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THERMOREGULATION IN SPINAL MAN

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It has never been unequivocally established whether or not the spinal cord, chronically isolated from the brain above the thoracic sympathetic outflow, plays any part in temperature regulation. Animals, kept in rooms at an air temperature of 80° F (26·5° C), after chronic traumatic lesions of the spinal cord, behave essentially as poikilotherms (Pflüger, 1878; Pembrey, 1897; Freund & Strassmann, 1912; Sherrington, 1924), but if they are kept at 60–70° F (15·6–21·1° C) they appear to regain some measure of temperature regulation against cold (Thauer, 1939; Clark, 1940). Studies on man are equivocal. Some authors claim that patients with acute traumatic lesions in the lower cervical segments are, initially, hyperthermic but later regain an ability to sweat (Foerster, 1936). Others state that they are hypothermic (Holmes, 1915).

The experimental studies reported in this paper were made on human male patients, who, with one exception, had complete lesions due to injuries at various levels in the spinal cord, in order to study further the nature of the thermo-regulatory processes in spinal man. The subjects comprised one normal man, four patients with lesions at segmental levels between C6 and T1, one with a lesion below T4 and one with a lesion below T8. In all patients many months had elapsed since their injuries to the spinal cord. These subjects were exposed at rest and naked for 1–2 hr at air temperatures of 18–20, 28 and 35–37° C. These temperatures and the design of the experiments were chosen to effect a comparison with the data of Hardy & du Bois (1938, 1941) and so to bring out any alteration in the thermoregulatory processes due to the cord lesions.

METHODS

The patients were all in good physical condition. None had bedsores nor urinary infection of any consequence. The cervical lesion patients and the T4 patient were bed-ridden and subject, therefore, to a smaller range of fluctuation in environmental temperatures than the other men. The wards

of this hospital were neither centrally heated nor air-conditioned, and hence the microclimate of these patients probably shows a considerable variation between winter and summer. Their physical characteristics are given in Table 1.

The same experimental procedure was adopted in each study. The subjects were stripped and rested nude for 1 hr on hospital beds at an environmental temperature of 27° C. Thereafter they were transferred either to an environmental temperature of 18–20 or 35–37° C for 1½–2 hr. The cold temperature conditions were obtained in a room open to the outside air, but shielded from direct and most reflected radiation from the sun on the colder days of the summers of 1954 and 1956. Air in the room was circulated slowly by means of a small electric fan to avoid gradients. The study at 35–37° C was made in a specially constructed 'hot box' (Guttmann, 1947). The heat source was a series of infra-red electric heaters in the roof of the box, but the patient was shielded from direct radiation. The air heated in this way was circulated slowly. Humidity in the box was constant at 40–50% during exposure of the cervical subjects, but tended to rise to 60–70% when the subjects who sweated were studied. The temperatures given of all the environmental conditions are globe thermometer readings, as they take account of radiation. Globe thermometer readings in the hot box were approximately 2° C higher than the air temperature but were within 1° C in the neutral and cold temperatures. The presence of sweat was detected by the quinizarin dye method, which has been shown to be very sensitive (Guttmann, 1941, 1947).

Rectal temperature was measured every 15 min with a clinical rectal thermometer. Skin temperature was measured by means of a copper-constantan thermocouple, stretched over a Y shaped applicator; the e.m.f. was recorded on a Cambridge Instrument galvanometer specially constructed for skin temperature measurements. Skin temperatures (T_s) were measured at the forehead, chest, side, fingers and toe. Temperatures were also measured on the anterior surface of the body above and below the level of the lesion. Average skin temperatures were calculated from the formula:

$$T_s = 0{\cdot}7T \text{ of trunk} + 0{\cdot}1T \text{ of foot} + 0{\cdot}1T \text{ of finger} + 0{\cdot}1T \text{ of forehead}$$

Weight of sweat loss could be determined with adequate accuracy by loss of weight of only the normal subject. Metabolism was measured twice in each air temperature condition by means of a Benedict-Roth type of apparatus and was determined over 5 min intervals. K_{int}, the heat conductance of the tissues, and K_{air}, the conductance of the air, were determined from the equation due to Hardy & Soderstrom (1938):

$$K_{int} = \frac{M \pm S}{T_r - T_s} \quad \text{and, similarly,} \quad K_{air} = \frac{M \pm S}{T_s - T_a}$$

where M = rate of metabolic heat production (cal/m²/hr),

S = rate of heat storage (cal/m²/hr),

T_r = rectal temperature (° C) and T_s skin temperature (° C).

