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REGIONAL SIMILARITIES AND DIFFERENCES IN THERMOREGULATORY VASO- AND SUDOMOTOR TONE

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SUMMARY

1. Skin nerve sympathetic activity was recorded simultaneously from the following pairs of nerves: left and right median, median and peroneal, left and right peroneal, posterior cutaneous antebrachial and superficial radial, posterior cutaneous antebrachial and median. The recordings were performed on healthy subjects exposed to different ambient temperatures. Electrodermal responses and pulse plethysmograms were recorded from the neural innervation zones.

2. Vasoconstrictor impulse bursts recorded simultaneously from the *median* and *peroneal* nerves during exposure to a cold environment showed a striking similarity with respect to the timing and strength of individual bursts. A similar strong correlation was observed also among sudomotor bursts recorded simultaneously from the *posterior cutaneous antebrachial* and *superficial radial* nerve during exposure to a warm environment.

3. On some occasions, such as during exposure to a moderately warm environment or emotional stress, a temporal correlation was also observed between vasoconstrictor bursts recorded from the *median* and sudomotor bursts recorded simultaneously from the *posterior cutaneous antebrachial* nerve.

4. The double nerve recordings provided evidence that in the distal glabrous skin areas reflex thermoregulatory functions are mainly executed via vasoconstrictor fibres whereas sudomotor fibres are brought into action only at relatively high temperature. On the contrary, in the hairy skin on the dorsal side of forearm and hand reflex thermoregulation is to a large extent executed via sudomotor fibres.

INTRODUCTION

When sympathetic muscle nerve activity (m.s.a.) is simultaneously recorded from two muscle nerves in different extremities the two neurograms show a striking similarity both with respect to the timing and the mean voltage amplitude of individual sympathetic bursts (Sundlöf & Wallin, 1977). On the other hand, in simultaneous recordings of sympathetic muscle nerve activity and sympathetic skin nerve activity (s.s.a.) no similarities have been noted either with respect to the timing, or the amplitude of the sympathetic bursts (Wallin, Delius & Hagbarth, 1973, 1974). This has been taken as evidence that the sympathetic ganglia contain at least two different neuronal populations exposed to separate preganglionic drives.

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One aim of the present study was to make simultaneous recordings from different skin nerves in man to see whether the skin nerve impulse bursts destined for different skin areas are synchronously timed. Another aim with the simultaneous recordings was to see whether there are regional differences as regards the extent to which the sudomotor and vasoconstrictor systems participate in thermo-regulatory functions. This latter aim was prompted by earlier indirect evidence that the vasomotor and sudomotor outflows to different skin areas are not equally involved in the maintenance of thermostasis (Hertzman, 1959; McCook, Wurster & Randall, 1965).

METHODS

Material

The healthy subjects volunteering for the investigations were eight males and three females, ranging in age from 21 to 32 years.

In eleven experiments skin nerve sympathetic activity was recorded simultaneously from two different extremity nerves. The pairs of nerves chosen for such simultaneous micro-electrode recordings were: the two median nerves (one experiment); the two peroneal nerves (two experiments); the median and the peroneal nerve (four experiments), the median and the posterior cutaneous antibrachial nerve (three experiments); the superficial radial and the posterior cutaneous antibrachial nerve (one experiment). The median nerve recordings were made at elbow level from nerve fascicles supplying palmar and digital skin fields whereas the peroneal recordings were made at knee level from fascicles supplying the skin on the dorso-lateral side of the foot. For the posterior cutaneous antibrachial nerve the recording site was 5–10 cm proximal to the elbow and the fascicles impaled had innervation zones on the dorsal side of the forearm. The superficial radial nerve was impaled a few cm proximal to the wrist and the fascicular innervation zones were on the dorsal side of the hand.

Technique

Detailed accounts of the recording and display systems have already been given (Bini, Hagbarth, Hynninen & Wallin, 1980). The tungsten electrodes, amplifiers, filters and integrating circuits used for the two-channel nerve recordings were duplicates of the equipment previously described. Once a stable recording site of sympathetic skin nerve activity had been found in one of the nerves, the search for another recording site in the second nerve began. The intrafascicular recording sites accepted were those from which it was possible to record either spontaneous or reflexly evoked bursts of neural impulses succeeded by transient plethysmographic or electrodermal responses in the innervation zone of the impaled fascicle. Attempts were made to adjust the positioning of the electrodes so that optimal signal-to-noise ratios were obtained in both recordings. Care was taken to ascertain that neither in the input stages nor in the tape recorder there was any significant 'cross-talk' between the two micro-electrode recording channels. Since a previous study dealt with the correlation between the neural sympathetic activity and the accompanying electrodermal and plethysmographic events (Bini *et al.* 1980) only the mean voltage ('integrated') neurograms are shown in the present paper (with omission of electrodermal and plethysmographic recordings). The double nerve recording sites could often be maintained for several hours. Unless otherwise stated the subject was resting in a comfortable position while the *environmental temperature* was changed in the ways previously described (Bini *et al.* 1980). When the subject was lying inside the hypothermia box the arm on which the recordings were made passed through an opening in the box and was (from the axillary region) exposed to a constant room temperature of 22–24 °C. As in the previous study *arousal stimuli* were elicited by sudden noises, taps or electrical skin stimuli on a free extremity, *mental stress* was obtained by asking the subject to solve arithmetical problems and *muscle work* was performed as strong isometric contractions of muscles in a free extremity.

