Effect of Experimental High environmental Temperature and Humidity on Steers

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Effect of Experimental High environmental Temperature and Humidity on Steers

Shinichi TERUI, Seishi ISHINO, Kazuo MATSUDA*, Yoshio SHOJI**, Kaichi AMBO** and Tsuneyuki TSUDA**

Tohoku Branch, National Institute of Animal Health, Aomori, Japan
** *Faculty of Agriculture, Tohoku University, Sendai, Japan*

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Summary

To elucidate the cause of death of cattle which was observed in the grazing field where sometimes a sudden rise of air temperature was recorded, three steers were exposed experimentally to severe heat stress untill death or nearly to death.

The settled ambient temperature and relative humdity in the artificial climate room were 42°C, 80 per cent, 36°, 80 per cent, and 33°C, 80 per cent, and No. 1, No. 2 and No. 3 steers were exposed respectively to above conditions.

No. 1 died in 4 hours, No. 2 died on the 4th day and No. 3 collapsed on the 7th day. Panting, severe elevation of rectal temperature, reduction of movement of digestive tracts, hypersalivation and dysfunction of skin reflex were noticed in No. 1 and No. 2.

A remarkable decrease of white blood cell and P_{CO_2} value were detected, and an increase of serum creatinine value, serum LDH and GOT activities were also noticed accordant with the elevation of rectal temperature. At necropsy, the cloudy discloration which looked like boiled meat appearance was characteristic in the skeletal muscle at the hind part of the bodies. Systemic severe congestion and hemorrhage, slight looseness of hepatic cell cords and degeneration of epithelial cells of the uriniferous tubules were observed. But these pathological changes were seen only in No. 1 and No. 2.

Judging from the clinical symptomes, hematological and pathological findings, the No. 1 and No. 2 were diagnosed as heatstroke and No. 3 was a collapse due to anorexia and heat stress.

The direct and indirect devastating effects of high temperature and high humidity upon the physical conditions of the steer were explained.

Many case reports and experimental data have been published about the physiological responses and ecological behaviours of cattle exposed to heat stress. However, there were only a few reports concerning the physiological and pathological changes or the relationships between environmental conditions and sensitiveness for microbial infections of cattle exposed to so severe thermal stress as to bring death.

* Present Adress: Azabu Veterinary College, Sagamihara, Japan.

We have previously reported that the outbreak of acute disorder in grazing cattle usually infected with Piroplasma (Theirelia) in early summer in the northern Tohoku district could possibly be due to the harmful effect of such a meteorological conditions as a sudden rise of air temperature in the grazing field. Our experience also revealed that cattle raised in a barn for about two and a half months from spring to summer often died from the sunstroke when exposed suddenly to the strong summer solar radiation for only one and a half days. Contrary to this, other cattle accustomed to grazing for about two and a half months from spring to summer showed only slight abnormality under a similar solar exposure for several days.

To analyse the physiological factors involved in the above mentioned observations, the measurement of sensitivity change to various etiologic agents under several environmental conditions will be the necessary experimental approach. As a preliminary experiment, therefore, healthy cattle were exposed to heat stress so as to bring resultant death in an artificial climate room. The clinical, hematological and pathological findings and results of expired gas analysis are described in this paper.

Materials and Methods

1. *Experimental Cattle*: Three Holstein-Friesian steers, weighing 201, 202 and 204 kg were employed for the present experiment. All of them were raised at the same pasture and proved to be healthy from clinical and hematological examinations.

2. *Experimental Condition*: Each of the experimental animals was named as No. 1, No. 2 and No. 3 and exposed to the following environmental condition in the artificial climate room; room temperature 42°C and humidity 80 per cent (No. 1), 36°C and 80 per cent (No. 2), and 33°C and 80 per cent (No. 3). The preliminary examinations were made on all three steers in this room for two or three days under the condition of room temperature 20°C and humidity 60 per cent.

3. *Experimental Methods*:

1) *Clinical Findings*: Vigor, appetite, salivation, rumination, peristole and peristalsis (stetoscopically), skin reflex, rectal temperature, heart rate, respiration rate, volume of feed intake and quantity of water intake were examined hourly in No. 1 and every six hours in No. 2 and No. 3.

2) *Hematological Findings*: Venous blood collected from the jugular vein was used for the examination. Each component of blood and serum were examined with the following methods.

Red blood cell (RBC) and White blood cell (WBC): Bürker-Türks method.

Hematocrit (Ht): Microcapillary method.

Hemoglobin (Hb): Cyanmethemoglobin method.

Hemogram: Giemsa stained blood film.

Blood sugar (BS): o-Aminobiphenyl method.
Total serum protein (TP): Atago's refractive protein meter.
Serum creatinine (Creat): Folin-Wu method.
Glutamic Oxaloacetic Transaminase (GOT): Reitman-Frankel method.
Lactic acid Dyhydrogenase (LDH): Cabaud Wroblwski method.
Alkaline Phosphatase (ALP): King-King method.
Calcium (Ca): o-Cresolphtaleincomplekinson method.
Inorganic Phospholus (IP): Fiske-Subbarow method.
Chloride (Cl): Schales-Schales method.
Magnesium (Mg): Atomic absorptive method.
Sodium (Na) and Pottasium (K): Flame photometoric method.
pH, Po_2 and Pco_2: Astrup method.

These examinations were carried out at the interval of three hours in No. 1, and six hours in No. 2 and No. 3 animals.

3) *Heat Production*: The expired gas was collected in a Douglas bag and was analysed with the gas-chromatograph method.

4) *Pathological Findings*: Necropsy was carried out as soon as possible after death caused by heat stress in No. 1 and No. 2, but was made immediately after the death by exsanguination in No. 3. Main organs and tissues were fixed in 10 per cent formalin. Histopathological specimens were prepared routinely.

Experimental Results

1. *Clinical Findings*

1) *No. 1 animal (Room temperature 42°C and humidity 80 per cent)*: Room temperature and humidity were elevated from nine o'clock on the experimental day. Room humidity reached 80 per cent after one hour, but temperature reached 42°C after five hours.