In the steady state, i.e. when T_r and T_s have reached steady values and M is constant, these estimates present no difficulty. In cold conditions, as occurred here in the high spinal lesion cases, T_s reached a steady level in the last hour, but T_r fell steadily. We have therefore assumed that the rate of heat flow between T_r and T_s is metabolic rate plus the rate of change of heat content of deep tissues, viz., $T_r \times 0{\cdot}83 \times 2W/3$ (it being assumed, with Hardy & Soderstrom (1938), that in cool conditions T_r represents 2/3 of the total tissues). In hot conditions the situation is more difficult. T_r rises steadily and so does average T_s, even though tending towards an asymptote in the last hour. Moreover, it is certain that T_r does not, as in cold or neutral air temperatures, represent the average temperature of 2/3 of the total tissues. Hence where heat is being stored in deep and superficial tissues at different rates, as in the cervical lesion cases in hot conditions, we have refrained from trying to calculate K_{inf} or K_{air}.

TABLE 1. Physical characteristics of subjects

Patient	Site of lesion	Height (ft. in.)	Height (cm)	Weight* (lb.)	Weight* (kg)	Surface area (m^2)
Hardy & du Bois (1941) man	—	5 10½	179	170	77	1·95
Normal control	—	5 11	180	175	79	1·97
Case 1	T8 (complete)	5 8	173	145 (161)	66	1·76
Case 2	T4 (complete)	5 9	175	132 (147)	60	1·70
Case 3	C6–7†	6 0	183	112 (147)	51	1·65
Case 4	C7 (complete)	5 10	178	108	49	1·60
Case 5	C6 (complete)	5 7	170	100 (125)	95	1·50
Case 6	C5–6 (complete)	6 0	183	112 (140)	51	1·65

* The weights of all of the high spinal lesion men could not be obtained because of the difficulty in dealing with men paralysed to this extent. The same loss, 20% of pre-accident weight, is, therefore, assumed in these cases.

† Incomplete lesion at C6–7; spinothalamic tract cut; touch present to L1, 2, 3, on right side but absent on left side.

RESULTS

Neutral air temperature (27° C)

At this ambient temperature the normal control and the patients with lesions at different levels in the spinal cord were able, when exposed naked at rest for 1 hr, to maintain the central body temperature at a constant level. The control subject differed from the patients in that his rectal temperature fell from 37·3 or 37·5° C, the ambulatory level, to 36·9° C in the first half hour, and then remained constant. The patients, being confined to bed, did not show the initial fall. Patients and the control subject were quite comfortable in this air temperature.

The temperature of the extremities, especially of the normal man, altered rapidly and markedly from the pre-experimental level in the first 30 min, and thereafter was relatively constant (see Fig. 1). In the last half hour there was the expected gradient in temperature over the body. Toe temperatures were close to air temperatures, viz. 26–27° C; finger temperatures were 30–32° C and trunk and forehead were 34–35° C. Temperatures at the extremities of the patients were quite different (Figs. 2–4). The toes of the T4 and T8 subjects were the same as the fingers, 30–32° C. The cervical lesion patients had *both* toes and fingers as warm as the trunk.

Average skin temperature of the normal man was somewhat lower than that of the patients because of the lower toe and finger temperatures of the former. The rates of metabolism of the control and the patients were similar. Hence the calculated values of K_{int} and K_{air} are also similar (Table 2). The estimated values in the normal control agree well with those of Hardy and du Bois (1941).

