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Differential Vasomotor Adjustments in the Evaporative Tissues of the Tongue and Nose in the Dog Under Heat Load

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Abstract. 1. Nasal and lingual blood flow were synchronously recorded with respiratory rate and arterial blood pressure in 14 anesthetized spontaneously breathing dogs in which blood temperature was raised by radiant heat. The blood flow responses of the infraorbital and sphenopalatine arteries to increasing heat load were characterized by a continuous increase which resulted from significant decreases in local vascular resistances. The final values during panting exceeded the resting values by 3 times. In contrast, lingual blood flow remained nearly unaffected as long as panting did not occur. With the onset of panting, lingual blood flow increased in close correlation with the increase in respiratory rate. This increase also resulted from a significant decrease in local vascular resistance.

2. The preoptic-anterior hypothalamic region was heated with a water perfused thermode in 10 other dogs at normal (38.4° C) and elevated (39.4° C) blood temperature. Hypothalamic heating at a normal blood temperature induced vasodilatation only in the nasal vessels, while lingual blood flow and respiratory rate were nearly unaffected. However, in animals at an elevated blood temperature hypothalamic heating stimulated the full heat defense response, i.e. a marked increase in both nasal and lingual blood flow associated with polypnea.

3. The results suggest that under normal conditions, in which the dog is breathing with the mouth closed, the graded enhancement of convective heat transfer to the respiratory mucous surfaces of the nose enables a continuous control of evaporative heat loss from these surfaces. During panting, when the dog is breathing with the mouth opened, the additional increase of heat transfer to the surface of the protruded tongue further increases the efficiency of evaporative heat loss. In addition the results confirm the hypothesis that the upper brainstem coordinates the differential patterns of circulatory adjustments in evaporative tissues.

Key words: Nasal and lingual blood flow – Hypothalamus-temperature regulation – Regional vasomotor differentiation – Panting.

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Introduction

The dog dissipates excessive body heat mainly by evaporation which takes place at the surface of the upper airway tracts. This requires an adequate ventilation and moistening of these surfaces as well as an appropriate convective heat transfer from the body core to these surfaces by the circulation. The nasal mucosa has been suggested as the primary site of evaporation by Schmidt-Nielsen et al. (1970). This structure is particulary suited for evaporation due to the fact that its large surfaces are in direct contact with the airstream (Dawes and Prichard, 1953; Scott, 1954) and that large serous type glands are found in the nasal cavities as the source of water for evaporation (Blatt et al., 1972).

Open mouth panting with evaporation from the tongue and oral mucosa is additionally employed when maximum heat dissipation is necessary (Krönert and Pleschka, 1976a). Previous experiments have shown that, with the onset of panting, lingual blood flow increased in strict correlation with the increase in respiratory rate (Kindermann and Pleschka, 1973; Krönert and Pleschka,1976b; Roth and Pleschka,1977), indicating a common control of both heat dissipation from the tongue and heat transfer to its evaporative surfaces. It was further demonstrated that the augmentation of blood flow to the tongue was associated with an increase in arterio-venous temperature difference

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indicating that the tongue acts as a heat exchanger (Krönert and Pleschka, 1976b).

Since, in prepanting stages, the dog dissipates heat mainly by evaporation from the nasal mucosa, the question arises to which extent and how convective heat transfer to this surface is controlled. Surprisingly little is known about the circulatory adjustments taking place in the nasal vessels at different degrees of heat load. In particular, nothing is known as to how blood flows through the nasal and lingual mucosa are coordinated during heat load. To shed more light on these questions, blood flows of the lingual, the infraorbital, and the sphenopalatine arteries were synchronously measured in anesthetized dogs in which blood temperature was increased by external heating. In addition, local hypothalamic heating was carried out in order to ascertain whether the upper brainstem is involved in the control of the thermoregulatory adjustments of nasal and lingual blood flow.

Methods

The results were obtained in 24 dogs of both sexes with an average weight of 28.5 ($\pm$ 3.7) kg. The animals were anesthetized with 30 mg·kg^{-1} i.v. administered sodium pentobarbital. If necessary, additional doses of 100–200 mg of the anesthetic were given during the surgical procedure.

