REGIONAL SKIN TEMPERATURES ASSOCIATED WITH TOTAL SYMPATHETIC BLOCKADE IN CONSCIOUS DOGS

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It is well established that sympathetic nerve blockade can increase skin temperature and blood flow in the upper and lower limbs [1–8]. In contrast, little information is available about the role of the autonomic nervous system in influencing skin temperature on the trunk.

During spinal anaesthesia with an analgesic level of T10 in man, skin temperatures on the chest and abdomen decreased, with variable changes in skin blood flow [5,8].

To determine whether a reflex increase in sympathetic discharge from segments above the blocked regions, causing vasoconstriction, is the dominant mechanism in the decrease in skin temperature on the trunk above and within analgesic body regions, we evaluated regional skin temperature in trained conscious dogs, in which sympathetic influences were eliminated by high extradural anaesthesia. To address the potential influences of baseline efferent sympathetic tone, investigations were undertaken in cold and warm constant temperature environments.

MATERIALS AND METHODS

The investigations were performed in six trained short-fur dogs (five German boxers and one boxer mongrel, weights 19–25 kg) housed in the local animal care facility. Four of the animals had had one or both common carotid arteries exteriorized in skin loops several months before the experiments. The effects of total sympathetic blockade—produced by extradural anaesthesia—on regional skin temperatures, arterial pressure and heart rate were evaluated on different days in each dog during exposure to either a cold or a warm constant ambient temperature slightly below or above the thermoneutral range (which is approximately 25 °C in dogs [9]).

Measurements

Cutaneous temperature was measured using temperature sensors of our own design. Briefly,
standard PT 100 resistors (100 Ω at 0 °C, length 2 mm, width 2 mm) were imbedded in a thin layer of epoxy resin. These resistors are linear within ±0.01 °C over a temperature range from −50 °C to +100 °C and in the actual temperature range during the experiments deviated less than 10⁻³ °C. The imbedded resistors were placed in the centre of a plastic ring (radius 4.5 mm, height 2 mm) and minimally covered with latex foam, so that the resistor protruded from one surface against the dog’s skin. By applying a step change in temperature, it was found that the sensors had time constants of approximately 30 s. The sensors were attached to the animal’s skin using a drop of collodion applied to the plastic ring, and connected to a Z-80 microprocessor (operating system CP-M, Infos, FRG) using an interface of our own design.

Before each individual study, the sensors were calibrated against a mercury thermometer in a closed bottle at room temperature. A terminal provided an analogue and digital display of the measured temperatures. Recorded temperatures were rounded to the nearest 0.1 °C. Skin temperatures were always measured on the same skin area at eight different regions (fig. 1) and were recorded with rectal and room temperatures.

Arterial pressure was measured electromanometrically (Statham 23 ID transducer) via a catheter inserted into a carotid loop artery (four dogs) or into a femoral artery (two dogs). Heart rate was measured by a cardiotachometer, triggered by the R-wave of the electrocardiogram (ECG). Signals were amplified and recorded on a Beckman polygraph.

**Extradural anaesthesia**

An extradural catheter was introduced through a 17-gauge Tuohy needle in an intervertebral space (usually between L5 and L6) under sterile conditions during anaesthesia using an ultra-short acting barbiturate (methohexitone 4 mg kg⁻¹ i.v.). The extradural space was identified by the “loss of resistance” technique using a saline-filled syringe. A thin polyethylene catheter was then advanced for 3 cm into the lumbar extradural space through the needle and the needle withdrawn over the catheter. For extradural anaesthesia, 2% lignocaine 9–11 ml (stored at room temperature) was injected. All animals showed evidence of blockade of the entire sympathetic nervous system; that is, the blockade extended caudally from the first thoracic segment. In all dogs the third eyelid, which is innervated by sympathetic nerve fibres from the upper three thoracic segments [10,11], was paralysed and moved laterally over the eye. None of the dogs responded to noxious stimulation (needle probing) applied to the first intercostal space. Besides complete paralysis of the hind legs, all dogs exhibited diaphragmatic breathing, indicating that the intercostal muscles were at least partially paralysed. Finally, the reflex increase in arterial pressure secondary to clamping of both common carotid arteries was essentially abolished in the two animals in which this response was tested. To assess further the spread of the injectate within the extradural space, 10 ml of a water soluble radiocontrast medium (Solutrast 200M, Lomberg, FRG) was injected through the extradural catheter once the experiments had been completed—that is after the block had resolved. Radiopaque material was observed by x-ray in the extradural space of the upper thoracic spine in each dog.

