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BODY TEMPERATURE, SHIVERING, BLOOD PRESSURE AND HEART RATE DURING A STANDARD COLD STRESS IN AUSTRALIA AND ANTARCTICA

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SUMMARY

1. Four men of European descent were exposed naked to an air temperature of 10° C for 2 hr in Australia, and again after 24 weeks' residence at Mawson, Antarctica.

2. Their ability to maintain rectal temperature during the test cold exposure significantly improved at Mawson. Shivering and cold diuresis did not change. The response of skin temperature did not change significantly except for a small increase in toe temperature. Bradycardia caused by the cold exposure was significantly greater at Mawson, but the rise in blood pressure did not change. Spontaneous fluctuations in rectal temperature that occurred during the cold exposure were intensified at Mawson.

3. The results confirm those of a previous study at Mawson, and are attributed to general acclimatization to cold. It is suggested that tissue insulation increased as a result of enhanced vasoconstriction.

INTRODUCTION

Acclimatization to cold is readily induced in animals (Hart, 1963) but not in man (Edholm, 1960; Rodahl, 1960), and the nature of human acclimatization is still uncertain. Recent work in polar regions has shown significant changes, suggestive of general acclimatization, in men's responses to a test cold exposure. Decreased heat production during the winter was observed by LeBlanc (1956) in the Arctic, and by Milan, Elsner & Rodahl (1961) and Wyndham & Plotkin (1963) in the Antarctic; and Davis (1963) observed decreased shivering in the Arctic. Skin temperatures were increased (Milan *et al.* 1961; Wyndham & Plotkin, 1963) or

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unchanged (LeBlanc, 1956; Davis, 1963), and rectal temperature was unchanged except in LeBlanc's study, in which it decreased.

In 1959 men's responses to a standard 90 min cold exposure were investigated (Budd, 1962, 1964) at Mawson (lat. 67° 36′ S, long. 62° 53′ E), a station of the Australian National Antarctic Research Expeditions (ANARE). A highly significant improvement occurred in the subjects' ability to maintain rectal temperature, and was attributed to general acclimatization. Heat production, shivering, and skin temperature did not change significantly, although extremity temperatures tended to be lower in Antarctica. The evidence suggested that a smaller heat debt was incurred in Antarctica, and it was concluded that tissue insulation had increased, as a result of vasomotor changes.

This paper reports further experiments done at Mawson in 1964, using the same methods as in 1959. The results confirm and extend the previous findings, and provide evidence of acclimatization for concurrent studies of adrenal function, and of the response to infused noradrenaline (Budd & Warhaft, 1966), made on the same subjects.

METHODS

Four members of the 1964 Mawson party were exposed to a standard test cold exposure before and during their year in Antarctica. Two complete series of exposures were carried out: series 1 in Melbourne in midsummer, 2 weeks before the subjects sailed for Antarctica, and series 2 at Mawson in late winter, 24 weeks after their arrival. In each series the same four subjects were exposed twice. An additional series (series 3) was begun in early spring, 31 weeks after arrival, but was not completed; only one subject (*B*) was exposed twice, and one (*F*) was not exposed at all.

TABLE 1. Characteristics of the subjects

Subject	Age (yr)	Occupation	Height (cm)	Weight (kg) Series 1	Series 2	Series 3	Mean
B	25	Glaciologist	170	64·2	67·8	67·7	66·6
D	25	Diesel mechanic	178	85·5	83·7	82·8	84·0
F	29	Surveyor	175	60·2	67·2	*	63·7
M	36	Officer-in-charge	183	79·8	80·8	82·8	81·1
Mean	29	—	176	72·4	74·9	—	73·9

* Not observed.

Subjects. The subjects were healthy men of European descent, aged between 25 and 36 yr (Table 1). Two of them had previously wintered in Antarctica (*M* in 1961, *B* in 1962), but all had spent the year 1963 in a temperate climate.

Exposure to cold at Mawson. The main features of the Mawson climate during the period of the study (February to October) are shown in Fig. 1. Average air temperature was −15° C and average windspeed 11·2 m/sec (25 miles/hr). Sunshine was recorded for an average of 1·8 hr/day, although this is an underestimate because the Campbell–Stokes sunshine recorder used did not record when the sun was less than 10° above the horizon. Drifting snow

was recorded on 58 % of the days. By contrast, during the 3 months before the expedition's departure the air temperature in Melbourne averaged 20° C. Indoor air temperature at Mawson was usually near 20° C, and most men dressed lightly.

