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FINGER BLOOD FLOW IN ANTARCTICA

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SUMMARY

1. Finger blood flow was estimated, by strain-gauge plethysmography, before and during a 1 hr immersion in ice water, on twenty-five men throughout a year at Wilkes, Antarctica. A total of 121 satisfactory immersions were made.

2. Blood flow before and during immersion decreased significantly in the colder months of the year, and the increase caused by cold-induced vasodilatation (CIVD) became less as the year progressed. The time of onset, blood flow at onset, and frequency of the cycles of CIVD showed no significant relation to the coldness of the weather (as measured by mean monthly wind chill) or the time in months. Comparisons of blood flow before and after five field trips (average duration 42 days), on which cold exposure was more severe than at Wilkes station, gave similar results.

3. The results suggest that vasoconstrictor tone increased. This interpretation agrees with previous work on general acclimatization in Antarctica, but contrasts with work elsewhere on local acclimatization of the hands.

INTRODUCTION

Cold adaptation of the hands, in the form of an increased resistance to the numbing effect of cold, has been demonstrated in the Arctic (Mackworth, 1953) and the Antarctic (Massey, 1959), and has also been induced in the laboratory (Mackworth, 1955). That this improved tolerance might be due to changes in finger blood flow has been suggested by investigations of a number of cold-exposed occupational and racial groups, whose hands, when immersed in ice water, have shown either a greater blood flow (Brown, Bird, Boag, Boag, Delahaye, Green, Hatcher & Page, 1954) or an earlier onset and greater magnitude of rewarming by cold-induced vasodilatation (Yoshimura & Iida 1952; Elsner, Nelms & Irving, 1960; Nelms

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& Soper, 1962) than those of control groups. Observations of finger blood flow, before and during immersion in ice water, were therefore made throughout a year in Antarctica. The results, reported here, contrast in several respects with those of previous investigations of local adaptation.

METHODS

Finger blood flow was estimated, by strain-gauge plethysmography, in twenty-five members of the 1966 party of the Australian National Antarctic Research Expeditions (ANARE) to Wilkes (lat. 66° 15′ S, long. 110° 31′ E.). The first experiment was done in early February, 10 days after the party's arrival in Antarctica, and the last in mid-December of the same year. One hundred and thirty-two experiments were done, each man being tested on 3–7 occasions. The subjects were healthy men of European descent, aged between 22 and 58 years. All but one had spent the previous year in a temperate climate; the other had wintered on Macquarie Island in the subantarctic.

Exposure to cold. The climate of Wilkes is cold and windy (Fig. 1). Average daily air temperature in 1966 was below 0° C on 354 days of the year and below −18° C on 64 days; the yearly means were −10° C for air temperature and 4·4 m/sec (10 miles/hr) for windspeed.

The station huts were heated to comfortable indoor temperatures of 20° C or more. Most of the huts were connected by enclosed unheated corridors, so that men could move between them without being exposed to the weather. However, no one remained continually indoors. Construction workers and mechanics were often outdoors, and frequently had to handle cold metal. Camp duties were shared by all, and most men in turn spent a week as a labourer with the construction crew.

Five major field trips inland, of duration 17–74 days (mean 42 days), were made. The men taking part travelled by tractor, and slept and ate in caravans which were inadequately heated except on the final trip, when new heaters were installed. On these trips the men were exposed to cold more often than at Wilkes, and the weather was more severe—the wind was stronger, and the air temperature was commonly below −22° C and sometimes below −40° C. In addition to these trips, five shorter trips were made by dog sledge, and most men went on day excursions whenever their duties and the weather allowed.

Experimental procedure. In order to take full advantage of the co-operation offered by the subjects, a fixed experimental plan was not used, and no attempt was made to test the same subjects in every month. Table 1 shows that the tests on each subject were spread over most of the year. Fifteen subjects had their first test in the first 2 months of the year, and twenty-four within the first 4 months. Most subjects were tested on five or more occasions.