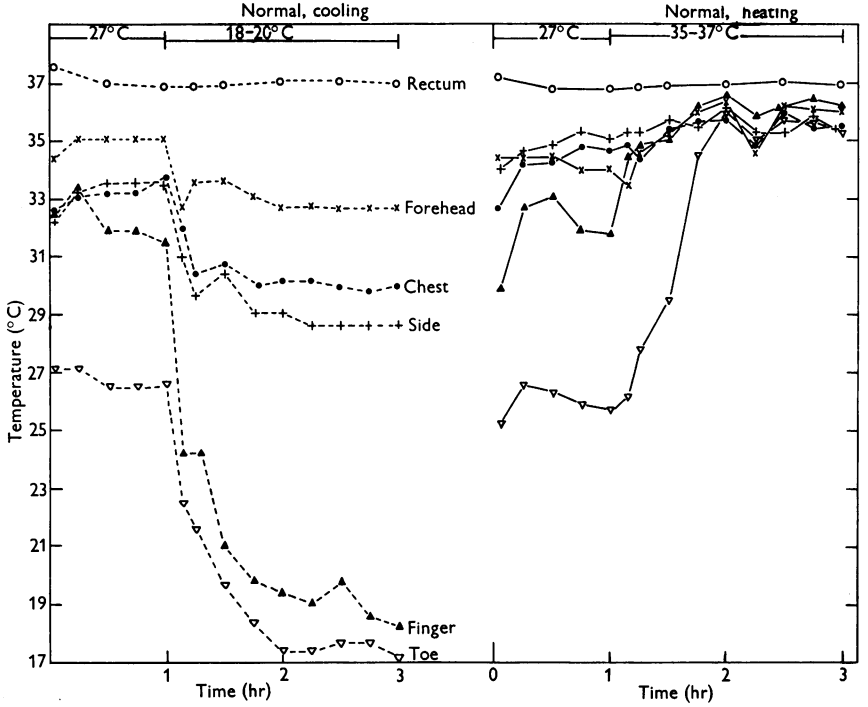

Fig. 1. Temperatures of normal man during 1 hr of rest at 27° C, followed by 1–2 hr of exposure at 18–20° C and 35–37° C.

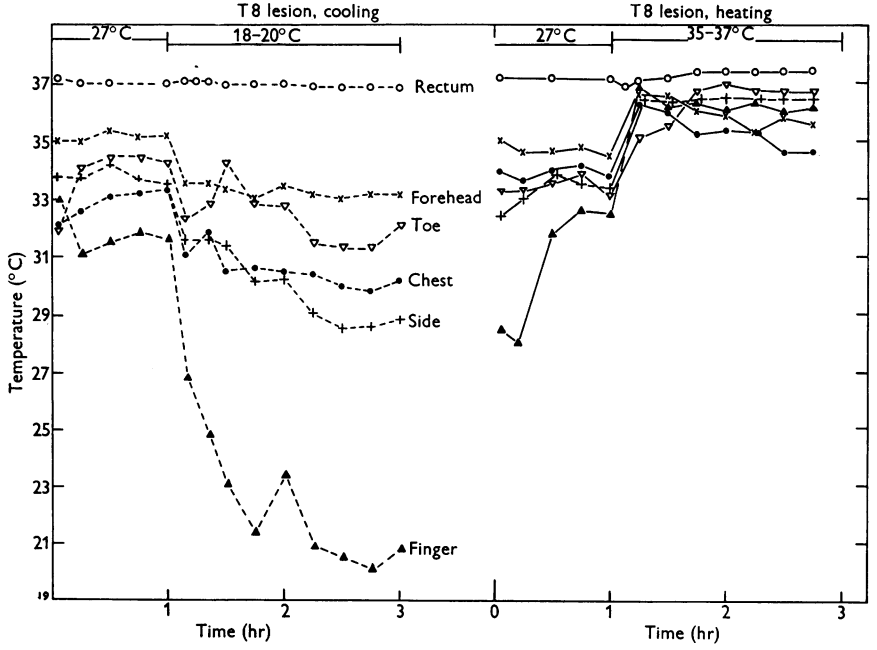

Fig. 2. Temperatures of patient case 1, with lesion below T8.

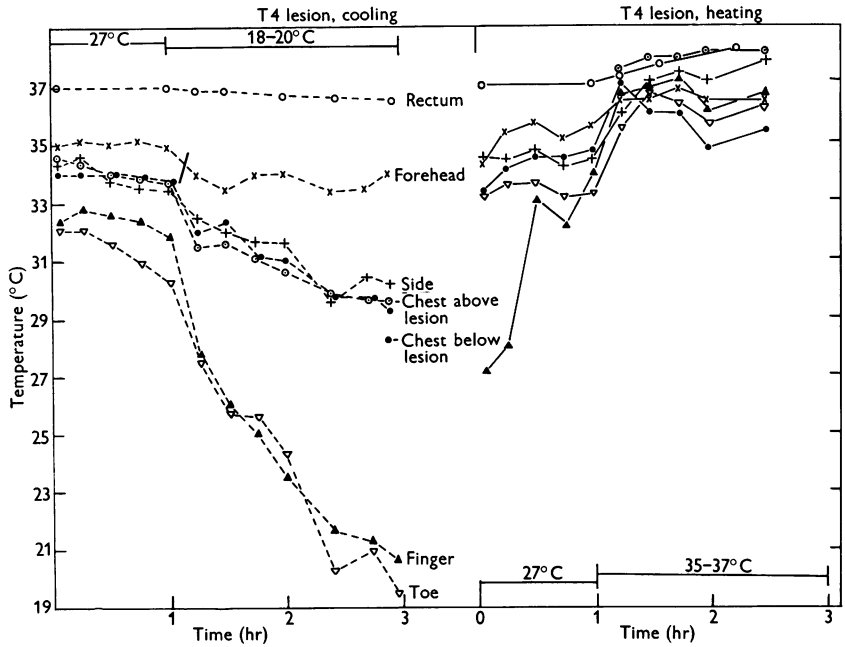

Fig. 3. Temperatures of patient case 2, with lesion below T4.

