Regional Blood Flow Antagonism Induced by Central Thermal Stimulation in the Conscious Dog

Thermal stimuli applied to the hypothalamic or the spinal temperature sensors in anesthetized dogs evoke thermoregulatory changes of skin blood flow which are accompanied by opposite changes of blood flow in the intestine[1,2]. As further shown in anesthetized rabbits, antagonistic changes of regional cutaneous and visceral sympathetic activity correspond to this circulatory phenomenon[3,4]. Thermal stimulation of the hypothalamus as well as of the spinal cord evokes adequate responses of all known thermoregulatory effectors[5,6]. Therefore, it has been assumed that regional antagonism of vasomotor activity is produced by the autonomic nervous system as a thermoregulatory response.

The experimental results on which this conclusion has been based have been obtained under experimental conditions which may restrict or alter the full pattern of responses to central thermal stimulation, especially because of anesthesia and succinyl choline paralysis. Therefore it seemed necessary to confirm the observed vasomotor pattern under more natural conditions, i.e. in the conscious animal.

Method. Two mongrel dogs weighing 20 and 22 kg were prepared for the experiments by chronic implantation of vertebral canal thermodes and, in 1 animal, by additional implantation of a set of hypothalamic thermodes; the methods of implantation and localization are described in detail elsewhere[7]. During the subsequent 2–3-weeks of recovery from the surgical procedures, the animals were trained to rest quietly on a table. 1 day before the start of the experiments, probes of an electromagnetic flowmeter (Statham Multiflo M-4000) were applied in N_2O-Halothane anesthesia under aseptic conditions to the superior mesenteric artery (intestinal blood flow) and to the anterior tibial artery (skin blood flow) of the left hind leg, which was partly immobilized by means of a plaster bandage in order to prevent dislocations of the flow probe. A catheter filled with heparine solution was inserted into the left subclavian artery for arterial pressure recording[8]. Starting 24 h later, the conscious animals were investigated daily for about 1 week. The animals were placed on their right sides. Ambient temperature was thermostatically controlled in such a way that it ranged around the lower limit of thermoneutrality, when central heating was performed, and at the upper limit of thermoneutrality, when central cold stimuli were applied. For thermal stimulation, the thermodes were perfused with water at various, constant temperatures between 19 and 37.5 °C for cooling and 43–48 °C for heating. The stimulation periods lasted 5 min and were preceded and followed by control periods of at least 5 min duration, during which the thermodes were perfused with water at core temperature. Mean blood flow in the anterior tibial artery and in the superior mesenteric artery and arterial mean pressure were recorded as circulatory parameters. Further the temperatures of the hypothalamus, of the vertebral canal, of the rectum and of the ingoing perfusion fluid were measured with thermocouples. Since zero blood flow in the investigated arteries could not be determined in the chronic preparation, changes of regional blood flow were assessed only qualitatively.

Results. The circulatory responses to thermal stimulation of *spinal temperature sensors* are demonstrated in Figure 1 by original recordings. As shown on the left side of the figure, vertebral canal cooling at 30 °C perfusion temperature, which lowered vertebral canal temperature to 33.8 °C ,reduced skin blood flow. Conversely, as shown on the right side, heating with 45 °C perfusion temperature, which elevated vertebral canal temperature to 43 °C, augmented skin blood flow. In both situations, blood flow of the superior mesenteric artery changed oppositely to skin blood flow. Arterial mean pressure showed only short-lasting changes not related to thermal stimulation.

