

Occurrence of the spinal reflex due to skin pressure in sudomotor and cutaneous vasoconstrictor nerve system of humans

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Abstract

The effects of skin pressure applied to one side of the waist on sudomotor and vasoconstrictor nerve activity were compared with the effects on sweating and cutaneous blood flow in humans. The sweat rate and cutaneous blood flow were measured on left and right dorsal feet. Skin sympathetic nerve activity (SSNA) was recorded by microneurography from a microelectrode inserted in left and right peroneal nerves. Skin pressure was applied in a supine position to the area over the left or right anterior superior iliac spine under warm (T_a : 30–36 °C) and cool (T_a : 19–23 °C) conditions. Sudomotor and vasoconstrictor bursts were identified for quantitative analysis. The skin pressure increased the contralateral/ipsilateral ratio of the sweat rate. It also increased the contralateral/ipsilateral ratio of the cutaneous blood flow and the contralateral/ipsilateral ratio of the sudomotor burst amplitude. However, skin pressure did not induce any significant changes in the contralateral/ipsilateral ratio of the vasoconstrictor burst amplitude. The results indicate that an asymmetrical reflex effect of skin pressure on vasoconstrictor nerve activity was absent, suggesting that, whereas the ipsilateral suppression of sweating elicited by skin pressure was mediated by the sudomotor nerve system, the ipsilateral suppression of cutaneous blood flow was not mediated by the vasoconstrictor nerve system. Thus, the occurrence of the spinal reflex due to skin pressure is not uniform between the sudomotor and the vasoconstrictor nerve systems, which represent different organizations at the level of spinal cord.

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1. Introduction

Somatic afferent stimulation may affect various autonomic functions. For example, heart rate increases reflexively when a thermal, mechanical or chemical stimulus is applied to the skin of anesthetized cats (Kaufman et al., 1977). Cutaneous vasodilatation occurs as a result of a reduction in vasoconstrictor nerve activity in response to various types of noxious stimuli in cats (Jänig, 1975; Jänig and Kümmel, 1981; Kümmel, 1983). Sweating in cat footpads is influenced by a noxious stimulation of the skin (Jänig and R ath, 1977). Cold shivering is inhibited by electric stimulation of the cutaneous nerve fibers (Kosaka et al., 1975). Gastrointestinal motility is inhibited by stimulating

the abdominal skin such as by pinching in rats (Kametani et al., 1979). Bladder contractions are enhanced by the stimulation of perineal skin when the bladder is quiescent, while rhythmic micturition contractions of the bladder are inhibited by the stimulation of perineal and chest skin when the bladder is expanded (Sato et al., 1977, 1983). Catecholamine secretion at the adrenal medullary gland is also influenced by stimulation of the skin (Araki et al., 1984).

The responses to skin pressure have been examined extensively in humans. It has long been known that skin pressure elicits a decrease in sweating, a fall in skin temperature, a reduction in the metabolic rate, a decrease in secretion from the parotid glands and an inhibition of gastric motility (Takagi and Kobayashi, 1955). In general, skin pressure applied to one side of the body produces an ipsilateral suppression and contralateral facilitation of the effector organ response (Takagi and Kobayashi, 1955).

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Eccrine sweating is pronounced in regard to the effect of skin pressure. Skin pressure applied to one side of the body inhibits sweating on the ipsilateral side with a strong segmental relation between the site of pressure and the area of sweat suppression (Takagi, 1960; Ogawa et al., 1981a). However, it is not clear whether skin pressure affects the cutaneous blood flow, although it is known, as described above, that skin pressure applied to one side of the body reduces skin temperature on the ipsilateral side (Takagi and Kobayashi, 1955; Shinoda, 1997). Thus, it is hypothesized that skin pressure causes an asymmetrical effect on cutaneous blood flow.

The purposes of the present study were (1) to examine quantitatively whether cutaneous blood flow is affected by skin pressure and, if so, (2) to elucidate the mechanism involved. Accordingly, we recorded sudomotor and vasoconstrictor nerve activity by microneurography simultaneously from left and right peroneal nerves and the sweat rate and cutaneous blood flow in the left and right dorsal foot areas supplied by these nerves. The asymmetrical effect on these parameters produced by skin pressure applied to one side of the waist was analyzed using the contralateral/ipsilateral ratio.

2. Methods

2.1. Subjects

Nine healthy male students, aged 21–24 years, were studied. Each subject was informed of the purpose and the protocol of the experiment and gave their consent to participate. The study was approved by the Human Research Committee of the Research Institute of Environmental Medicine, Nagoya University.