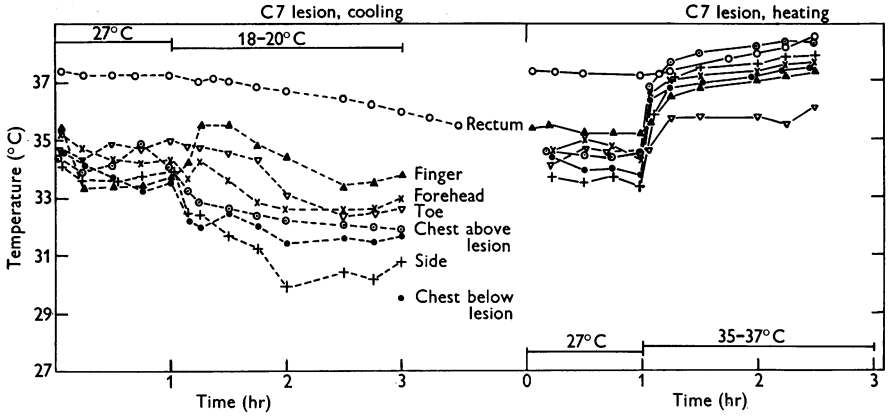

Fig. 4. Temperatures of patient case 4, with lesion below C7.

Air temperature of 18–20° C

The normal control and the patient with the lesion below T8 were able to regulate central body temperature adequately at this ambient temperature, judged by the fact that rectal temperatures were maintained at the same level as in the neutral air temperature during 2 hr of exposure. These levels were 37·0 and 36·9° C respectively (Figs. 1, 2). The other patients did not show an

ability to maintain central body temperature at a constant level. Rectal temperature in the T4 patient fell from 37·0 to 36·5° C in 2 hr (Fig. 3). The rectal temperatures of the cervical-lesion cases, nos. 3–5, fell respectively from 37·3 to 35·5° C in $2\frac{1}{2}$ hr; from 36·8 to 35·8° C in $1\frac{1}{2}$ hr; and from 37·0 to 35·8° C in $1\frac{1}{2}$ hr (Fig. 4).

TABLE 2. Body temperatures and heat conductance values for all subjects

	Mid-hour T_r (° C)	T_r (per hr)	Mid-hour T_s (° C)	$T_r - T_s$	M (cal/m²/hr)	S (cal/m²/hr)	K_{int}	K_{air}	E
			Neutral air temperature (27° C)						
Hardy & du Bois (1941)	36·8	0	32·5	4·3	35·0	0	8·8	6·0	12
Control	36·9	0	32·7	4·2	37·4	0	9·2	6·2	10
T8	37·0	0	33·6	3·4	38·6	0	11·2	5·9	—
T4	37·0	0	33·8	3·2	35·2	0	11·0	5·2	—
C6–7	37·3	0	33·9	3·4	36·8	0	10·8	5·3	—
C7	36·9	0	33·3	3·6	38·0	0	10·5	6·0	—
C6	36·8	0	33·7	3·1	33·6	0	10·8	5·1	—
C5–6	37·0	0	33·4	3·6	33·8	0	9·4	5·3	—
			Cold air (18–20° C)						
Hardy & du Bois (1941)	36·8	0	30·5	6·3	$R+C+E=M+S=55$		8·7	6·0	10
Control	37·0	0	27·2	9·8	40·4	0	4·0	5·8	10
T8	36·9	0	29·7	7·2	63·5	0	8·8	5·9	—
T4	36·6	−0·2	28·5	8·1	42·7	−6·7	6·1	5·2	—
C6–7	36·5	−0·8	31·5	5·0	45·3	−18·5	12·8	5·8	—
C7	36·1	−0·6	30·2	5·9	45·6	−16·6	10·5	5·0	—
C6	—	—	—	—	—	—	—	—	—
C5–6	36·1	−0·7	38·7	5·5	42·6	−19·3	11·3	5·3	—
			Hot air (35–37° C)						
Hardy & du Bois (1941)	37·0	0	35·2	1·8	35·0	0	25·0	6·0	42
Control	37·0	0	35·4	1·6	60·3	0	37·7	—	70
T8	37·4	0	35·7	1·7	55·6	0	32·7	—	—
T4	38·1	+1·1	36·3	1·8	41·6	+37·1	—	—	—
C6–7	38·0	+1·0	37·3	0·7	42·8	+33·6	—	—	—
C7	38·1	+1·2	37·2	0·9	68·3	+61·2	—	—	—
C6	38·1	+1·0	37·4	0·7	42·1	+40·1	—	—	—
C5–6	—	—	—	—	—	—	—	—	—