The animal died four hours after the exposure to 42°C and 80 per cent. The clinical symptoms were shown in Fig. 1. Rectal temperature rose as a straight line proportionally to the elevation of room temperature, and reached 45°C before death. The respiratory rate was accelerated with the elevation of room temperature, but suddenly decreased when rectal temperature reached a point of 43°C. Gastro-abdominal type respiration was accompanied by tachypnea and hyperpnea, but at the maximum peak of respiration rate, hyperpnea appeared accompanied with two steps respiration. No significant change was noticed in the heart rate except for irregularity. At the fatal stage, however, the heart rate increased very rapidly followed by a sharp decrease. In addition, an agonizing behaviour appeared with the elevation of room temperature, for instance, frequent bellowing, repeated swinging his head to every direction, panting and hypersalivation. Depression of vigor, decreased appetite and reduced ruminal and intestinal movements became evident, and then a small amount of diarrhea faeces, reduced skin reflex of the back

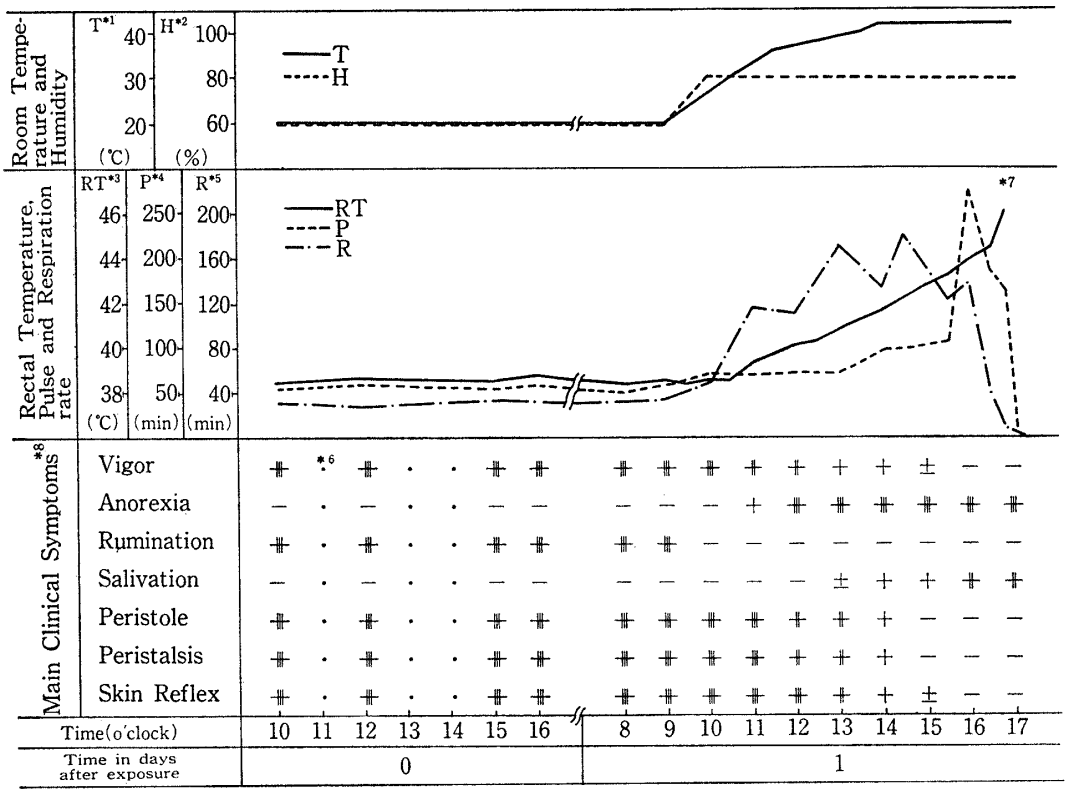

FIG. 1. Experimental condition and main clinical symptoms of No. 1 steer.
*1 Temperature, *2 Humidity, *3 Rectal temperature, *4 Pulse, *5 Respiration rate, *6 Not examined, *7 Dead, *8 Clinical symptoms, +: mild, ++: moderate, +++ severe, ±: equivocal, −: not observed

and hind quarters were noticed within several hours before death.

2) *No. 2 animal (Room temperature 36°C and humidity 80 per cent)*: At the 4th day after the exposure, the animal died by prolonged heat stress as shown in FIG. 2. The sequential changes in his rectal temperature, respiratory rate, heart rate and other clinical symptoms closely resembled those of No. 1. However, these findings were more mild than those of No. 1. Though the rectal temperature was maintained at 42°C through 2nd and 3rd days, it rose to 45°C on the 4th day. The reduced skin reflex was localized around the nates and the upper part of hind legs.

3) *No. 3 animal (Room temperature 33°C and humidity 80 per cent)*: Respiration rate increased suddenly with the elevation of environmental temperature as shown in FIG. 3. However, rectal temperature reached 42°C at the 3rd day after exposure to this condition. From the beginning of the exposure to the 3rd day, a very small elevation of rectal temperature and slow decrease of once increased respiration rate were noticed. Heart rate showed a slight increase both at the first and 6th day of exposure. After the 4th day, vigor, appetite and rumination diminished completely, and the movement of rumen and intestine could not be detected, and then diarrhea occured and skin reflex at the surface of the nates