Volunteers were called for at meal times, and at the mid-morning and mid-afternoon tea breaks; consequently almost all tests began in the 30 min after a meal or a hot drink. Most of the tests were done between 1100 hr and 1700 hr. The subject reached the laboratory by a short walk along unheated corridors. The laboratory temperature was maintained between 20 and 25° C, and the subject adjusted his clothing so as to be comfortably warm during the test.

The first 10 min after the subject's arrival in the laboratory were spent in attaching the strain gauge, thermistor, and occlusion cuff to the right index finger. The subject then sat with his right hand resting comfortably on a padded support level with the lower end of the sternum, and two observations, at 2 min intervals, were made of finger blood flow and skin temperature. The finger was then immersed, to the middle of the second phalanx, in a vacuum flask of crushed ice and water fitted with an electrically-driven stirrer, and the observations were continued at 2 min intervals. In most experiments immersion time was 60 min, and in all but a few it was more than 30 min.

Technique. Finger blood flow was estimated by strain-gauge plethysmography (Whitney,

1953). The strain gauge (Electro-medical and Engineering Company, Melbourne) was a 280 mm length of silicone rubber tubing of 0·50 mm bore and 0·25 mm wall thickness, which was filled with mercury and closed at each end by platinum pegs which served as contacts. No temperature compensation was used. Changes in gauge resistance were measured by a low resistance Wheatstone bridge circuit energized by a 6 V lead–acid accumulator which was kept fully charged, and recorded on a servo recorder (Heath Servo Recorder Model EUW-20A. Heath Company, Benton Harbor, Michigan) with a balancing time of 0·1 sec/2·5 cm deflexion. The gauge was calibrated before each experiment by clipping it to a

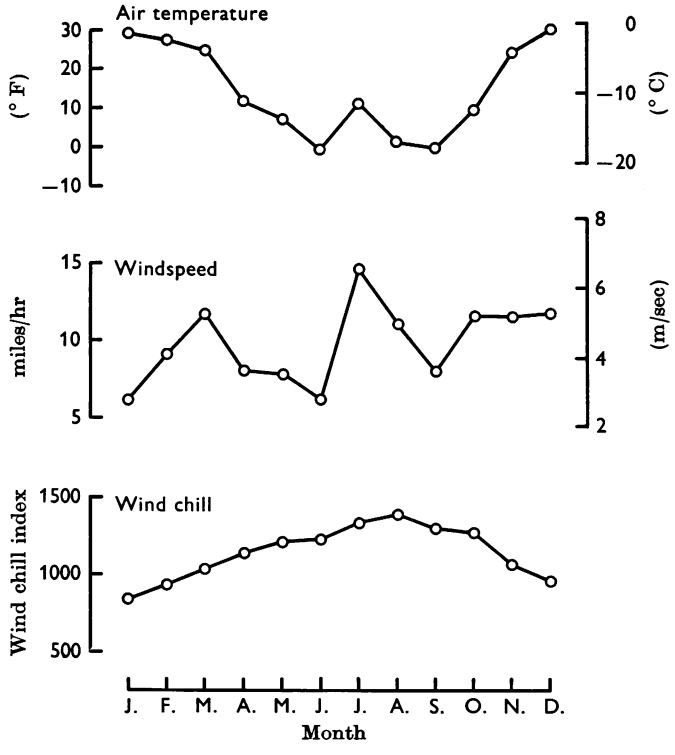

Fig. 1. Average monthly air temperature, windspeed and wind chill at Wilkes in 1966.

vernier caliper, which was held vertical on a retort stand. Successive increments of 1·00 mm in the caliper opening were made, until after some 7 mm the pen had traversed (at an angle of 90° to the base line) the full scale of the chart. The trace confirmed that the response was linear, and the average response for 1·00 mm caliper opening was taken as the calibration factor for the gauge. On a few occasions the gauge was also calibrated after the experiment: no change was detected.