Mid-hour T_r and T_s were during the last hour of exposure, when relatively steady state was attained in most experiments

$$K_{int}=\frac{M\pm S}{T_r-T_s} \qquad K_{air}=\frac{M\pm S}{T_s-T_a}.$$

Associated with these differences in the ability to maintain central body temperatures in cold air there were marked differences in the thermoregulatory responses observed. The temperatures of the various areas studied in the normal man fell rapidly in the first 15–30 min and then reached a relatively steady level in the second hour of exposure. There were marked differences in the extent of fall in temperature of the different areas. Toes and fingers fell by 12–13° C to within 1–2° C of the ambient temperature, whereas the forehead fell by only 2° C. The patient with the T8 lesion displayed one marked variation

from the normal response: toe temperatures did not fall so far and, in fact, fell only to that of the trunk temperature. In the cervical-lesion patients neither toe nor finger temperatures fell below those of the trunk. In one of the three cervical-lesion patients the toe was actually the warmest part of the body. The patient with a lesion below T4 had a marked fall of both toe and finger temperatures, which contrasted with those of other patients, indicating that the lesion had not severed completely the sympathetic fibres to the lower extremities.

The extent of muscle involved in shivering in relation to time and pattern was very different in the normal man compared with the cervical-lesion patients. The control subject and the T8 man (Table 3) began to shiver in single groups of muscles in different parts of the body by the 20th minute and by the 30th-40th minute there were periods lasting 1–3 min of very vigorous shivering involving the major parts of the muscle mass (but only in muscle above the level of the lesion in the T8 man). Both subjects complained of feeling chilled before each bout of shivering but felt warm immediately afterwards and for some minutes subsequently. The higher level lesion patients began to shiver a few minutes earlier, at about 12–15 min, in the muscles of the shoulder girdles and the neck and face (Table 3). By the 30th–40th minute there was continuous 'chattering of the teeth' and a feeling of deep chill. By the end of the exposure these patients felt uncomfortably and continuously cold. Metabolism of all the subjects studied was raised by shivering. Heat production of the cervical and T4 patients was increased by 50%. The T8 patient doubled his rate of energy production. Each time the control subject was connected to the metabolic apparatus during a bout of shivering the latter promptly ceased. The metabolism recorded therefore was the resting phase with muscles tense owing to cold.

The calculated values of K_{int} were greater in the cervical-lesion patients than in the others and were actually not below the values at 27° C. This is due to the fact that, in the first place, at steady state the temperature gradient T_r to T_s does not fall so markedly and, secondly, the rate of heat transfer (metabolism + loss of stored heat, i.e. $T_r \times 0{\cdot}83 \times 2W/3$) is relatively large. The value of K_{air} in the control subject is less than that given by Hardy & du Bois (1941) in their studies.

K_{air} values are approximately the same in the normal man and in the patients and are similar to those in the neutral temperature zone. They are close to the value of 6·0 cal/m²/hr/° C of Hardy & du Bois (1941).

Air temperature of 35–37° C

At this ambient temperature the normal subject and the T8 lesion patient both controlled the central body temperature adequately. Rectal temperatures of the control were steady at 37·0° C, those of the T8 man rose from 37·1 to

37·4° C in the first 45 min and remained steady thereafter (Figs. 1, 2). Rectal temperatures of all the other patients rose to 38·5–38·6° C in 1½ hr of exposure (Figs. 3, 4).