2.2. Recording of skin sympathetic nerve activity (SSNA)

A tungsten microelectrode with a tip of 1 μm in diameter and an impedance of 3–5 $\text{M}\Omega$ was inserted percutaneously into the skin fascicle of the peroneal nerve at the level of the popliteal fossa. SSNA was identified as neural activities with the following properties (cf. Sugeno et al., 1998): (1) spontaneous, irregular burst activities that are not synchronous with the cardiac beat, and are (2) evoked reflexively by mental stress, sensory stimuli or deep breathing, (3) followed at a constant latency of 2–3 s by a sweat response (sweat expulsion and/or electrodermal response) or by vasoconstriction (transient reduction of cutaneous blood flow). SSNA signals were amplified with a preamplifier ($\times 20,000$; Kohno Instrument, Kohno II, Nagoya, Japan), processed with band-pass filters (500–5000 Hz; E-3201A, NF Circuit Design, Yokohama, Japan) and displayed on a storage oscilloscope (5113, Tektronix, Beaverton, OR), with monitoring through a loudspeaker. The processed signals were then discriminated to improve the S/N ratio, full-rectified, integrated with a time

constant of 0.1 s and displayed as a mean voltage neurogram on a thermal chart recorder (Recti-Horiz, NEC-San-Ei, Tokyo, Japan). The SSNA signals were otherwise stored on a digital data-recorder (PC116, Sony, Tokyo, Japan). The stored data were later replayed as a mean voltage neurogram on a thermal array recorder (RTA-1200, Nihon Kohden Kogyo, Tokyo, Japan) at a paper speed of 0.25 cm s^{-1} .

2.3. Measurement of sweat rate

A sweat capsule with an area of 8 cm^2 was attached to the central part of the left and right dorsal feet. The capsule was ventilated with dry air at a flow rate of 1.5 l min^{-1} . The effluent air was sensed with a capacitance hygrometer (HMI-23, Vaisala, Helsinki, Finland) and the humidity change was recorded continuously on a pen-recorder (RS-66, Rikadenki, Tokyo, Japan) and stored on the data-recorder. The humidity change was calibrated to the sweat rate ($\text{mg cm}^{-2} \text{min}^{-1}$) by infusing a given amount of distilled water into the air stream. The sweat rate data, together with electrodermal activity and cutaneous blood flow data, were stored on a computer at a sampling frequency of 1 Hz.

2.4. Measurement of electrodermal activity

Skin potential activity was recorded from plate electrodes attached using conventional ECG paste to the skin of the middle part of the left and right dorsal feet. Skin potential was amplified using a bioelectric amplifier (AB-621G, Nihon Kohden Kogyo) with a time constant of 2 s. Skin potential data, monitoring with the thermal chart recorder, were stored on the data-recorder.

2.5. Measurement of cutaneous blood flow

A laser-Doppler flowmeter (LDF) was utilized (ALF-21, Advance, Tokyo, Japan) with a time constant of 0.1 s. LDF probes with glass fiber openings at 0.9 mm were attached using adhesive tape to the central part of the left and right dorsal feet and to the anterior eminence of the left and right soles. LDF signals were displayed on the thermal chart recorder and stored on the data-recorder.

2.6. Application of skin pressure

Pressure was applied to the skin over the anterior superior iliac spine by the examiner. It was applied vertically to avoid pressing the contralateral buttock. The application was made with an approximate strength of 5 kg using a specially designed stick made from vinyl chloride with an external diameter of 2.0 cm and a length of 40 cm, which has a pad of compressed cotton with an area of approximate 20 cm^2 at the end (0.25 kg cm^2).

The pressure was applied for 5 min in the majority of the trials and for 4 min in a few trials. As a general rule, skin pressure was applied to the right and the left side alter-

natively. Between the trials, there was a resting period of 5 min or more. One to six pressure applications were performed in an experiment.

2.7. General procedures

2.7.1. Warming experiment

The subject, wearing short pants and a short-sleeved cotton shirt, assumed a supine position on a bed in a noise-proof room, the ambient temperature (T_a) of which was controlled. The bed had a wooden frame and was covered with a soft sponge mattress about 5 cm thick with a rubber surface, providing a sufficient cushioning effect. At T_a of 26–28 °C (relative humidity 30–40%), sweat capsules, LDF probes and electrodes for recording electrodermal activities were attached. Then, a microelectrode was inserted into left and right peroneal nerves to record SSNA. After the baseline recording was made, T_a was increased slowly to 36–40 °C. The trial of pressure application to the skin was not made until the spontaneous occurrence of sympathetic bursts and effector responses was attained to be steady.

2.7.2. Cooling experiment

In this series, electrodermal activity and cutaneous blood flow were measured as effector responses: sweat rate was not measured. After the baseline recording, T_a was lowered to 19–22 °C. When shivering or piloerection appeared to arise, the cooling was discontinued. Thus, all experiments were conducted under conditions where shivering and piloerection were absent.

2.8. Data analysis

2.8.1. Effector responses

The effect of skin pressure were evaluated using contralateral/ipsilateral ratio, thus only asymmetrical effects were analyzed. We inferred that symmetrical effects are difficult to be evaluated, because thermoregulatory responses may fluctuate

spontaneously by the central mechanism. Thermal sweating, for example, may fluctuate at rest with the periods of 7–12 min (Takagi et al., 1966).

