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Are there any Differences in the Task of Cold Defense Mechanisms Between Hypothalamic and Extra-hypothalamic Centers in Body Temperature Regulation?*

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Abstract: To determine the responsibility of lower pons as a extrahypothalamic thermoregulatory center, midpontine decerebration was made on unanesthetized rabbits continuously measuring rectal and skin temperature and oxygen consumption. Cold air stress induced remarkable drop of ear temperature in midpontine rabbits, while less drop was observed in normal rabbits. Oxygen consumption during skin cooling increased up to 37% in normal rabbits and only 16% in midpontine rabbits. Rectal temperature raised by 2.0−2.5°C in both groups, which is interpreted as an excess of heat gain. The difference in origins of excessing heat gain between midpontine and normal rabbits are further discussed in the text.

Key words: Midpontine decerebration, Body temperature regulation, Extrahypothalamic center

As previously reported, cold defenses especially peripheral vasoconstriction and heat production were not remarkably affected by a midpontine decerebration (5). The degree of thermoregulatory responses of midpontine rabbits induced by spinal cooling were only slight, so that it was assumed that thermocentres located lower to midpontine area have a considerable capability to regulate body temperature. Many reports on the extrahypothalamic thermoreception also support this inference (4), (8).

However, previous studies were made on a relatively short afferent pathway of

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thermoreception, i. e. from lumbar spinal cord to lower pons. In the present study, cold stimulation to total skin during thermal transient (air temperature) was made to midpontine rabbits whereby rectal temperature (Tre), ear temperature (Tea), and oxygen consumption (Vo_2) were continuously measured.

Twenty rabbits were decerebrated at midpontine, pretrigeminal level. Oxygen consumption measurement and temperature monitoring were as described elsewhere (5).

Fig. 1 and Fig. 2 show typical responses to low ambient temperature. In midpontine rabbits, the mean value of Vo_2 caliculated from 30 minutes measurement before air cooling was 9 ml/kg.•min, and that of Vo_2 during cold stimulation (averaged from one hour measurement) was 10.5 ml/kg.•min. Thus Vo_2 ratio (air cooling/before cooling) was found to be 1.16. A drop of Tea was 8°C against 32°C decrease in ambient temperature, so, drop ratio is 0.25. In normal rabbits, the Vo_2 ratio was 1.37 and drop ratio of Tea was 0.91.

During the experiment, the rise of Tre in midpontine rabbits was similar to that of normal rabbits. It is assumed that in both groups, heat balance response was towards