Quantitative estimations of long-term changes in skin nerve activity were made by first feeding the mean voltage neurograms through a voltage-to-frequency converter (Tektronix Function Generator – FG 502) and then counting the number of standard pulses per minute. The strength of individual bursts was evaluated by manual measurement of the amplitude of the burst deflections in the mean voltage neurograms as displayed by the inkwriter.

RESULTS

Synchrony of vasoconstrictor bursts in different skin nerves

There was a striking similarity between the two integrated neurograms in all those experiments in which spontaneous skin nerve sympathetic activity was simultaneously led off from the *two median*, the *two peroneal* or the *median and peroneal nerves*. Fig. 1 *A* and *B* shows examples of such double nerve recordings of sympathetic bursts occurring spontaneously in resting subjects at an environmental temperature

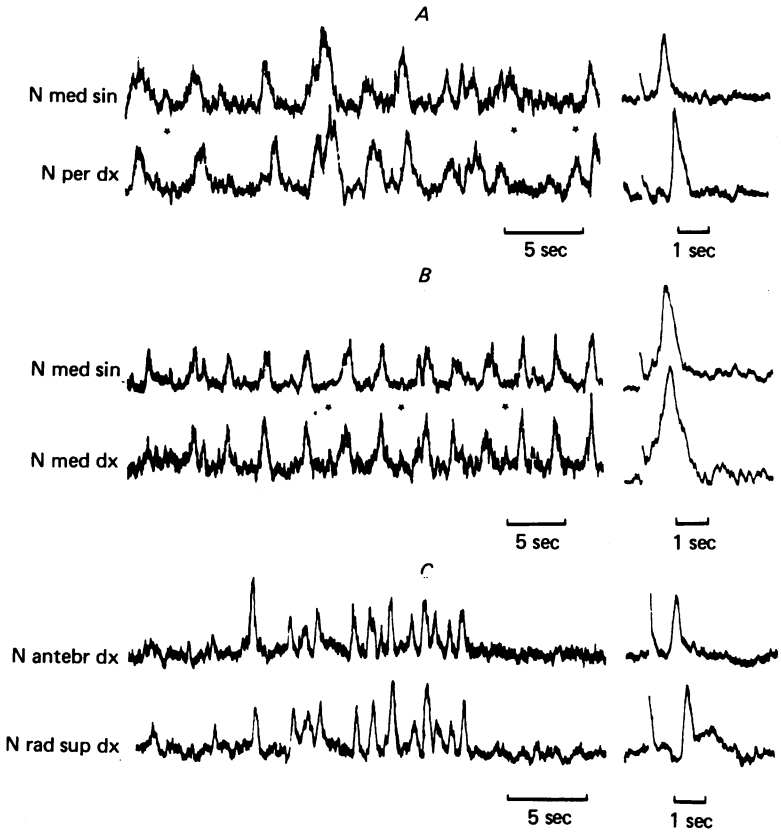

Fig. 1. Synchrony of sympathetic bursts in different skin nerves. *A* and *B*, mean voltage neurograms showing bursts of skin vasoconstrictor impulses simultaneously led off from the left median and the right peroneal nerve (*A*) and from the two median nerves (*B*). *Left*, bursts occurring 'spontaneously' in a resting subject at an ambient temperature of 18 °C. *Right*, reflex burst elicited by electrical skin stimulus. Asterisks indicate small bursts that were seen in one nerve only. *C*, bursts of sudomotor impulses simultaneously led off from the left antebrachial and the left superficial radial nerve. *Left*, bursts occurring during muscle work performed at an ambient temperature of about 28 °C. *Right*, reflex elicited by electrical skin stimulus. Note latency difference of about 0·3 sec between bursts in median and peroneal nerve (*A*) and latency difference of about 0·4 sec between bursts in antebrachial and superficial radial nerve (*C*): in the latter case the conduction distance between elbow and wrist recording sites was 38 cm, corresponding to a peripheral conduction velocity of about 1 m/sec. Note also shorter duration of sudomotor (*C*) as compared to vasoconstrictor bursts (*A*, *B*).