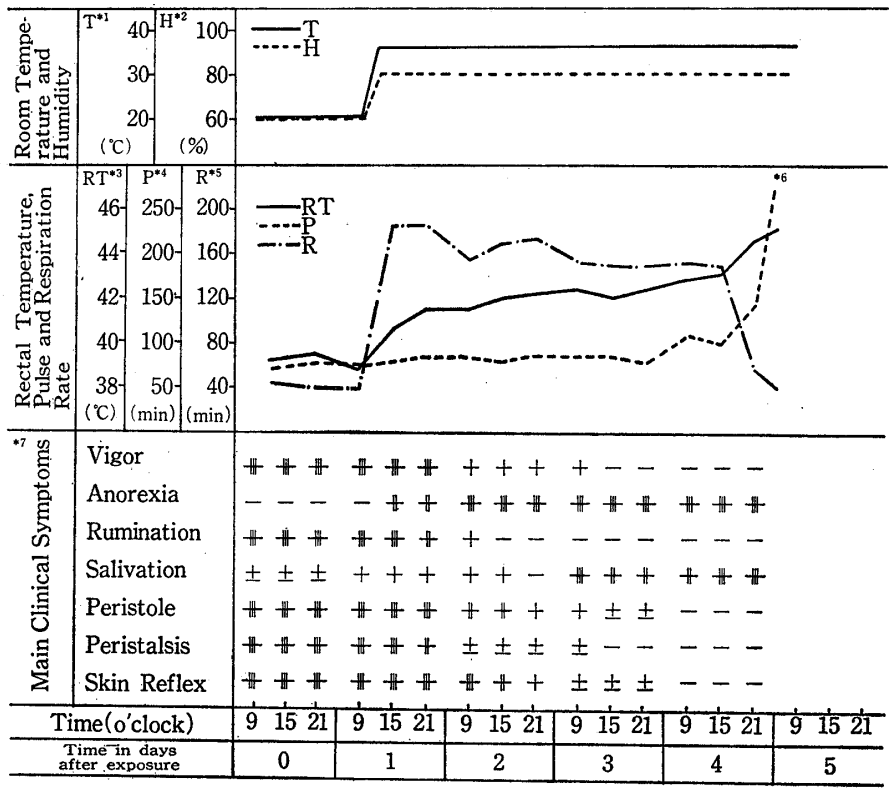

FIG. 2. Experimental condition and main clinical symptoms of No. 2 steer.
*1 Temperature, *2 Humidity, *3 Rectal temperature, *4 Pulse, *5 Respiration rate, *6 Dead, * Clinical symptoms, +: mild, ++: moderate, +++: severe, ±: equivocal, −: not observed

disappeared. At the 7th day, the animal was sacrificed as it was decided that he was at the fatal stage judging from the clinical symptoms.

2. *Hematological Findings* (FIGS. 4–7)

A slight increase of Ht value accordant with the time lapse after the exposure was detected in No. 1 and No. 2, however, no significant change was observed in No. 3.

A remarkable decrease of WBC count at the fatal stage was characteristic in every case. Percentage of neutrophils showed a slight gradual decrease accordant with the time lapse after the exposure.

The TP value increased transitorily in No. 1 and No. 2, but it decreased gradually in the fatal stage. The TP value of No. 3 decreased gradually with the time lapse after the exposure.

The BUN value showed a slight gradual decrease in No. 1 after the heat exposure. After the 3rd day the BUN value in No. 2 clearly increased. However, in No. 3 a slight increase of the BUN value was observed till 9 o'clock on the 5th day, but thereafter decreased gradually.

In each case, the increase of Creat value was observed after the exposure, and was evident especially in No. 2.

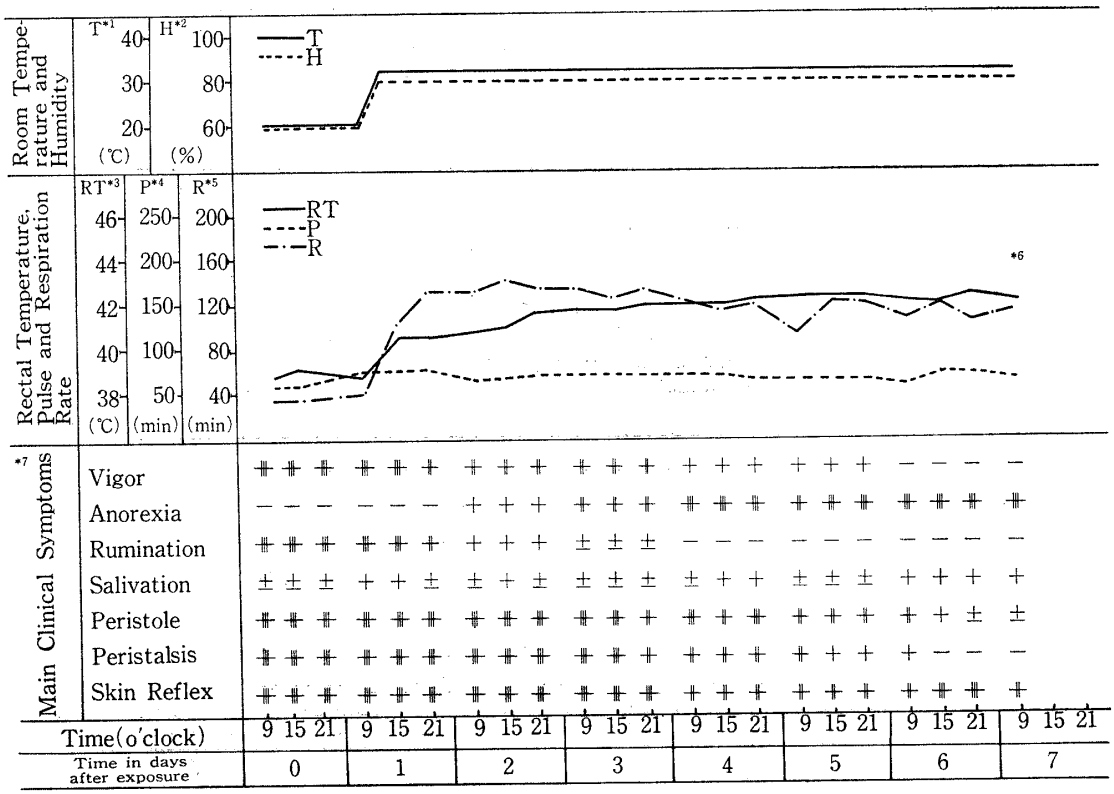

FIG. 3. Experimental condition and main clinical symptoms of No. 3 steer.
*1 Temperature, *2 Humidity, *3 Rectal temperature, *4 Pulse, *5 Respiration rate, *6 Scarificed, *7 Clinical symptomes, +: mild, ++: moderate, +++: severe, ±: equivocal, −: not observed

In BS value, no significant change was observed in No. 1, but in No. 2 and No. 3 a tendency of slight decrease was noticed after the exposure.

GOT activity showed a remarkable increase accordant with time lapse in No. 1 and No. 2, but a slight gradual decrease in No. 3. ALP activity increased in No. 1, but a gradual slight decrease was observed in No. 2 and No. 3. A remarkable increase of LDH activity was detected in No. 1, however, only a slight increase was noticed in No. 2 and No. 3.