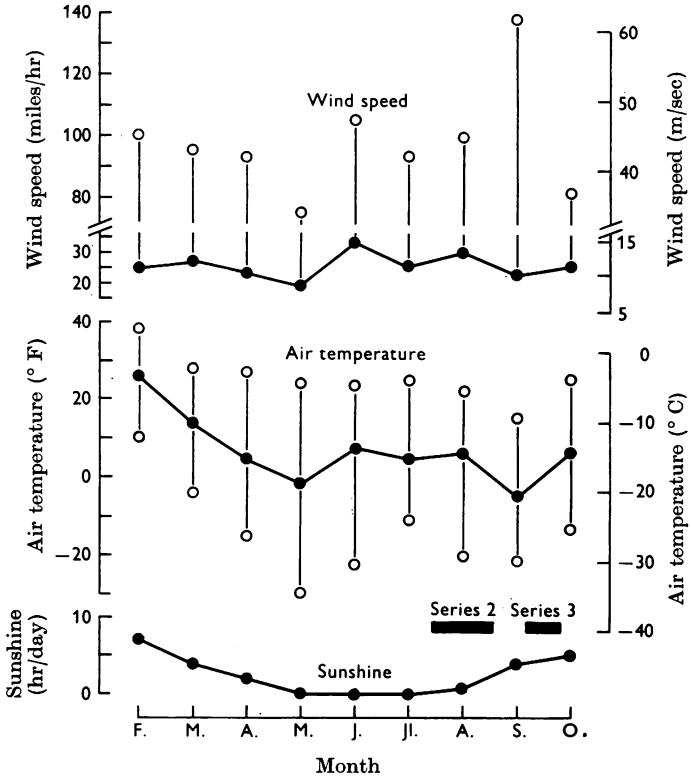

Fig. 1. Average (●) and extreme (○) monthly windspeed, air temperature and sunshine at Mawson during the period of the investigation in 1964. The horizontal bars show the time of series 2 and 3. Sunshine was not recorded when the sun's elevation was less than 10° above the horizon. (Courtesy of Commonwealth Bureau of Meteorology.)

Living conditions at Mawson have been described in detail elsewhere (Budd, 1964). The station consists of more than thirty huts, 10–400 m apart, and in moving between them men were exposed to the weather 1–2 min, at least a dozen times every day. With the notable exception of subject *B*, they seldom bothered to don outdoor clothing for such short exposures unless a blizzard was blowing, and consequently became superficially chilled each time. Camp duties, such as supplying the huts with fuel and water, disposing of waste, and looking after the sledge dogs, entailed regular short exposures to cold, and field trips entailed prolonged exposures. Subjects *B* and *F* made a 6 week journey of 800 km in autumn, in an average air temperature of −28° C and wind speed 10·3 m/sec (23 miles/hr), and subject *D* went on four trips of a few days each. The outdoor clothing normally worn does not provide complete protection from the cold (Milan *et al.* 1961; Budd, 1966), and during periods of light work, such as driving tractors or repairing equipment, the subjects often felt cold. Subject *B*, however, was exceptional in that he dressed very heavily, and always wore his

customary outdoor clothing when moving between huts. He reported that even on long field trips he never felt cold, except on the exposed part of his face.

Test cold exposures. The Melbourne experiments were done in a refrigerated room provided by the Commonwealth Scientific and Industrial Research Organization's Division of Building Research. The Mawson experiments were done in the hospital hut, which was warmed to the desired temperature by electric heaters.

Experimental procedure was the same in each series. All experiments were done in the morning, after the subject had had a sleep of at least 8 hr. He ate a standard breakfast (of low protein content) and then came to the laboratory, where he emptied his bladder, undressed, and was weighed, in a warm room. Thermistors were attached to the left index finger, forearm, shoulder, forehead, abdomen, medial thigh, and big toe. A rectal thermistor, mounted on a stiff catheter, was inserted to a depth of 10 cm, and a mask was fitted for the collection of expired air. The subject then moved to the adjacent cold room, where he lay, wrapped in blankets, on a mattress of 5 cm nylon mesh suspended 50 cm from the floor. A sphygmomanometer cuff was fitted to the right arm and a stethoscope diaphragm taped to the right cubital fossa.

When preparations were complete and the subject comfortable (usually 15–20 min), the 'warm phase' observations were begun. After 30 min the blankets were rapidly removed, and for the next 2 hr (the 'cold phase') the subject lay quietly, naked but for a short pair of cotton underpants, at an air temperature of 10° C. At the end of this period he dressed in the warm room and again emptied his bladder. All thermistors were removed except those on the abdomen and thigh and the one in the rectum: these were retained for 45 min (the 'rewarming phase'), during which period observations were made every 15 min. In Melbourne the rewarming phase was finished before the subject ate or drank anything, but at Mawson the 30 min and 45 min observations were usually made while he was eating lunch. Air temperature during rewarming was some 5° C higher in Melbourne than at Mawson.

During the cold phase the air temperature was read every 5 min from a thermometer mounted at the level of the subject, and the average calculated. Relative humidity was determined with a whirling hygrometer, halfway through the cold phase. Air movement in the positions vacated by the subject's feet, trunk and head was measured with a katathermometer at the end of the exposure and the average calculated. Mean radiant temperature of the surroundings was determined at the same time with a globe thermometer. The technique in each case was that described by Bedford (1946). The results (Table 2) show that the ambient conditions were well controlled, and that the test cold stress was the same in each series. Before each series all thermometers and thermistors were calibrated, in a well-stirred water-bath, against a standard thermometer, which was itself calibrated by the National Standards Laboratory in Sydney.

