawn for blood gas analyses. The Pco_2 of the blood samples was measured with a blood gas analyzer (Model-580, Corning, USA). Dead space ventilation was calculated from the modifired Bohr's formula[9] as follows: $V_D = V_t \cdot (PA_{CO2} - PE_{CO2})/PA_{CO2}$ [V_D: dead space ventilation, V_t : tidal volume, PA_{CO2}: partial pressure of CO_2 in the arterial blood, PE_{CO2}: partial pressure of expired CO_2], and alveolar ventilation was taken as the difference between the respiratory minute volume and dead space ventilation.

Rectal temperature was continuously monitored with a thermistor probe inserted at 20 cm into the rectum.

Results and discussion

1) Cardiovascular function

The effects of the environmental temperature and exercise load on the heart rate and mean arterial blood pressure are shown in Fig. 2. The heart rate during the resting period was within a narrow range of 50–65 beats/min, regardless of the environmental temperatures to which the animals had been exposed. The heart rate sharply increased by the exercise loads. And the extent of the increase in the heart rate was proportional to the walking speed. No appreciable effect of the environmental temperature on the increase in the heart rate during the exercise period was recognized. Similar results for the heart rate responses to exercise were observed in our previous experiment on sheep[10]. In all the trials of the present experiments, the increase of the heart rate by exercise reverted to the resting level within 5 min after the end of the exercise.

During the resting period, the value of the blood pressure varied within a range of 90–115 mmHg. No appreciable effects of the environmental temperature on the blood pressure levels were observed. During the 3 experimental periods, the values of the blood pressure in the 80 m/min walking trials were somewhat higher than those in the 40 m/min walking trials, but no significant differences were recorded. The blood pressure responded to the exercise loads in the thermoneutral environment by increasing for only a few minutes and then decreasing below the resting levels. In the hot environment, the blood pressure responded by fluctuating within the range of resting levels for some time and then decreasing.

The changes in the cardiac output (L/min) and stroke volume (L/beat) during the resting, exercise and recovery periods are shown in Fig. 3. The values of the cardiac output and stroke volume during the resting period were significantly higher at 33ºC than at 18ºC ($p<0.05$). Similar results as those observed in the present study were also reported in oxen by Whittow[13]. Since the differences in the heart rate between the environmental temperatures of 18ºC and 33ºC were not significant, it was assumed that the increase in the cardiac output at 33ºC might be due to the increase in the stroke volume. The cardiac output sharply increased by exercise, and then reverted to the resting level within 40 min after the end of the exercise.

In most of the cases, the stroke volume increased by the exercise, but decreased when the animal was exam-