Associated with these differences in the extent of rise of rectal temperature are clear-cut differences in the regulatory responses examined. Skin temperatures of all areas, except the toe, of the normal man rose to between 35 and

TABLE 3. Time of onset of thermal responses

Time (min)	Control	Spinal lesion
Cold (18–20° C)		
9	Gooseflesh	—
16	—	Neck muscle shivering
20	Shivering, abdomen and thighs	Shivering, chin and left biceps
24	General shivering	Shivering, left deltoid and right biceps
30	Waves of cold and warmth, feet very cold	Feels cold on face, shoulder and cheeks; also a 'deep-seated' feeling of cold
40	—	Teeth chattering continuously
46	Feels warmer following severe shivering	—
60	Very cold, shivering + + (greatly increased)	
75	—	—
85	Feels warmer	—
106	Very cold again	—
Heat (35° C)		
0	Sweating around lips, soles and palms	—
5	Sweat around eyebrows, lower neck and armpits	—
10	Sweat on chin	—
15	Sweat on sternum and dorsal surfaces feet	—
20	Pin-points of sweat on cheeks	—
25	Pin-points of sweat around nipples	—
30	Sweat on forehead, temples and ankles	Cheek and forehead red; no sweat
40	Sweat on inner thigh and around navel	Increased respiratory rate
60	Massive sweating first on sternum, a band around the waist and dorsal surfaces of feet, shins, pubes, inner thighs	Panting definite; very restless
70	Sweat on outer thighs and legs up to knees	Skin red and dry, especially on forehead. Very restless and panting + + (greatly increased)
80	Confluence of sweat	—
90–100	Generalized sweating and apprehensive	—

36° C in the first 15–20 min and then remained relatively steady for the rest of the exposure. Toe temperature took longer to rise to this level and its doing so coincided with the outbreak of generalized sweating. Average T_s in the steady state was 35·4° C. Skin temperature of the T8 lesion patient rose in much the same way but to a higher level initially. At the 40th minute, however, sweating became generalized and profuse above the level of the lesion

and the forehead and trunk temperatures fell drastically. Temperature of the toe and the skin below the lesion continued to rise above 36° C. The mean skin temperature of the *T* 8 patient during sweating was 35·7° C. Skin temperatures of the cervical-lesion patients rose rapidly above 37° C and then tended slowly towards asymptotes at temperatures between 37 and 38° C in the last hour of exposure. Temperatures of all areas, except the toe, were within 1° C of each other. Toe temperatures did not rise above 36° C. This may have been due to the fact that the toe was directly adjacent to the observation window which was opened and closed repeatedly to make observations. The skin of the cervical-lesion patients became scarlet and no sweat could be detected by the quinizarin dye method. This contrasted with the deep blue colour of the sweat areas in the control, and the T 8 and T 4 patients.

Skin temperature and rectal temperature of the control and of the T 8 lesion patient both reached steady levels in the last hour and hence K_{int} could be calculated with confidence. The values 37·7 and 32·7 cal/m²/hr/° C are higher than those of Hardy & du Bois (1941) and are doubtless due to the higher rates of metabolism, 60·3 and 55·6 cal/m²/hr (Table 2).

Both T_r and T_s of the cervical-lesion patients continued to rise at different rates throughout the exposure. It is very difficult to estimate, under these conditions, the proportion of metabolic heat which is being stored in central tissues and that which is transferred to the skin and superficial tissues and stored there, because it appeared unlikely to us that the apportioning of 2/3 of the body tissues to T_r in calculating deep body heat is applicable under these hot conditions. Hence we did not consider it served any useful purpose to calculate K_{int} in these non-steady state conditions.

Average skin temperature of the normal man was 1–2° C below the globe thermometer reading in the hot box and heat was gained therefore by radiation and convection. The gain calculated from the temperature gradient $T_a - T_s$ and the cooling constant (at 27° C) is small and probably did not exceed 10 cal/m²/hr. This gain and the metabolic heat, 60 cal/m²/hr, must, in the steady-state situation, be balanced by the evaporation of sweat. Sweating in the control was at the rate of 200 g/hr, and this would account for a heat loss of 70 cal/m²/hr. Therefore heat gain and heat loss were approximately balanced. The cervical-lesion patients did not sweat and average skin temperatures rose eventually to, or just above, the globe thermometer reading. Hence heat loss by radiation, convection and evaporation was negligible. One would expect therefore that most of the metabolic heat would be stored and result in a rise in T_r and T_s. T_r, in fact, rose by 1·0–1·2° C/hr and T_s more slowly. Calculating stored heat from rise in T_r and T_s and the usual weighting factors (i.e. $(2T_r + T_s)/3$ the gain in heat would account for 80–90% of metabolic heat production. The difference may be due either to heat loss by the respiratory tract or to the factors apportioning heat to T_r and T_s being incorrect.

All the subjects, the control and the patients, complained of a sense of suffocation in the last half hour in the hot box. The high levels of metabolism coincided with restlessness and apprehension on the part of the subjects. All the cervical-lesion patients demanded that the experiments cease at $1\frac{1}{2}$ hr of exposure. At this juncture the cervical-lesion patients had rectal temperatures of 38·5° C and had developed a fast panting type of respiration (Fig. 5).