TABLE 2. Ambient conditions during the test cold exposures. Mean values and ranges for all observations in each series

	Series 1	Series 2	Series 3
Number of exposures	8	8	4
Air temperature (° C)	10·2 (9·7–11·7)	10·4 (9·7–10·7)	10·2 (9·8–10·7)
Mean radiant temperature (° C)	10·0 (9·2–10·5)	10·3 (9·6–10·4)	9·6 (9·0–10·0)
Air movement (cm/sec)	15 (13–16)	15 (8–20)	12 (10–14)
Relative humidity (%)	72 (67–80)	62 (50–74)	84 (80–90)

Techniques. Rectal and skin temperatures were read in the same order every 5 min during the warm and cold phases, by means of thermistors and a null-reading Wheatstone bridge. The smallest change that could be measured was 0·06° C for rectal temperature and 0·1° C

for skin temperature. Shivering was estimated each minute during the cold phase, by the same observer in every exposure, and recorded according to a 6-point scale (0–5), corresponding to none, mild, moderate, severe, very severe, and violent. The time of onset of shivering was carefully noted. Heart rate was determined every 5 min during the warm and cold phases by counting the radial pulse, for 15 sec at Mawson and 30 sec in Melbourne. Blood pressure was measured with a mercury sphygmomanometer every 5 min during the warm and cold phases, by the same observer in every exposure, using the standard technique described by Wood (1956).

Oxygen consumption was determined every 15 min during the warm and cold phases. On each occasion, expired air was collected for 10 min in a Douglas bag, the volume measured with a dry gas meter, and the oxygen and carbon dioxide content determined with a Scholander 0·5 ml. analyser. Unfortunately, at Mawson it proved impossible to obtain an air-tight fit of the face mask because the subjects had grown beards, which they were unwilling to shave off. The resulting leaks made it necessary to discard all the Mawson results for oxygen consumption. Subjective impressions of cold during the exposure were noted in an interview immediately afterwards.

Urine flow rates. All urine passed was collected over the following four periods:

1. 'Pre-exposure period'. The 12 hr before the subject's arrival at the laboratory.
2. 'Exposure period'. The 3½ hr between arrival and the end of the cold phase.
3. The 4 hr following the end of the cold phase.
4. The next 6 hr.

The results for collections 3 and 4 are combined, in this paper, to give a single 'post-exposure period'. Collection times varied somewhat, but the exact time of beginning and ending each collection was noted, and the volume accurately measured, to give a reliable value for the average rate of urine flow. A 100 ml. sample was taken from each collection, acidified, and stored for later analysis in Australia.

Analysis of results. The results from all four subjects were used in the averages and statistical analyses, except for skin temperature of the finger, forearm, abdomen and thigh, in which the warm-phase and cold-phase results from only three subjects were used. Subject *B*'s results at these sites were discarded because they were grossly affected by his habit (which no other subject shared) of flexing his legs, and resting his hand and forearm on his abdomen, during the cold exposures.

Differences between series 1 and 2, and between subjects, were tested by means of a two-factor analysis of variance (Snedecor, 1961), and correlations between variables were tested by analysis of covariance (Wishart, 1950). The warm-phase results used were the averages of all observations of blood pressure and heart rate, and of the last two observations (10 and 5 min before the beginning of the cold exposure) of skin and rectal temperature. The cold-phase results were the averages of all observations of blood pressure, heart rate and shivering, and the final values (after 120 min of cold exposure) of skin and rectal temperature. In addition, the results for skin and rectal temperature after 90 min in the cold were analysed, to permit comparison with the results of the 1959 study at Mawson. Absolute values were used, except in the case of rectal temperature, which was expressed as change from the corresponding warm-phase value.

RESULTS

Body temperature

Warm phase. Rectal temperature was significantly lower at Mawson (Table 3), the mean values being 37·05° C in series 1 and 36·68° C in series 2. Finger temperature was significantly higher (Fig. 2) in series 2 (31·6° C)

TABLE 3. Body temperatures. Significance of differences between series in analyses of variance

		Cold phase	
	Warm phase	90 min*	120 min*
Rectal temperature	$0{\cdot}005 > P$	$0{\cdot}05 > P$	$0{\cdot}10 > P > 0{\cdot}05$
Skin temperature:			
Toe	N.S.	$0{\cdot}05 > P$	$0{\cdot}10 > P > 0{\cdot}05$
Shoulder	N.S.	N.S.	N.S.
Forehead	N.S.	N.S.	N.S.
Finger†	$0{\cdot}01 > P$	N.S.	N.S.
Forearm†	N.S.	N.S.	N.S.
Thigh†	N.S.	N.S.	N.S.
Abdomen†	$0{\cdot}10 > P > 0{\cdot}05$	N.S.	N.S.

N.S. not significant.
* Time since onset of test cold exposure.
† Three subjects only: subject *B* excluded.

than in series 1 (25·5° C). Abdomen temperature was 0·4° C warmer in series 2, the difference approaching significance ($0{\cdot}05 < P < 0{\cdot}10$).