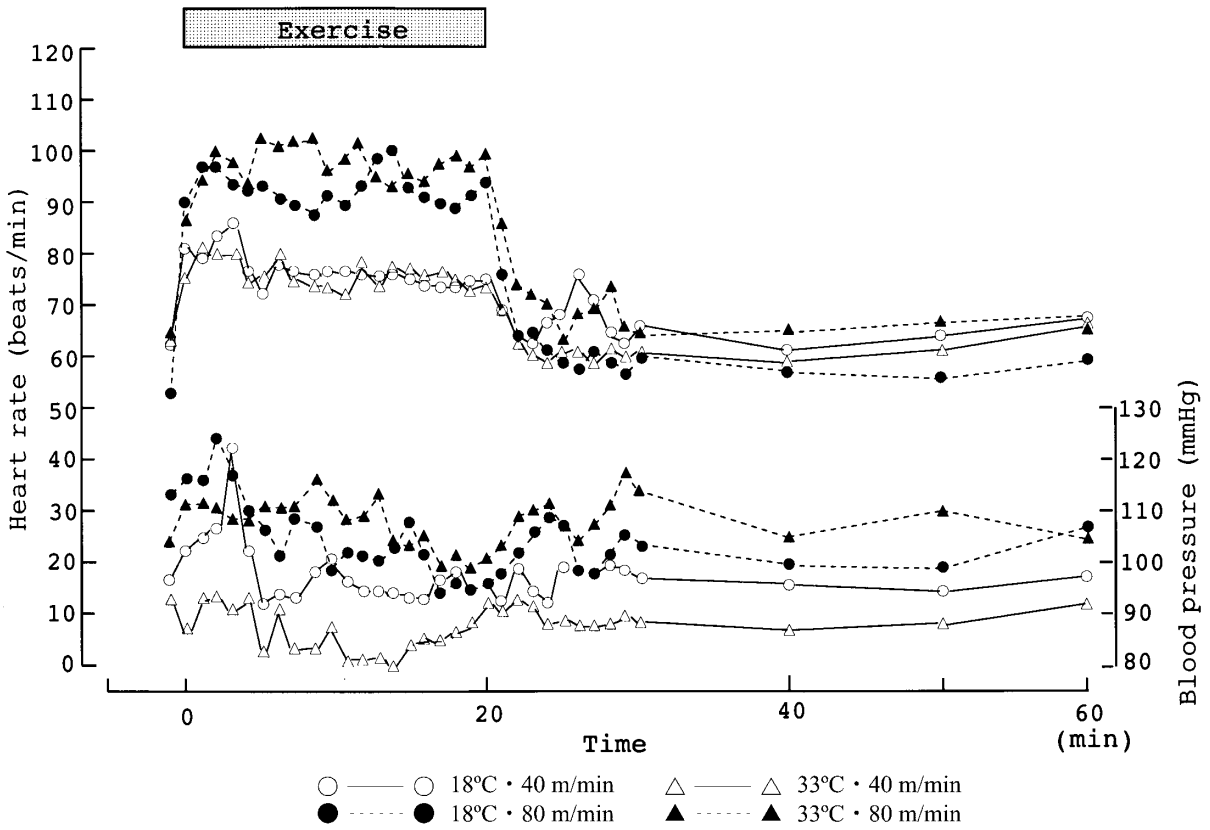

Fig. 2. Effects of environmental temperature and exercise load on heart rate and blood pressure

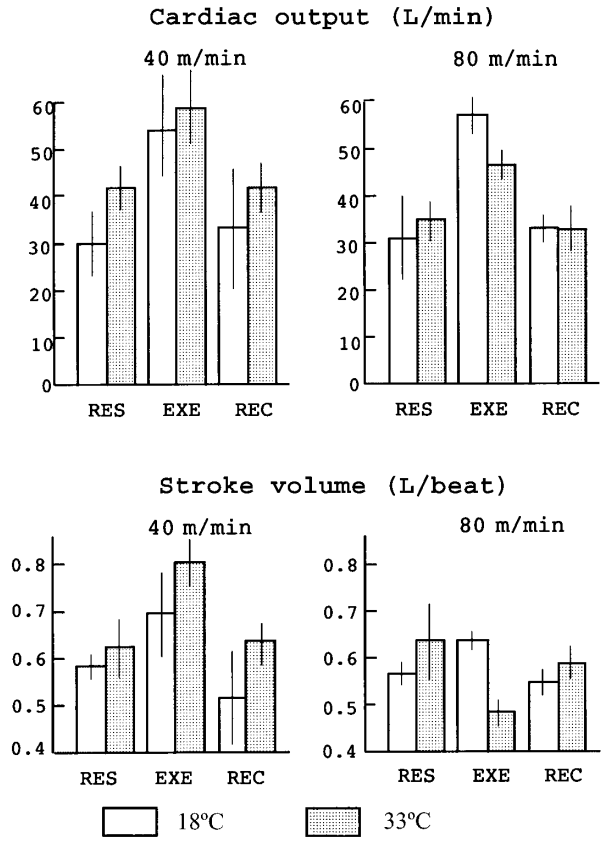

Fig. 3. Changes in cardiac output and stroke volume during resting (RES), exercise (EXE) and recovery (REC) period