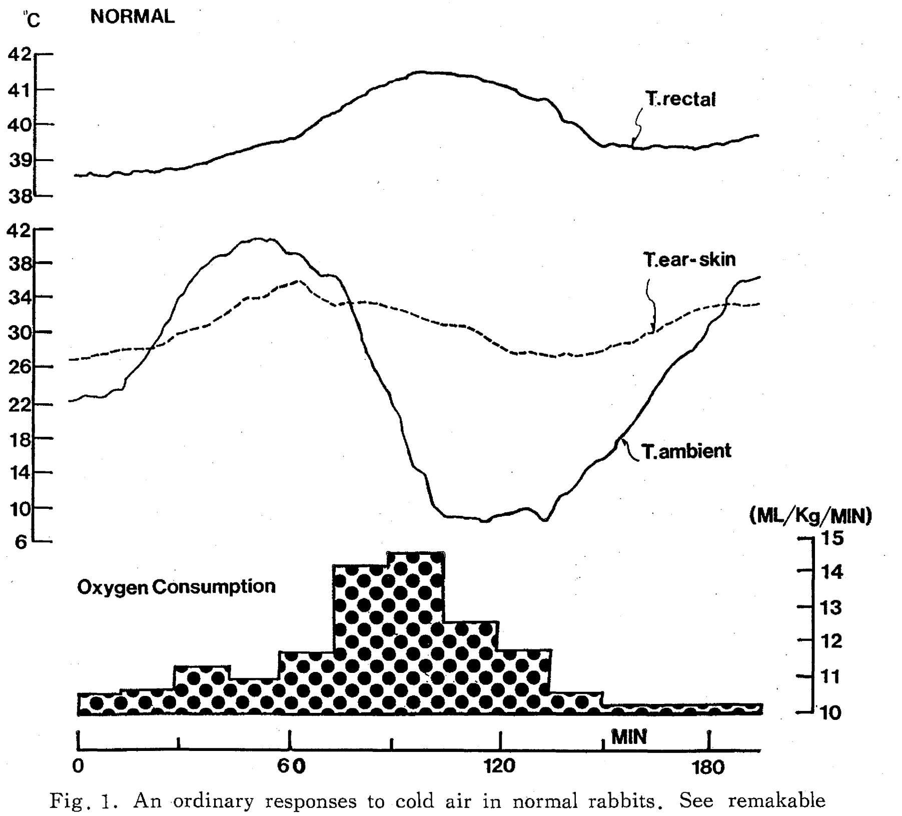

Fig. 1. An ordinary responses to cold air in normal rabbits. See remakable increase of oxygen consumption with a less fall of ear temperature accompanied by raise of rectal temperature.

positive balance during the experiment. Nevertheless, the mechanism gaining that positive balance seems different. A rise of Tre in normal rabbits was achieved mainly by heat production (viz., increased Vo_2); on the other hand, Tre increase in midpontine rabbits was accompanied by extreme lowering of Tea coupled with small increase of Vo_2 (Table 1).

Compairing these data with the results reported by Kosaka et al. (5), it is obvious that the Tea drop elicited by skin cooling and spinal cooling were different (see table 2). Tea is determined by blood flow and vasomotor tone of the ear which is regulated by sympathetic activities (2). Moreover, the sympathetic vasomotor activity is controlled by thermal inputs from core and skin (6), (7), (8), (9), and also from hypothalamus(3).

To achieve a drop in Tea, the vasomotor center in lower pons medulla oblongata should receive effective thermal inputs from receptive areas. In the case of midpontine decerebration, there are no interruption to convey these thermal inputs upwards from lumbar spinal cord to lower pons. Nevertheless, a drop of Tea was greater in skin-cooled groups than in spinal-cooled groups.

These results lead us to presume that;

1. Thermal signals arisen at skin, terminated into lower pontine area through spinal cord and trigeminal nerves, have more pivotal role for heat gain responses.
2. There are some inhibitory pathways from upper pons to lower pons which suppress the vasomotor responses. (see Fig. 3)

According to recent study of Amini-Sereshki et al., (1) there is the tonic inhibitory system from midbrain and upper pons to lower pons in rats. Therefore, it might be

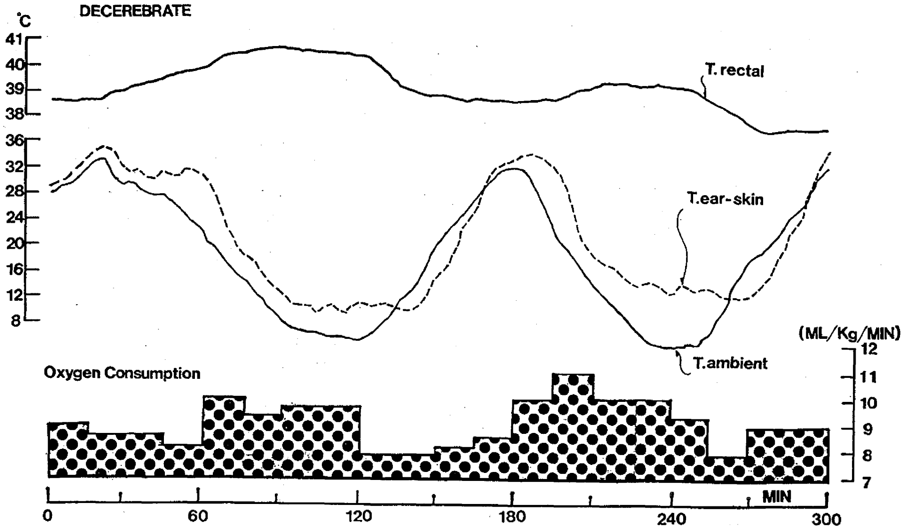

Fig. 2 A typical thermoresponses to transient ambient temperature in midpontine-decerebrated rabbit. Rectal temperature is raised in cool air. Note the drop of ear temperature in cooling, compared to normal rabbits.