Cold phase. The ability to maintain rectal temperature improved significantly at Mawson (Fig. 3). In series 1 an initial fall was followed by a transient rise and then a continuing fall, to a final (120 min) value of −0·33° C. In series 2 the rise was earlier and higher and was sustained longer, thereafter falling to a final value of −0·16° C. The incomplete series 3 closely resembled series 2. The difference between series 1 and series 2 was significant ($P < 0{\cdot}05$) after 90 min of cold exposure, and approached significance ($0{\cdot}05 < P < 0{\cdot}10$) after 120 min. There were significant differences between subjects in the ability to maintain rectal temperature, and each subject responded in a characteristic fashion (Fig. 4). Wide fluctuations in rectal temperature occurred at intervals throughout the cold exposures at Mawson (Fig. 4), in contrast to the more even rate of change in Melbourne; they occurred in all subjects, and in series 3 as well as in series 2. In one instance rectal temperature fell by 0·5° C in 10 min, then rose by 0·4° C in the following 5 min.

The only significant change in skin temperature was that of the toe (Fig. 2) which was 0·8° C warmer in series 2 after 90 min of cold exposure ($P < 0{\cdot}05$), and 0·5° C warmer after 120 min ($0{\cdot}05 < P < 0{\cdot}10$). Forehead temperature remained within 1° C of its initial value throughout the cold exposure, in both series.

Rewarming phase. For the first 30 min after the end of the cold exposure, whilst the skin temperature rose quickly, rectal temperature fell steeply, to almost 1° C below warm-phase values (Fig. 5). Both then rose slowly, but were still well below warm-phase values after 45 min. There were striking differences between series in the first 15 min, when rectal temperature fell much further from its 120 min value in series 2 (0·62° C) than

in series 1 (0·26° C), and skin temperature rose less in series 2 (2·9° C) than in series 1 (4·0° C). After 30 min rectal temperature was similar in each series, but skin temperature remained about 1° C lower in series 2 than in series 1.

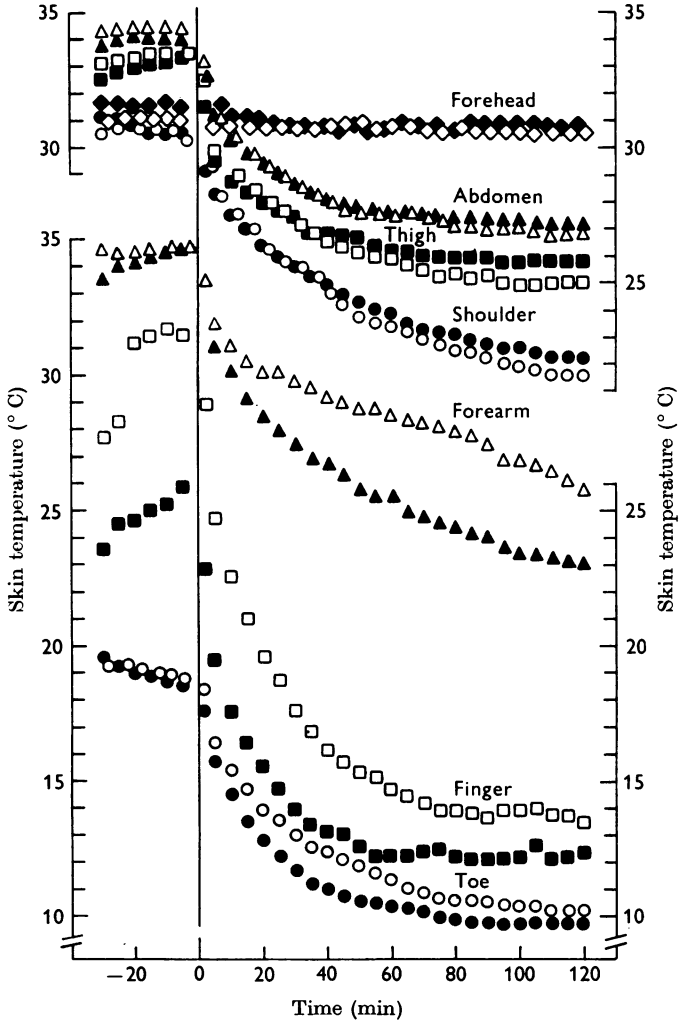

Fig. 2. Skin temperature. Mean values in Series 1 (◆■●▲) and Series 2 (◇□○△). Subject *B* is excluded from the averages for finger, forearm, thigh and abdomen. Zero time indicates the commencement of the cold phase.

Shivering and oxygen consumption

The intensity of shivering, and the manner in which it developed throughout the test cold exposure, were similar in both series (Fig. 6).

Shivering began after 6 min of cold exposure in series 1, and after 5 min in series 2. It increased rapidly in the first 10 min and then more gradually until 80 min, after which it remained fairly steady until the end of the exposure. The average 'shivering score' for all subjects was 2·42 units in series 1 and 2·33 units in series 2; two subjects shivered more in series 2 than in series 1 and the other two shivered less.