of about 18 °C. During these recordings the finger and toe plethysmograms (not illustrated) showed signs of relatively strong vasoconstrictor tone. The large majority of the bursts occurred synchronously in the two nerves and also the relative strength of individual bursts (converted to an amplitude parameter in the mean voltage neurograms) varied in parallel. In Fig. 2 the amplitude of the bursts led off from the median nerve is plotted against the amplitude of corresponding bursts led off from

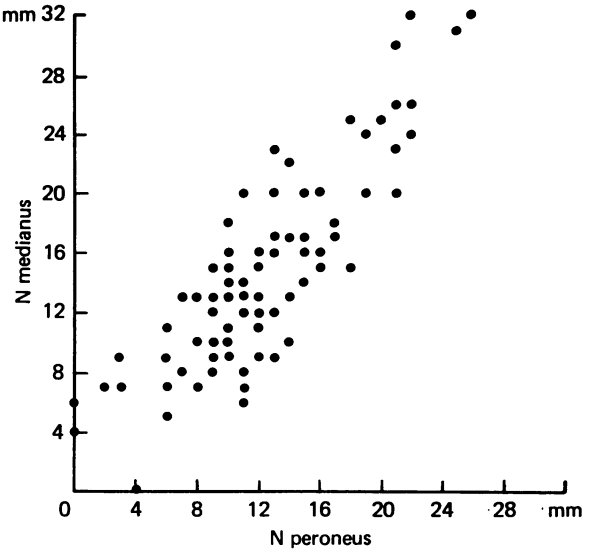

Fig. 2. Quantitative relationship between amplitude of corresponding vasoconstrictor bursts recorded from median and peroneal nerve at an ambient temperature of 18 °C. Correlation coefficient for linear regression, $r = 0{\cdot}83$. Slope of line significantly different from zero, $P < 0{\cdot}01$. Data derived from 75 consecutive bursts in a recording like that illustrated in Fig. 1*A*, left.

the peroneal nerve. Even though there were a few minor bursts which were seen in one nerve only (indicated by asterisks in Fig. 1*A*, *B*) there was an obvious linear relationship between the relative amplitudes of individual bursts in the two nerves.

For both the spontaneously occurring bursts and bursts appearing in response to sudden arousal stimuli, there was a time lag of about 0·3 sec between the bursts led off from the median and peroneal nerves. As illustrated by the records to the right in Fig. 1*A* and *B* the latency of the reflex bursts in the median nerves was about 0·6 sec whereas the corresponding value in the peroneal recordings was about 0·9 sec.

The predominant vasoconstrictor origin of the spontaneous sympathetic skin nerve activity in recordings like those exemplified in Fig. 1*A* and *B* was evidenced by the fact that a moderate rise in environmental temperature in all subjects caused a marked reduction of the neural activity accompanied by plethysmographic signs of vasodilatation. The temperature-induced changes in vasoconstrictor outflow occurred in parallel in all double recordings involving the median and peroneal nerves. Fig. 3 illustrates the simultaneous changes occurring in the vasoconstrictor outflow in the two median nerves when the environmental temperature was first raised to 30 °C and then rapidly lowered to 23 °C. There is a marked parallelism between the two traces

in the diagram to the left, giving quantitative values for the long-term variations of the skin nerve activity in the two nerves. In both nerves it decreased during warming and increased during cooling. The traces to the right show excerpts from the two simultaneous recordings at three different stages of environmental temperature.