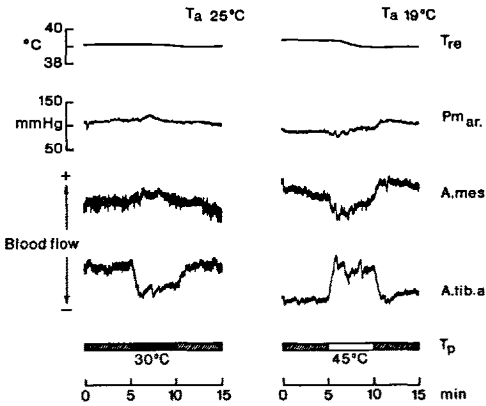

Fig. 1. Antagonistic changes of blood flow in intestine (A. mes. sup.) and skin (A. tib. ant.) induced by selective cooling (left) and heating (right) of the spinal cord in a conscious dog. T_a, ambient air temperature; T_{re}, rectal temperature; Pm_{ar}, arterial mean pressure; T_p, perfusion temperature. Cooling (black bar), heating (white bar), perfusion with water at core temperature (hatched bars).

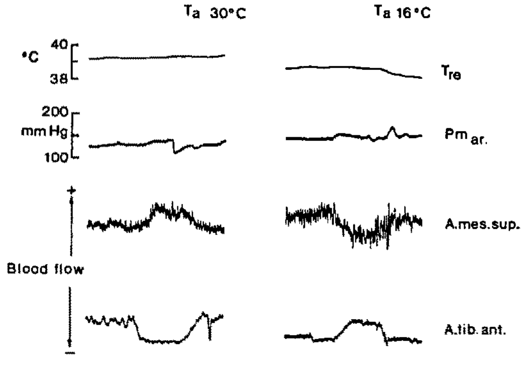

Fig. 2. Antagonistic changes of blood flow in intestine and skin induced by selective cooling and heating of the hypothalamus in a conscious dog. Parameters as in Figure 1.

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[1] R. Kullmann, W. Schönung and E. Simon, Pflügers Arch. *319*, 146 (1970).

[2] W. Schönung, H. Wagner, C. Jessen and E. Simon, Pflügers Arch. *328*, 145 (1971).

[3] O.-E. Walther, M. Iriki and E. Simon, Pflügers Arch. *319*, 162 (1970).

[4] M. Iriki, W. Riedel and E. Simon, Pflügers Arch., in press (1971).

[5] J. D. Hardy, *Brain Sensors of Temperature Regulation.* Brody memorial lecture No. VIII (Univ. of Missouri, Columbia 1969).

[6] R. Thauer, in *Physiological and Behavioral Temperature Regulation* (Ch. C. Thomas, Springfield 1970), p. 472.

[7] C. Jessen and E. Th. Mayer, Pflügers Arch. *324*, 189 (1971).

[8] D. C. Heitz, R. A. Shaffer and M. J. Brody, J. appl. Physiol. *26*, 664 (1969).

The vascular responses to spinal heat stimulation were, on principle, the same at all applied stimulus intensities, even if panting was additionally evoked by strong heating. During vertebral canal cooling, a response like that shown in Figure 1 was observed only at perfusion temperatures around 30 °C, which correspond to an average vertebral canal temperature of 35.5 °C[7]. Stimulation with higher perfusion temperatures seemed too weak to induce a definite vascular response. At stronger cooling, shivering, in addition to skin blood flow reduction, was evoked, which seemed to obscure the vascular response in the intestine by changes of muscle blood flow[1].

Figure 2 shows the effects of thermal stimulation of the *hypothalamic temperature sensors*. In the example demonstrated, the thermodes were perfused during cooling with water of 30 °C, which lowered hypothalmic temperature to 33.8 °C, and during heating with water of 44.5 °C, which elevated hypothalamic temperature to 40.7 °C. The circulatory responses induced by these stimuli were obviously identical with those evoked by thermal stimulation of spinal temperature sensors. Increase of skin blood flow during heating as well as reduction during cooling were accompanied by opposite changes of intestinal blood flow. Arterial mean pressure was not affected by hypothalamic thermal stimulation.

In the case of hypothalamic thermal stimulation, the demonstrated responses were observed, only with quantitative differences, at all stimulus intensities. Depending on stimulus intensity panting was additionally evoked during heating and shivering during cooling.