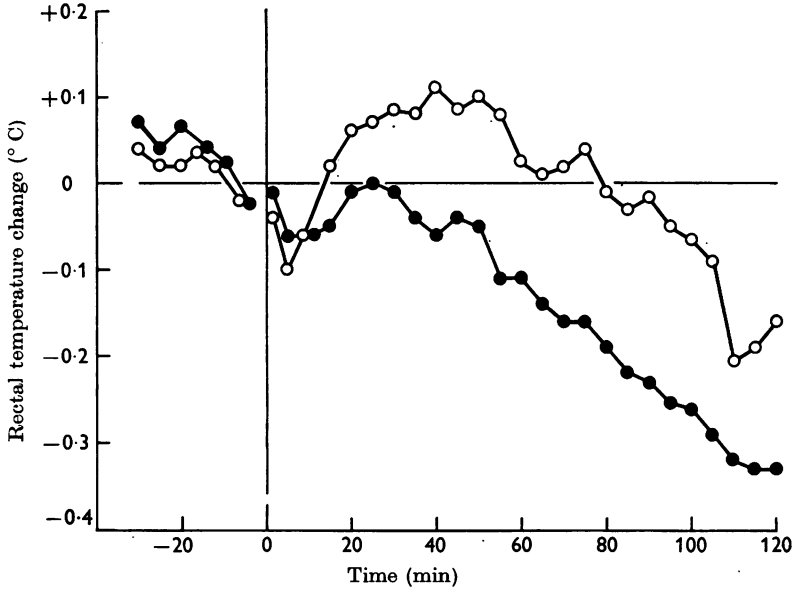

Fig. 3. Rectal temperature change in series 1 (●) and series 2 (○). Mean values of the eight exposures in each series. Zero time indicates the commencement of the cold phase.

Oxygen consumption was determined reliably only in series 1. The average warm-phase value was 246 ml./min (38·0 kcal/m².hr), and the average maximum value in the cold phase was 501 ml./min (77·4 kcal/m².hr). The pattern of the increase in oxygen consumption was very similar to that of shivering (Fig. 6). Moreover, of each subject's two replicate exposures, the one with the higher shivering score was also the one with the greater oxygen consumption.

Blood pressure and heart rate

Systolic and diastolic blood pressure rose in response to the test cold exposure (Fig. 7), the increase in diastolic pressure being twice as great as that of systolic pressure (Table 4). The increase was similar in both series, but systolic blood pressure in both the warm and cold phases was significantly lower in series 2 than in series 1. Average heart rate slowed

in both series, but by a significantly greater amount in series 2 (7 beats/min) than in series 1 (2 beats/min); there was no significant difference between series in the warm phase.

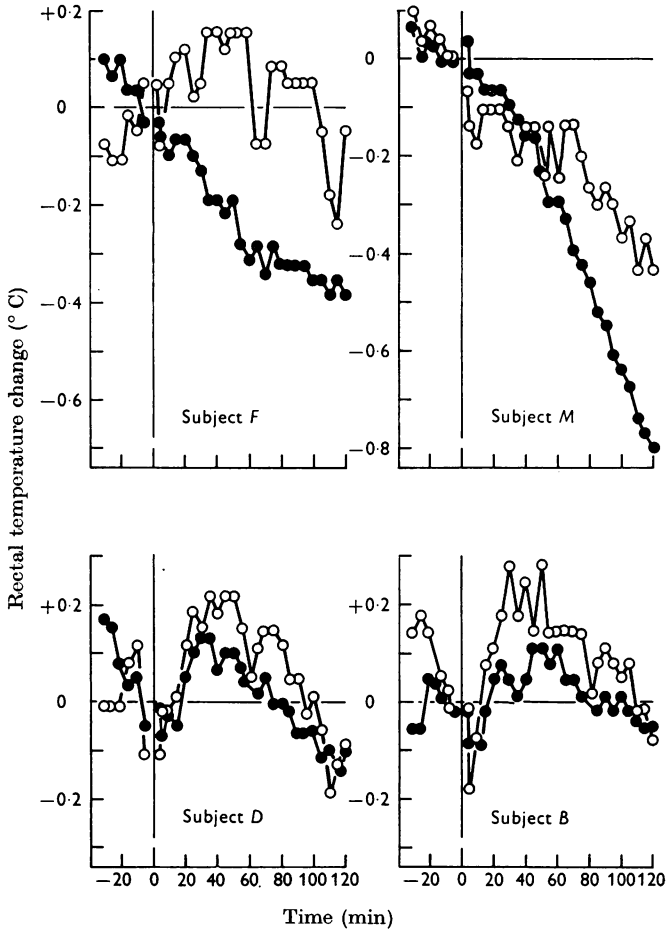

Fig. 4. Rectal temperature change in series 1 (●) and series 2 (○). Individual values (mean of two replicates) in each series. Zero time indicates the commencement of the cold phase.

Urine flow rate

For three of the subjects urine flow rate during the 'exposure period' was 2–3 times greater than in the 'pre-exposure' and 'post-exposure' periods (Fig. 8), but for one subject (*M*) it was most often 8 times greater. There were no consistent differences between series: the increase during the 'exposure period' was greater at Mawson than in Melbourne for subjects *F* and *B*, less for subject *D*, and unchanged for subject *M*.