The sweat rate and blood flow data were averaged for every 10 s. The contralateral/ipsilateral ratio was then calculated from each 10-s value. The average of the ratio was compared between the period of pressure application and the control period before the application. The change in the contralateral/ipsilateral ratio due to skin pressure was expressed as a percentage by dividing the average value for the period of skin pressure by that for the control period; the significance of the change was tested using a non-paired *t*-test.

2.8.2. Identification and analysis of sudomotor and vasoconstrictor bursts

In multi-unit recordings, SSNA is composed of sudomotor, vasoconstrictor and pilomotor nerve activities (Bini et al., 1980). Assuming that the pilomotor activities were weak or absent, we identified sudomotor and vasoconstrictor bursts as follows (Bini et al., 1980; Sugeno et al., 1990, 1998): *sudomotor bursts* are those followed by skin potential responses at a latency of approximately 1.5 s and/or by sweat expulsions at a latency of approximately 2.5 s (Fig. 1); *vasoconstrictor bursts* are those followed by transient vasoconstrictions at a fixed latency of approximately 3–5 s (Fig. 1). Theoretically, vasoconstrictions should be determined on the dorsal foot, which is supplied by the peroneal nerve. However, there was a general tendency for vasoconstrictions to be less distinctive on the dorsal foot than on the sole, possibly because of counteraction from the concomitant vasodilatation. Accordingly, we determined the vasoconstrictions mainly on the sole, with reference to those on the dorsal foot (Fig. 1). Sympathetic bursts that were followed both by a sweat response and vasoconstriction (*mixed bursts*) were excluded from the analysis (e.g., bursts marked by SV* in Fig. 1).

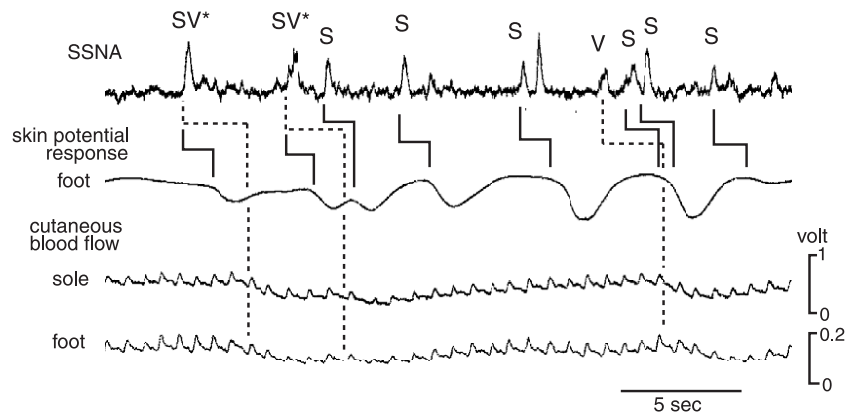


Fig. 1. Identification of sudomotor and vasoconstrictor bursts in SSNA. The solid and broken lines indicate the correspondence between the burst activity and effector response (sweating and vasoconstriction, respectively). S, sudomotor burst; V, vasoconstrictor burst; SV*, burst of mixed type (composed of sudomotor and vasoconstrictor nerve activities).

In the cooling experiments, *sudomotor bursts* were usually very rare so that they were not analyzed. In the cooling experiments, *vasoconstrictor bursts* were defined as synchronous sympathetic bursts that were not followed by skin potential responses.

Sudomotor and vasoconstrictor bursts having an amplitude smaller than 10% of the maximum were excluded from the analysis.

For quantifying the burst activity, the amplitude of the individual sudomotor and vasoconstrictor bursts was measured, and the contralateral/ipsilateral ratio was calculated. The average of the ratio was compared between the period of pressure application and the control period before the application. The change in the ratio due to the application of pressure was expressed as a percentage. The significance of the change was tested using a non-paired *t*-test.

2.8.3. Statistical analysis

In addition to the above-described non-paired *t*-test, a one-sample *t*-test was used when testing the average of the change in the contralateral/ipsilateral ratio for all trials. The level of significance was taken as 5%. Values are expressed as the mean \pm S.D.

3. Results

Twenty trials in 4 experiments and 14 trials in 4 experiments were performed in the warming and the cooling series, respectively. In 14 of 20 trials in the warming series and 10 of 14 trials in the cooling series, a complete set of data was available (Table 1). In the warming experiments (T_a : 30.4–35.9 °C), each trial gave 6–33 (21.1 ± 7.4) sudomotor bursts and 2–16 (8.4 ± 4.1) vasoconstrictor bursts, whereas in the cooling experiments (T_a : 19.2–23.0 °C), each trial gave 19–47 (26.3 ± 8.0) vasoconstrictor bursts (Table 1).

3.1. Sweat response

The skin pressure applied to one side of the waist reduced the sweat rate on the dorsal foot of the ipsilateral side (Fig. 2). The contralateral/ipsilateral ratio of the sweat rate was increased by skin pressure in all 14 trials of the warming experiments (Fig. 3). The increase was highly significant for each trial. The average of the increase for all trials ($40.8 \pm 43.6\%$) was also significant ($P=0.003$).