ined in a 80 m/min walking trial at 33ºC. There was some inconsistency about this aspect in our previous experiment on sheep[10] that was conducted under similar experimental conditions, in which the stroke volume increased by both the exercise at low and high walking speeds in a hot environment of 33ºC. It was reported, however, in men[8] that the cardiac output and the stroke volume decreased by an intensive exercise at a high environmental temperature. These phenomena can be interpreted as follows: The increase in the cardiac output in a hot environment can contribute to the acceleration of sensitive heat loss from the skin surface due to the increase of the blood flow. However, when an exercise is performed, the circulation blood volume increases more due to the blood flow demand of the muscle for activity in addition to that of the skin for heat radiation. Therefore, sometimes the venous return to the heart decreases and consequently the cardiac output and/or the stroke volume decrease. While the cardiac output increased remarkably in the hot environment and during the exercise loads, the mean blood pressure did not increase in the hot environment or during the exercise loads except for a few minutes at the beginning of the exercise at 18ºC, as shown in Fig. 2. This phenomenon might be related to the changes in the peripheral circulatory resistance, namely, an increase of the total peripheral resistance might have contributed to the constant level of the blood pressure. However, the cause of the initial increase in the blood pressure might be related to the delayed vasodilator reflex of peripheral vessels. As indicated in Table 1, the values of the total peripheral resistance during the resting period were rather lower in the hot environment than in the thermoneutral one, and they also decreased during the exercise period. These findings are similar to those observed in heat-exposed oxen[13] and exercised horses[1]. It is considered that these changes were associated with the vasodilation of the skin to promote heat loss from the skin surface.

2) Respiratory function

Changes in the respiratory function caused by heat exposure and the exercise are shown in Figs. 4 and 5. The respiration rate during the resting period ranged from 15 to 20 per min at the temperature of 18ºC, while the rate increased 4-fold at 33ºC. Regardless of the thermal environments, the increase of the respiration rate by exercise was about 10/min and 30/min at a walking speed of 40 m/min and 80 m/min, respectively. The environmental temperature did not affect the increase of the respiration rate by the exercise. Under the heat stress of 33ºC, rapid and shallow pantings were observed. The resting value of the respiration volume increased from 23 L/min

Table 1. Effects of environmental temperature and exercise load on total peripheral resistance (dynes-sec cm^{-5})*

Periods	18ºC		33ºC	
	40 m/min	80 m/min	40 m/min	80 m/min
Resting	317.1 ± 74.7	316.9 ± 78.2	181.2 ± 23.1	241.4 ± 6.1
Exercise	141.2 ± 25.3	134.1 ± 2.7	128.4 ± 27.3	183.1 ± 9.0
Recovery	249.1 ± 86.0	259.2 ± 5.8	173.6 ± 16.7	257.6 ± 6.1

*1332×(Mean arterial pressure [mmHg])/(Cardiac output [mL/sec]).
Values are means ± SD of 2 steers.