Surgical Procedures, Measurements and Recordings. Catheters were placed in the femoral vein for administration of anesthetics and in the thoracic aorta for recording of blood pressure. Blood pressure was measured with a Statham P23db transducer connected to a transducer coupler of a Brush recorder. To monitor blood temperature, a thermocouple was introduced into the thoracic aorta via a brachial artery and connected to an electric thermometer (Ellab Copenhagen). Blood flow was measured with 3 non-occlusive electromagnetic flowmeters (Statham SP 2202). The flow probes were calibrated by means of a motor driven syringe (for details see Kullmann et al., 1970) and had a measurement error of 2–5% depending on the probe diameter. To obtain lingual blood flow, a probe of appropriate size was placed around one of the lingual arteries proximal to the exit of the tonsillar artery and the hyoid rami. In order to attach the flow probes to the nasal vessels, the dog's head was first fixed in a stereotaxic device, and the maxilla of one side was exposed above the first and second molar. Then the pterygopalatine fossa was opened by drilling a hole in the maxillary bone. The orbital fat was removed and the infraorbital artery was dissected free from the adjacent maxillary division of the trigeminal nerve under stereomicroscopic control (Zeiss). Branches to the orbital gland and the posterior and middle dorsal alveoli were cut after appropriate ligature. In general a flow probe of 2.0 mm size in diameter was found suitable for the infraorbital artery. The sphenopalatine artery was dissected free rostral to the exit of the major palatine artery and a flow probe (usually 1.5 mm in diameter) was attached. The dog's core temperature could be raised either by heating pads or radiant heat, while heating of the preoptic anterior hypothalamic region was performed using a water perfused thermode discribed in detail elsewhere (Krönert and Pleschka, 1976b). For thermode insertion the dorsal skull was exposed and, under X-ray control, the rostral edge of the sella turcica was chosen as the target point for the center of the thermode array, which was inserted through drill holes in the skull until its tip reached the base of the skull. Through indwelling thinner tubes, the thermode tubes were perfused with water at 48° C at a flow rate of about 200 ml·min^{-1}. Respiratory rate was assessed by measuring the transthoracic impedance changes in phase with breathing with a Brush impedance coupler. This signal was fed into a Brush biotachometer to provide an analog signal equivalent to respiratory rate per minute. All measurements were recorded continuously on a 8 channel Brush polygraph.

Experimental Protocol. Two groups of animals were studied.

In *group A* which consisted of 14 dogs the following values were recorded at resting body temperature and after progressive increases in blood temperature by 0.5° C: mean blood flows of the lingual, the sphenopalatine and the infraorbital arteries, arterial pulse pressure, heart rate and respiratory rate. Perpheral resistance of each vascular bed was calculated from blood flow and mean blood pressure. The latter was evaluated from the pulse pressure.

Mean values with standard deviations were calculated from the corresponding data to evaluate the average responses. The differences between the various periods were tested for statistical significance using the Wilcoxon matched-pairs signed-ranks test at a level of $2P \leq 0.05$. Differences in the number of measurements are due to the differences in the core temperatures at which the experiments were started and to the individual differences in the panting thresholds.

In *group B* which consisted of 10 dogs the blood flows to the nose and tongue were recorded together with mean blood pressure and respiratory rate before, during and after hypothalamic heating at a resting and at a slightly elevated blood temperature. Heart rate was evaluated from the pulsatile flow recordings. Due to technical problems, the blood flows of the sphenopalatine and the infraorbital arteries could not be synchronously measured. Accordingly, the blood flow values of the infraorbital and lingual arteries were derived in 5 dogs, and the blood flow values of the sphenopalatine and again the lingual arteries in 5 other dogs.

Mean values and standard deviations were calculated from steady state values before, during and after thermal stimulation of the hypothalamus. Again the differences between the various periods were tested for statistical significance with the Wilcoxon matched-pairs signed-ranks test at the level of $2P \leq 0.05$ and $P \leq 0.05$ respectively (McConack, 1965).

Results

The Effects of Body Core Heating on Nasal and Lingual Blood Flow and Respiratory Rate

The graph in Fig. 1 summarizes the average values of lingual and nasal blood flow and of the respiratory rate as related to blood temperature.