3.2. Cutaneous blood flow response

Both in the warming and in the cooling experiments, the effect of skin pressure on cutaneous blood flow was rather weak (cf. Fig. 2). The skin pressure increased the contralateral/ipsilateral ratio of the cutaneous blood flow in 13 out of 14 trials for the warming series (Fig. 3) and in 8 out of 10 trials for the cooling series (Fig. 4). The increase was

Table 1

The trials of pressure application and the number of bursts analyzed

Name of trial	Experiment	Subject	Pressure site	Number of bursts	Number of bursts
<i>Warming experiment</i>					
1R1	1	DT	R	13/16	7/7
1L1			L	8/6	6/7
2R1	2	YY	R	19/23	19/12
2L1			L	25/25	6/8
2R2			R	13/16	7/7
2R3			R	12/20	2/2*
3R1			3	NK	R
3L1	L	11/14			12/12
3R2	R	22/21			9/9
3R3	4	MH	R	34/33	15/16
4R1			R	18/20	7/4*
4L1			L	26/31	5/4*
4R2			R	32/30	15/15
4L2	L		25/25	8/8	
<i>Cooling experiment</i>					
5R1	5	KM	R	–	47/47
5L1			L	–	17/23
5R2			R	–	24/22
5L2			L	–	23/23
6R1	6	YM	R	–	26/30
7R1			7	MM	R
7L1	L	–			27/25
7L2			L	–	24/24
8R1			8	MH	R
8L1	L	–			27/29

The numbers left and right of the slash (/) indicate the numbers of bursts obtained before and during the pressure application, respectively. The three trials in the warming series were eliminated from the vasoconstrictor burst analysis because the burst was scant (*).

significant in eight trials of the warming experiments and in six trials of the cooling experiments. The change averaged $17.7 \pm 18.2\%$ ($P=0.003$) for the warming experiments and $17.2 \pm 15.9\%$ ($P=0.008$) for the cooling experiments, demonstrating an asymmetrical effect of skin pressure on cutaneous blood flow.

3.3. Sudomotor burst activity

In the warming experiments, sudomotor bursts were predominant over the vasoconstrictor or mixed bursts (Fig. 5A). The rate of sudomotor bursts was $7.5 \pm 2.9 \text{ min}^{-1}$ for the control period and $8.8 \pm 3.7 \text{ min}^{-1}$ for the period of pressure application, the difference not being significant ($P=0.317$).

Although the amplitude of sudomotor bursts varied greatly during the control period as well as the pressure application, it appeared that skin pressure reduced the amplitude of the bursts of the ipsilateral side (Fig. 5A). The contralateral/ipsilateral ratio of sudomotor burst amplitude was found to be increased by skin pressure (Fig. 3). The increase was significant in all 14 trials. The average of the change ($33.2 \pm 10.4\%$) was also significant ($P<0.001$).

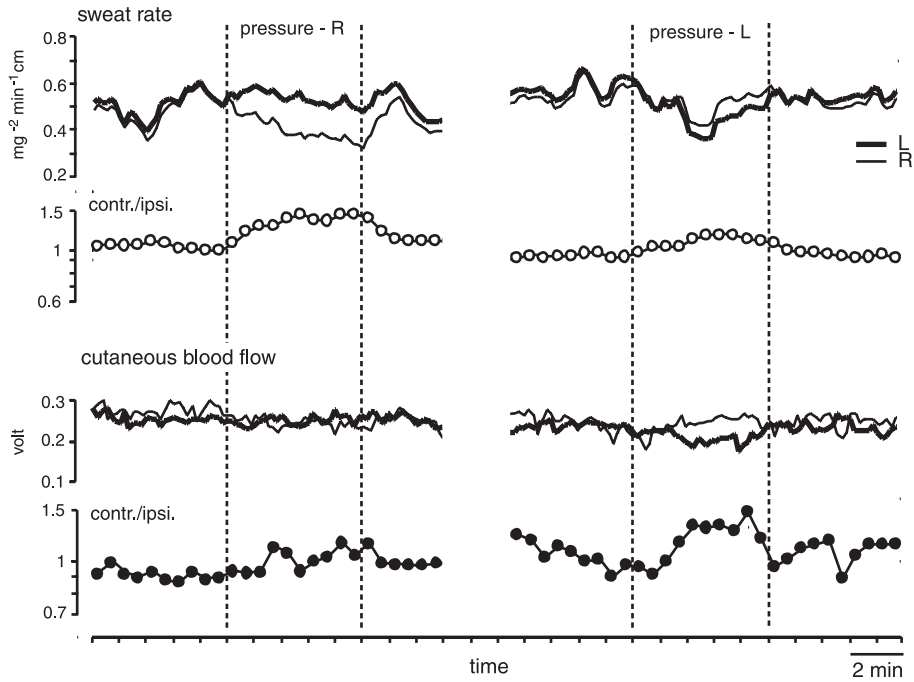


Fig. 2. Effects of skin pressure on sweating and cutaneous blood flow in representative trials of a warming experiment. Skin pressure to R and L sides appears to suppress ipsilaterally the sweating and the cutaneous blood flow on the dorsal foot, although its effect on cutaneous blood flow is rather weak. The contralateral/ipsilateral ratios are increased by the skin pressure both in the sweating and the cutaneous blood flow. Contr./ipsi., contralateral/ipsilateral ratio.