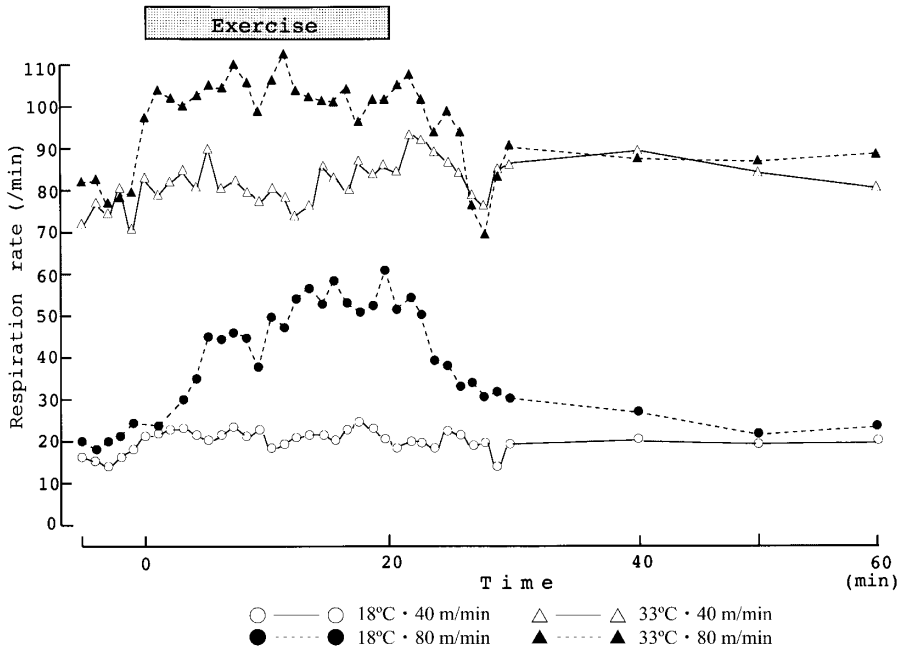

Fig. 4. Effects of environmental temperature and exercise load on respiration rate

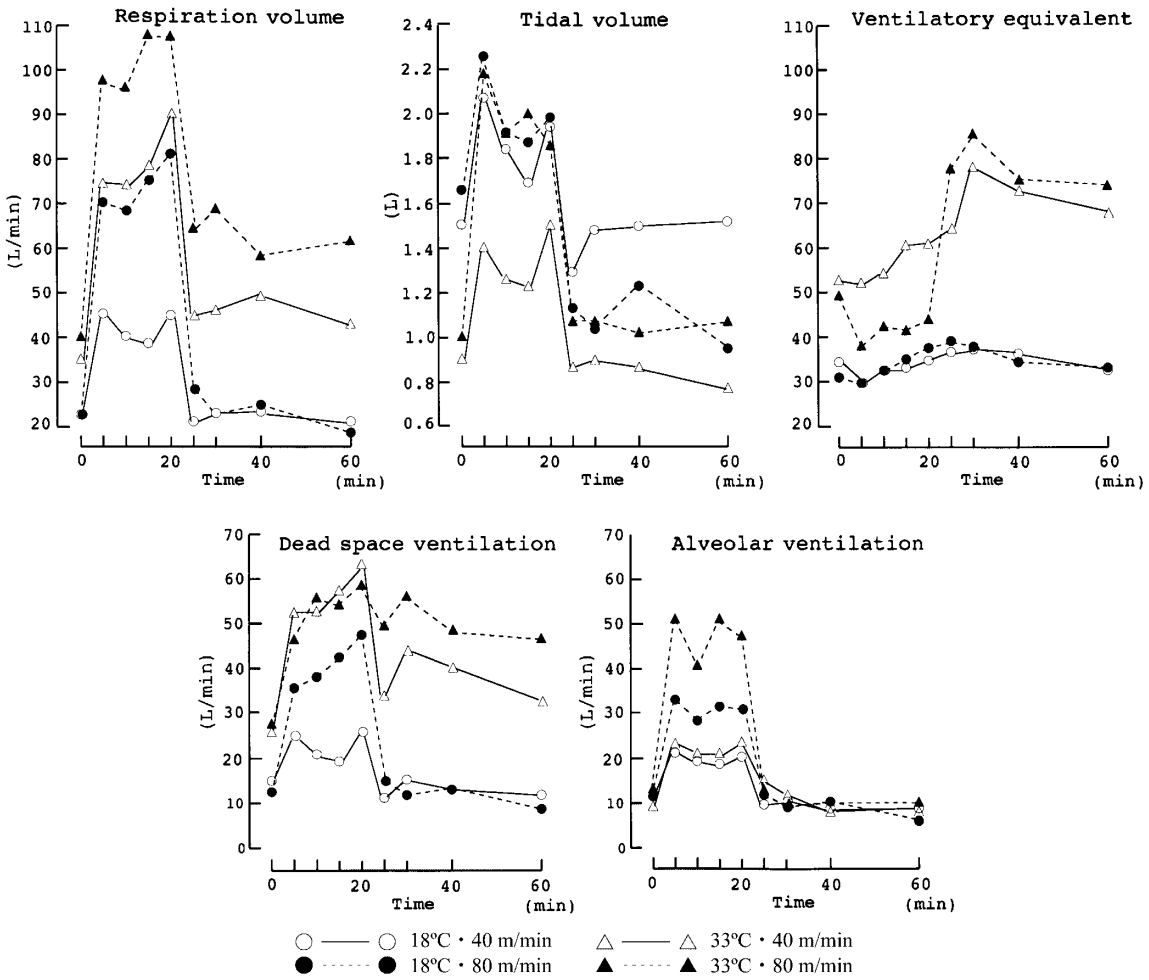

Fig. 5. Effects of environmental temperature and exercise load on respiratory function

