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CUTANEOUS VASOMOTOR CONTROL IN THE HUMAN HEAD, NECK AND UPPER CHEST

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Two types of vasomotor control are known to be present in human skin. In one case vasodilatation is achieved by inhibiting the activity of nerves which constrict the blood vessels, and in the other vasodilatation is achieved by increased activity in nerves which cause dilatation of the vessels. Thus, in the hand the vasoconstrictor mechanism is dominant (Lewis & Pickering, 1931; Gaskell, 1956), whereas in forearm skin the vasodilator mechanism is dominant (Grant & Holling, 1938; Edholm, Fox & Macpherson, 1957).

The type of vasomotor control in other skin areas has received little attention, although the presence of a vasoconstrictor fibre supply to the vessels of the head and neck was suggested over a century ago (Bernard, 1858).

The present series of experiments was undertaken to investigate the relative importance of the vasoconstrictor and vasodilator mechanisms in certain areas of the skin of the head, neck and upper chest in man. The opportunity was also taken to study certain other aspects of the reactivity of these areas. Preliminary reports on this work have already been published (Fox, Goldsmith & Kidd, 1960*a*, *b*).

METHODS

Subjects and procedure

The subjects were ten healthy males. A subject was seated in a stirred temperature-controlled bath with the legs and lower trunk immersed in water to a constant depth. With the exception of the head and neck, the rest of the body not covered by water was insulated against heat loss by an inflatable jacket and layers of cotton wool. Each experiment commenced with the water in the bath at 34° C. Blood flow, measured by venous-occlusion plethysmography (Edholm, Fox & Macpherson, 1956), provided a direct indication of the cutaneous vasomotor state in one forearm. Changes in heat flow in the skin of the head, neck or upper chest were simultaneously monitored by small surface calorimeters placed symmetrically on either side of the mid line.

Each experiment followed the same general pattern, in which the subject's oral temperature was raised to about 38° C on two successive occasions. After the first heating phase, which served as a control period, the subject was cooled by a cold shower over the trunk and

by lowering the bath temperature. When the body temperature had returned to normal the cutaneous nerve supply to one side was blocked with 2 % Xylocaine (Duncan, Flockhart; lignocaine, B.P.) solution containing adrenaline 1/60,000. After an interval to observe the effect of the nerve block, the subject's temperature was raised for a second time and the responses on the two sides again compared. A rise in heat flow after blocking the nerve supply to an area indicated the presence of the vasoconstrictor mechanism. The absence of any increase in heat flow, and a failure to dilate compared with the unblocked or control side during the subsequent period of body heating, indicated the presence of the vasodilator mechanism. The effectiveness of the cutaneous nerve block was checked during the experiment, and again on completion, by light touch and pin prick. In some experiments the absence of sweating in the blocked area was observed by means of the iodine and starch technique (Minor, 1927).

Later experiments were shortened by omitting the initial heating period and comparing the reactivity of the two sides of the body to the inhalation of amyl nitrite from 5-minim (0·3 ml.) vitrellae broken in a mask and held over the mouth and nose of the subject. The responses obtained from these tests have also been used to compare the reactivity of different areas of the head, neck, upper chest, forearm and hand.

Some results from two other series of experiments are also included in this paper because they are relevant to the discussion on the mechanism of the vasomotor control. In one of these the vasomotor and sudomotor responses to gustatory stimulants, and especially chillies (capsicum) were compared in skin areas of the face. In the other series the relative rates of thermal sweating in the different areas of the head were compared in experiments on four subjects exposed nude in a climatic chamber to an environment in which the dry-bulb temperature was 40° C and dew-point 30° C; the sweat was collected in small capsules containing absorbent tissue and silica gel, and the sweat losses were calculated from the weight changes.

The skin calorimeter

Each calorimeter consisted of a light hollow Perspex cylinder, 20 mm in diameter and 20 mm in length, the centre of the base of which was formed by a Hatfield–Turner heat-flow disk (Hatfield, 1950) of 12 mm diameter and 1·5 mm thickness, as shown diagrammatically in Fig. 1. The output from the disk was measured with a potentiometer and galvanometer capable of discriminating changes in heat flow of $\pm 0{\cdot}02$ kcal/m²/hr. The internal temperature of the calorimeter was kept constant by water circulated at 32° C through insulated