Table 1.

COLD DEFENSES AGAINST SKIN COOLING	NORMAL	MIDPONTINE
Tre	↑	↑
Tea (vasoconstriction)	↓ (+)	↓↓ (++)
V_{O_2}	↑↑	↑

Table 2.

COLD DEFENSES AGAINST SPINAL COOLING	NORMAL	MIDPONTINE
Tre	→	→
Tea	↓↓	↓～→
V_{O_2}	↑↑	↑↑

Summarized from data of Kosaka *et al.*[5]

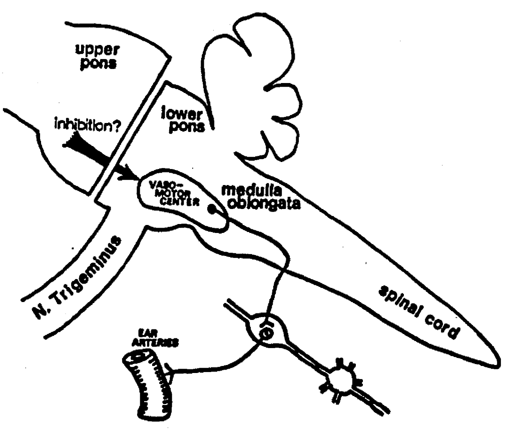

Fig. 3 A possible inhibitory pathway at pontine area for vasomotor centre

concluded that the thermoregulatory center of lower pons has major role for heat saving responses by means of great capability of vasoconstriction of ear in rabbits; however, the possibility of former hypothesis is not yet to be discarded.

An increase in V_{O_2} of both groups under the low ambient temperature, and likewise, a decrease under high ambient temperature were observed. It was shown that, in spinal cooling, there was no statistical differences of V_{O_2} between normal and decerebrated rabbits (5). However, midpontine rabbits in this study showed smaller changes of V_{O_2} in response to air cooling in contrast to the response obtained in spinal cooling. Therefore we assumed that thermal inputs from spinal cord to lower pons would elicit nearly enough effector mechanism for metabolic heat production and signals arisen at skin, passing through spinal cord, would distribute a fairly part of them to lower pons. In addition, there might be a fascilitatory pathway for heat production from upper center to lower pons.

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ウサギ橋下部の体温調節中枢としての意義

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橋中央部除脳ウサギ（三叉神経起始部より頭側で切断）について，無麻酔下に環境温の変化による冷刺激を与え，直腸温，耳介皮膚温・酸素消費量の判定を行った．除脳ウサギの耳介皮膚温低下は正常群に比して大であり，前回報告した脊髄冷却時の反応とは逆の結果が得られた．酸素消費量の増加は除脳ウサギで変化が少なく正常群の約半分であった．このことも前回の結果とは異っており，即ち冷刺激が脊髄に与えられた時の酸素消費量の増加は除脳群・正常群の間で有意の差がなかった．除脳による上位中枢からの入力の消失によって生じたこれらの変化を説明するためには，いくつかの推論が考えられる．

最初に耳介皮膚温の低下については

1)　脊髄からの冷入力により惹起される反応よりも皮膚からの入力による反応の方が橋下部においては優勢である．

2)　上位中枢から橋下部に対しては体温調節反応の抑制性の入力があるため，除脳によりこれらの反応が亢進する．

の2つの仮定がなされうる．

次に酸素消費量の増加については，脊髄からの入力のみでも，ほぼ充分な効果器の作用発現が認められるが，皮膚入力が加われば，上位中枢を介する促進が行なわれるものと考えられる．

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