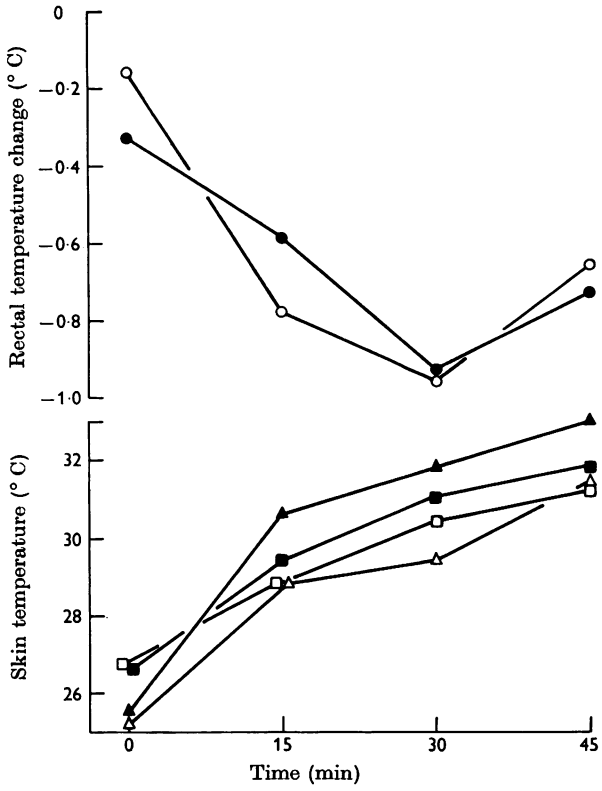

Fig. 5. Rewarming phase. Rectal temperature change (● ○) from warm-phase values, and skin temperature of abdomen (■ □) and thigh (▲ △), in series 1 (● ■ ▲) and series 2 (○ □ △). Mean values of the eight exposures in each series. Zero time indicates the end of the cold phase.

DISCUSSION

General acclimatization to cold

The main finding of this study is that at Mawson the ability to maintain rectal temperature during acute cold stress improved. The absence of any change in shivering makes it unlikely that heat production changed, because shivering was directly related to heat production in series 1, and in all series of the 1959 study at Mawson (Budd, 1964). The results are not attributable to changes in body fat or physical fitness: the changes in rectal temperature response were not correlated with changes in body weight, which themselves are usually correlated with changes in fatness (Edwards, 1950; Lewis, Masterton & Rosenbaum, 1960); and it has been shown (Adams & Heberling, 1958; Heberling & Adams, 1961; Keatinge, 1961) that an improvement in physical fitness—which almost certainly

developed at Mawson—does not affect the ability to maintain rectal temperature during acute cold stress.

These results confirm those obtained at Mawson in 1959. The nature of the change in the response of rectal temperature was the same in both studies. Its average rate of development, per week in Antarctica, was similar—the ability to maintain rectal temperature during the first 90 min of the test cold exposure improved at the rate of 0·011° C per week in 1959, and 0·009° C per week in the present study. Since in the more extensive 1959 study the improvement was shown to be related to the environmental cold stress, and rapidly decayed when the subjects returned to the warm climate of Australia, it is reasonable to conclude that in both studies the changes observed indicated general acclimatization to cold.

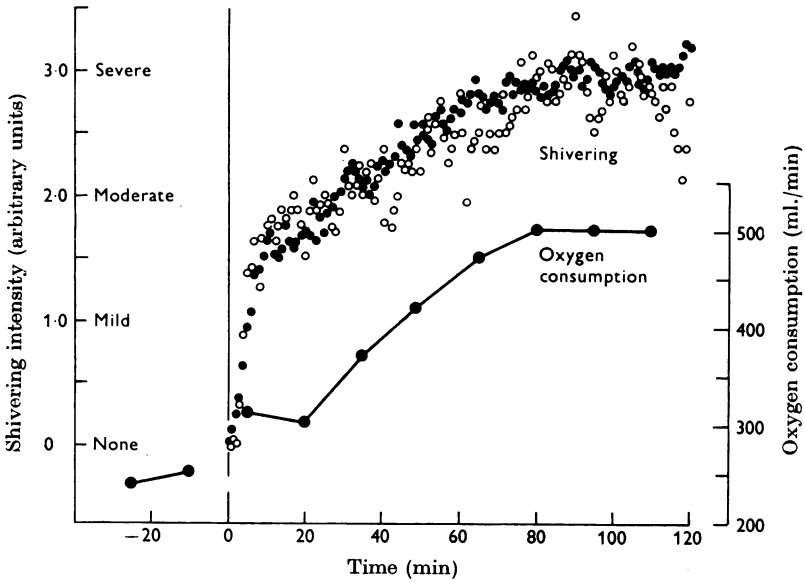

Fig. 6. Shivering and oxygen consumption. Mean values in series 1 (●) and series 2 (○). Oxygen consumption was determined in series 1 only. Zero time indicates the commencement of the cold phase.

A transient rise in rectal temperature often occurs when men are abruptly exposed to cold, and is considered to be caused by either a reduced return of cooled venous blood (Aschoff, 1944; Bazett, Love, Newton, Eisenberg, Day & Forster, 1948) or a compensatory vasodilatation in the intestinal blood vessels (Grayson, 1950, 1951), as generalized vasoconstriction develops in the peripheral vessels. Similarly, the steep fall in rectal temperature that usually occurs when chilled men are rewarmed is considered (Bazett *et al.* 1948; Behnke & Yaglou, 1951) to be caused by

TABLE 4. Blood pressure and heart rate. Mean values of the eight observations in each series

	Series 1	Series 2
Systolic blood pressure (mm Hg)		
Warm phase*	134	125
Cold phase*	141	131
Change	7	6
Diastolic blood pressure (mm Hg)		
Warm phase	77	81
Cold phase	91	92
Change	14	11
Heart rate (beats/min)		
Warm phase	65	66
Cold phase**	63	59
Change**	−2	−7

* Difference between series significant at the 5 % level.
** Difference between series significant at the 1 % level.