Finger temperature was measured with a thermistor and a Wheatstone bridge, but the results were unsatisfactory because of poor thermistor contact and instability of the circuit, and they were therefore discarded.

The same technique was used in all experiments. The full length of the strain gauge was wound around the distal phalanx of the finger (usually for 4–5 turns) and held in position by two 5 mm squares of 'Blenderm' adhesive tape (Minnesota Mining and Manufacturing (Australia) Pty. Ltd., Sydney). The thermistor, which was mounted in the end of a 4 cm

glass probe, was strapped to the lateral aspect of the finger-tip, distal to the strain gauge, by another 5 mm square of tape. An inflatable cuff 25 mm wide was wound around the proximal phalanx, and a sphygmomanometer cuff was fixed around the upper arm over the clothing. Both cuffs were attached to 10 l. air cylinders via 2-way taps, to permit rapid inflation and deflation. The venous occlusion pressure (in the cylinder attached to the finger cuff) was maintained at a level (usually 80 mm Hg) just below that which obstructed blood flow during cardiac diastole, any obstruction being apparent on the recording as a characteristic square-cut pulse wave. The arterial occlusion pressure (in the cylinder attached to the arm cuff) was maintained at more than 200 mm Hg.

TABLE 1. The months in which each subject was tested. Each test is shown by an asterisk

Subject no.	Month J.	F.	M.	A.	M.	J.	J.	A.	S.	O.	N.	D.	Total
1	.	.	*	*	.	.	*	*	.	.	*	.	5
2	.	.	*	.	*	.	.	.	*	.	*	.	4
3	.	*	.	.	*	*	*	*	.	*	.	*	7
4	.	*	.	.	.	.	*	*	.	.	.	.	3
5	.	.	.	*	.	.	*	.	*	.	*	.	4
6	.	.	.	.	*	*	*	.	*	.	.	*	5
7	.	*	.	.	*	.	*	.	*	.	*	.	5
8	.	*	.	.	*	.	*	.	*	.	.	*	5
9	.	.	.	.	*	*	*	*	.	.	*	.	5
10	.	.	*	.	*	*	.	*	*	.	.	*	6
11	.	*	.	.	*	*	*	.	*	.	*	.	6
12	.	*	.	.	*	.	*	.	.	.	*	.	4
13	.	.	.	.	*	.	*	.	*	.	.	*	4
14	.	.	*	.	*	.	*	*	.	.	*	.	5
15	.	*	*	.	*	.	*	.	*	.	*	.	6
16	.	.	*	.	.	*	*	*	.	*	*	.	6
17	.	*	.	.	*	.	*	*	.	*	.	*	6
18	.	.	.	.	.	.	*	.	.	*	.	*	3
19	.	.	.	.	*	*	*	.	*	.	*	.	5
20	.	*	.	.	*	.	*	*	.	.	*	.	5
21	.	.	.	.	*	.	*	*	.	.	*	.	4
22	.	.	.	.	*	.	*	*	.	.	*	.	4
23	.	*	.	*	.	*	*	*	.	.	*	.	6
24	.	.	.	.	*	*	.	.	.	.	*	.	3
25	.	.	.	.	*	.	*	*	*	.	*	.	5
Total	.	10	6	3	19	9	22	13	11	4	17	7	121

When making an observation the arm cuff was first inflated to occlude arterial inflow. Sometimes this would be followed by a decrease in finger volume, possibly due to redistribution of fluids in the absence of arterial pressure. The venous occlusion cuff on the finger was then inflated, often producing a marked 'jump' in the tracing—presumably by displacement of tissue and fluids into the distal phalanx. As soon as this change ceased (which was always within 10 sec of inflation of the finger cuff) the arm cuff was released and the subsequent volume change attributed to the flow of blood into the finger. When the curve was approaching a maximum the venous occlusion cuff was deflated. The whole procedure occupied about 20 sec. The base line of the recorder had to be reset as soon as the finger was immersed in ice water, owing to the lack of temperature compensation of the strain gauge.