Starting at a blood temperature of 38.0° C the unilateral lingual blood flow to the tongue was 8.9 ml·min^{-1} while the flow of both the infraorbital and the sphenopalatine arteries were nearly the same (11.1 and 11.4 ml·min^{-1} respectively).

It can be seen that a rise of blood temperature of 1.5° C resulted in a significant increase in blood flow to the nasal vessels, while lingual blood flow differed little from the resting value. In addition, the augmentation of nasal perfusion was accompanied by a slight rise in respiratory rate. With a further increase in blood temperature, nasal blood flow and respiratory rate

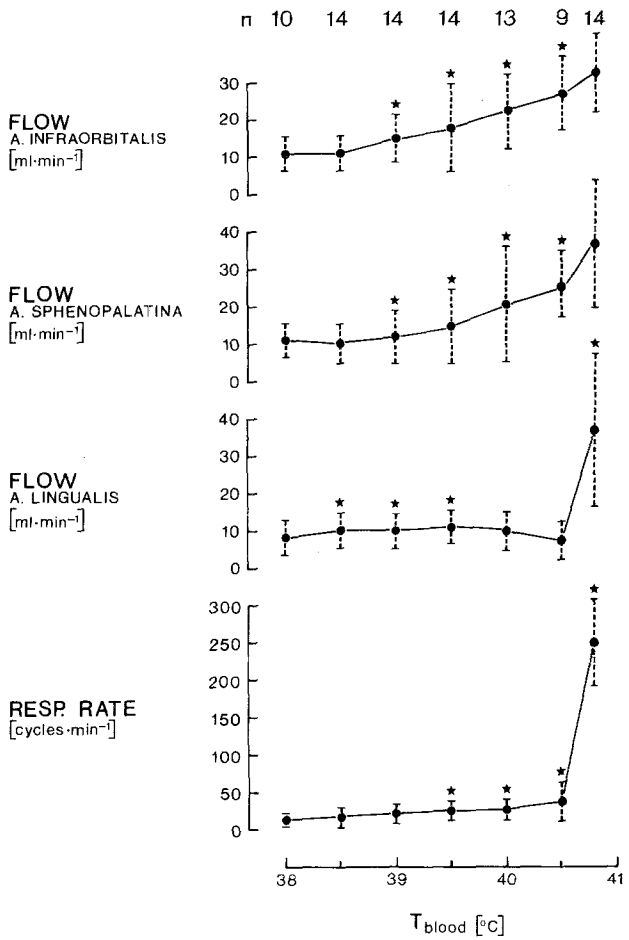

Fig. 1. The response of infraorbital, sphenopalatine and lingual blood flow and of respiratory rate to increasing blood temperature. Mean values are indicating by solid dots, standard deviations by vertical bars. Values significantly different from the preceding values are indicated by an asterix