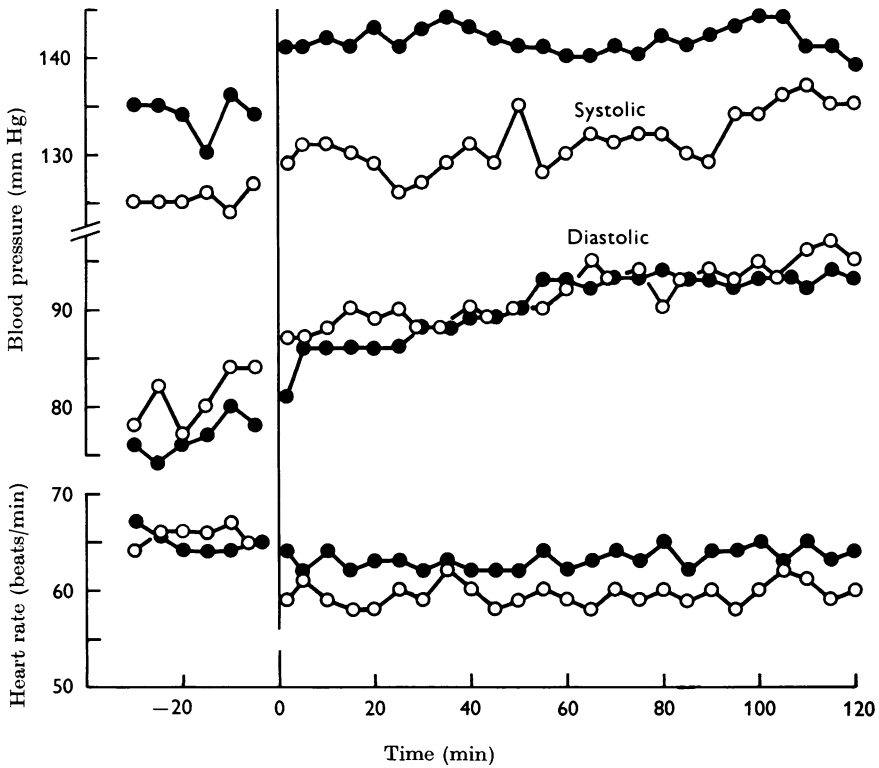

Fig. 7. Blood pressure and heart rate. Mean values in series 1 (●) and series 2 (○). Zero time indicates the commencement of the cold phase.

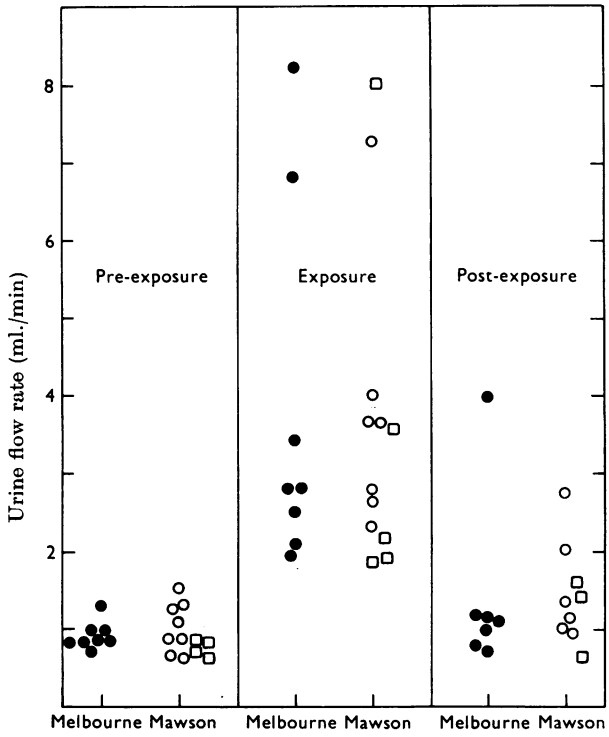

Fig. 8. Average rate of urine flow during the pre-exposure, exposure, and post-exposure periods, in series 1 (●), series 2 (○) and series 3 (□). Each point represents one observation.

the renewed flow of blood through the cold peripheral tissues as vasoconstriction relaxes. It was therefore suggested (Budd, 1964) that the changed response of rectal temperature observed after acclimatization at Mawson in 1959 indicated a more prompt, intense and sustained peripheral vasoconstriction than had occurred before acclimatization, and that this enhanced vasoconstriction, probably accompanied by more efficient heat exchange between arteries and venae comites, was a likely cause of the increased tissue insulation implied by the results for rectal temperature and heat production.