Figure 2 shows examples of tracings at high and low flow rates. The angle θ was measured with a protractor, and blood flow calculated by means of the formula—

$$\text{Blood flow (\% of finger volume/min)} = \tan\theta \frac{200S}{CL},$$

where S = chart speed (mm/min), C = calibration factor (pen deflexion (mm) for 1 mm extension of gauge), and L = length of strain gauge (mm).

The calculation is based on the assumption (Whitney, 1953) that the percentage change in finger volume is proportional to twice the percentage change in finger circumference.

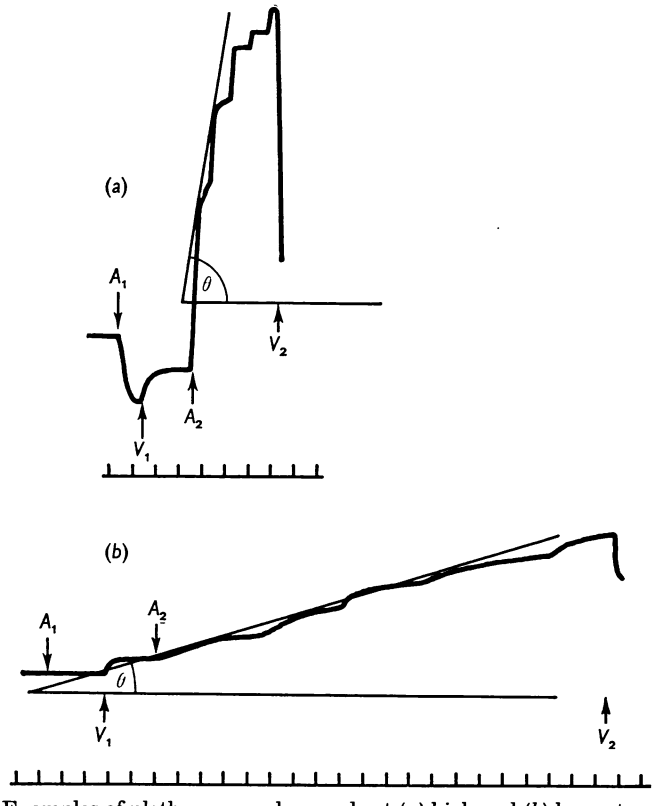

Fig. 2. Examples of plethysmograph records at (*a*) high and (*b*) low rates of finger blood flow. Time in seconds is shown below each record. The arrows at A and V indicate the times of (1) inflation and (2) deflation of the arterial and venous occlusion cuffs respectively. Individual pulse waves are apparent at the higher flow rate, but not at the lower.

Analysis of results. The results of seven of the 132 observations were discarded because of technical shortcomings, and those of four others were discarded because the period of immersion was less than 30 min. Each of the remaining 121 experiments was plotted against time in minutes, and the following six variables were calculated:

(1) The average of the two pre-immersion observations.

(2) Average blood flow during the immersion. This was done by dividing the area under the curve of blood flow (integrated by the trapezoidal rule) by the time (min).

(3) Change in flow (the net response to immersion). This was determined by subtracting average pre-immersion flow from average immersion flow.

(4) Time to the onset of cold-induced vasodilatation (CIVD).

(5) Blood flow at the time of onset of CIVD.

(6) The frequency of the cycles of CIVD. This was estimated by counting the number of times the plotted curve of blood flow against time contacted or crossed the mean value

(±5%) for that experiment; in experiments lasting less than 1 hr (say x min) this number was multiplied by $60/x$ to estimate the hourly frequency. The average number of cycles of CIVD/hr is approximately half the number of crossings.