**Experimental programme**

All dogs, trained to lie unrestrained on their right side on a cushioned table during the course of the experiments, were studied on different days in the morning in the fasting state at constant ambient temperature around either 22 °C (cold environment) or 27 °C (warm environment). At least 2 days elapsed before a particular dog was studied again. With all catheters (i.v., i.a., extradural) and temperature sensors in place, we waited for at least 1 h after the dogs had
completely recovered from anaesthesia before the recording started. All experiments followed the same programme, the dogs being randomly assigned to either the cold or the warm environment:

(1) A further control period of 15 min to ascertain that all variables were in a steady state.
(2) Extradural injection of lignocaine within 2 min and recording of variables for 45 min. Ambient temperature was maintained within ±0.2 °C.

No fluids or drugs were given during the definitive experiments.

In some dogs the effects of extradural injection of saline 10 ml at room temperature, or of deep i.m. injection of 2% lignocaine 10 ml through a catheter placed in the back musculature were also studied to exclude significant effects secondary to potential changes in spinal cord temperature or increases in blood concentrations of anaesthetics.

Data analysis

Data are reported as averages (SEM) at 5-min intervals before and during extradural anaesthesia. Since the complete extent of nerve block was usually established within 25–35 min, differences between the control state and the denervated state were tested with Student’s two-tailed $t$ test for paired data 45 min after the injection of lignocaine; that is, when the changes had reached a steady state. Statistical significance was assumed when $P$ was less than 0.05.

RESULTS

Sympathetic blockade always resulted in an increase in skin temperature in both the front and
Rectal temperatures were not significantly different between the two groups in the control state and did not change significantly after sympathetic block in either the cold (38 (0.2) °C to 38 (0.19) °C; \( P = 0.5 \)) or the warm (38.05 (0.04) °C to 38.31 (0.3) °C; \( P > 0.2 \)) environments.

The regional differences in the response to sympathetic blockade become even more apparent when the actual skin temperatures are considered (fig. 3). In a cold environment the well described temperature gradient along the body axis was apparent, with the highest skin temperature on the chest and the lowest on the paws. This typical pattern was reversed after sympathetic block, which elicited the expected marked increases in skin temperature in the front and hind limbs, but also a distinct decrease in the temperature on the thoracic and abdominal skin regions. In the warm environment there was the expected general increase in control skin temperature and, at first glance, there did not seem to be much of a difference between the innervated and denervated state. Nonetheless, statistical analysis revealed that, even under these conditions, there was an increase in skin temperature on the limbs, and a decrease on the trunk, after sympathetic block.

The majority of the dogs shivered slightly in the cold environment before sympathetic block, but never panted in the warm environment.

Systolic and diastolic arterial pressures decreased significantly after sympathetic blockade in both environments (fig. 4). In the warm environment systolic arterial pressure decreased from 128 (6) mm Hg to 112 (2) mm Hg and diastolic from 89 (3) mm Hg to 79 (3) mm Hg 45 min after extradural block. In the cold environment control arterial pressure was greater but also decreased significantly after sympathetic block (systolic: 143 (7) mm Hg to 120 (7) mm Hg; diastolic: 98 (4) mm Hg to 83 (6) mm Hg). Despite the decrease in arterial pressure, heart rate was not changed significantly by extradural block. Mean arterial pressure (93) (4) mm Hg and heart rate (91 (2) min v. 94 (1) min) were similar in both environments after denervation.

No complications resulted from the procedure. Arterial blood-gas measurements performed in four dogs 45 min after extradural block showed neither hypercarbia nor hypoxia.