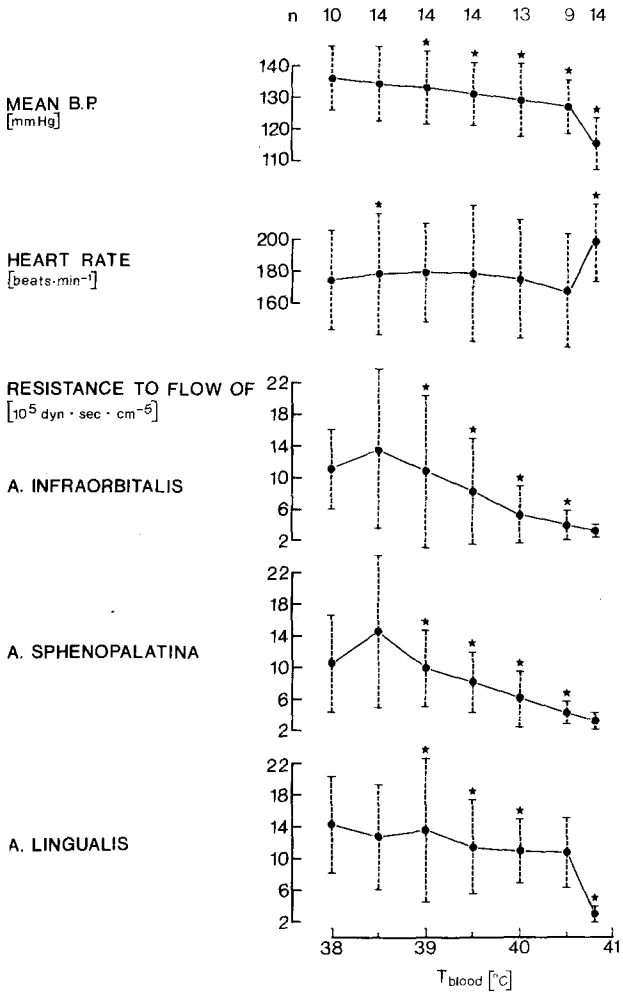

Fig. 2. The response of mean blood pressure (mean B. P.), heart rate and resistance to flow of the infraorbital, sphenopalatine and lingual arteries to increasing blood temperature. Mean values, standard deviations and significant differences are as indicated in Fig. 1

continued to rise, while lingual blood flow remained low or even slightly decreased.

However, with the onset of panting which occurred above a blood temperature of 40.5° C on the average, there was a marked increase in lingual blood flow. During continuous panting at an average respiratory rate of 248 cycles·min^{-1}, lingual blood flow was 37.5 ml·min^{-1} as compared to 8.9 ml·min^{-1} at rest. The corresponding values for blood flow in the infraorbital and sphenopalatine arteries during panting were 29.0 and 33.0 ml·min^{-1}.

Figure 2 shows that an unequivocal decrease in peripheral resistance of the vascular beds of the infraorbital and sphenopalatine arteries occurred with the rise of blood temperature causing an increase in nasal blood flow despite the decreasing systemic pressure. As expected, there was a marked decrease in peripheral resistance of the lingual vascular bed at the onset of panting. The increase in heart rate, which occurred at the highest blood temperature studied was probably a reflex response to the fall of arterial pressure.

The Effects of Hypothalamic Heating on Nasal and Lingual Blood Flow and Respiratory Rate

The original recordings shown in Fig. 3 demonstrate the effects of hypothalamic heating on the sphenopalatine and lingual blood flow as well as on blood pressure and respiratory rate at different blood temperatures.

It is striking, that hypothalamic heating provoked a marked increase in sphenopalatine blood flow within 3 min while lingual blood flow, respiratory rate and