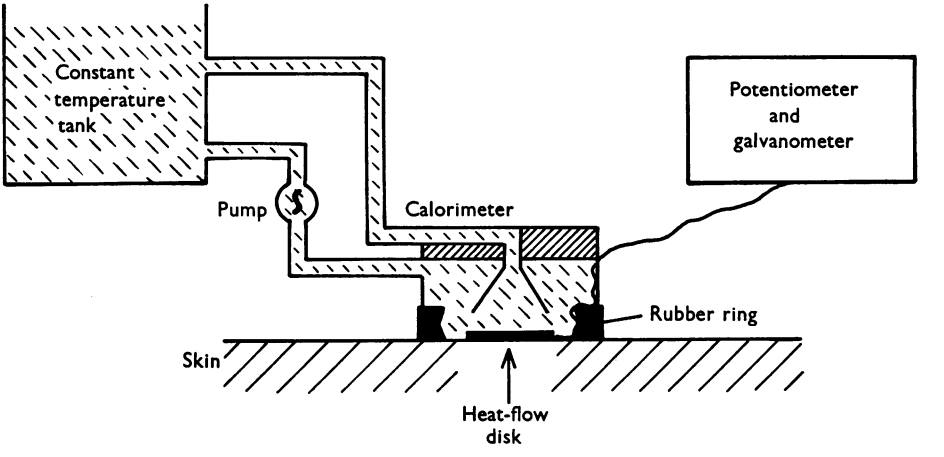

Fig. 1. Diagrammatic representation of the skin calorimeter showing the general lay-out and important components.

flexible tubes. This temperature was chosen as being just below the lowest skin temperature of subjects in a neutral thermoregulatory state, and ensured that there was always a positive heat flow through the skin to the capsules.

The calorimeter must be closely applied to the skin but the vessels must not be under appreciable compression or tension (Vere, 1958). To hold the device to the skin with light even pressure, and yet retain a limited degree of mobility for the subject, a rubber ring was first firmly glued to the selected site with 'Newskin' (Harwoods Laboratories). The base of the calorimeter was then pushed into this ring, which fitted tightly and was self-locating. A thin film of greaseless water-soluble jelly between calorimeter and skin was used to avoid a sudden change in thermal conductivity across the interface with the onset of sweating.

Areas of skin studied. Calorimeters were placed symmetrically on either side of the mid line to observe skin blood flow changes in the areas shown in Table 1, which also gives the nerves blocked. In general, motor nerves contain some vasoconstrictor fibres to muscle vessels (Barcroft, 1953) and care was therefore taken to avoid motor blocks. This proved difficult in the lip and in one experiment there was quite noticeable paresis; however the result did not appear to differ from the remaining experiments.

TABLE 1. The skin areas studied and the cutaneous nerves blocked

Area studied	Position of calorimeters	Cutaneous nerve blocked	Site of block
Scalp	15 cm above supraorbital ridge, 1·5 cm from mid line	Supraorbital	Just above the eyebrows
Forehead	4 cm above supraorbital ridge, 2·5 cm from mid line	Supraorbital	
Ear	(1) Cranial aspect of eminentia scaphoideum (2) Lateral aspect of lobe	Greater auricular	Posterior border of sternomastoid
Nose	Over nasal cartilage and alae, close to mid line	External nasal	Junction of bone and nasal cartilage
Lip	Glabrous portion of slightly everted lower lip	Mental	Emergence from mental foramen
Chin	Just medial to mental foramen	Mental	Emergence from mental foramen
Cheek	Over malar bone, just inferior to infraorbital foramen	Infraorbital	Emergence from infraorbital foramen
Submandibular	Just inferior to lower border of mandible	Anterior cutaneous of neck	Over belly of sterno-mastoid
Neck	Midway between jaw and clavicle	Anterior cutaneous of neck	Over belly of sterno-mastoid
Chest	(1) 2·5 cm below junction of medial and middle third of clavicle	Infraclavicular	Over clavicle
	(2) Between 2nd and 3rd ribs, 8 cm from mid line anteriorly	Anterior cutaneous branches of thoracic	Emergence lateral to sternum

The relation between blood flow and heat flow

The factors which determine the rate of heat flow through the skin are governed by the basic equation for heat transfer:

$$H = \frac{\Delta t}{I},$$

where H is the heat flow (kcal per unit surface area), Δt is the temperature difference across the skin, and I is the thermal insulation of the skin.

Thus, for a given rate of blood flow the heat flow will be augmented if the temperature difference across the skin is increased by a rise in the temperature of the blood reaching the

skin. Likewise, if the temperature of the blood remains constant, but its rate of flow through the skin rises, the thermal insulation diminishes and the heat flow rises.

There are, therefore, two variables which combine to determine the magnitude of the changes in heat flow and only one of these reflects changes in the vasomotor state of the skin. This means that the changes in heat flow registered by this device only provide unequivocal evidence of changes in vasomotor state when the body temperature is constant. In general, during body heating the increase in heat flow due to vasodilatation will be augmented by that due to the rise in body temperature. However, in the initial stages of body heating, deep body temperature usually falls, owing to the influx of cooler blood from the vasodilatation in the superficial tissues, and measurements of heat flow may then underestimate the degree of change in the cutaneous blood flow. The relative magnitudes of change in heat flow in different areas during vasodilatation also depend in part both on the sizes of the regional temperature gradients and on the masses of tissue involved. For example, the pinna of the ear, having a low heat capacity, cools down during vasoconstriction and warms up during vasodilatation more rapidly than an area such as the forehead. Considerable care is therefore required when interpreting changes in heat flow during body heating. For this study it was fortunately possible to overcome many of these difficulties by the simultaneous comparison of behaviour of the two sides of the body, using one side as the control.