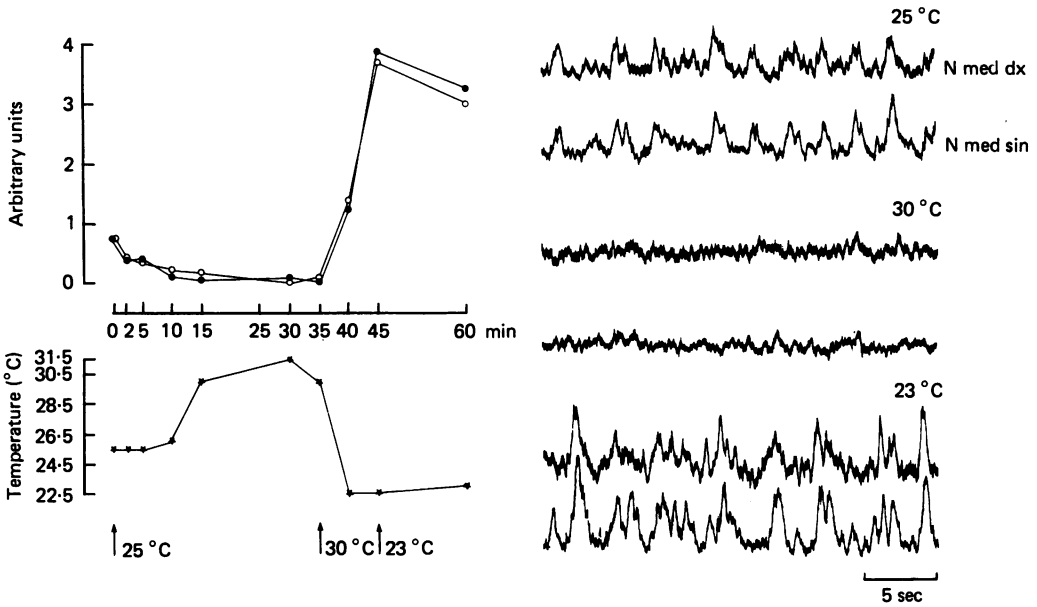

Fig. 3. Variations in ambient temperature producing parallel changes in vasoconstrictor activity simultaneously recorded from the two median nerves. *Left*, upper two curves showing long term variations in vasoconstrictor burst activity in left (○) and right (●) median nerves in response to the gradual changes in ambient temperature shown by the lower trace. See Methods regarding the technique used to estimate the total amount of sympathetic activity per minute. *Right*, short excerpts from the mean voltage neurograms which provided the data for the diagrams. The excerpts derive from those periods when the ambient temperature was 25, 30 and 23 °C (as indicated by arrows below diagram).

An enhancement of the median and peroneal nerve activity similar to that induced by cooling could also be induced by manoeuvres inducing mental stress or emotional excitement. During such manoeuvres there was also a close correlation between the timing and strength of individual bursts in the median–median and median–peroneal paired recordings.

Synchrony of sudomotor bursts in different skin nerves

One double recording was made from the superficial radial nerve and the posterior cutaneous antebrachial nerve. As previously noted (Bini *et al.* 1980) there are signs of regional differences in thermoregulatory function between the posterior antebrachial nerve and the median or peroneal nerves. In contrast to the median and peroneal nerves it is, for example, unusual to find spontaneous sympathetic activity at normal room temperature in the antebracial nerve. Repetitive sympathetic bursts are produced, however, by strong isometric muscle contractions or a rise in room tem-

perature. In the double recording the findings were similar in the superficial radial and the antebrachial nerves. There was no spontaneous activity but bursts appeared in both nerves during muscle work and increased room temperature. The bursts were time locked in the two nerves, with the bursts in the antebrachial nerve (led off from the elbow level) preceding those in the superficial radial nerve (led off from the wrist level) by about 0·4 sec (Fig. 1*C*, left). A similar difference in latency, corresponding to a peripheral conduction velocity of about 1 m/sec was found also for single bursts which could be elicited in both nerves by sudden arousal stimuli (Fig. 1*C*, right). As shown in Fig. 1*C* there was a close correlation not only with respect to the timing of individual bursts in the two nerves but also with respect to the relative amplitudes of concurrent bursts.

That the bursts led off from these nerves contained sudomotor impulses was evidenced by the fact that individual bursts were succeeded by transient reductions of electrodermal resistance in the neural innervation zones. At high environmental temperatures there was also plethysmographic signs of sustained vasodilatation in the forearm skin, but there were no transient plethysmographic responses following individual bursts. As previously shown (Bini *et al.* 1980) sudomotor bursts have a shorter mean duration than vasoconstrictor bursts and a comparison of the records in Fig. 1*A–C* reveals that the bursts in *C* have a shorter mean duration than those in *A* and *B*.

In most subjects emotional excitement or mental stress also produced repetitive sympathetic bursts in the posterior antebrachial and superficial radial nerves. Like the bursts appearing during muscle work and during rising environmental temperature these bursts were accompanied by transient electrodermal responses but no transient plethysmographic responses.

Regional differences in thermoregulatory vaso- and sudomotor tone

As mentioned above there were signs of regional differences in the thermoregulatory vaso- and sudomotor tone between different skin nerves. This was investigated more thoroughly in simultaneous recordings from the *posterior antebrachial* and *median nerves* while exposing the subject to more extreme *variations in external temperature* (in the hypothermia box). During slow increases in box temperature (up to 45 °C) similar results were obtained in all three experiments (Fig. 4). At normal room temperature (and up to about 27 °C) little or no sympathetic activity could be discerned in the posterior antebrachial nerve, but at 35–40 °C repetitive bursts appeared (accompanied by electrodermal responses), which increased in frequency and strength as profuse sweating started between 40 and 45 °C. The accompanying sympathetic events in the median nerve were the following: the spontaneous repetitive bursts, present at room temperature, diminished during moderate warming to about 30–35 °C (while the finger plethysmogram showed signs of vasodilatation) but with a further rise in temperature an increasing number of bursts appeared, which like the bursts in the posterior antebrachial nerve were accompanied by electrodermal responses. That the latter type of bursts were mainly composed of sudomotor impulses was evidenced not only by accompanying electrodermal responses but also by the fact that they had a smaller temporal dispersion than the initial vasocon-

strictor bursts (c.f. median nerve neurograms at 27 and 43 °C in Fig. 4). In all three subjects the sympathetic nerve activity rose during profuse sweating to higher quantitative values in the posterior antebrachial than in the median nerve (see diagram in Fig. 4).