In order to assess the linear trend of blood flow over the year, the regression of each variable on time in months was calculated for each subject, using an electronic digital computer. For each of the six variables, the individual regressions of the twenty-five subjects were compared and, if their residual variances and slopes were found to be homogeneous, pooled to form a common regression (Williams, 1959). Variables 4 and 6 were transformed before analysis to $\sqrt{(X+\frac{1}{2})}$, where X is a single observation (Steel & Torrie, 1960). The regression analyses were repeated with wind chill (Siple & Passel, 1945)—an index which combines the cooling effects of windspeed and air temperature—as the independent variable.

These analyses give an over-all impression of the change in blood flow throughout the year, but they deal with a mixed group of subjects who were not all exposed to cold to the same extent. In order to obtain a clearer impression of the effects of cold, the results of the experiments on the men who took part in each of the five major field trips were examined separately. In each case blood flow had been estimated shortly before the trip, and as soon as possible after it—usually within 3 weeks and always within 6. There were twenty-one such pairs of experiments. The differences between the observations before and after the field trips were tested statistically by Student's t test.

RESULTS

Immediately after immersion, blood flow fell to a low level. After a variable period, cold-induced vasodilatation (CIVD) occurred and blood flow rose, to a level which was often higher than the pre-immersion value; this was followed either by regular cycles of vasoconstriction and vasodilatation (Fig. 3*a*) or by rapid irregular changes with no clear pattern (Fig. 3*b*).

The yearly average finger blood flow (Fig. 4) was 15·0% of finger volume/min in the pre-immersion period and 18·8% during immersion, an increase of 3·8%. CIVD occurred, on the average, 6·5 min after immersion, when the blood flow was 4·0%. The frequency of cycling averaged 4·5 cyc./hr.

Regressions on time. The regressions on time for the individual subjects were of homogeneous variance and slope only in the case of pre-immersion flow, change in flow, and frequency of CIVD (as shown by the number of crossings), hence only in these three variables was it possible to calculate a common regression. The common regressions of pre-immersion flow and frequency of CIVD were not significant, but that of change in flow was ($P < 0{\cdot}05$): the regression coefficient was $-0{\cdot}6527$% finger vol./min per month. In the case of the remaining three variables, the regressions of the twenty-five individual subjects were examined separately. Few were significant ($P < 0{\cdot}05$): five for immersion flow, one for flow at the onset of CIVD, and none for time to onset of CIVD. Except in one case of immersion flow, the slope of the significant regression coefficients was negative.

Summarizing, the increase in finger blood flow (over pre-immersion values) that occurred during immersion declined as the year progressed. The absolute level of blood flow during immersion decreased in four subjects and increased in one; and blood flow at the onset of CIVD decreased in one subject. No significant trend, either upwards or downwards, was apparent in the other subjects and variables.