Differences in structure between one skin area and another may also affect the relation between heat flow and blood flow, in particular the effective depth from the surface of the skin at which the blood is flowing and the presence of vascular arrangements giving a counter-current heat-exchanger effect (Bazett, Love, Newton, Eisenberg, Day & Forster, 1948; Scholander & Krog, 1957). This becomes important when considering the significance of differences in heat flow of one area as compared with another.

RESULTS

Direct comparison of heat flow and blood flow measurements

Control experiments were made to compare simultaneously in the forearms the changes in skin heat flow, as measured by the calorimeter, with the changes in total blood flow, as measured by venous-occlusion plethysmography. The close correspondence between the magnitude and timing of skin heat flow and total blood flow during three successive periods of indirect body heating, in which the body temperature was raised progressively to produce a graded vasomotor response, is illustrated in Fig. 2.

Cutaneous nerve block experiments

On the basis of the relative importance of the two vasomotor mechanisms, the areas studied have been classified in two groups:

(1) areas of skin in which vasomotor control is principally mediated by variations in vasoconstrictor tone—ear, lip and nose; and

(2) areas of skin in which vasomotor control is principally mediated by active vasodilatation—scalp, forehead, chin, submandibular area, neck and upper part of the chest.

Vasomotor control in the ear, lip and nose. The response in the ear to cutaneous nerve block from one experiment is shown in Fig. 3. Heat-flow

measurements from the two ears were similar during the preliminary control period and following the inhalation of amyl nitrite. Immediately after blocking the greater auricular nerve on one side there was a marked rise in heat flow, indicating the release of a large component of vasoconstrictor tone. At this point, the subject was cooled by a cold shower over the trunk, which evoked vasoconstriction in the skin of the control ear but not in the skin of the nerve-blocked ear. Since the heat flow on the control side reached a higher level than on the blocked side during indirect body heating, a small component of active vasodilatation cannot be excluded, but the result shows clearly that in the ear vasomotor control is principally mediated by the release of vasoconstrictor tone.

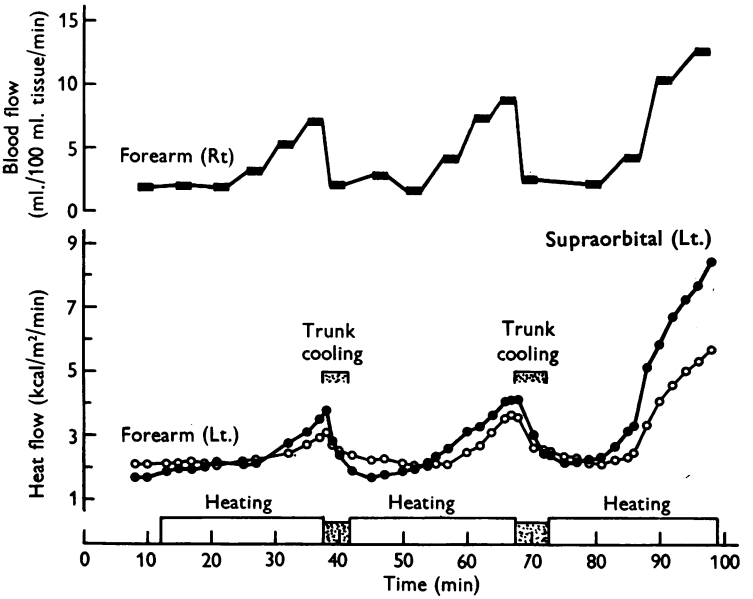

Fig. 2. Control experiment comparing heat-flow and blood-flow measurements simultaneously in the two forearms during periods of body heating and cooling. Heat-flow changes in the supraorbital region are also shown.

An experiment in which the mental nerve to one side was blocked is shown in Fig. 4. Following the injection there was an immediate rise in skin heat flow in the glabrous portion of the lower lip on that side, indicating the release of vasoconstrictor tone. The possibility of a small component of active vasodilatation cannot be excluded.

Similarly, blocking the external nasal nerve to one side of the nose produced a large rise in skin heat flow. However, in this case during the subsequent period of indirect heating the heat flow from the control side