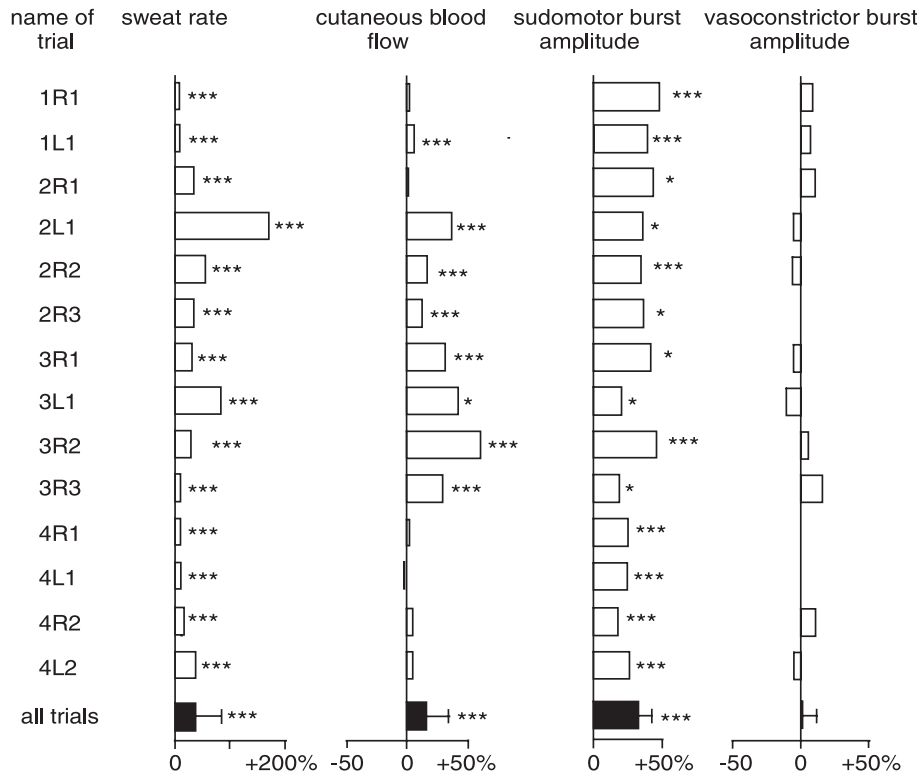


Fig. 3. The change in the contralateral/ipsilateral ratio of sweat rate, cutaneous blood flow, sudomotor burst amplitude and vasoconstrictor burst amplitude caused by skin pressure in the warming experiments. The ratios of the sweat rate, cutaneous blood flow and sudomotor burst amplitude were increased by skin pressure. No significant change was noted for the ratio of vasoconstrictor burst amplitude. In the trials 2R3, 4R1 and 4L1, vasoconstrictor burst amplitude was not analyzed. The names of trials are the same as those presented in Table 1. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.005$.

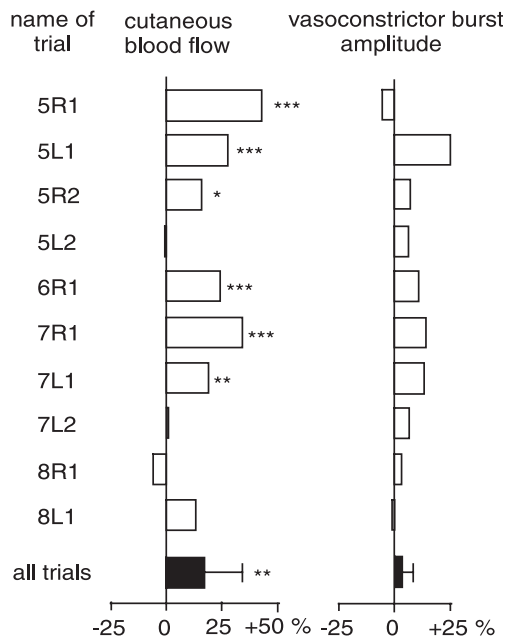


Fig. 4. The change in the contralateral/ipsilateral ratio of the cutaneous blood flow and the vasoconstrictor burst amplitude caused by skin pressure in the cooling experiments. The ratio of the cutaneous blood flow was increased by skin pressure. No significant change was noted for the ratio of the vasoconstrictor burst amplitude. The names of trials are the same as those presented in Table 1. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.005$.