This hypothesis is equally applicable to the results of the present study, and is supported by the changes observed during the rewarming phase. Relaxation of vasoconstriction during rewarming was accompanied by a greater fall of rectal temperature at Mawson than in Melbourne, suggesting that at Mawson the vasoconstriction had been more intense. The results for skin temperature during the cold phase, however, appear to contradict

the hypothesis. Increased tissue insulation would seem to imply lower skin temperatures, yet the only change approaching significance was that toe temperature was slightly warmer at Mawson; nor were there any significant changes in skin temperature in the 1959 study. However, other workers (Adams & Covino, 1958; Heberling & Adams, 1961; Milan *et al.* 1961; Davis, 1963; Wyndham & Plotkin, 1963) have also observed skin temperatures that seemed to be inconsistent with their results for deep body temperature and heat production, and Davis (1963) concluded that measurements of skin temperature had been of little value in his studies of acclimatization. A number of explanations have been proposed (Adams & Covino, 1958; Milan *et al.* 1961; Carlson, 1963; Budd, 1964), based on changes in shivering, in the distribution of skin temperature, and in countercurrent heat exchange between arteries and venae comites. Moreover, the expectations for skin temperature in these studies have been based on Burton's simplified theory (Burton, 1934) of heat flow in the animal body, which presupposes that the subjects are in thermal equilibrium. In most cases the subjects were not in thermal equilibrium but were still cooling, and this fact might well account for the paradoxical results obtained.

Results suggestive of increased tissue insulation have been obtained in studies of seasonal acclimatization in Japan (Yoshimura, 1960) and Korea (Hong, 1963), and in a study of men exposed to wet-cold conditions on an Antarctic island (Budd, 1965). Moreover, it seems quite probable, as suggested by Carlson (1963), that the decreased heat production (LeBlanc, 1956; Milan *et al.* 1961; Davis, 1963; Wyndham & Plotkin, 1963) and decreased shivering (Davis, 1963) reported in some previous studies of acclimatization may also have been due to increased tissue insulation, which would have reduced the requirement for increased heat productiou in the cold.

The abrupt fluctuations in rectal temperature that occurred at intervals throughout the cold exposures at Mawson (Fig. 4) were many times greater than the least change measurable with the instruments used, and were not due to changes in technique or observers. They are attributed to variations in vasomotor tone, an abrupt fall in rectal temperature signifying peripheral vasodilatation and a subsequent rise a renewed vasoconstriction (Bazett *et al.* 1948). Similar fluctuations were observed at Mawson in 1959, sometimes correlated with changes in skin temperature; and the same phenomenon has been described by Grayson (1949), and by Cannon & Keatinge (1960). These recurrent episodes of vasodilatation bear an interesting resemblance to the periodic abrupt changes in vasomotor tone described by Burton & Taylor (1940), and to the episodes of cold vasodilatation that occur in the extremities during local cooling

(Burton & Edholm, 1955). Periodic changes in slope, and even some small fluctuations, are apparent in the curve of rectal temperature in some of the Melbourne exposures as well (Fig. 4), suggesting that the wider fluctuations observed at Mawson were simply an exaggeration, caused by the enhanced vasoconstriction of acclimatization, of a normal cyclic variation in the vasomotor response to cold.

Other observations

The high systolic blood pressure in series 1 was probably due to a combination of apprehension about the experiment and stress associated with the impending departure of the expedition, as noted in a previous study (Budd, 1965). When men are exposed to whole-body cooling their heart rates may rise (Adolph & Molnar, 1946), fall (Arnett & Watts, 1960), or remain unchanged (Keatinge & McCance, 1957), and all three responses may be seen in the one investigation (Budd, 1964, 1965). In the present study the test cold exposure caused bradycardia in only one subject in series 1 but in all subjects in series 2, the difference being statistically significant. The cause is conjectural, but a possible interpretation is that the bradycardia was a reflex response to enhanced vasoconstriction.

The effect of the test cold exposure on urine flow rates is partly obscured by the inclusion, in the 'exposure period' collection, of urine passed during the preceding 90 min in a warm environment, and possibly also by the effects of variations in fluid intake at breakfast. Nevertheless, it seems clear that cold diuresis occurred—as might be expected (Adolph & Molnar, 1946)—and that it did not change when the subjects became acclimatized. The latter finding contrasts with Wyatt's observation (in Edholm & Lewis, 1964) that cold diuresis tended to be less in the Antarctic winter than in the autumn and spring.

Subjective impressions during the test cold exposures were recorded only qualitatively in this and the previous Mawson study, so that comparisons between series depended largely on the subject's memory. However, in another study (Budd, 1965) subjective impressions of cold were recorded quantitatively, by means of a 5-point scale, every 5 min throughout a 2 hr cold exposure—with the same result. In none of the three studies did acclimatization produce any clear change in the discomfort felt.

The constancy of forehead temperature throughout the cold exposure in both series supports the view (Hertzman & Roth, 1942; Froese & Burton, 1957) that there is little or no vasoconstriction in this area in response to cold.

The small but significant decrease in warm-phase rectal temperature in series 2 suggests a possible relation to climate, as was observed by Driver (1958) and Palmai (1962); but Massey (1956) and Lewis (1958)

found no relation between deep body temperature and climate, and in the 1959 Mawson study warm-phase rectal temperature varied significantly between series but showed no relation to any of the environmental factors measured.

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