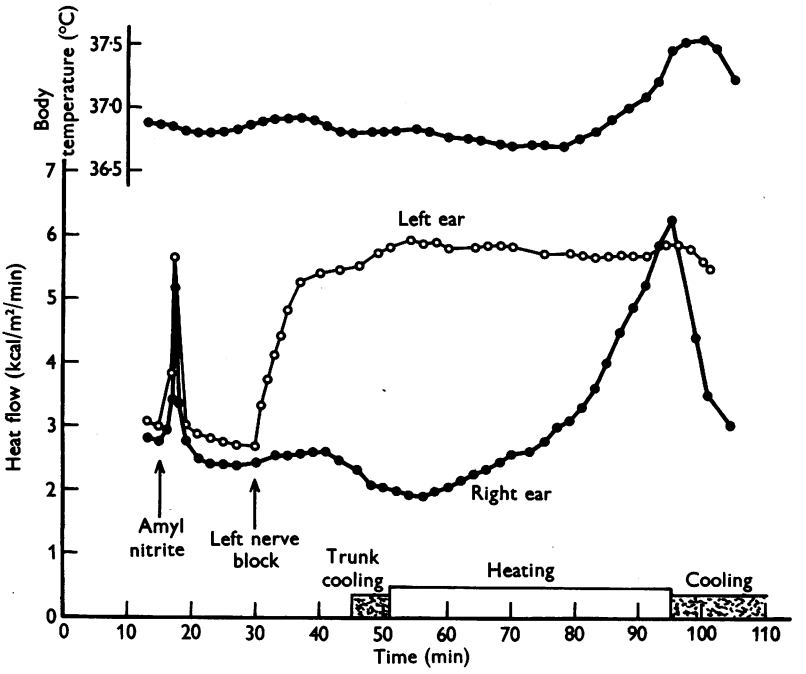

Fig. 3. The effect on heat flow from the left ear of blocking the greater auricular nerve. The effects of an inhalation of amyl nitrite before nerve block, and of body heating and cooling after nerve block, on the heat flows from both ears are also shown.

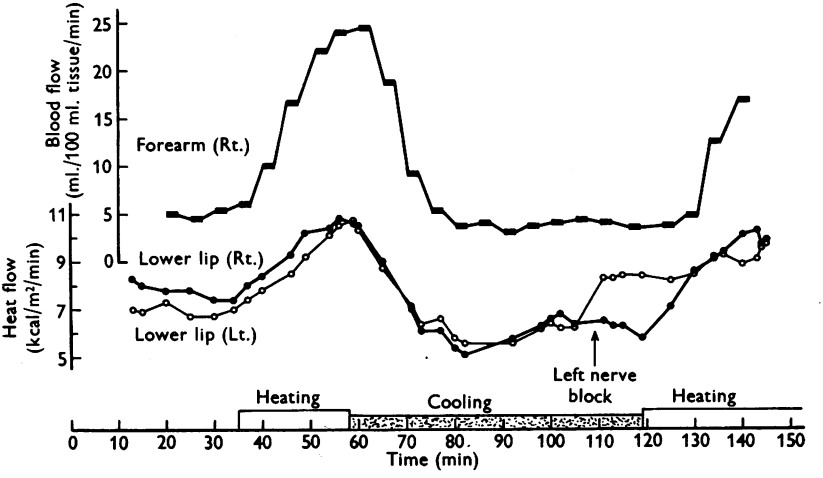

Fig. 4. The effect on heat flow from the left lower lip of blocking the left mental nerve. Changes in heat flow from the two sides of the lip during periods of body heating and cooling are shown and compared with the changes in total forearm blood flow.

clearly exceeded that from the nerve-blocked side, indicating the presence of a component of active vasodilatation.

Vasomotor control in the scalp, forehead, chin, submandibular area, neck and upper chest. In all these areas blocking the cutaneous nerve supply resulted in only a small increase in heat flow due to the release of vasoconstrictor tone, but during indirect heating the response from the nerve-blocked side fell far short of that from the control side, indicating the presence of a large component of active vasodilatation. This is illustrated by the results obtained from nerve block of the supraclavicular and anterior cutaneous branches of the 2nd and 3rd thoracic nerves, as shown in Fig. 5.

In two experiments designed to investigate the vasomotor control of the cheek, in which the infraorbital nerve was blocked on one side of the face, considerable difficulties were encountered because the areas covered by the calorimeter and retaining ring were necessarily close to the infraorbital foramen, and a direct spread of adrenaline to part of the area covered by the heat-flow disk made it impossible to draw any satisfactory conclusion.

The maximum rise in skin heat flow resulting from cutaneous nerve block in each of the areas studied is summarized in Table 2. Between those cutaneous areas having high vasoconstrictor tone (ear, lip, nose) and those with low vasoconstrictor tone (scalp, forehead, chin, submandibular area, neck and upper chest) the difference in response to cutaneous nerve block is highly significant ($P < 0{\cdot}001$).

The evidence for the presence of active vasodilator control in certain areas is given in Table 3, which shows the heat flows recorded from control and treated sides immediately before and after body heating. In almost every experiment there was an increase in heat flow on the nerve-blocked side as well as on the control side. In general the increases in the nerve-blocked areas are attributed to the direct effect on heat flow of a rise in body temperature (see p. 300). The differences between experiments are attributed to differences in the degree of body heating. In the areas with a large component of vasoconstrictor control, e.g. ear and lip, the heat flows on the two sides after body heating were almost equal, which is evidence for the absence of any major component of active vasodilator control. In the skin areas not showing a large component of vasoconstrictor tone the heat flow after body heating was usually considerably lower than on the control side, which is evidence for the presence of active vasodilator control.