The Ca value decreased gradually at a slight degree accordant with the time lapse in all cases. IP value showed gradual decrease after the exposure in No. 1, but in No. 2 and No. 3, there were no significant changes. The Na value showed a slight increase in No. 1, but slight gradual decrease in No. 2 and No. 3. The K value increased immediately after the exposure in No. 1, and on the 4th day in No. 2. However, a slight decrease of the K value was observed from the 5th day to the end of the experiment in No. 3. The Mg value did not exhibit any significant change in every case after the exposure. The Cl value showed a slight increase after the exposure in No. 1, and no change in No. 2, but in No. 3 decreased until the 2nd day, and thereafter increased.

The changes of pH, Pco_2 and Po_2 of jugular blood are shown in FIG. 7. A

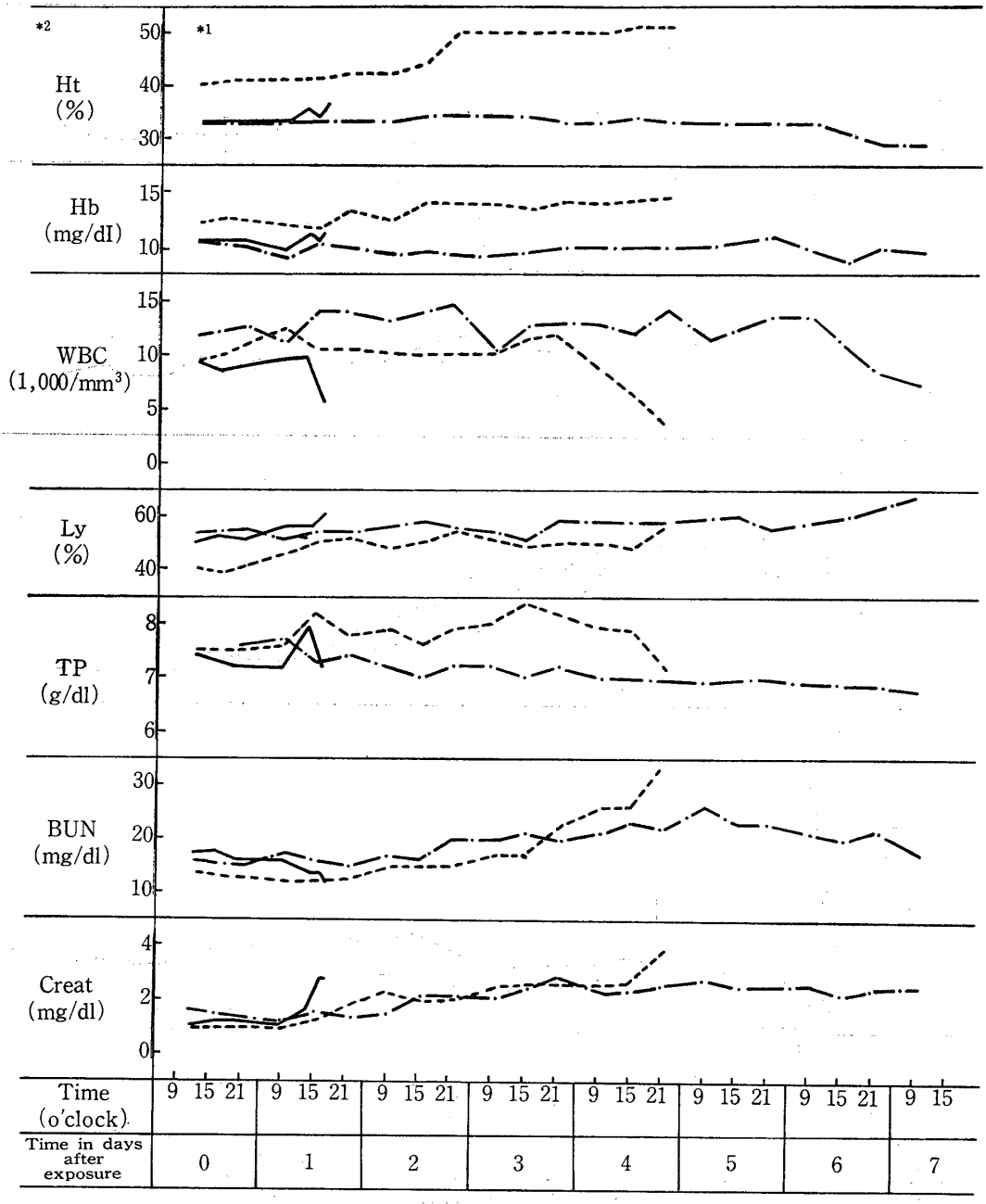

FIG. 4. Excursion in blood and serum components of experimental steers (1).
*1 ——: No. 1 steer, – – – –: No. 2 steer, –·–·–: No. 3 steer
*2 Ht: Hematocrit, Hb: Hemoglobin, WBC: White blood cell, Ly: Lymphocyte, TP: Total serum protein, BUN: Urea nitrogen, Creat: Creatinine

slight increase of the pH value occurred at the middle stage of the exposure in all cases. The Pco_2 value showed a remarkable decrease in every case accordant with the time lapse. The Po_2 value of No. 1 showed a remarkable increase at the fatal stage, however, a tendency of decrease was noticed accordant with the time lapse after the exposure in No. 2 and No. 3.