Neither the extradural injection of physiological saline nor the i.m. injection of lignocaine had any appreciable effect on regional skin temperatures, arterial pressure or heart rate.
DISCUSSION

The present study was designed to investigate the role of the sympathetic nervous system in influencing skin temperature on the trunk, a variable that has been measured clinically to indicate the level of sympathetic blockade with regional anaesthesia [5, 8, 12]. Under conditions of constant ambient temperature both above and below the thermoneutral range and with rectal temperature constant, regional skin temperature significantly increased on the limbs, but decreased on the trunk after complete sympathetic block. This finding is inconsistent with the hypothesis that a primary increase in sympathetic outflow from unblocked body regions is the dominant factor in the decrease of trunk skin temperature within and above analgesic areas observed after spinal anaesthesia. Furthermore, our results indicate that the sympathetic nervous system substantially contributes to the maintenance of the normal skin temperature gradient along the body’s long axis.

Critique of methods

Apart from tissue conductance and, hence, blood flow, skin temperature depends on ambient and core temperatures [13, 14]. Thus only under conditions of constant ambient and constant core temperatures can changes in skin temperature reflect changes in skin blood flow. Since we have evaluated regional skin temperatures at both constant ambient and constant body temperatures, changes in skin temperatures should, at least qualitatively, have reflected corresponding changes in skin blood flow. This is supported by the observation in man at constant environmental temperature that skin temperature correlates closely with finger and hand blood flow at skin temperatures between 28 and 34 °C [13, 15], a range studied in our experiments.

The present studies were carried out in dogs rather than in man, because it would have been impossible to maintain constant environmental and core temperatures under operating room conditions, a uniform problem in previous studies [5, 16]. In addition, it did not seem appropriate to perform repeated complete sympathetic blocks and invasive monitoring under rigid laboratory conditions in human subjects. Instead, we have chosen the dog as the experimental model because it can be studied in the conscious state and its mechanisms for temperature regulation are well understood. While man and dog are regarded as qualitatively similar with respect to thermoregulation in cold and neutral environments, they differ in their mechanisms for heat dissipation in hot environments—that is, above 30 °C. Whereas man can dissipate heat by sweating, the dog has no sweat glands except under the footpads, and dissipates excess heat by panting [10, 17]. In this respect, dogs are similar to those humans with congenital absence of sweat glands [18]. Since the-
present experiments were carried out at relatively low ambient temperatures, panting was not observed. Thus while caution must be exercised when extrapolating results from any animal model to man, the absence of sweat glands on the dog’s trunk was thought to facilitate the physiological interpretation of our observations.

Extradural anaesthesia, in our experiments, definitely eliminated sympathetic efferent activity. This was demonstrated by paralysis of the third eyelid (the equivalent of Horner’s sign), the sympathetic innervation of which derives from the upper three thoracic segments [10,11]. It was further confirmed by the absence of the normal pressor response to carotid clamping, an accepted sign for block of the sympathetic outflow to the blood vessels [19], in the dogs in which this response could be tested. The observed increase in skin temperature in the front limbs also indicates complete sympathetic block [6]. Finally, the dogs were analgesic to noxious stimulation applied as high as the first intercostal space. The haemodynamic effects of sympathetic denervation by extradural anaesthesia, to our knowledge, have not been studied previously in the conscious dog. Extradural blockade decreased mean arterial pressure by 11 and 16 mm Hg to 93 and 94 mm Hg in the warm and cold environments, respectively. These values are virtually identical to those observed in dogs after complete autonomic block with hexamethonium, and similar to the decrease of 16 mm Hg observed after extradural block with a sensory level of T1 in human volunteers [6]. Despite the decrease in arterial pressure, heart rate did not change significantly in our study, again similar to observations made in man [6]. The heart rate after denervation in our dogs was identical to the rate observed after complete adrenergic blockade by propranolol and phenoxybenzamine [20]. Arterial pressure was the same in the denervated state at both ambient temperatures. In the absence of efferent sympathetic baroreflex control, reflex cardiovascular influences on the skin circulation by afferent thermoreceptor activity [21] and arterial or low pressure baroreceptors are excluded [22].