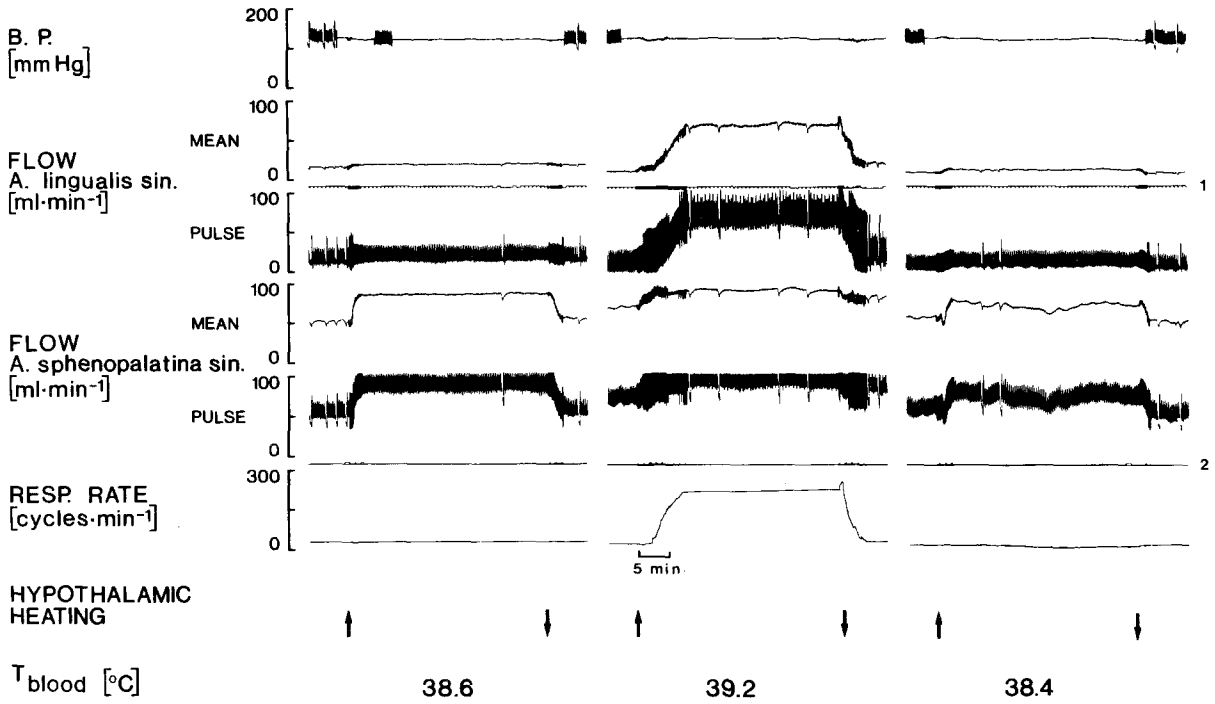

Fig. 3. The effect of hypothalamic heating on blood pressure *(B. P.)*, lingual *(Flow A. ling. sin.)* and sphenopalatine blood flow *(Flow A. sphenopalatina sin.)* and on respiratory rate at different blood temperatures in an anesthetized, spontaneously breathing dog. Marker 1 indicates 1 s, marker 2 1 min time intervals

blood pressure remained nearly unaffected at a blood temperature of 38.6° C. This increase was maintained as long as the thermal stimulus to the hypothalamus lasted. When perfusion of the thermode was stopped, sphenopalatine blood flow returned to its initial value within 3 min.

When blood temperature was increased to 39.2° C there was an increase in sphenopalatine blood flow, but not in lingual blood flow in accordance with the results obtained in group A. With this slightly elevated blood temperature however, hypothalamic heating now induced the full response of respiration and circulation within a period of 8 min. It consisted of a marked increase in lingual blood flow and respiratory rate together with a small further increase in sphenopalatine blood flow. Again this response was maintained as long as the thermal stimulus lasted. When the thermal stimulus was switched off there was a decrease in respiratory rate and blood flow to the tongue and nose within about 4 min to initial values.

When the blood temperature of the animal had finally restabilized at 38.4° C the effects of hypothalamic heating were the same as those observed previously at 38.6° C.

The results from 10 animals are summarized in Table 1. At a mean blood temperature of 38.4° C, hypothalamic heating resulted in a significant increase in blood flow to the nose which amounted to 62% for the sphenopalatine and 85% for the infraorbital artery. This central heat stimulus did not significantly affect lingual blood flow nor respiratory rate at this blood temperature. The mean values show, that the increase in nasal blood flow was due to a significant decrease in resistance to flow in the corresponding vascular beds.

With regard to the response time of the nasal blood flow changes to the changes in hypothalamic temperature, new steady state values were reached within a mean time of 2.5 (± 0.7) min after the initiation of hypothalamic heating and within a mean time of 3.6 (± 1.8) min after its cessation.

Under the light thermal preload obtained by elevating the blood temperature to 39.4° C, hypothalamic heating not only induced a further significant increase in nasal blood flow, but also significant increases in both, lingual blood flow and respiratory rate. Taking into account the fact, that the nasal blood flow changes started from an already elevated level, the 210% increase in lingual blood flow is very striking. In contrast, the enhancement of sphenopalatine blood flow was 54% and that of the infraorbital 20%. The increase in blood flow to the tongue and nose during hypothalamic heating could be accounted for by significant decreases in the regional peripheral resistances. There were only minor changes in blood pressure and heart rate during the thermal stimulation at both normal and elevated blood temperatures. Both circu-

Table 1. Mean values ($\bar{x}$) and standard deviations (s) of blood pressure (mean B.P.), heart rate, blood flow, and resistance (R.) to blood flow in the lingual (A. ling.), the sphenopalatine (A. sphen.) and the infraorbital artery (A. infra.) and of respiratory rate before (off), during (on) and after (off) hypothalamic heating at different blood temperatures (T_{blood} °C)