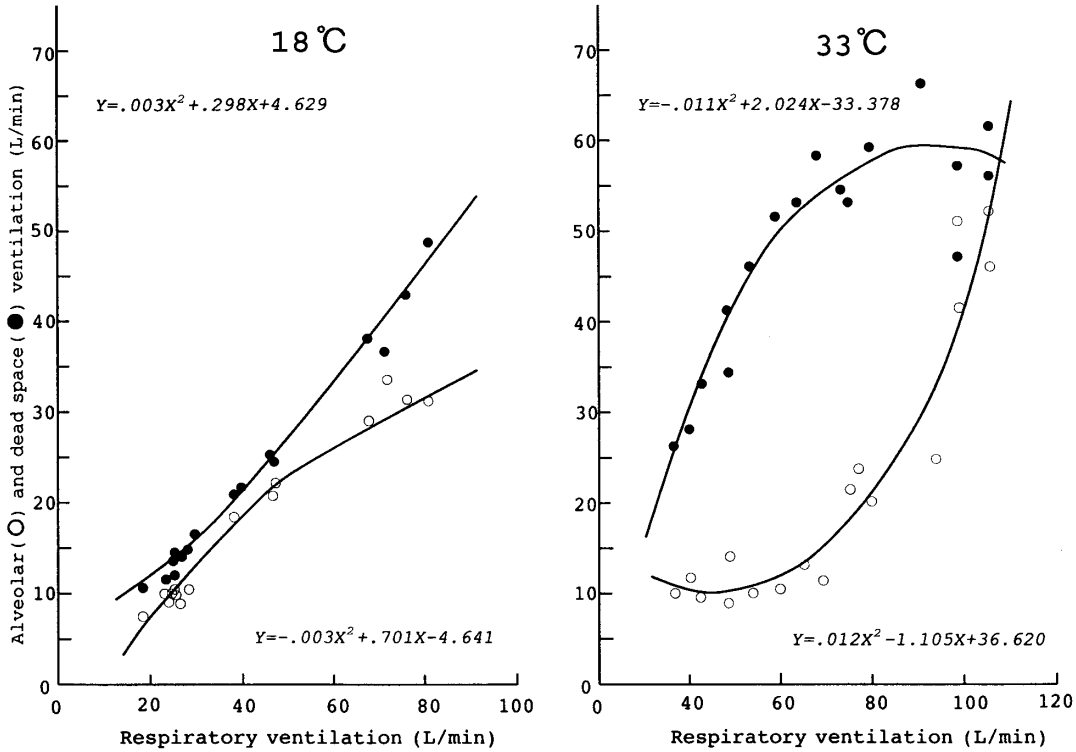

Fig. 6. Changes in alveolar and physiological dead space ventilation with increasing total respiratory ventilation of steers in environments of 18ºC (left) and 33ºC (right)

to 40 L/min by heat exposure, while the value of the tidal volume decreased from 1.6 L/min to 1.0 L/min by the exposure.

The ventilation equivalent is defined as the ventilation volume which is required for absorbing 1 L of oxygen, and it is one of the indices for measuring the efficiency of respiration. The ventilation equivalent during the resting period at 18ºC was in the range of 30–35. These values were somewhat higher than those (25–30) observed in sheep in our previous experiment[10], suggesting that the respiration efficiency of steers is lower than that of sheep in a thermoneutral environment. However, in the hot environment of 33ºC, the ventilation equivalent in steers increased to 48–52, while in sheep to about 160. These results indicate that the evaporative heat loss from the upper respiratory tract is more important in sheep than in steers in a hot environment. Brockway et al.[2] reported that the respiratory heat loss in sheep increased with the elevation of the environmental temperature, while the evaporative heat loss from the skin surface remained constant regardless of environmental temperatures.