3.4. Vasoconstrictor burst activity

In the warming experiments, pure vasoconstrictor bursts were scarce ($2.0 \pm 1.1 \text{ min}^{-1}$ during the control and $1.9 \pm 1.0 \text{ min}^{-1}$ during the pressure application, no significant difference; Fig. 5A). In the cooling experiments, on the other hand, vasoconstrictor bursts were predominant ($13.6 \pm 6.8 \text{ min}^{-1}$ during the control and $13.8 \pm 8.6 \text{ min}^{-1}$ during the pressure application, no significant difference; Fig. 5B) over the sudomotor bursts, so that a sufficient number of vasoconstrictor bursts were available (Table 1).

The skin pressure did not appear to alter the amplitude of vasoconstrictor bursts either on the ipsilateral or on the contralateral side (Fig. 5B). In the warming experiments, the contralateral/ipsilateral ratio of the burst amplitude was not changed by the application of skin pressure (Fig. 3). The skin pressure increased the ratio in 6 out of 11 trials and decreased it in 5 trials, but the magnitude of the change was small as compared with the sudomotor bursts, and no trials showed a significant difference. The average of the change ($2.4 \pm 9.9\%$) was not significant either ($P = 0.444$).

In the cooling experiments, the contralateral/ipsilateral ratio of the vasoconstrictor burst amplitude was also not changed by the skin pressure (Fig. 4). Although the ratio was increased in eight trials, the magnitude of the increase was small and so not significant in any trial. The average of the change for all cases ($8.2 \pm 8.8\%$) was also not significant ($P = 0.167$).

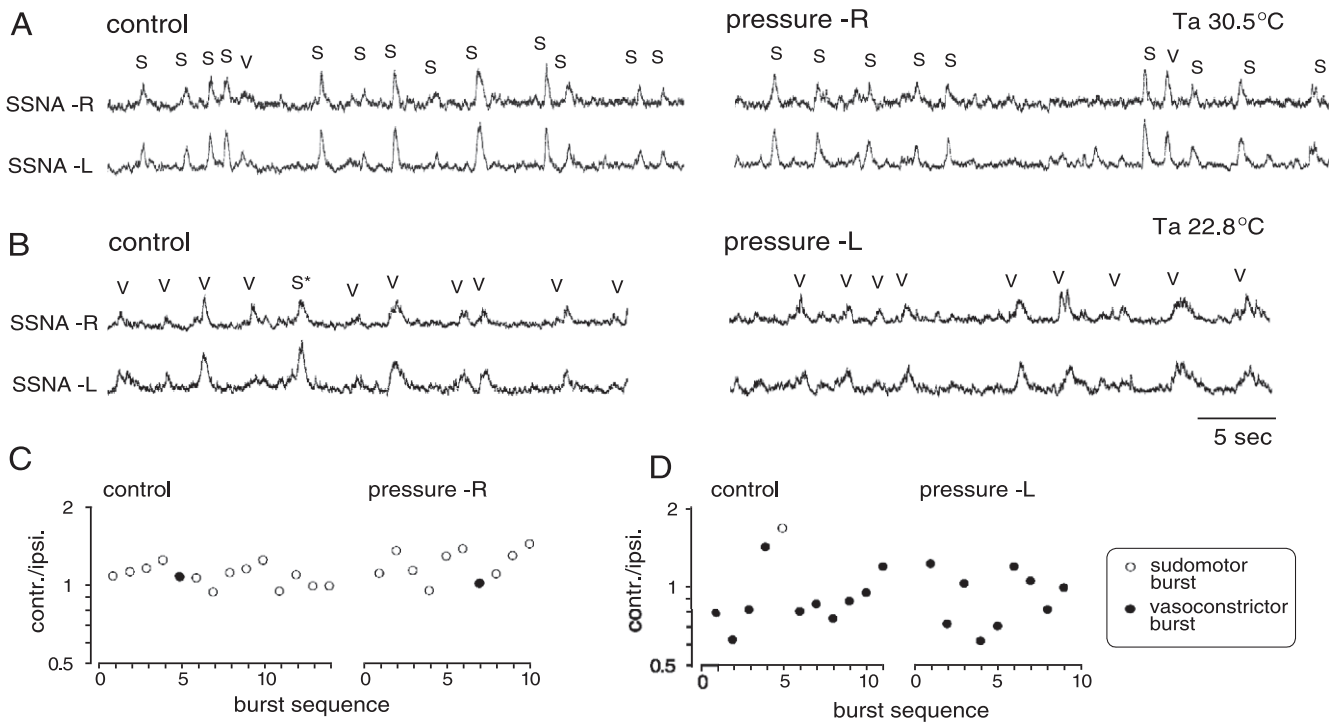


Fig. 5. Sudomotor and vasoconstrictor bursts during the control period and the period of skin pressure in representative trials of a warming (A) and a cooling (B) experiments. C and D are the contralateral/ipsilateral ratios calculated from the sudomotor and vasoconstrictor bursts presented in A and B, respectively. S and O, sudomotor burst; V and ●, vasoconstrictor burst. SSNA, skin sympathetic nerve activity.