T_{blood} (°C)	$\bar{x}$		38.4			39.4		$n = 10$
	s		0.4			0.4		
Hypothalamic heating		off	on	off	off	on	off	
Mean	$\bar{x}$	131.4	125.6	125.8	121.9	108.3	121.9	$n = 10$
B.P.	s	19.6	22.5	18.0	12.5	41.6	11.2	
(Torr)	$2P$		<0.01	<0.05		n.s.	$=0.05$	
	$\bar{x}$	169.4	168.0	166.0	174.0	167.4	169.8	$n = 10$
Heart rate	s	42.6	27.0	29.0	28.4	26.8	31.1	
($b \cdot min^{-1}$)	$2P$		n.s.	n.s.		0.05	n.s.	
	$\bar{x}$	11.1	12.9	11.8	12.0	37.3	14.0	$n = 10$
Flow A. ling.	s	4.8	5.0	4.8	4.3	19.3	6.1	
($ml \cdot min^{-1}$)	$2P$		<0.01	<0.05		<0.01	<0.01	
	$\bar{x}$	11.66	9.39	10.32	9.32	3.17	9.26	$n = 10$
R. A. ling.	s	6.44	5.28	5.78	3.88	1.58	6.74	
(10^5 dyn $\cdot$ s $\cdot$ cm^{-5})	$2P$		<0.01	<0.05		<0.01	<0.01	
	$\bar{x}$	28.8	46.6	30.8	38.4	59.2	42.2	$n = 5$
Flow A. sphen.	s	19.6	26.1	19.7	19.0	20.3	23.5	
($ml \cdot min^{-1}$)	P		$=0.05$	$=0.05$		$=0.05$	$=0.05$	
	$\bar{x}$	6.02	3.11	4.91	3.00	1.86	2.87	$n = 5$
R. A. sphen.	s	4.47	2.03	2.94	1.22	0.64	1.23	
(10^5 dyn $\cdot$ s $\cdot$ cm^{-5})	P		$=0.05$	$=0.05$		$=0.05$	$=0.05$	
	$\bar{x}$	18.0	33.4	20.8	28.0	33.6	26.6	$n = 5$
Flow A. infra.	s	10.0	14.9	10.8	11.0	11.5	13.0	
($ml \cdot min^{-1}$)	P		$=0.05$	$=0.05$		$=0.05$	$=0.05$	
	$\bar{x}$	6.94	3.31	5.96	4.04	2.87	4.79	$n = 5$
R. A. infra.	s	3.14	1.37	3.50	1.93	0.88	3.15	
(10^5 dyn $\cdot$ s $\cdot$ cm^{-5})	P		$=0.05$	$=0.05$		$=0.05$	$=0.05$	
	$\bar{x}$	17.1	20.0	17.6	31.9	252.4	33.0	$n = 10$
Resp. rate	s	6.6	12.9	9.2	10.7	57.2	9.5	
($cycles \cdot min^{-1}$)	$2P$		n.s.	n.s.		<0.01	<0.01	

lation and respiration responded almost immediately to central thermal stimulation and the peak response occurred at 5.7 ($\pm$ 3.2) min after the onset of hypothalamic heating and 6.6 ($\pm$ 6.9) min after its cessation.

Discussion

The present investigation, dealing with the thermoregulatory adjustments of vessels supplying evaporative tissues of the nose and tongue, has revealed blood flow responses to increasing heat load which were characterized by continuous blood flow adjustments in the nasal vascular beds in contrast to a clear threshold dependent response of the lingual arteries.