Responses to inhalation of amyl nitrite

In all areas the response to inhalation of amyl nitrite rapidly reached a peak within 2 min and thereafter returned to the resting value some

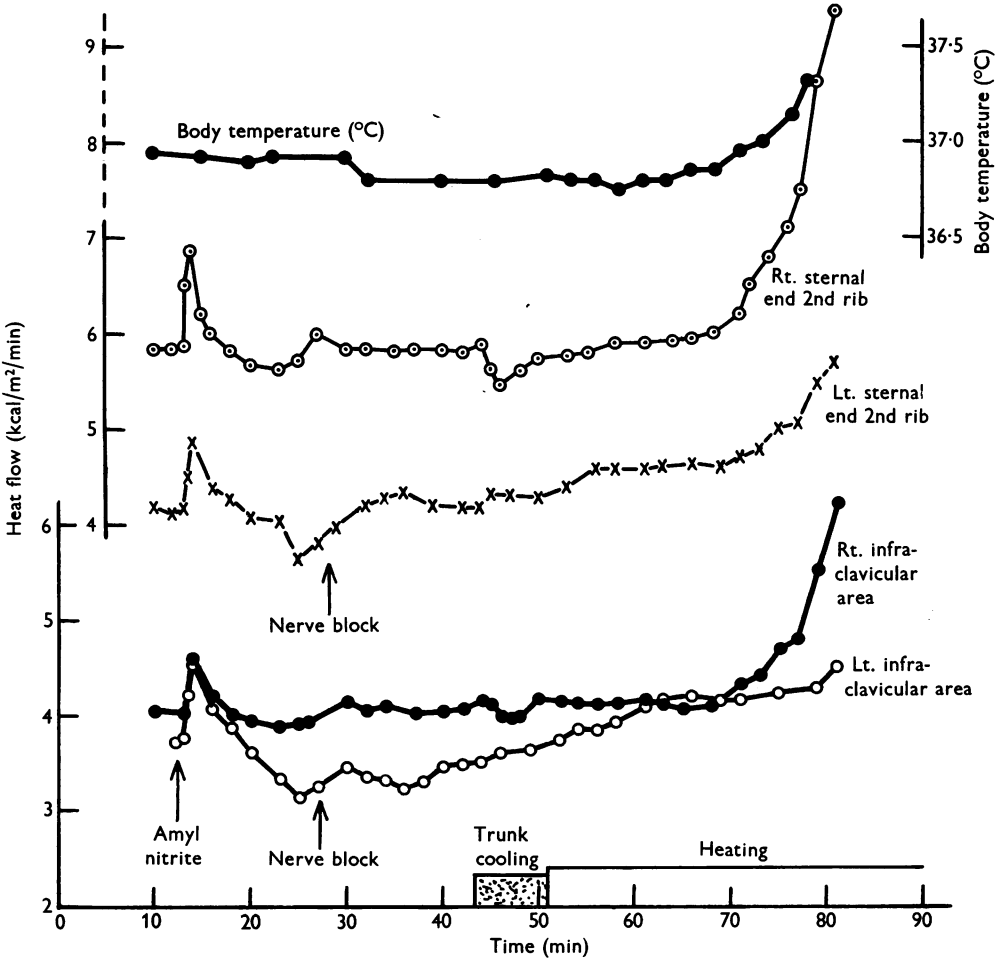

Fig. 5. The effect on heat flow from regions of the upper chest of blocking the cutaneous nerve supply. The failure to dilate during body heating and the absence of vasoconstrictor reflexes in the nerve-blocked areas are clearly shown.

TABLE 2. Comparison of increases in skin heat flow (kcal/m²/min) in various areas of the body in response to cutaneous nerve block

Ear	Lip	Nose	Chin	Scalp	Fore-head	Sub-mandibular	Neck	Infra-clavicular	Anterior chest
2·0	1·6	1·0	0·8	0·6	0·8	0·3	0·2	0·3	0·2
5·8	2·2	1·8	0·9	—	0·7	0·7	0·4	0·4	0·5
2·2	2·5	—	0·6	—	0·4	—	—	—	—
3·3	2·5	—	—	—	—	—	—	—	—
Mean									
3·33	2·2	1·4	0·77	0·6	0·63	0·5	0·3	0·35	0·35

TABLE 3. Comparison of the skin heat flows (kcal/m²/min) from the nerve-blocked and control sides before and after heating