4. Discussion

The main results of this study were that skin pressure applied to one side of the waist (1) increased the contralateral/ipsilateral ratio of the sweat rate on the dorsal foot; (2) increased the contralateral/ipsilateral ratio of the cutaneous blood flow on the dorsal foot; (3) increased the contralateral/ipsilateral ratio of the sudomotor burst amplitude in the peroneal nerve; but (4) did not affect the contralateral/ipsilateral ratio of the vasoconstrictor burst amplitude in the peroneal nerve. These results imply that the asymmetrical effect of skin pressure was absent in the vasoconstrictor nerve activity.

The present experiments were conducted in the supine position because the microneurographical procedure limits the posture of the subject. It is likely, therefore, that both left and right buttocks (and back) had been exposed to some extent to the skin pressure previous to the pressure stimulation. Thus, it seems reasonable to conclude that, in the present study, the pressure stimuli acted only as additional stimuli to the background pressure so that the effect might have been underestimated.

4.1. Effects on sudomotor functions

It has been known that skin pressure applied to one side of the body reduces sweating in certain areas on the ipsilateral side, which is referred to as hemihidrosis (Takagi and Sakurai, 1950; Takagi, 1960). The area in which the sweating is reduced is sharply bordered by the median line on the face and the trunk. Skin pressure is usually effective at any site on the body surface, although it is more effective in the axillary or the gluteal regions. The hemihidrotic effect is proportional to the pressure intensity per unit area and the surface area (Tadaki et al., 1981), thus establishing a quantitative relationship between the pressure stimulation and the hemihidrotic effect. Skin pressure applied to both left and right sides of the body reduces sweating on both sides. When pressure is applied to one side of axillary region, the sweating is reduced on the ipsilateral side of the upper body, including the face, chest and back, whereas when it is applied to the one side of the gluteal region, the sweating is reduced on the ipsilateral side of the lower body, including the hip and the leg.

As the pressure site is moved downward along the lateral line of the trunk, the extent to which the sweating is reduced descends from rostral to caudal (Ogawa et al., 1981a). Such a segmental nature of the response strongly suggests that the hemihidrosis is caused by the spinal reflex mechanisms and is not mediated by the central mechanism for thermoregulation (Ogawa et al., 1979). By the analysis of sweat expulsions, which reflect the postganglionic sudomotor burst activity (Sugenoya et al., 1990), it is found that the skin pressure, when applied to one side of the body, does not essentially influence the synchronous occurrence of sweat expulsions, but reduces the height of sweat expulsions on

the body side ipsilateral to the pressure application (Ogawa and Bullard, 1972). Thus, it is postulated that the sudomotor outflow descending from central thermoregulatory mechanisms is modified at the level of the spinal cord by afferent signals generated by skin pressure.

It is generally believed that the hemihidrotic reflex is mediated by the afferent fibers belonging to group II or III neurons (Takagi, 1960) and that these afferent fibers connect via interneurons (Petras and Cummings, 1972), with an inhibitory effect, to ipsilateral preganglionic sudomotor neurons, the cell bodies of which are located largely in the intermediolateral nucleus. This presumption is supported by the present observation suggesting that skin pressure reduced ipsilaterally the sudomotor burst amplitude. However, the neuronal circuits involved in the autonomic spinal reflex, including the functions of interneurons, have not been clarified (cf. Jänig, 1996).

Skin pressure often produces a facilitation of sweating outside the areas of sweat suppression, particularly in symmetrical areas (Ogawa et al., 1981b). Such a sweat facilitation on the contralateral side is presumed to be caused by the reciprocal innervation of preganglionic sudomotor neurons in the spinal cord or by a compensatory mechanism mediated by the central thermoregulatory mechanism or both. This implies that the increase in the contralateral/ipsilateral ratio of the sweat rate due to skin pressure may be comprised of both the ipsilateral decrease and the contralateral increase. Based on previous studies (Ogawa et al., 1981a), however, it is reasonable to assume that the increase in the ratio was accomplished predominantly by the ipsilateral decrease and only partly by the contralateral increase.

The present results indicate that the contralateral/ipsilateral ratio of the sweat rate and the sudomotor burst activity increased identically (Fig. 3), thus leading to the conclusion that the asymmetrical change in the sweat rate due to skin pressure is mediated by the sudomotor nerve system. An early microneurographic study also concluded that skin pressure reduced ipsilaterally the amplitude of sudomotor bursts (Sugiyama et al., 1992).

4.2. Effects on vasoconstrictor functions

Whether skin pressure affects the cutaneous blood flow has not been established in humans. Takagi (1960) observed considerable inter-individual variation in regard to the effect of skin pressure on skin temperature. He also recorded plethysmograms from the hands and feet, and concluded that the effect of skin pressure on cutaneous blood flow is variable. Shinoda (1997) investigated, using thermography, the responses of skin temperature distribution over the anterior aspect of the whole body surface to electric acupuncture stimulation (a stimulation equivalent to skin pressure; Ogawa et al., 1981b) applied to various sites along the lateral chest line. The results demonstrated, under cool conditions where no thermal sweating

occurred, that electric acupuncture stimulation (e.g., skin pressure) reduced skin temperature in a localized area on the ipsilateral side, with a distinct segmental property. This observation suggests that skin pressure reduces cutaneous blood flow ipsilaterally. The present study indicated using a laser-Doppler method that skin pressure increased the contralateral/ipsilateral ratio of the cutaneous blood flow (Figs. 3 and 4). This confirms the effect of skin pressure on cutaneous blood flow, although whether the skin pressure reduced the blood flow on the ipsilateral side or facilitated that on the contralateral side is not clear.