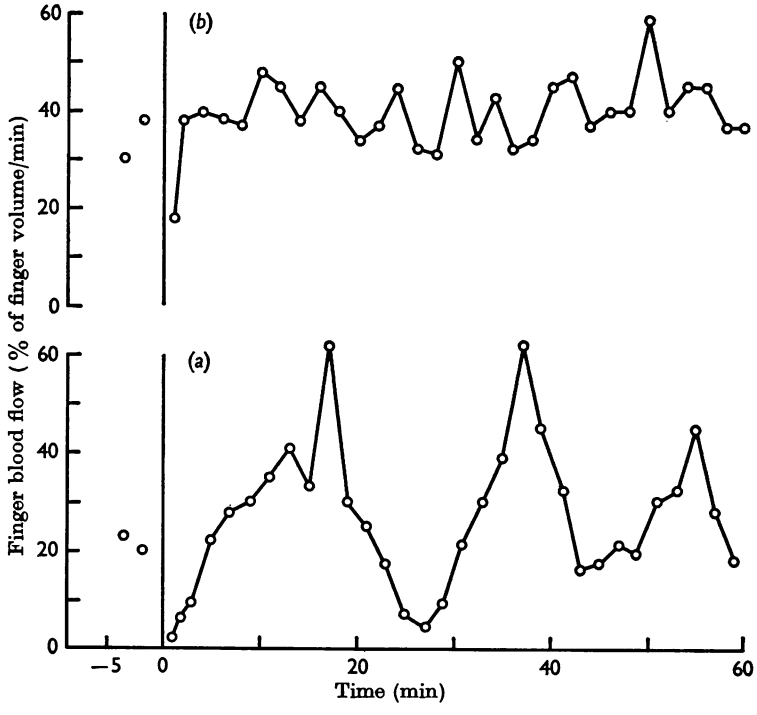

Fig. 3. Examples of finger blood flow showing (*a*) regular cycles of cold-induced vasodilatation, and (*b*) rapid irregular changes. The finger was immersed in ice water at zero time.

Regressions on wind chill. The common regressions on wind chill were significant for pre-immersion flow and immersion flow. Both regressions were negative, indicating that blood flow was less in the colder months; the slope of the regression of pre-immersion flow (−0·0160 % finger vol./min per unit of wind chill) was almost twice that of immersion flow (−0·0090). The common regression for frequency of CIVD was not significant. In the three remaining variables, heterogeneous variance made it impossible to calculate a common regression; no more than two of the twenty-five individual regressions were significant in any of these variables

Effect of field trips. The results of the experiments before and after field trips (Table 2) agree with those of the regressions on wind chill. Blood flow

tended to decrease to a greater extent in the pre-immersion period than in the immersion period; the decrease approached the 5% level of significance for the former, and the 10% level for the latter. No consistent change occurred in any of the other measures of CIVD.

Effect of frostbite. Two subjects suffered minor frostbite on field trips. One developed blisters on the tips of all his fingers but retained normal sensation; the other lost a fingernail, and for the next month had difficulty in doing up buttons because of numbness of all fingers of the right hand.