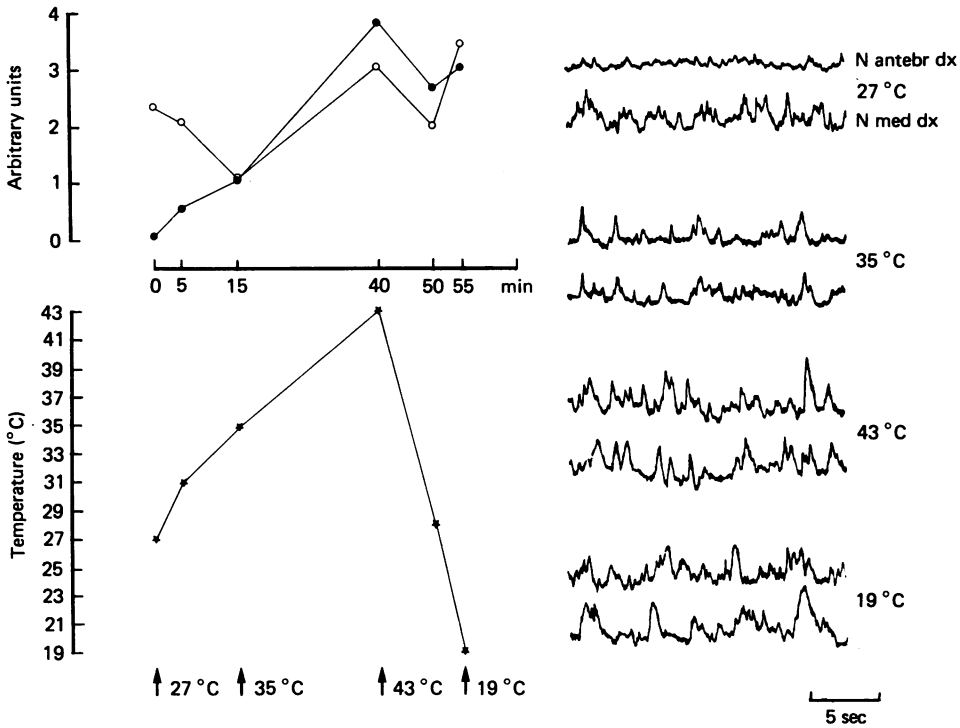

Fig. 4. Variations in ambient temperature with differential effects on vasoconstrictor activity in median and on sudomotor activity in antebrachial nerves (impulses destined to reach glabrous and non-glabrous skin respectively). *Left*, upper two traces showing long term variations in sympathetic activity in right antebrachial and right median nerve in response to the gradual changes in ambient temperature shown by the lower trace. *Right*, short excerpts from the mean voltage neurograms providing the data for the diagrams. The excerpts derive from those periods when ambient temperature was 27, 35, 43 and 19 °C (as indicated by arrows below diagram). As judged by simultaneous skin resistance and plethysmographic recordings the activity increasing in the two nerves during warming was mainly of sudomotor origin, whereas the activity subsiding in the median nerve in the early part of the warming session was mainly composed of vasoconstrictor impulses.

During the rapid fall in temperature (from 45 to 15 °C) there was in all subjects an initial reduction of the SSA in both nerves. As the temperature fell below 25–30 °C the median nerve activity increased in strength and the finger plethysmogram showed signs of increasing vasoconstriction. In the experiment illustrated in Fig. 4 there was, at the end of the cooling period, also an increase in this activity led off from the posterior antebrachial nerve, accompanied by plethysmographic signs of vasoconstriction in the forearm skin. In the other experiments, there were plethysmographic but no obvious neural signs of increased vasoconstrictor outflow to this skin region.

In general, however, neurograms obtained at the end of the cooling session were difficult to interpret since they often contained movement artefacts caused by shivering.

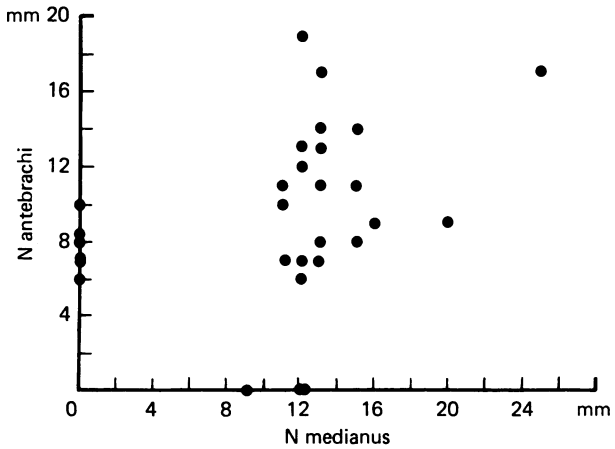

Fig. 5. Quantitative relationship between amplitude of corresponding bursts recorded from antebrachial (ordinate) and median nerve (abscissa) at an ambient temperature of 43 °C. Correlation coefficient for linear regression, $r = 0{\cdot}33$. Slope different from zero, significance $P < 0{\cdot}1$. Data derived from thirty consecutive bursts like those illustrated in Fig. 4, right (43 °C).