Despite the increase of the contralateral/ipsilateral ratio of cutaneous blood flow, the contralateral/ipsilateral ratio of the vasoconstrictor burst amplitude was not affected by the skin pressure. This discrepancy suggests that the effect of skin pressure on cutaneous blood flow was not mediated by vasoconstrictor nerves. Alternative explanation is that the ipsilateral reduction of cutaneous blood flow was caused by the ipsilateral reduction of active vasodilatation. However, what nerves mediate the reduction is the subject of consideration.

Kellogg et al. (1995) have demonstrated that cholinergic nerves mediate cutaneous active vasodilatation through the release of an unknown co-transmitter, although it is not determined whether these cholinergic nerves are sudomotor nerves. Accordingly, it is likely that the ipsilateral reduction of cutaneous blood flow was mediated by such cholinergic nerves. The ipsilateral reduction of cutaneous blood flow was observed in the cool environments as well as in the warm environments. This indicates that the cholinergic nerves, if involved, should be activated in such cool environments. It is known that sudomotor nerves are activated in thermoneutral or cool environments (Ogawa and Bullard, 1972).

The cutaneous vasodilator nerve system would be another candidate for a nerve system that could mediate the skin pressure-induced change of cutaneous blood flow. Vasodilator nerve system has not been identified in humans, but is believed to exist based on indirect evidence (Noll et al., 1994; Pèrgola and Kellogg, 1994; Wallin et al., 1998; cf. Jänig and Häbler, 1999; Johnson and Proppe, 1996). However, a vasodilator nerve-mediated mechanism is less probable at least in the present study because it is unlikely that cutaneous vasodilator nerves are activated in such a cool environment as employed in the present study.

The present study indicated that the contralateral/ipsilateral ratio of the vasoconstrictor burst amplitude was not affected by skin pressure. However, this does not eliminate the possibility that the skin pressure applied to one side of the waist elicited a symmetrical effect on the cutaneous blood flow. It is noted that in some subjects, skin pressure applied to one side of the body, specifically when it is strong, may elicit decreases in the sweating rate on left and right sides (unpublished data). In the present analysis,

however, it cannot be determined whether such changes were elicited.

4.3. Somatosympathetic reflex

The mechanisms behind the sympathetic reflexes evoked by somatosensory stimuli have been investigated extensively in intact and spinal animals. The somatosympathetic reflex consists of two components: an early spinal component and a late supraspinal (medullary) component (Sato and Schmidt, 1971, 1973). The groups II, III and IV neurons form an afferent pathway of the somatosympathetic reflex. The spinal reflex is characterized by segmental responses, whereas the supraspinal reflex is characterized by generalized responses. As described earlier, the hemihidrosis in humans is typical of the spinal reflex. However, as is demonstrated in the present study, a spinal reflex that exhibits an asymmetrical effect is not inevitable for all the sympathetic functions.

Various types of somatosensory stimuli elicit sudomotor responses in spinal animals (Ito et al., 1978; Jänig and Kümmel, 1981; Kümmel, 1983) as well as in brain-intact animals (Karl et al., 1975; Jänig and Räth, 1977; Kümmel, 1983). Numerous animal experiments have demonstrated that the same type of cutaneous stimulus, either noxious or innocuous, tends to elicit a spinal reflex both in the sudomotor and vasoconstrictor systems (Ito et al., 1978; Jänig, 1975; Jänig and Kümmel, 1981; Jänig and Räth, 1977; Karl et al., 1975; Kümmel, 1983). Nevertheless, no definitive evidence has emerged from these animal experiments as to whether the asymmetrical reflex is elicited in the sudomotor nerve system (governing plantar sweating in animals) or in the vasoconstrictor nerve system.

It is not possible with the present data to conclude why and how skin pressure elicits an asymmetrical reflex effect in the sudomotor system but not in the cutaneous vasoconstrictor system in humans. Certainly, this difference in reflex response would reflect a difference in the organization at the spinal cord level between the sudomotor and vasoconstrictor systems. In addition, the difference in the organization may be associated with the function of each sympathetic control system.

Regardless of the mechanism involved, the ipsilateral suppression of thermal sweating elicited by skin pressure may be advantageous for saving water by reducing sweat loss from regions near the pressed site where evaporation is less effective. It is still unclear whether the suppression of cutaneous blood flow on the pressed side has some physiological roles, although it may contribute to reducing heat transfer from the object through the skin under very hot conditions.

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