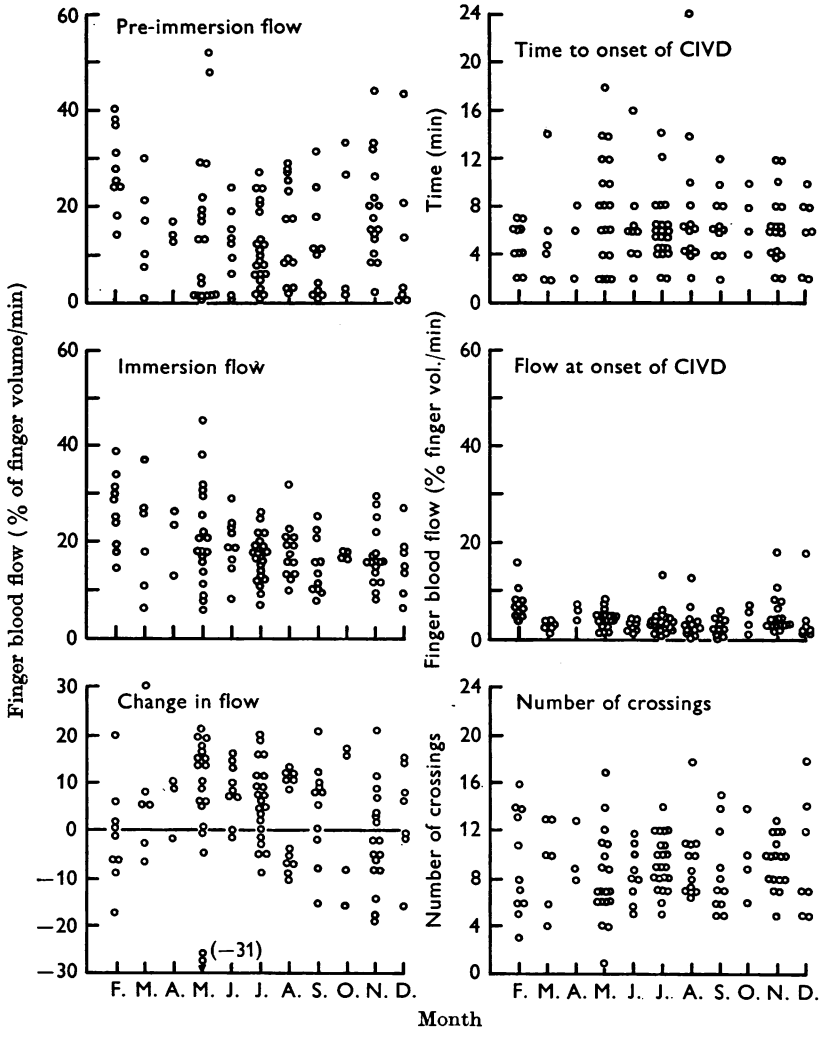

Fig. 4. Individual monthly values of finger blood flow and cold-induced vasodilatation. Each point represents the results of one experiment. The subjects tested were not always the same in each month.

These injuries caused no apparent change in the level and pattern of finger blood flow, nor in the amount of pain experienced during immersion in ice water, for such changes as were observed were shared by the other men on the trip, who had not been frostbitten.

Subjective responses. Complaints of pain during immersion were sporadic and showed no clear trend throughout the year. Nausea, sweating and dizziness occurred in four experiments, which had to be abandoned as a

TABLE 2. Finger blood flow and cold-induced vasodilatation (CIVD) before and after field trips. Averages of twenty-one pairs of observations

	Mean values (n = 21)			Significance of difference	
	Before trip	After trip	Difference	t	P
Pre-immersion flow (% finger vol./min)	21·0	13·0	−8·0	1·93	0·10 > P > 0·05
Immersion flow (% finger vol./min)	20·8	18·6	−2·2	1·62	0·20 > P > 0·10
Change in flow (% finger vol./min)	−0·2	+5·6	+5·8	1·53	0·20 > P > 0·10
Time of onset of CIVD (min)	7·4	7·3	−0·1	0·05	P > 0·95
Blood flow at onset of CIVD (% finger vol./min)	4·3	4·2	−0·1	0·06	P > 0·95
No. of crossings	7·5	7·6	+0·1	0·07	P > 0·90

result. In each case one cycle of vasodilatation had occurred and the second vasoconstriction was just beginning. Two of these episodes occurred during the first immersion for the particular subject, one during the second and one during the third. The previous and subsequent immersions were uneventful.

DISCUSSION

The results of this investigation are in marked contrast with those of certain previous studies of local adaptation to cold (Yoshimura & Iida, 1952; Brown *et al.* 1954; Elsner *et al.* 1960; Nelms & Soper, 1962). Instead of increasing, finger blood flow—both before and during immersion—decreased in the colder months of the year; and the increase caused by cold-induced vasodilatation became less as the year progressed. No significant change occurred in the time of onset of CIVD, the blood flow at its onset or the frequency of the cycles. These findings seem to imply an increase of vasoconstrictor tone. That the increase was also apparent after field trips, which necessitated more prolonged and intense cold exposure than did life at Wilkes station, suggests that its cause was chronic cold exposure. Massey's finding (1959) that finger temperature fell in Antarctica—especially after long sledging journeys—supports this interpretation.

The contrast with previous work might be due to either of two causes.

The first is that at Wilkes a single group of men was tested repeatedly, whereas most of the other studies mentioned consisted of comparisons between two or more groups, who may have differed not only in cold exposure but in other respects (such as race, diet, and physical fitness) as well. The second is that the changes in finger blood flow at Wilkes may have been part of a general adaptation to cold exposure of the whole body, rather than a local adaptation to chilling of the hands alone. The second explanation might well be the more important, for general acclimatization has been shown to occur in the Antarctic (Budd, 1964, 1965; Budd & Warhaft, 1966), and its mechanism appears to consist of enhanced vasoconstriction.

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