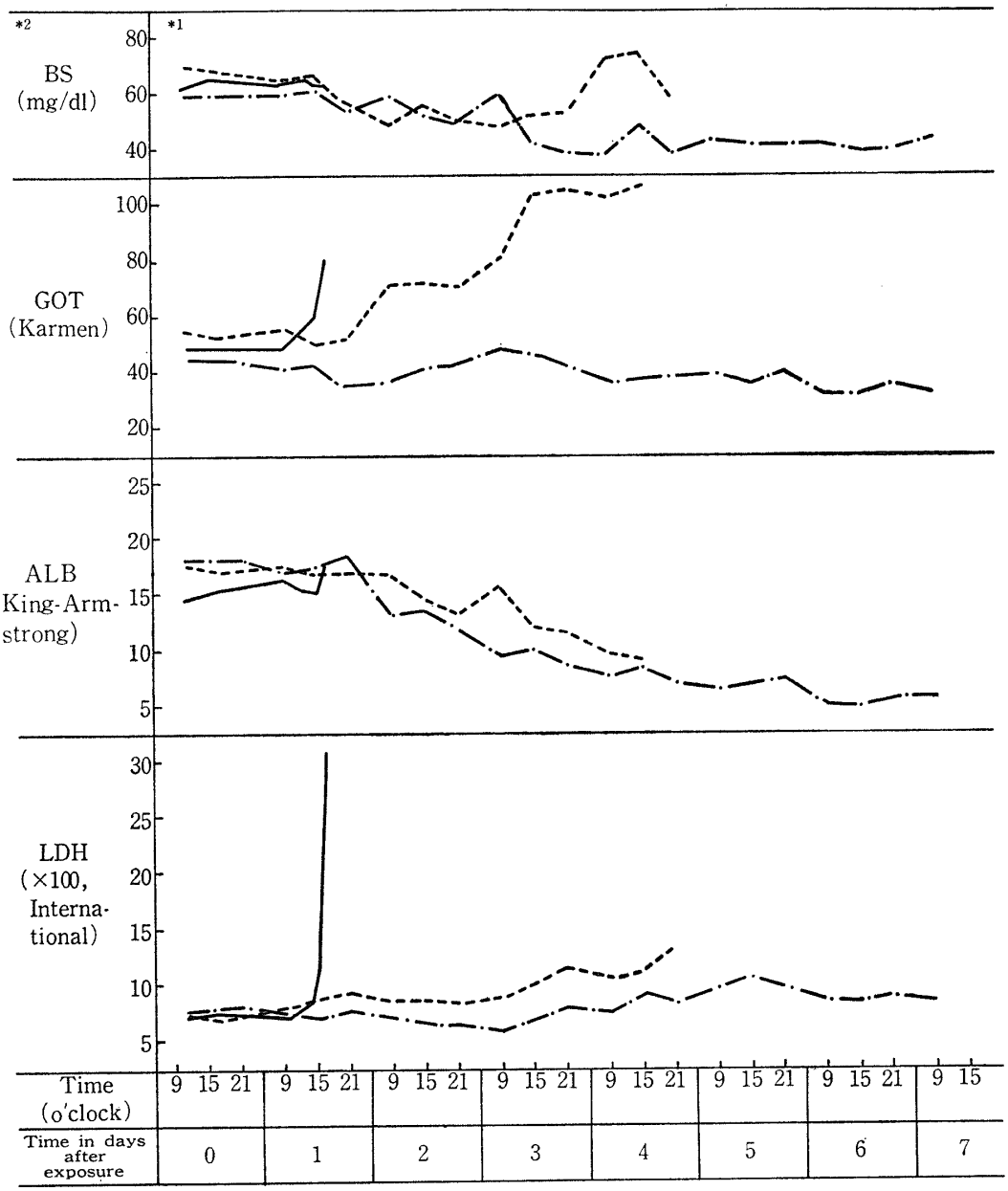

FIG. 5. Excursion in blood and serum components of experimental steers (2).
*1 ——: No. 1 steer, – – – –: No. 2 steer, –·–·–: No. 3 steer
*2 BS: Blood sugar, GOT: Glutamic oxaloacetic transaminase, ALP: Alkaline phosphatase, LDH: Lactic acid dehydrogenase

3. *Expired Gas* (FIG. 8)

The expired gas volume was proportional to their respiration rate in every case. In No. 1 and No. 2, remarkable increase of gas accordant with a remarkable increase of respiration rate after exposure was followed by a sudden decreases at the fatal stage. In No. 2, however, two peaks, one at the first day and the other at the 3rd–4th day, of expired gas volume were observed. In No. 3, the peak of gas volume was detected at the 3rd-4th day. Heat production showed similar changes

