

THERMOREGULATION IN HENS EXPOSED TO MEASURED CHANGES OF TEMPERATURE

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Among the warm-blooded animals, birds possess the highest body temperature and the highest level of energy metabolism. A relatively stable homoiothermia is observed only in large birds [2], while in small birds the variations of body temperature may reach 10–12°, despite their well-developed chemical thermoregulation [3, 8]. Of the mechanisms of physical thermoregulation, the absence of perspiration is indicated by the well-marked polypnea [6]. Investigators have expressed highly contradictory views on whether or not birds exhibit vasomotor reactions and respond to cold by shivering [4–7].

The object of the present investigation was to study thermoregulatory reactions in hens.

EXPERIMENTAL METHOD

Experiments were carried out on 8 Leghorn hens. Changes in body temperature (in the rectum) and skin temperature (comb, dorsum beneath the left wing, and leg, in the hollow of the plantar surface). The functional capacity of the chemical and physical thermoregulatory mechanisms was evaluated from the changes in body temperature during standard heating and cooling (exposure of 1 h to a temperature of 36–37° or 2–5°). In some experiments the respiration rate was counted and the electromyograph (EMG) of the muscles in the lateral region of the thigh was recorded. Respiration was recorded by means of a rubber cuff connected by pneumatic transmission to a piezoelectric crystal. A 4-channel EEG-4 electroencephalograph with an ink-writing mechanism was used for the recordings. The muscle potentials recorded with needle electrodes (distance between electrodes 1–1.5 cm) were fed into the input of a type UBN amplifier and recorded on photographic film by a type MPO-2 oscillograph. The temperature and the oxygen consumption were determined periodically over a period of 1–2 h during adaptation to the laboratory surroundings and then for several hours in the course of the experiment at intervals of 1 h between determinations. The respiration and EMG were recorded at intervals of 5–10 min.

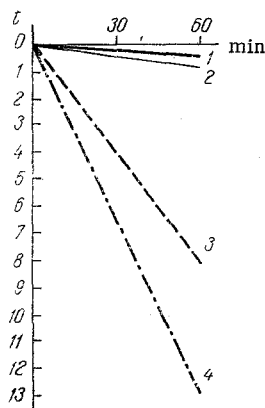


Fig. 1. Changes in body temperature of hens in various parts during cooling. 1) Rectal temperature; 2) skin temperature beneath the wing; 3) comb; 4) leg.

EXPERIMENTAL RESULTS AND DISCUSSION

During exposure to heat (11 experiments), heat dyspnea developed stepwise between the 4th and 10th minutes, and it was subsequently observed with short breaks throughout the period of exposure in the chamber. The body temperature rose from the original level ($41.5 \pm 0.1^{\circ}$) by 1.1° ($42.6 \pm 0.4^{\circ}$) after 30 min and by 1.5° ($43 \pm 0.3^{\circ}$) after 1 h. The skin temperature beneath the wing rose by the same degree (Fig. 1). The body temperature at the moment dyspnea began was 0.5° higher than its original level ($P = 0.01$). The reaction of polypnea was thus less perfect in the hens than, for example, in carnivores. At the same time, heat dyspnea in hens is one of the principal mechanisms for actively raising the heat loss, for the vasomotor regulatory reaction during overheating was hardly brought into operation. The axial gradient —

*Standard error of the mean.

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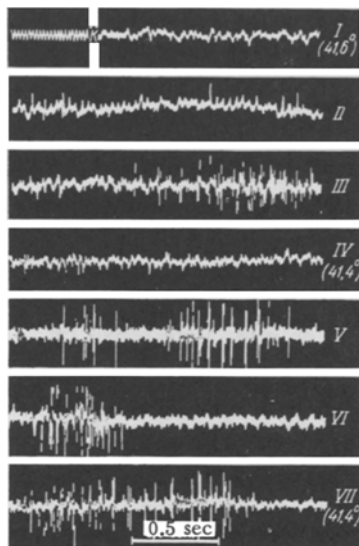


Fig. 2. Changes in bioelectrical activity of the thigh muscles of a hen exposed to the weak action of cold. K) Calibration 50 V; I) initial EMG; II) 10 min; III) 15 min; IV) 30 min; V) 40 min; VI) 50 min; VII) 60 min after beginning of exposure in a refrigerator. Body temperature in parentheses.