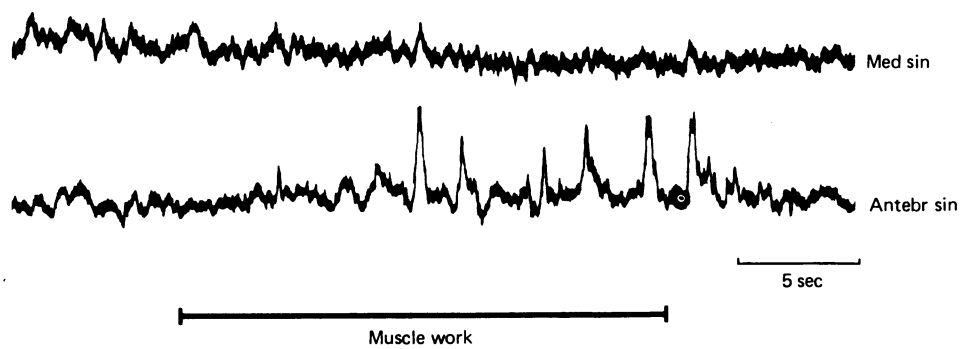

Fig. 6. Muscle work (a Jendrassik-like manoeuvre performed during the horizontal bar at an ambient temperature of 22–24 °C) producing differential effects on vasoconstrictor activity in the median and on sudomotor activity in the antebrachial nerve. As judged by simultaneous skin resistance and plethysmographic recordings the declining activity in the median nerve (upper) was mainly composed of vasoconstrictor impulses destined to distal glabrous skin areas, whereas the increasing activity in the antebrachial nerve (lower) was mainly composed of sudomotor impulses destined to reach the non-glabrous skin of the forearm.

During those periods when repetitive sympathetic bursts were present both in the median and the posterior antebrachial nerves many of the bursts occurred synchronously in the two nerves, but a number of bursts were seen in one nerve only and the amplitude correlation between synchronous bursts in the two neurograms was less pronounced than for the median–median and the median–peroneal nerve pairs (cf. Figs. 3, 4 and diagrams in Figs. 2 and 5).

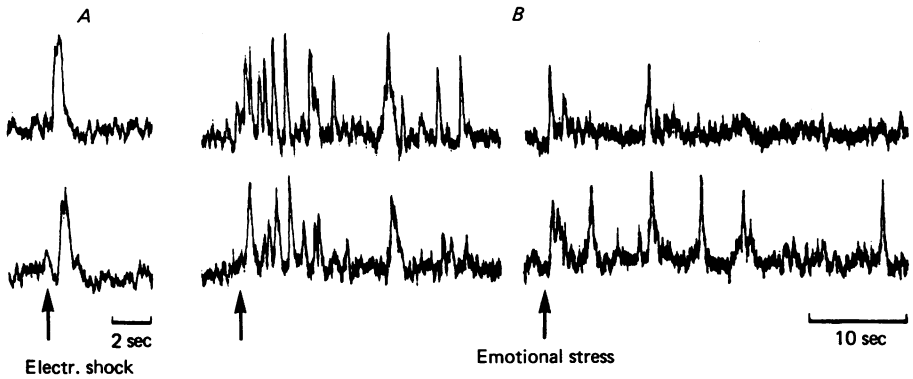

Fig. 7. Mean voltage neurograms showing bursts of sympathetic impulses simultaneously led off from left antebrachial (upper) and left median nerve (lower) at an ambient temperature of 22–24 °C. *A*, reflex burst triggered by electric shock (on right arm). *B*, two examples of recurrent bursts appearing when subject is asked to solve an arithmetical problem and the moment the mental tasks are given. As judged by previous electrodermal and plethysmographic recordings in similar situations the bursts in the median nerve (destined to reach glabrous skin areas) were composed of a mixture of sudomotor and vasoconstrictor impulses, whereas the bursts in the antebrachial nerve (destined to reach the forearm skin) were composed mainly of sudomotor impulses (cf. Bini *et al.* 1980).