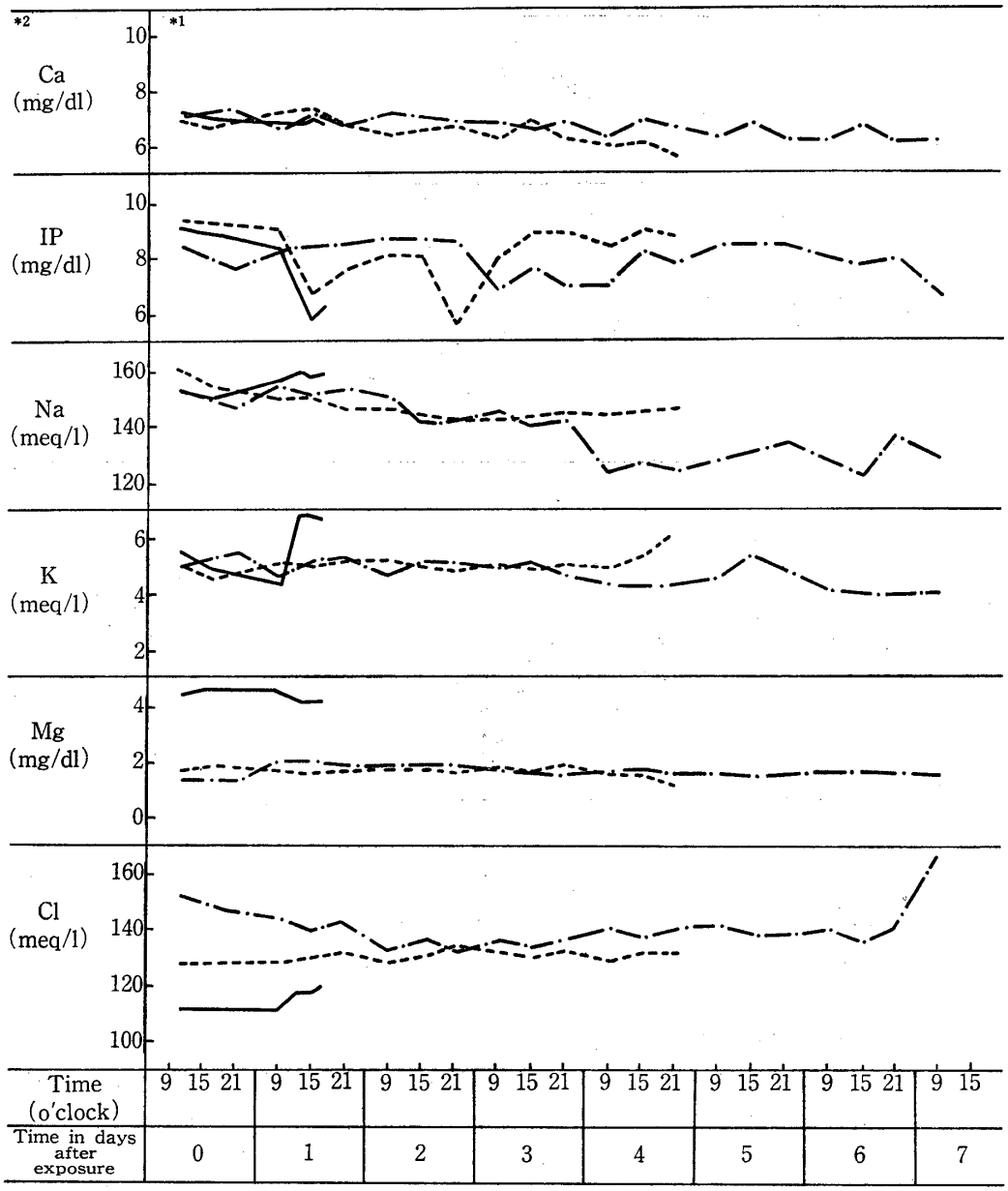

FIG. 6. Excursion in blood and serum components of experimental steers (3).
*1 ——: No. 1 steer, – – – –: No. 2 steer, –·–·–: No. 3 steer
*2 Ca: Calcium, IP: Inorganic Phosphorus, Na: Sodium, K: Pottasium, Mg: Magnesium, Cl: Chloride

to those of respiratory minute volume. No significant change of RQ was observed in No. 1 and No. 2, but gradual decrease was noticed only in No. 3.

4. *Pathological Findings*

1) *Macroscopic Findings*: Both No. 1 and No. 2 which died from heat stress showed the brightless of hair coats, decreased body weight and congested conjunctival, buccal mucosa and vaginal mucous menbranes in general. Especially the cloudy discoloration which looked like boiled meat appearence was character-

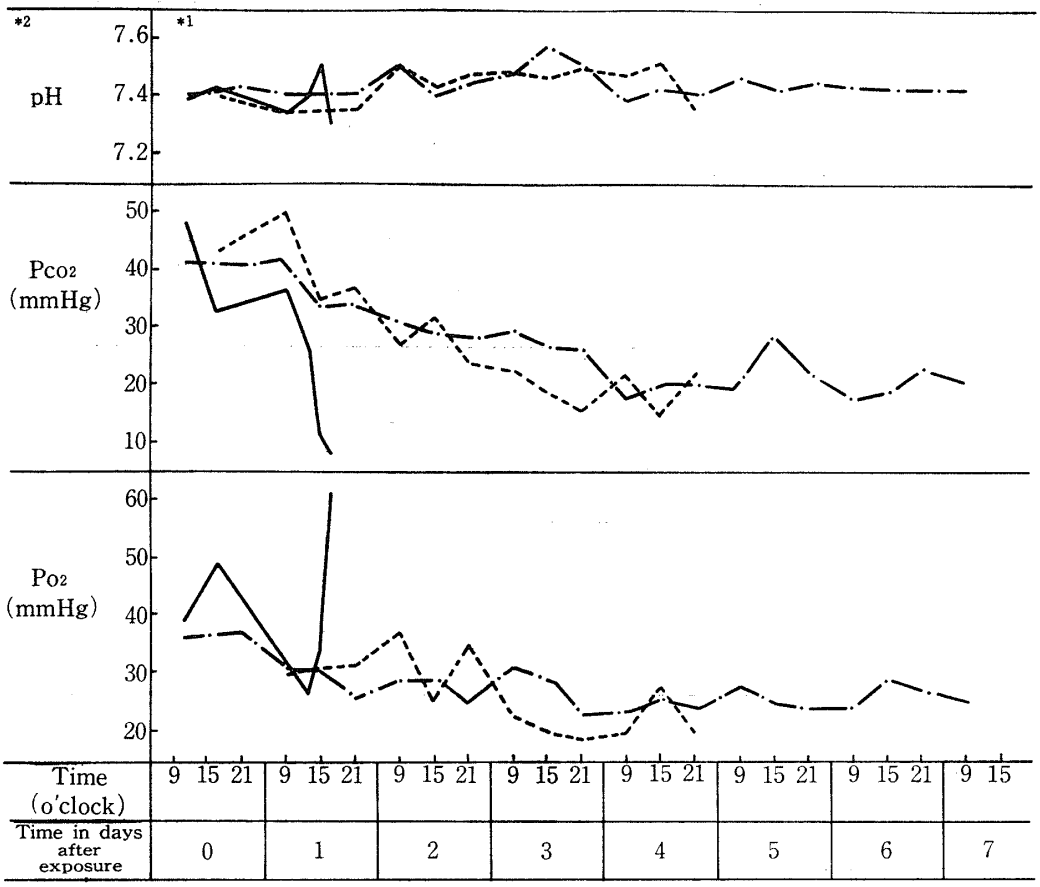

FIG. 7. Excursion in blood and serum components of experimental steers (4)
*1 ——: No. 1 steer, – – – –: No. 2 steer, –·–·–: No. 3 steer
*2 P_{CO_2}: Pressure of CO_2 in blood, P_{O_2}: Pressure of O_2 in blood

istic in the skeletal muscle at the hind part of the bodies. The dehydration of the connective tissues, the involution of the fatty tissues and congestion of the small blood vessels were found in the subcutoneous tissues. In addition, slight hemorrhaging of the pericardium and congestion of such organs and tissue as the lung, spleen, kidney, small intestine, urinary bladder, thymus, adrenal gland and brain were noticed. The surface of liver showed extreme turbid colour. The above mentioned findings were not so much evident in No. 2 as in No. 1. Noticeable pathological changes were not observed in No. 3.