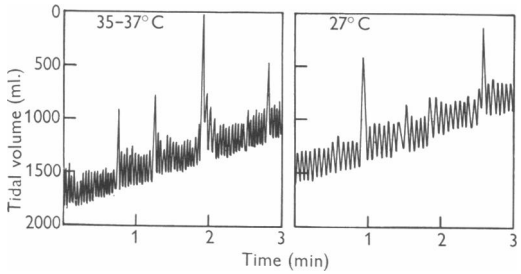

Fig. 5. Spirometer tracing of patient case 4, in the neutral environment and at 35–37° C. Note the increased rate and smaller tidal volume in heat.

DISCUSSION

It is quite clear from these studies that patients with complete transection lesions of the spinal cord above the level of the thoracic sympathetic outflow, who have been bed-patients at the air temperatures which prevail in the non-air-conditioned wards in Britain, are unable to regulate body temperature against heat and cold as well as normal individuals. At an air temperature of 36–37° C, rectal temperature of naked patients with cervical lesions rose to 38·5° C in $1\frac{1}{2}$ hr, and at an air temperature of 18–20° C fell to 35·5° C in $1\frac{1}{2}$–2 hr. Normal man and the patient with the T8 lesion were able to regulate body temperature quite adequately over this range of air temperature. We have reservations about the general applicability of the responses of the T4 patient to others with complete lesions, as we believe that certain of the sympathetic fibres to the toe were present. He had, however, the same rate of rise in rectal temperature in heat but cooled less rapidly in cold air.

The manner in which certain of the heat regulatory mechanisms of the patients failed in the control of body temperature, compared with the normal, are also apparent in this study. On exposure to air temperatures of 35° C, the subjects of Hardy & du Bois (1941) regulated body temperature mainly by means of sweating, but a three- to fourfold increase in conductance of heat through the peripheral tissues was also reported. In cool air (22° C) the subjects of Hardy & du Bois initially lost heat from the periphery, but later maintained heat balance by an increase of metabolism due to shivering. Conductance of heat did not decrease compared with the neutral temperature zone, at 28° C. Our normal control subject had essentially the same responses as those of Hardy & du Bois, except

that conductance at 18–20° C was approximately half the value at 28° C. The difference probably lies in the fact that Hardy & du Bois calculated conductance during the non-steady state—i.e. when stored heat was being lost and therefore $M+S$ was quite large. On the other hand, we were unable to get a value for rate of heat production during shivering of our normal control and the value of M that we obtained is probably much lower than the true average. Had the average rate of M been doubled during the steady level state, and this is quite likely, then the value for conductance would have been closer to that of Hardy & du Bois.

The cervical-lesion cases were quite unable to sweat at 35–37° C. Skin temperature therefore rose to approximately the same level as air temperature and rectal temperature rose rapidly. Metabolism was increased considerably above that observed at the neutral temperature. In part it is probably due to restlessness and apprehension, which we were not able to allay, and probably in part due to the Van Hoff-Arrhenius effect of temperature on tissue metabolism. We are not able in these experiments to distinguish between the contribution of these two factors to the rise in metabolism. From the rate of rise in average body temperature, change in body heat was calculated, using the conventional factors,

$$\frac{2T_r}{3}+\frac{T_s}{3},$$

apportioning heat to deep and superficial areas. This value was relatively close to the rate of metabolism, which is surprising as we did not expect these factors to represent the proportion of tissue at T_r and T_s respectively. The rate of heat storage (S) was 80–90% of rate of production of heat (M); it is probable that the difference could well be accounted for by heat loss from the respiratory tract. Unfortunately we were unable to weigh the cervical-lesion patients accurately or to assess the evaporation through the respiratory channel, and hence have no quantitative data on the effect of the panting which occurred when rectal temperatures of all three of these patients exceeded 38° C. The respiratory rate rose from 15 to 25 min, and tidal volume fell from 500 to 350 ml. The rate of rise of rectal temperature did not appear to be reduced by panting. This could be taken to mean that panting is not an effective channel of heat loss in man. It is probably a direct effect of heat on the temperature regulating centre and similar in this regard to what is seen in the dog on exposure to heat.