Site		Control side			Nerve-blocked side			
		Before	After	Diff. (*a*)	Before	After	Diff. (*b*)	$\frac{b}{a} \times 100$
Ear		1·90	14·80	12·90	11·60	16·10	4·50	35
		0·10	10·20	10·10	3·60	8·50	4·90	49
		2·00	6·20	4·20	5·80	5·90	0·10	2
	Mean	1·33	10·40	9·07	7·00	10·17	3·17	
Lip		5·80	10·00	4·20	8·40	9·70	1·30	31
		5·70	10·80	5·10	7·50	9·20	1·70	33
		7·60	9·80	2·20	9·90	9·80	−0·10	0
		5·50	8·80	3·30	9·10	9·20	0·10	3
	Mean	6·15	9·85	3·70	8·73	9·48	0·75	
Nose		2·90	7·50	4·60	5·10	6·90	1·80	39
		2·40	8·50	6·10	4·00	7·80	3·80	62
	Mean	2·65	8·00	5·35	4·55	7·35	2·80	
Chin		4·50	8·50	4·00	5·60	6·90	1·30	32
		7·10	7·80	0·70	7·00	7·10	0·10	14
		4·60	5·40	0·80	5·30	5·40	0·10	12
	Mean	5·40	7·23	1·83	5·97	6·47	0·50	
Scalp		2·80	4·50	1·70	3·10	4·40	1·30	76
Forehead		4·80	9·90	5·10	6·30	8·70	2·40	47
		4·80	11·50	6·70	4·30	5·90	1·60	24
		5·80	9·00	3·20	3·90	5·10	1·20	37
	Mean	5·13	10·13	5·00	4·83	6·57	1·73	
Submandibular		4·20	10·20	6·00	5·80	8·10	2·30	38
		5·40	9·00	3·60	5·10	6·90	1·80	50
	Mean	4·80	9·60	4·80	5·45	7·50	2·05	
Neck		4·50	8·60	4·10	2·00	5·10	3·10	76
		6·60	9·00	2·40	4·50	5·10	0·60	25
	Mean	5·55	8·80	3·25	3·25	5·10	1·85	
Infraclavicular		1·70	4·40	2·70	3·00	3·80	0·80	30
		4·20	6·20	2·00	3·70	4·50	0·80	40
	Mean	2·95	5·30	2·35	3·35	4·15	0·80	
Anterior chest		3·80	7·10	3·30	2·70	3·00	0·30	9
		5·70	9·40	3·70	4·30	5·70	1·40	38
	Mean	4·75	8·25	3·50	3·50	4·35	0·85	

5–8 min later (see Figs. 3 and 5). The mean maximum increases in skin heat flow in the various areas tested following inhalation of amyl nitrite are shown in Table 4.

A vasodilator response was seen in all areas of the face, neck and upper chest, whereas a small decrease in skin blood flow occurred in the forearm. In the hand either a decrease or a biphasic response was seen, with an initial decrease followed by a slight vasodilatation. The response in the skin areas in which vasoconstrictor release is the dominant mechanism is significantly greater than in the predominantly active vasodilator areas

($P < 0{\cdot}001$). The response of the cheek is like that of the predominantly vasoconstrictor-release areas.

TABLE 4. Comparison of increases in skin heat flow (kcal/m²/min) in various areas of the body in response to inhalation of amyl nitrite

Ear	Lip	Nose	Cheek	Chin	Sub-mandibular	Neck	Infra-clavicular	Anterior chest	Fore-head	Fore-arm
2·6	1·9	1·7	2·6	0·8	1·2	0·7	1·3	1·1	0·2	−0·2
3·0	1·9	1·8	3·0	0·6	1·0	0·7	0·6	1·3	0·5	−0·2
3·6	2·5	3·0	1·6	0·7	1·2	1·2	0·8	1·1	0·5	−0·1
2·8	2·8	3·2	1·7	0·8	1·1	1·2	0·4	1·2	0·8	−0·1
2·1	1·6	0·9	1·8	0·6	1·3	1·1	0·8	0·6	0·9	−0·2
2·3	1·9	0·9	2·1	0·3	1·3	0·9	1·2	0·9	0·8	−0·2
2·7	2·7	0·9	2·2	0·4	1·1	1·3	0·5	0·8	0·2	−0·2
2·8	2·8	1·0	1·1	0·7	1·1	1·7	0·9	0·9	0·7	−0·1
2·9	2·9	—	0·9	0·8	—	—	0·9	0·6	0·3	—
2·8	3·4	—	1·2	0·9	—	—	0·9	0·8	0·3	—
3·1	—	—	1·1	1·0	—	—	—	1·0	—	—
2·7	—	—	—	1·5	—	—	—	0·5	—	—
2·6	—	—	—	1·2	—	—	—	—	—	—
—	—	—	—	1·2	—	—	—	—	—	—
—	—	—	—	0·7	—	—	—	—	—	—
—	—	—	—	1·4	—	—	—	—	—	—
—	—	—	—	1·2	—	—	—	—	—	—
—	—	—	—	1·3	—	—	—	—	—	—
Mean										
2·77	2·44	1·68	1·75	0·89	1·16	1·1	0·83	0·9	0·52	−0·16
S.D. 0·36	0·58	0·67	0·67	0·33	0·10	0·33	0·28	0·25	0·26	0·05

Responses to gustatory stimulation

In ten experiments gustatory sweating was elicited by chewing chillies, and the vasomotor effects in various areas of the face were studied. An experiment in which in turn gum, then a slice of freshly cut lemon and, finally, chillies were chewed is shown in Fig. 6. Chewing gum *per se* produced no response in the skin of the cheek. The slice of lemon produced a small vasodilatation in the skin of the cheek, and chewing chillies a larger response, and both were associated with sweating of the opposite cheek. There was no response in the forearm until the subject was heated.