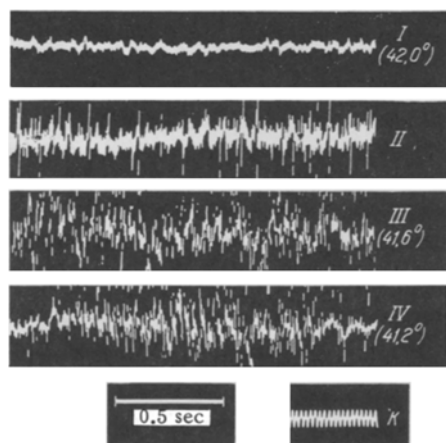


Fig. 3. Changes in bioelectrical activity of the thigh muscles of a hen during severe exposure to cold. I) Initial EMG; II) 10 min; III) 30 min; IV) 45 min after beginning of exposure in a refrigerator. Rest of legend as in Fig. 2.

thermoregulatory muscle tone (to $100 \mu\text{V}$). However, throughout the period of exposure to cold (except the first 10–15 min) the intervals between shivering attacks were longer than the intervals of increased thermoregulatory muscle tone.

The efficiency of chemical thermoregulation in the hens was probably due to the small relative body surface and the high insulating properties of the feathers. In addition, during cooling peripheral vasoconstriction played a definite role. At the end of exposure to cold a marked fall was observed in the temperature of the legs – by 13° ($P < 0.01$) and the comb – by 8° ($P < 0.01$), and this naturally led to a reduction of

the difference between the body temperature and skin temperature – was stable (on the average 1.8° before and 1.7° after heating). For animals with a well-developed vasomotor thermoregulatory mechanism, disappearance of the axial gradient during exposure to heat is a characteristic feature [1]. In hens a vasomotor reaction to exposure to heat was observed only with the blood vessels of the comb. The comb temperature rose by 1.8° ($37.7 \pm 0.4^\circ$) after 30 min, and by 2.6° ($38.5 \pm 0.8^\circ$) after 1 h from its initial level ($35.9 \pm 0.1^\circ$). The axial gradient relative to the skin of the comb fell slightly (5.6° before and 4.5° after exposure to heat).

Hence, vascular reactions to overheating appeared over a limited area and were of low intensity. As a result, the body temperature of the hens rose significantly during exposure to moderate external heat, despite the presence of a polypnea reaction.

During controlled exposure to cold (22 experiments) the body temperature was practically unchanged: after 30 min it fell to $41.1 \pm 0.2^\circ$ and after 1 h to $41.0 \pm 0.2^\circ$. Lengthening the exposure in the chamber to 2 h did not affect the result. The resistance to cold was due mainly to highly active chemical thermoregulatory reactions. The oxygen consumption of hens may exceed its initial level by 2.5 times as the temperature falls below the thermoneutral point (26°) [8].

The results of investigation of the electromyogram showed that the muscles are undoubtedly a powerful source of increased heat formation during cooling. From 10 to 15 min after the beginning of exposure to cold, volleys of intensified spike activity ($100\text{--}300 \mu\text{V}$, 40–60 cps, 0.5–1.5 sec) began to appear on the EMG, superimposed against the background of weaker potentials ($25\text{--}50 \mu\text{V}$), close in amplitude to the original background (Fig. 2). The paroxysmal character of the increased activity is typical of shivering. With an increase in the intensity of the cold (moistening the feathers with water, application of ice bags) the attacks of shivering increased (Fig. 3). In these cases, the body temperature fell by $1\text{--}3^\circ$. The thermoregulatory reaction was brought into operation before the body temperature fell. The shivering could be detected by palpation, and at the end of the exposure, visually. The appearance of shivering was preceded by a marked increase in

the heat loss. The development of vasoconstriction in an area covered by feathers was not significant. The decrease of skin temperature beneath the wing by 0.8° ($P > 0.05$) suggested merely a tendency for the reaction to appear. Consequently, during the action of cold, the vasomotor reaction was clearly brought into operation in the extreme parts of the body, the efficiency of the chemical thermoregulatory mechanism was increased, and this helped to maintain the normal level of the body temperature during exposure to moderate degrees of cold.

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