The thermoregulatory effects of heat stimulation of both hypothalamic and spinal temperature sensors were reflected by the courses of rectal temperature. Its slight fall during central heating, as visible in the figures, was apparently caused by increased heat loss from the skin following cutaneous vasodilatation at cool ambient conditons. During central cold stimulation no such changes of core temperature attributable to thermoregulatory adjustments of skin blood flow could be observed, probably because at warm ambient conditions the possible fluctuations of conductive heat loss from the body surface were reduced.

Conclusions. The pattern of regional blood flow adjustments during central thermal stimulation in the conscious dog is in agreement with the vascular responses observed in the anesthetized, paralyzed animals. Therefore, with respect to the underlying mechanisms, the same conclusions are justified. The results reported thus indicate that evocation of antagonistic changes of activity in sympathetic efferents of the cutaneous and intestinal resistance vessels may occur in the conscious dog as a typical thermoregulatory response of the autonomic nervous system.

Zusammenfassung. Am wachen Hund wurde bei selektiver thermischer Reizung von Rückenmark und Hypothalamus ein regionaler Durchblutungsantagonismus für das kutane und intestinale Gefässgebiet als thermoregulatorische Antwort nachgewiesen.

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Evidence of Direct Nervous Connections Between the Neuromasts of the Lateral Line System of Fishes

According to text books, the sense organs (neuromasts) of the lateral line system of fishes are innervated by the so-called lateralis components of certain brain nerves. These include a pro-otic group which consists of components of the trigeminal nerve and the facial nerve, and a meta-otic group which is associated with the vagus nerve and, in some cases, with the glossopharyngeal nerve. These lateral nerves run to different series of neuromasts and supply single organs of these series by means of side branches.

In the course of studies on the anatomy and development of the lateral line system in the Genus *Tilapia* (Cichlidae)[1], evidence was obtained which indicated that apart from the above-mentioned lateralis nerves there exist nervous bundles which connect the neuromasts directly with one another. Studies of the classic literature revealed, surprisingly, that direct cellular connections between the neuromasts of the lateral line system of fishes had already been intensively discussed in the early papers. The most important of these publications, in so far as the present question is concerned, is that by CLAPP[2] on the lateral line system of *Batrachus*. No paper before or after has portrayed the structures in question with such detail. Her excellent figures (pl. XX, compare Figures 22 and 23) clearly show that the neuromasts, which are spread over the various parts of the head and the trunk of *Batrachus* are not only supplied by the commonly known lateralis nerves but are also endowed with direct connections, one with another. CLAPP calls these connections 'connecting strands' and remarks that that they 'had the appearance of the commissures connecting the ganglia of the sympathetic system, and from the fact that it resisted the action of nitric acid, I inferred that it was nerve tissue'. However, having discussed her observations on the connecting strands as well as the interpretations by other authors, she left the question of their nature open.

Other authors of this period either considered the connecting strands to be lymphatic canals or else suggested that they were simply remnants of the embryonic development and were devoid of function[1]. It was SOLGER[3] who, alone, in 1880, definitely asserted that the connecting strands were nerve bundles, 'marklose, von kernführender Schwann'scher Scheide umschlossene Nervenfasern'. This conclusion was based upon observations of *Acerina* and *Lota*. In 1882 SOLGER[4] referred to 'nervöse Verbindung der Einzelorgane zu einer Organkette'. This is an interesting formulation which indicates a vague idea about the physiological implications of the fibers.

In the following decades, the controversy concerning the 'connecting strands', important as it was, fell gradually

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[1] A more detailed publication is being prepared. Compare also O. H. SPIESER, Anatomische Untersuchungen an den Hirnnerven von *Tilapia* (Cichlidae, Teleostei). Diss. Math.-Naturw. Fakultät Tübingen (1970).

[2] C. M. CLAPP, J. Morph. *15*, 223 (1899).

[3] B. SOLGER, Sitz. ber. naturf. Ges. Halle Jg. *1880*, 105 (1880).

[4] B. SOLGER, Zool. Anz. *5*, 660 (1882).