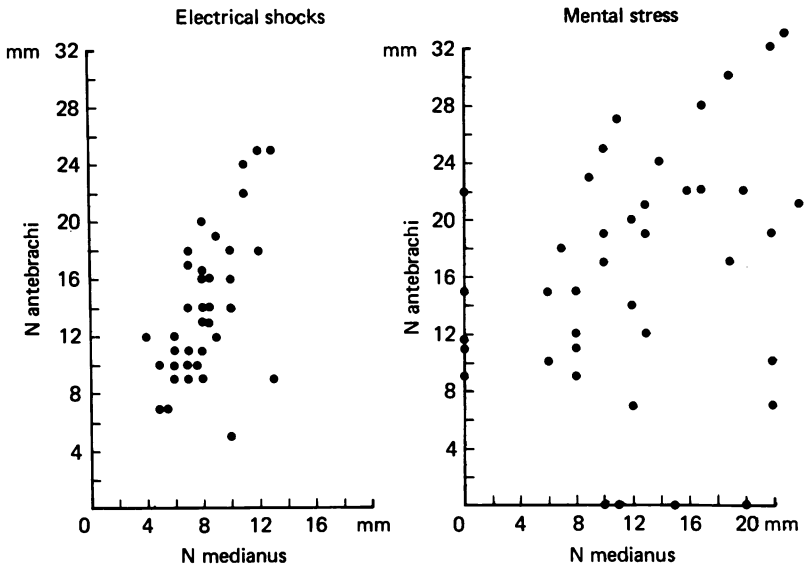

Fig. 8. Left, quantitative relationship between amplitude of corresponding reflex bursts in the antebrachial (ordinate) and median nerve (abscissa) in response to thirty-seven randomly applied electric shocks (single example in Fig. 7*A*). Correlation coefficient for linear regression, $r = 0{\cdot}59$. Significance for slope different from zero, $P < 0{\cdot}01$. Right, quantitative relation between amplitude of corresponding bursts in the two nerves occurring during recurrent periods of mental stress like those illustrated in Fig. 7*B*. Correlation coefficient for linear regression, $r = 0{\cdot}27$. Significance for slope different from zero, $P < 0{\cdot}1$.

An increase in the skin nerve activity led off from the posterior antebrachial nerve accompanied by a decrease of the median nerve activity could be produced not only by a moderate rise in external temperature but also by *muscle work* (Fig. 6). As shown in the previous study, however, the effect of muscle work was highly dependent on the environmental temperature and the preexisting sudo- and vasomotor tone (Bini *et al.* 1980). At high environmental temperature (35–40 °C) the manoeuvre produced signs of increasing sudomotor outflow in both nerves, whereas in a chilly environment (15 °C) it produced signs of decreasing vasoconstrictor tone not only in the median but (in some experiments) also in the posterior antebrachial nerve.

Opposite s.s.a. responses in the two nerves such as those produced by a moderate rise of external or internal temperature, were not seen during manoeuvres involving *arousal or mental stress*. Electrical skin shocks of constant strength repeated at irregular intervals triggered bursts of sympathetic impulses in both nerves, varying in amplitude from one stimulus to the next (Fig. 7*A*). As illustrated by the diagram in Fig. 8 (left) these random variations in burst amplitude occurred in parallel in the two nerves. The mental stress in connection with the solving of an arithmetical problem also produced repetitive bursts in both nerves (Fig. 7*B*). Many of these individual bursts occurred synchronously in the two nerves but there was not such a close correlation between burst amplitudes as there was for the triggered bursts (cf. diagrams in Fig. 8).

DISCUSSION

Regional differences in thermo-regulatory vasomotor and sudomotor tone. As previously shown (Hertzman, 1959; McCook *et al.* 1965) there are in healthy subjects marked regional differences in the sudomotor and vasomotor responses to changing environmental temperature. Using impedance plethysmography to measure bilateral blood flow in forearms, hands and digits Montgomery & Williams (1977) recently confirmed earlier claims that the more distal body segments exhibit relatively larger vasomotor responses to changes in ambient temperature than do more proximal body parts. According to Hertzman (1959) thermoregulatory sweating is more marked in the forearm and on the dorsum of the hand than in the palm, and for the skin on the calf it has been reported that a moderate rise in temperature may cause early appearance of reflex sweating without concurrent signs of vasodilatation (McCook *et al.* 1965). In the latter study it was also found that rapid cooling of the subjects caused stronger plethysmographic vasoconstrictor responses in the palm than in other areas.