The increase of the respiration volume by exercise reverted to the resting level almost within 5 min after the end of the exercise at 18ºC, while it did not revert to the resting level within 40 min after the exercise at 33ºC. Both the alveolar and the dead space ventilation increased by the exercise loads. And the increase of the alveolar ventilation reverted to the resting level almost within 5 min after the exercise, regardless of the thermal conditions. In contrast, the increase of the dead space ventilation, as well as the increase of the respiration volume, reverted to the resting level almost within 5 min after the exercise at 18ºC, but not within 40 min after the exercise at 33ºC. These results indicate that the delay in the recovery of the ventilation volume at 33ºC was mainly due to the delay in that of the dead space ventilation. In the hot environment of 33ºC, the dead space ventilation might have been maintained at a high level for a long period of time even after the exercise to dissipate the stored heat which was generated by the muscular activity during the exercise.

Fig. 6 shows the changes in the alveolar and dead space ventilation with increasing total respiratory ventilation (respiratory minute volume) in each thermal environment. At the environmental temperature of 18ºC, the values of the dead space ventilation were somewhat higher than those of the alveolar ventilation. And both values increased gradually with the increase of the respiratory ventilation. At the environmental temperature of 33ºC, the value of the dead space ventilation was significantly higher than that of the alveolar ventilation

($p<0.05$). And there were few changes in the alveolar ventilation until the total respiratory ventilation increased to almost 2 or more times compared to the initial value. However, when the respiratory ventilation exceeded 60 L/min, the dead space ventilation reached a plateau level, while the alveolar ventilation sharply increased. Similar results were reported in heat-stressed oxen by Hales[5], indicating that the partition of ventilation was markedly influenced by panting.

3) Metabolic responses

The effects of the environmental temperature and exercise load on the energy metabolism are shown in Fig. 7 . The heat production in relation to the metabolic body size during the resting period ranged from 0.17 to 0.22 kJ/min and it was not influenced by the environmental temperatures to which the animals had been exposed. Heat production increased by the exercise in proportion to the walking speed, and remained at a high level throughout the period. However, the value returned to the resting level within 5 min after the end of the exercise, regardless of the environmental temperatures and the walking speeds. The value of heat production during the exercise period tended to be higher at 33ºC than at 18ºC, presumably due to the vant'Hoff-Arrhenius effect. However, based on the results of the blood gas analysis[11], hyper-ventilation associated with shallow panting caused an increase in the CO_2 concentration in the expired gas. Therefore it can be considered that due to the increase in the CO_2 concentration, heat production might have been over-estimated by using the Brouwer's equation[3], and consequently the value was somewhat higher at 33ºC.

During the resting period, the body temperature tended to be higher at 33ºC than at 18ºC. And the body temperature increased gradually during the exercise period. The higher the walking speed was, the more rapidly the rectal temperature increased. No appreciable effects of the environmental temperature were recognized on the extent of increase in the body temperature during the exercise period, but the differences between the body