2) *Microscopic Findings*: Added to the macroscopical cloudy discoloration of the skeletal muscle, muscular layers of the tounge and oesophagus showed the looseness with swollen and undulant muscle fibers. On the other hand, the pattern of cross striations in each muscle fiber were still observed. The capillaries in the interstitial tissues of the myocardium and subendocardium were congested with hemorrhages. The adrenal gland and thymus also showed the same findings. The lung had filtration of serous fluid owing to severe congestion. The slight looseness of hepatic cell cords and the degeneration of epithelial cells of the

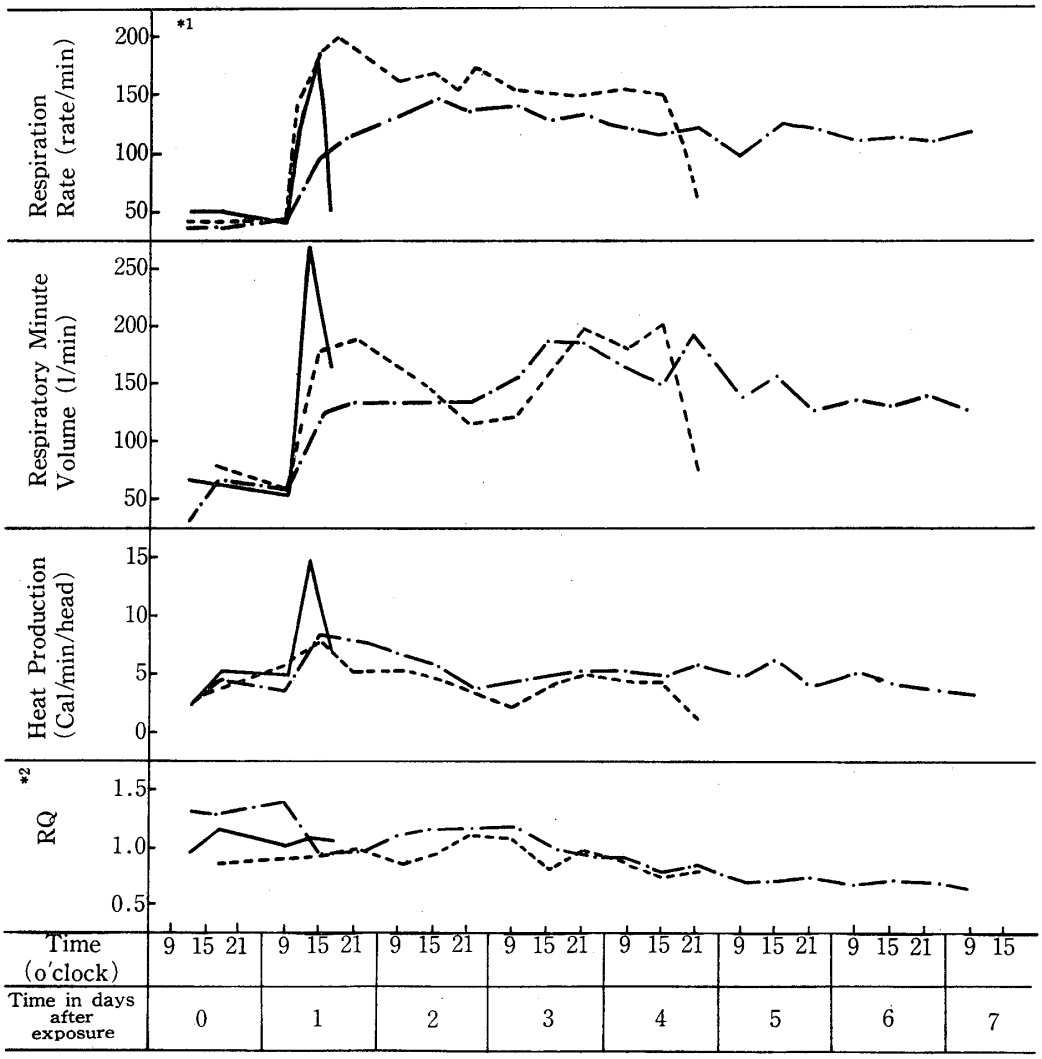

FIG. 8. Excursion in respiratory function of experimental steers
*1 ——: No. 1 steer, – – – –: No. 2 steer, –·–·–: No. 3 steer
*2 RQ: Respiratory quotient

uriniferous tubules were observed. These microscopic changes were seen only in No. 1 and No. 2.

Discussion

Concerning the effect of environmental temperature and humidity on cattle, many experiments were carried out in an artificial climate room by Blincoe and Brody (1), Brody *et al.* (2), Bearkley and Findley (3, 4, 5), Johnson *et al.* (6, 7). Kibler and Brody (8) and Ishii (9).

There were few detailed case reports concerning cattle exposed to severe thermal stress as to bring death.

Clinical symptoms observed in No. 1 and No. 2 steer resembled those in cattle

which had died from sunstroke in our field experiment (10), those cases were characterized by the increased respiration rate, and elevated rectal temperature at the fatal stage. Those findings were the same as those observed by the other authors.

It was speculated that the remarkable increase of heart rate noticed at the fatal stage in No. 1 and No. 2 would be due to cardiac weakness caused by heat stress. In No. 3, in spite of the increased respiration rate and elevated rectal temperature, heart rate remained normal. Therefore, the correlation between the level of heat stress and heart rate change was not obtained in the present experiment.

Hypersalivation was reported by Bearkley and Findley (4) in cattle exposed to heat stress. Ishii (9) noticed hypersalivation accordant with elevated rectal temperature in Holstein cows exposed to strong solar radiation, and even in those tethered under a shelter tree in summer. He suggested that the hypersalivation indicated a functional activation of the salivary gland to reduce the body temperature by the evaporation of saliva. But in his report whether a true increase in quantity of saliva or disturbance of saliva swallowing due to rapid or open mouth breathing was not differentiated.