From studies like those referred to above it is hard to draw definite conclusions as to whether the regional variations depend on differential outflows of vasomotor and sudomotor neural impulses or on differential effector organ responsiveness to the neural command signals. The present microneurographic findings provide direct evidence that in the distal skin areas innervated by the median and peroneal nerves, reflex thermoregulatory functions to a major extent are executed by vasoconstrictor fibres whereas sudomotor fibres in these nerves are brought into action only at relatively high ambient temperatures. In the skin on the dorsal side of the forearm and hand, supplied by the posterior antebrachial and superficial radial nerves, the thermoregulatory functions are instead to a large extent executed by sudomotor

fibres, the vasoconstrictor fibres in these nerves generally being brought into action only at such low temperatures that shivering also occurs. Since in most subjects, there does not seem to be any significant tonic vasoconstrictor activity in these nerves at normal room temperature, the plethysmographic signs of reflex vasodilatation in the forearm skin in response to warming can hardly be explained in terms of reduced vasoconstrictor tone. Alternative possibilities are that the skin nerve activity that increases in these nerves in response to warming is not composed only of sudomotor but also of vasodilator impulses or that the vasodilatation is due to the formation and action of bradykinin (for review see Rowell, 1974).

Synchrony of rhythms in different skin nerves. It is well known that in plethysmographic recordings from digits the traces obtained during rest show more or less frequently occurring transient vasoconstrictor responses and similar spontaneous waves are seen also in skin resistance records. These transient responses are known to occur with a high degree of synchrony in the four extremities (Burton, 1939) and they have by some authors been attributed mainly to randomly occurring arousal stimuli (for references see Figar, 1965). However, it has also been shown that the vasoconstrictor responses tend to occur in a rhythmical fashion with rates increasing up to about 10/min in a cold environment and it has been suggested that the responses indicate the arrival of volleys of vasoconstrictor impulses entrained by central rhythm-generating mechanisms (Burton, 1970).

The present recordings of spontaneous vasoconstrictor impulse bursts in the *median and peroneal nerves* showed a striking similarity with respect to the timing of individual bursts, consistent with the view that the vasoconstrictor outflows in these nerves are governed by the same central rhythm-generating mechanisms. Besides the slow vasoconstrictor rhythms seen in plethysmography traces (Burton, 1970) the neural recordings show a series of faster rhythms, including 100/min rhythm with subharmonics (Bini *et al.* 1980). The inter-limb synchrony was also striking for these faster rhythms which cannot be seen in the plethysmographic recordings because of effector organ inertia.

There was in the median and peroneal nerves a close correlation not only with respect to the timing but also with respect to the strength of individual vasoconstrictor bursts (Fig. 2) and with respect to the long-term variations in vasoconstrictor tone in response to temperature changes (Fig. 3). This indicates that the vasoconstrictor fibres in these two nerves are subjected to similar central drives both as regards the mechanisms responsible for the temporal entrainment of the neural impulses and as regards the thermoregulatory control of the vasoconstrictor tone. However, no attempts have yet been made to ascertain whether local or segmental thermoregulatory reflexes may selectively affect the vasoconstrictor tone in either one of the nerves.

A close correlation both with respect to timing and strength was observed also for the sympathetic bursts simultaneously led off from the *posterior antebrachial* and *superficial radial nerve*. These bursts, apparently destined to sweat glands (and possibly also contributing to vasodilatation) seemed to be entrained by similar central rhythms as were the vasoconstrictor bursts (Bini *et al.* 1979). In the early part of the warming periods, when growing sudomotor activity in the *posterior antebrachial nerve* was accompanied by declining vasoconstrictor activity in the *median nerve*, the bursts

in the two nerves appeared with a high degree of synchrony (Fig. 4, right). This suggests that when the sudomotor tone in one nerve is approximately equal to the vasoconstrictor tone in another nerve the two systems tend to discharge synchronously with bursts of equal strength in response to the rhythmical supraspinal drives. As sympathetic tone in one of the nerves increases, however, the bursts grow in strength at the same time as faster entrainment rhythms appear in the neurogram. Because the tone in the other nerve is not changing in the same direction or to the same extent, bursts will be present in the former nerve which are not seen in the latter. This may in part explain why a number of the burst led off from the median and posterior antebrachial nerves during sweating did not appear synchronously in the two nerves or showed low amplitude correlation (Fig. 5). An indication that the sympathetic systems in the two nerves to a large extent are exposed to synchronous inherent rhythmical fluctuations of excitability is provided by the diagram in Fig. 8 (left) showing a close correlation in the amplitude variations of bursts in the two nerves triggered by randomly occurring arousal stimuli.

Effects of arousal stimuli and of mental or emotional stress. Besides the thermoregulatory and the rhythm generating mechanisms there are other central drives governing the sympathetic nerve activity to the skin. Arousal stimuli and mental or emotional stress are generally associated with a wide-spread activation of vasoconstrictor and sudomotor systems (cf. Sourek, 1965). Since changes in ambient temperature may give rise to 'pleasant' or 'unpleasant' emotional feelings (Hensel, 1973) some of the sympathetic responses to thermal stimuli may be expressions of emotional rather than of thermal reactions. In the present experiments the sudden fall in ambient temperature (down to 15 °C) was generally experienced as 'unpleasant' and the increase in neural vasoconstrictor activity could partly be due to the emotional stress (Fig. 4).

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