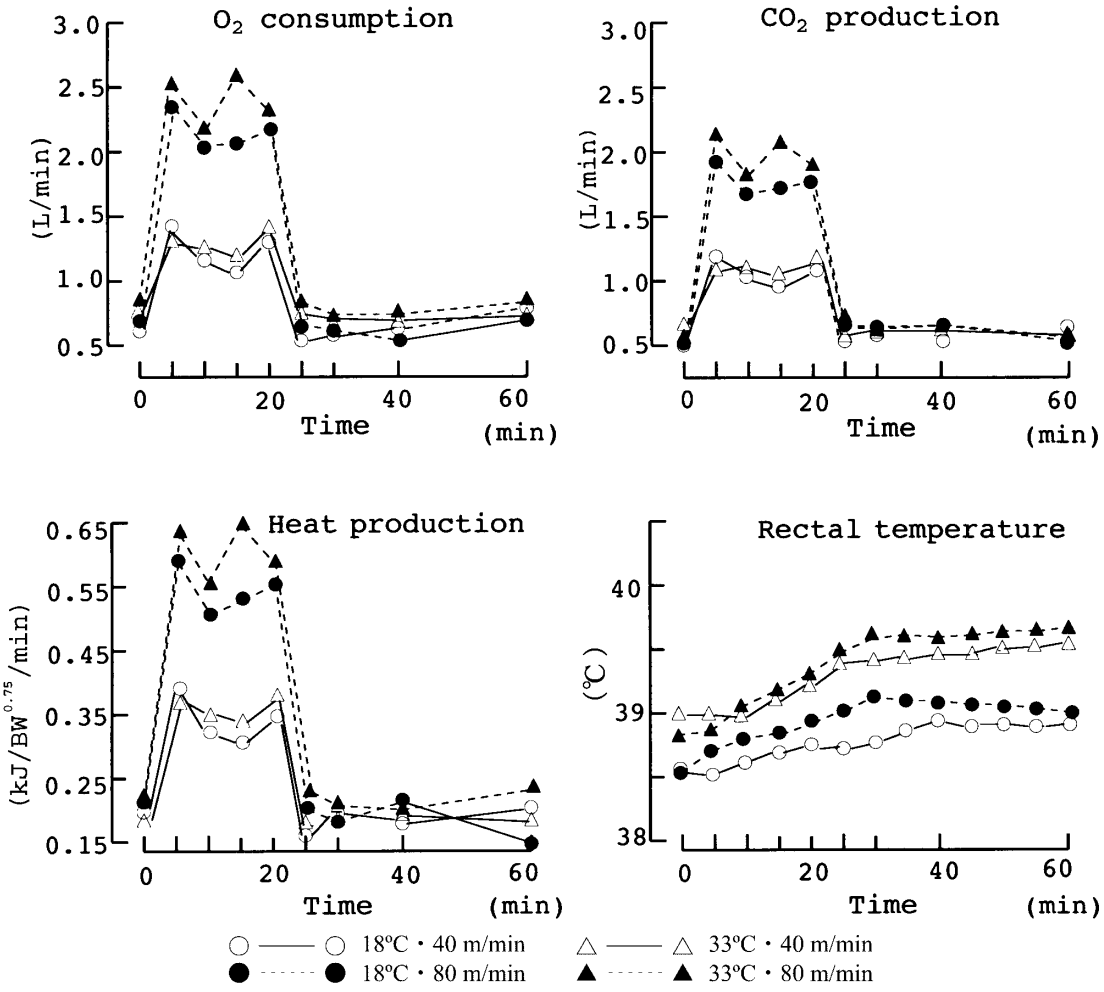

Fig. 7. Effects of environmental temperature and exercise load on metabolic responses

temperature at 33ºC and 18ºC were more pronounced during the recovery period.

Conclusion

Exercise and environmental heat load altered the cardiorespiratory and metabolic responses in steers. Cardiovascular function, including both heart rate and stroke volume, was accelerated by a walking load of 40 m/min, while it decreased by a walking load of 80 m/min. These results were probably associated with the reduction of the blood stream which returns to the heart due to the increase of the peripheral circulatory demand in motor organs. In a hot environment, the relative volume of the dead space ventilation in relation to the alveolar ventilation was significantly larger than that in a moderate environment. However, when the respiratory ventilation exceeded 60 L/min by the exercise load, the dead space ventilation reached a plateau level, while the alveolar ventilation abruptly increased. These reactions reflect the physiological limit of thermoregulation by heat dissipation from the upper respiratory tract. The decreased capability of heat dissipation from the upper respiratory tract is associated with a reduction of the dead space ventilation.

The results of this study demonstrated that exercise in a hot environment enhanced heat production, and that the peripheral circulation and respiratory activity fulfilled effectively their function to dissipate the internal body heat .

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