The vasodilator responses observed in various areas on chewing chillies are summarized in Table 5. The largest responses were seen in the skin of the nose and cheek, but all areas of the face, neck and upper chest showed some vasodilatation, whereas the forearm flows decreased.

The presence of gustatory sweating was always recorded, and in a few experiments measured. It was most profuse over the medial portions of the cheek and the bridge of the nose extending up to the forehead between the eyebrows and down to the nasolabial region. It was never observed on the chin, elsewhere on the forehead, submandibular region or ears.

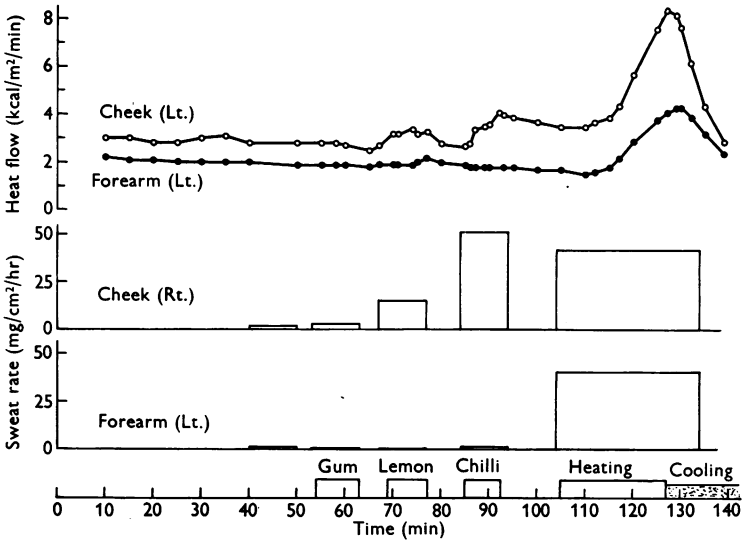

Fig. 6. The results from one experiment in which the effects of gustatory stimulants on sweating and heat-flow changes in the cheek and forearm were compared. The effect of a period of body heating is also shown.

TABLE 5. Comparison of increase in skin heat flow (kcal/m²/min) in various areas of the body in response to gustatory stimulation

	Nose	Cheek	Chin	Sub-mental	Forehead	Ear	Forearm
	0·8	1·1	0·4	0·7	0·4	0·4	−0·1
	1·4	0·9	0·6	—	0·3	0·2	−0·3
	1·8	1·5	—	—	0·5	0·2	−0·1
	—	1·4	—	—	—	0·2	−0·3
	—	1·0	—	—	—	—	−0·2
Mean	1·33	1·18	0·5	0·7	0·33	0·28	−0·2

TABLE 6. Comparison of sweat production in various areas of the body in response to thermal stimulation expressed as a percentage of total sweat production

Site	Number of observations	Sweat loss (% of total body loss)	S.D.
Forehead	18	0·049	0·016
Forearm	18	0·026	0·009
Chin	13	0·024	0·007
Nose	19	0·023	0·010
Cheek	16	0·021	0·007
Ear	19	0·0062	0·0023

Sweat responses with thermal stimulation

In nineteen experiments on four subjects exposed to a hot wet environment in a climatic chamber the rates of sweat production from equal areas of skin of the forehead, forearm, chin, nose, cheek and ear were compared.

Table 6 shows the mean values expressed as a percentage of the total sweat loss during exposure.

The loss from the forehead is significantly greater than that from the forearm, chin, nose and cheek ($P < 0{\cdot}001$), while the sweat loss from the ear is significantly less than that from any other area tested ($P < 0{\cdot}001$).

DISCUSSION

The results of the experiments in which the cutaneous nerve supply was blocked by local anaesthesia have shown that the areas studied can be divided into two main groups. The pinna of the ear, the lip and the nose are like the hand with the release of vasoconstrictor tone as the dominant mechanism of vasomotor control, whereas the skin of the scalp, forehead, chin, submandibular area, neck and upper chest have a dominant active vasodilator mechanism like the forearm. In all the areas in which the active vasodilator mechanism was found to be dominant a small component of vasoconstrictor release was also present and, conversely, in the nose there was evidence of some active vasodilatation.

It is important to emphasize that we may not have blocked all the vasomotor nerves to particular areas. It has already been pointed out that some of the sudomotor fibres to the cheeks are contained in both facial and trigeminal nerves (Wilson, 1936; Kuno, 1956).