Interpretation of results

We elected to study the effects of denervation on skin temperature at constant ambient temperatures both above and below the dog’s thermoneutral temperature of 25 °C [9]; that is, in a state where baseline efferent sympathetic tone can be assumed to be either increased (cold environment) or decreased (warm environment) [14,23]. In homeotherms, cooling usually elicits a marked decrease in blood flow, particularly in the peripheral parts of a limb [2, 13, 23]. Our data indicate that, in conscious dogs as in man [3, 16, 22], a vasoconstrictor tone is present in the limbs not only in a cold, but also in a warm environment. This conclusion is supported by the increase in limb skin temperatures under ambient temperatures both above and below the thermoneutral range.

The increase in skin temperature on the limbs is not unexpected, since several studies have reported an increase in blood flow after peripheral nerve block in man [1–3]. With extradural anaesthesia, blood flow to the upper extremity increases when a sensory level of T1 is reached [6], but decreases with a lower sensory level during both extradural [4,6] and spinal anaesthesia [8,16]. In contrast, almost no information is available with respect to regulation of trunk skin blood flow, primarily because there is no reliable method of measuring flow. Use of venous occlusion plethysmography is confined to the limbs and radiolabelled microspheres may yield potentially unreliable results when large arteriovenous anastomoses become patent in warmer environments [23]. It does not appear that the recent introduction of laser Doppler flowmetry will alter this situation [12,24].

During heat stress and active body warming, a significant vasoconstrictor control seems to be absent in man and an active vasodilator mechanism somehow coupled to sweat secretion prevails [18,25]. Little information is available about the role of the sympathetic nervous system in influencing trunk skin temperatures and blood flow. During exposure to ambient temperatures below the thermoneutral range, spinal anaesthesia with a sensory level to T10 has been reported to decrease skin temperature in the thoracoabdominal area and to decrease skin blood flow on the upper trunk to a variable degree [5,16]. No decrease in skin temperature has been found in another study, despite decreases in ambient temperature [12]. The former authors suggested that the decrease in trunk skin temperature could be explained by vasoconstriction of trunk skin vessels above, and potentially also within, the analgesic areas [16].
This is an attractive hypothesis because the sympathetic system seems to be poorly organized, with sympathetic efferents overlapping several dermatomes [26-28], and blood flow to the upper extremity has been reported to decrease during both extradural and spinal anaesthesia with a T4 sensory level [4,6,8] possibly as a result of baroreflex activation by the accompanying decrease in arterial pressure.

The findings of the present study, however, demonstrate that skin temperatures on the trunk can decrease above and even within the analgesic areas despite complete sympathetic block. Thus our results are incompatible with the hypothesis that a primary increase in sympathetic discharge is the dominant reason for the observed decrease in trunk skin temperature above and within blocked analgesic regions. Had there been significant vasoconstrictor tone present on the trunk in our experiments, we would have predicted an increased trunk skin temperature after sympathetic block, particularly in the cold environment. Instead, trunk skin temperature decreased despite constant ambient and rectal temperatures. Several mechanisms could explain this finding.

Marked muscular work can itself heat the overlying skin [29]. It is unlikely, however, that diminished muscle activity on the trunk after extradural block can explain the decrease in trunk skin temperatures. A similar decrease was observed not only in the cold but also in the warm environment, where the dogs did not shiver. Also, care was taken to place the thoracic temperature sensor directly over a rib with no underlying muscle.

Sympathetic block with an accompanying decrease in arterial pressure could have induced changes in the plasma concentrations of vasopressin or angiotensin. While we have not measured the concentrations of these substances, extradural anaesthesia per se is not associated with statistically significant changes in the plasma concentrations of renin, vasopressin or noradrenaline [30].

Direct effects of spinal cord cooling or of increases in the blood concentrations of local anaesthetic can be excluded as a reason for the observed changes in skin temperature, since neither the extradural injection of saline nor the i.m. administration of lignocaine produced any appreciable changes in skin temperature.

Finally, a redistribution of cardiac output towards the extremities, consistent with the observed increases in limb skin temperatures, could have passively decreased trunk skin blood flow and temperatures. We favour this explanation, because a similar decrease in trunk skin temperature was observed under conditions of both a presumably high and a low skin vasoconstrictor tone.

To the extent that our results can be extrapolated to the clinical situation, monitoring of trunk skin temperature does not appear to provide a reliable indication of the extent of sympathetic block on the trunk after central nerve block.

REFERENCES