The response of nasal blood flow to the enhancement of heat load was a continuous increase in the sphenopalatine and infraorbital flow rates from 11 ml $\cdot$ min^{-1} in each artery at rest to 33 ml $\cdot$ min^{-1} in the sphenopalatine and 29 ml $\cdot$ min^{-1} in the infraorbital artery during panting. In correlation with this increase in nasal blood flow, respiratory rate rose from 12 cycles $\cdot$ min^{-1} at rest to 35 cycles $\cdot$ min^{-1} at the prepanting blood temperature of 40.5° C. It is remarkable that in spite of anesthesia, an elevation of blood temperature by just 1.0° C was sufficient to increase nasal perfusion significantly. The final values during panting exceeded the resting values by 3 times. In contrast, lingual blood flow remained nearly unaffected by the increase in blood temperature as long as panting did not occur. However, with the onset of panting, after which respiratory rate increased up to 248 cycles $\cdot$ min^{-1}, lingual blood flow increased markedly from 9 ml $\cdot$ min^{-1} to 38 ml $\cdot$ min^{-1} (bilateral: 18–76 ml $\cdot$ min^{-1}) in close correlation with the increase in respiratory rate. The functional importance of the latter reaction is indicated by the fact that in the conscious animal at this time the mouth is opened and the moistened tongue protruded, moving synchronously with the respiratory movements to enhance evaporation.

The total blood flow distributed by the three paired arteries to the lingual and nasal region during panting was 197 $ml \cdot min^{-1}$, which represents roughly 10% of the cardiac output in the pentobarbital anesthetized dog (Göbel et al., 1977).

The increase of nasal blood flow by a factor of 3 from thermoneutral to maximum heat load conditions corresponds to the increase in nasal mucosal blood flow which has been observed in the conscious animal by Hales and Dampney (1975) on basis of blood flow per unit tissue weight. In their experiments, blood flow increased from 19.3 ($ml \cdot 100\ g^{-1} \cdot min^{-1}$) in a thermoneutral (T.N.) environment at a rectal temperature of 38.7° C to 60.8 ($ml \cdot 100\ g^{-1} \cdot min^{-1}$) during the first phase of rapid shallow panting (R.S.P.) coinciding with a rectal temperature of 40.2° C. However, blood flow to the tongue in their experiments increased from 5.8 ($ml \cdot 100\ g^{-1} \cdot min^{-1}$) at T.N. to 62.8 ($ml \cdot 100\ g^{-1} \cdot min^{-1}$) at R.S.P., i.e. by a factor of 11, which is clearly different from the factor of 4 in our experiments. Converting their values to absolute blood flow values by using a mean tongue weight of 79.7 g as reported in their paper, would result in a resting lingual blood flow of 4.6 $ml \cdot min^{-1}$ at T.N., and 50.6 $ml \cdot min^{-1}$ at R.S.P. both of which are less than in the present investigation. One reason for the differences of our values might be the anesthetic used in our experiments. According to the investigation of Brace (1978) pentobarbital itself elevates, for instance, forelimb blood flow in the resting dog and this could also be the case with regard to resting blood flow in the tongue. On the other hand we also found higher resting blood flows in conscious animals (Krönert and Pleschka, 1976b) than Hales and Dampney (1975) which suggests intraspecies differences between the investigated dogs as the cause for the discrepancy. The different methods used might also explain part of the discrepancy.

Taking into account the bilateral blood flow changes in our own experiments, and assuming a mean temperature gradient of 1.5° C between the arterial and venous blood as found for the tongue in the conscious animal by Krönert and Pleschka (1976b) it can be calculated that convective heat transfer from the core to the nasal mucosa is of the order of 13.5 Watt and to the tongue of 8.3 Watt at fully developed open mouth panting. The comparison of both values demonstrates the relevance of the tongue as an additional heat exchanger during panting. On the other hand, it would be expected that the extensive surface area of the nasal mucosa (Scott, 1954) is more efficient in dissipating heat than the smaller surface of the tongue and, therefore, the assumed overall arterio-venous temperature gradient of 1.5° C derived from measurements in the tongue is most probably smaller than the true arterio-venous temperature gradient of the nasal blood flow. In other words, a convective heat transfer larger than the calculated value may be reasonably assumed for the nose. With regard to the local distribution of lingual blood flow during panting it has been shown (Krönert and Pleschka, 1976a) that the opening of arterio-venous anastomoses provides a low resistance pathway for a high blood flow to the superficial veins where heat exchange probably occurs. In contrast, the role of arterio-venous anastomoses of the nasal mucosa in heat dissipation has not been quantitatively assessed, although their large number suggests that they must greatly influence the mucosal circulation. According to the studies of Dawes and Prichard (1953) their situation and potential diameter indicate that open arterio-venous anastomoses will divert blood away from the glandular capillary plexus and will also probably reduce the portion of the nasal blood flow received by the subepithelial plexus. However, under the condition of heat dissipation one can assume that concomitant to the opening of arterio-venous anastomoses glandular vasodilatation takes place providing sufficient blood supply for the secretory activity. Opening of both low resistance pathways for blood flow would meet all the requirements for evaporative heat loss: i.e. sufficient glandular secretion and enhanced heat transfer to the large venous plexus.