On exposure of the cervical-lesion subjects to cool air the temperatures of hands and feet do not fall as in the normal subject and conductance is at the same value as at 27° C. Furthermore, there is a severe limitation, due to the large area of muscle paralysis, upon the extent to which metabolism can be raised by shivering. Rate of heat production is increased by only 50% even when the teeth were 'chattering' continuously. This contrasts markedly with

the 100% increase in the T8 subject and the 200–300% reported by Adolph & Molner (1946) in normal subjects exposed to cold. The failure to increase the conductance of the peripheral tissue and the failure to raise metabolism in the face of an augmented rate of heat loss from the surface at 20° C compared with 27° C both result in a relatively rapid fall in rectal temperature. Presumably at the tissue conductance values of 10 cal/m²/hr/° C and at a maximum rate of metabolism of 40 cal/m²/hr, rectal temperature would continue to fall until a 4° C gradient existed between skin and rectal temperature. We did not feel justified in testing this point because of the discomfort experienced by the cervical-lesion patients at a rectal temperature of 35·5° C. The value of K_{air}, the cooling constant of Hardy & du Bois, is the same at 18–20° C as at 27° C. This fact lends us confidence that our experimental techniques, although much cruder than theirs, approach in these respects their exemplary efforts.

At 35–37° C air temperature the patient with a lesion below T4 did not maintain any better control of rectal temperature than the cervical-lesion cases. Rectal temperature rose at exactly the same rate as in these latter patients. There was some sweat on the neck and head but it is clearly insufficient to alter materially the rate of heat loss from the body surface as the rate of metabolism per unit surface area was the same as those of the cervical-lesion patients. In cool air this patient had better control than the cervical patients, as judged by the fact that rectal temperature did not fall so rapidly. The rate of metabolism was not different from that of the cervical-lesion patients but the temperatures of both toes and fingers fell to near air temperature and hence conductance was decreased. The fall in toe temperature must be interpreted to mean that the cord lesion was not complete and that some part of the sympathetic vasoconstrictor fibres to the toes was intact. This was the only clinical sign of the incompleteness of the lesion. The rate of cooling of patients with a complete lesion below T4 may therefore be greater than shown by this patient.

The patient with a lesion below T8 behaved essentially as a homoeotherm in hot and cold air. In cold air toe temperature did not fall but conductance of peripheral tissues was essentially the same as at 27° C. Shivering increased metabolism by 100% and hence heat loss was balanced by this means. In hot air sweating was vigorous on the skin above the lesion and the area was extensive enough for evaporation to balance the rate of heat production.

From these studies we can conclude that patients with chronic transverse lesions of the spinal cord at the lower cervical segments have relatively little control of rectal temperature against heat or cold when exposed naked. The T4 lesion patient is not better off in heat but appears to cool less rapidly in cold air. The T8 subject regulates as well as a normal man, despite the fact that there is no vasoconstriction to cold or sweating to heat in the lower extremities.

Our results on temperature regulation in man are in essential agreement with those of Sherrington (1924) on dogs. Our observations did not support

Foerster's theory (1936) that the spinal grey matter reacts to blood temperature in the same way as the hypothalamus and can promote heat-regulating mechanisms, such as sweating. We would agree with Erickson (1939), Ranson (1940) and others that the disparity between Foerster's hyperthermia and Holmes's hypothermia (1915) in patients with complete lesions of the cervical cord is explained mainly by the difference in the method of nursing—i.e. whether these patients are kept in hot or cold environments. Our patients were conditioned to ordinary ward temperatures: hence the conclusions do not exclude the possibility that, had the cervical-lesion patients been conditioned to live in lower temperatures (say, 15–20° C), they might then have shown better adjustment to cold exposure. In this connexion, it may be remembered that, in Clark's (1940) experiments, cervical spinal cats which had been kept for some time at 27–30° C were more easily chilled than were the same cats when previously accustomed to environmental temperature at 21° C.

SUMMARY

1. At a neutral air temperature, 27° C, patients with transverse spinal cord lesions below C6–T1, below T4 and below T8 maintain rectal temperature constant at approximately 37° C. Metabolic rate, K_{air} and K_{int}, are similar to those of normal man in the same conditions.

2. In cool air, 18–20° C, the normal man and the patient with T8 lesion maintain constant body temperature by cooling of the extremities, an initial fall in body heat and a rise in metabolism due to shivering. Cervical-lesion subjects cool rapidly, rectal temperature falling to 35·5° C in 2 hr owing to the fact that shivering raises metabolism by only 50% and extremities remain warm.

3. In hot air, 35–37° C, the normal and T8 men maintain a constant body temperature due mainly to evaporation of sweat. Cervical-lesion patients do not sweat over the body and rectal temperature rises rapidly to 38·5° C in $1\frac{1}{2}$ hr, when distress and panting are present.

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