The finding of a predominantly vasoconstrictor-release type of control in the ear agrees with the observation by Trotter & Davis (1909) that section of the greater auricular nerve in man produces bright flushing of the ear. There is also generally good agreement between the observations reported here and those of a recent study by Blair, Glover & Roddie (1961), based on measurements of change in skin temperature. These authors also conclude that in the ear and nose blood-flow changes are brought about mainly by variations in vasoconstrictor tone, whereas in the chest and cheek the changes are due to the activity of 'vasodilator' nerves. They did not, however, observe any vasoconstrictor tone in the predominantly active-vasodilator areas, perhaps because skin temperature measurements are less sensitive than the technique used here.

Using a gradient calorimeter Froese & Burton (1957) measured heat losses from the head as a whole and concluded that no vasoconstriction occurs in the head as a whole in response to cold. An absence of vasoconstrictor reflexes in forehead skin has also been reported by Hertzman & Roth (1942), using a photo-electric technique. Although the results of the present study indicate the presence of some vasoconstrictor control, it would seem to be more important in increasing blood flow above normal in response to body heating than in reducing it below normal in response

to cooling. It is also likely that the high heat flow from the head as a whole on exposure to cold is due, in part at least, to the ease with which cold vasodilator responses are elicited from this region (Fox & Wyatt, 1960).

It is of interest to consider whether the differences in vasomotor control found in these areas correlates with other differences between the areas, such as their response to amyl nitrite, gustatory stimulation, thermal sweating and cold vasodilatation.

Inhalations of amyl nitrite were used by Aldridge (1871) to study changes in the retinal vessels, and he noted that the flushing commenced at several points of the face, the most frequent being the nose, cheek, chin and ears, and that eventually the flush would extend to the front of the chest. The resemblance of this response to emotional blushing was described by Darwin (1890) and Lewis (1927), and a similar distribution of flushing is also observed following intravenous doses of bradykinin and histamine (Fox, Goldsmith, Kidd & Lewis, 1961). The largest responses to amyl nitrite inhalations in this study were found in the lip and ear, skin areas with a dominant vasoconstrictor-release mechanism, although the responsiveness of the ear is probably partly explained by its small tissue volume. If amyl nitrite does preferentially dilate areas with vessels supplied by vasoconstrictor nerves, a vasodilatation would also be expected in the hand, but possibly its absence here is due to the longer circulation time with greater destruction and dilution en route.

Thermal sweating was most profuse in the forehead, which is in agreement with a similar observation by Kuno (1930); the pinna of the ear has few sweat glands and produces only a little sweat. The dominance of the vasoconstrictor-release mechanism in the ear and in the glabrous portion of the lip is in accord with the hypothesis that active vasodilatation in the skin is mediated by sweat-gland activity, and therefore this type of vasomotor control is only found where sweat glands are present (Fox & Hilton, 1958).

Gustatory sweating can occur in most areas of the head and neck (Lee, 1954) but, as was noted in these experiments, the nose and cheek are among the regions in which overt sweating is most frequently observed. The present study shows that gustatory stimulation also causes vasodilatation and it was most marked in the nose and cheek, and least marked in the ear. Since the vasodilatation was particularly marked in the nose, an area in which the vasoconstrictor-release mechanism is dominant, the distribution of the gustatory response is apparently not determined simply by the pattern of vasomotor control, although the degree of vasodilatation in different regions probably is correlated with the intensity of sweating in those regions.

It has been concluded that arteriovenous anastomoses in the hand

controlled by the vasoconstrictor nerves are responsible for the changes in flow during cold vasodilatation (Grant & Bland, 1931). The cold-vasodilatation response is very readily elicited from the lip, ear and nose (Fox & Wyatt, 1960) and arteriovenous anastomoses have already been demonstrated in the human ear (Prichard & Daniel, 1956). It therefore seems likely that the lip and nose are also endowed with this particular vascular structure.

The functional significance of these differences in vasomotor innervation of facial areas is somewhat obscure. The glabrous portion of the lips represents a transitional area between true mucous membrane and skin, and it is possible that the vasoconstrictor-release mechanism is the dominant vasomotor mechanism in mucous membranes. In the same way a part of the vasoconstrictor release observed in the nose may be due to the proximity of the nasal mucous membrane. The most probable functional reason, however, would seem to be the greater protection afforded by the cold-vasodilatation phenomenon of vasoconstrictor-release areas to the more vulnerable parts of the face.

SUMMARY

1. The vasomotor control of the skin of the face, neck and upper chest has been studied in man, using surface calorimeters to monitor responses to cutaneous nerve block and the effects of administering vasodilator stimuli.

2. Vasodilator responses to inhalation of amyl nitrite and gustatory stimulation were examined and thermal sweat rates from various areas of the face were measured.

3. It is concluded that in man dilatation of the skin of the ear, lip and nose is mainly the result of release of vasoconstrictor tone but in the nose some active vasodilatation occurs in response to thermal and gustatory stimulation. Vasoconstrictor tone plays a relatively small part in the control of skin circulation in the scalp, forehead, chin, submandibular area, neck and upper chest, where dilatation is mainly achieved by active vasodilatation.

4. The relationship between the regional differences in vasomotor control, sweating and some circulatory responses is discussed.

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