In confirmation of the hypothesis of *Upper Brainstem Coordination* of the different patterns of circulatory adjustments of heat defense, local heating of the preoptic-anterior hypothalamic region induced a differentiated response of nasal and lingual blood flow as well as of respiratory rate depending on the thermal preload. Accordingly, hypothalamic heating at normal body temperature elicited vasodilatation only in the nasal vessels, while lingual blood flow and respiratory rate were nearly unaffected. Since the dog under normal conditions breathes with the mouth closed, this mechanism appears appropriate, enabling a continuous control of evaporation from the respiratory mucous surfaces of the nose by graded enhancement of convective heat transfer to these surfaces.

There are two pathways by which the cooled venous blood from the nose is drained; directly to the cavernous sinus of the brain via the angularis oculi and ophthalmic veins or bypassing the brain via the facial vein (Magilton and Swift, 1969). Since there is evidence that the nasal veins and thus the rate and route of blood flow is under control of the autonomic nervous system (Magilton and Swift, 1969), one could speculate that the control of the amount of the cooled nasal blood which is directed to the brain establishes a thermostatic mechanism for the brain (Magilton and Swift, 1967). It would appear logical that this thermostatic mechanism is primarily controlled by the hypothalamic thermosensors. Consequently extrahypothalamic thermosensors, like those of the spinal cord (Jessen and Mayer, 1971; Simon, 1974) or of the deep body tissues (Riedel et al., 1973) would predominantly be concerned with body temperature control. However, the hypothalamic thermosensors obviously contribute also to this input function since in animals submitted to a subliminal thermal preload hypothalamic heating stimulated the full heat defense response, i.e. not only a marked nasal vasodilation but also an enhancement of lingual blood flow concomitant to polypnea.

Concerning the mechanism by which the different modes of circulatory adjustments of nasal and lingual arteries are induced by the thermal stimulus, nervous regulatory influences as well as local differences in nerve fiber supply (Folkow, 1960) receptor distribution (Ahlquist, 1948; Green and Kepchar, 1959; Holtz and

Palm, 1966; Hall and Jackson, 1968) nerve effector relationship (Celander and Folkow, 1953; Peiper et al., 1967), and local humoral agents (Hilton and Lewis, 1955) have to be considered. Taking into account the amply described ability of the sympathetic system to differentiate its vasomotor outflow in homeostatic control conditions (Iriki and Simon, 1978), and considering the fact that the described flow changes were certainly part of such a homeostatic process, namely thermoregulation, a quantitatively differentiated control of the vasoconstrictor tone in these vascular beds is a most likely assumption. In addition there is growing evidence that neurogenic vasodilatation may generally contribute to remote control of local blood flow (Erici et al., 1952; Eccles and Wallis, 1974; Johansen, 1962; Hellekant, 1977; Malcomson, 1959; Jackson and Rooker, 1971). In particular, several investigations (Schönung et al., 1972; Riedel and Peter, 1978; Gregor et al., 1976; Pleschka et al., 1978) have presented evidence that thermoregulatory adjustments of skin and tongue blood flow are partly controlled by vasodilator efferents. Finally the vasodilatation of the tongue might simply reflect post-contraction hyperaemia independent of the sympathetic nervous system (Hilton et al., 1979). However, this mechnism seems to play a minor role, since Kindermann and Pleschka (1973) could induce lingual vasodilatation by increasing the core temperature in the paralyzed and artificially ventilated dog. Furthermore, Krönert and Pleschka (1976a) have clearly shown, that the thermally induced increase in lingual blood flow occurs predominantly in the mucosa and not in muscles.

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