A reductions of ruminal and intestinal movement were noticed in all cases in this experiment, but there are few reports about such symptomes except our previous report (10–12). Also a small amount of diarrheal faeces was noticed in each case just before the fatal stage, as cited also by Krum and Osborne (13), and Shapiro *et al.* (14). However, as this symptom contradicted the reduced ruminal and intestinal movements, it would have to be an involuntary faecal outlet due to the relaxant muscle sphincter internus and externus. This reduction of digestive tract and relaxation of muscle would be due to the abnormality of the nerve system which incur simultaneously reduced ruminal and intestinal movements and skin reflex.

In this experiment, a reduced skin reflex was noticed in No. 1 and No. 2 at the fatal stage, but whether this symptom was due to the paralysis of the sensory nerve system or that of the motor nerve system could not be confirmed. But this symptom correlated closely with the degeneration of the skeletal muscle in the hind part of the body.

The blood components of cattle kept at the temperature of 15–40°C and of 10–38°C were examined by Blincoe and Brody (1), and Brody *et al.* (2) respectively and no significant change in RBC and WBC count were found.

In this experiment, however, a remarkable decrease of WBC count was characteristic in every case. Although the WBC count is one of the most changeable components in the blood, the remarkable decrease of WBC count observed at the fatal stage suggested an abnormal *in vivo* distribution of WBC.

It is well known that the BUN value increases in renal disorder, dehydration, cardiac disorder, hyperthermia, pancreatic disorder, severe hemorrhaging and dysfunction of the thyroid gland. The remarkable increase of the BUN value in

No. 2 and No. 3 suggested a protein catabolism increase due to the heat stress. On the contrary, a decreased BUN value in No. 3 at the fatal stage would be due to a dysfunction of urea synthesis in liver.

An increased Creat value observed in this experiment accorded with those of the results of Blincoe and Brody (1) and Brody *et al.* (2). Blincoe and Brody (1) suggested that the rise of the blood Creat value was due to the activated endogenous nitrogen catabolism with a decline of feed consumption reinfroced by heat stress. However, the increased serum Creat value noticed in No. 1 and No. 2 may be due to the cell degeneration in the skeletal muscle.

The fact described by Brody *et al.* (2) that the decreased BS value accompanied the rise of room temperature was in accord with the present results of No. 2 and No. 3. It would be due to the decline of feed consumption and liver dysfunction.

Serum enzyme activity was examined in relation to heatstroke of men by Kew *et al.* (15) and Shibolet *et al.* (16), and also to heatstroke and sunstroke of dogs by Shapiro et al. (14) and Krum and Osborne (13).

A remarkable increased serum GOT activity noticed in No. 1 and No. 2 were in accord with the findings by Kew *et al.* (15), Shibolet *et al.* (16) and by Shapiro *et al.* (14). It is well known that serum GOT activity increases in cases of liver and kidney damage, myocardia infarction, damage of skeletal muscle and malignant tumors. However, the pathological observations suggested that the rise of GOT activity in our experiment would be due to the damage of the skeletal muscle. Kew *et al.* (15) and Shibolet *et al.* (16) reported that a remarkable increase of LDH activity was due to the damage of kidney and myocardium in the patients affected with sunstroke and heatstroke. However, judging from the pathological indications of only slight damage of kidney and myocardium led to the assumption that the increase of LDH activity in this experiment was due to the damage of the skeletal muscle.

Blincoe and Brody (1) and Brody *et al.* (2) reported about Ca, IP, Na and Mg values in the serum of cattle exposed to high ambient temperature. Krum and Osborne (13) also reported about the Na and K values in dogs affected with heatstroke. Our previous reports (11, 12) detailed about Ca, IP, Cl and Mg values in cattle exposed to heat stress.

In this experiment, the Ca value showed only a slight gradual decrease, similar to the previous reports (11, 12). However, Blincoe and Brody (1) did not detected any alteration. The Ca value decreases when the renal and parathyroid glands dysfunction and the absorptive dysfunction in the digestive tract occurs. As the findings in this experiment did not agree with the above statements, the meanings of decreased serum Ca value remained obscure. Brody *et al.* (2) reported that the IP value showed a slight increase when body temperature rose, but in our previous (12) and the present experiments, IP value had a tendency to decrease. We could not discuss about the meaning of this finding. The decreased Na value

reported by Blincoe and Brody (1) resembled the results in No. 2 and No. 3. The blood Na level increases in dehydration and renal dysfunction. The increase of the Na level noticed in No. 1 would be due to the loss of body fluid, because of the hypersalivation, hypersweating and increased vaporization by panting.

The pH value of the jugular blood rose gradually after the exposure to heat stress in each case, but decreased suddenly at the fatal stage in No. 1 and No. 2. The gradual decrease was observed after the 3rd day in No. 3. These results resembled the findings of Dale and Brody (17) and Blinca (18). The gradual decrease of Pco_2 value observed in each case resembled the findings by Blincoe and Brody (1) and Sato (19). They suggested that the decreased Pco_2 value was due to the hyperexcretion of CO_2 by panting. It was considered that the decreased Pco_2 value would be due to the remarkable increased respiration rate and expired gas volume in this experiment. The rise of pH value noticed in this experiment suggested respiratory alkalosis, judging from the above mentioned findings.

Fatty degeneration or necrosis in the liver, and cytoplasmic degeneration and necrosis in uriniferous tubules, myodegeneration or necrosis in skeletal muscle, hemorrhage and degeneration of nerve cell in brain were reported as common findings in human heatstroke and sunstroke (8, 16, 20–22). In addition, pathological findings of heat stress in dogs (13, 14) and monkeys (23) were characterized by cytoplasmic degeneration or necrosis in uriniferous tubules. However, degeneration and damage, except for skeletal muscle noticed microscopically in this experiment, were more mild than those of previous reports. These differences were probably due to the fact that in this experiment animals died or were slaughterd within a shorter period after the heat exposure than in other experiments.

Judging from the clinical, hematological, and pathological findings, the former two cases were diagnosed as heatstroke and the last case a collapse due to hyperanorexia and heat stress.

Typical responses in cattle exposed to heat stress could not be generalized from the above mentioned findings, because the findings described were obtained from only one steer in each experiment. But the devastating direct and indirect effect of high temperature and humidity upon the physical condition of steer were explained.

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