Effect of chronic local cold exposure on finger temperature responses[1]

THOMAS ADAMS AND R. ELPHIN SMITH
Department of Physiology and Biophysics, University of Washington School of Medicine, Seattle, Washington

ADAMS, THOMAS, AND R. ELPHIN SMITH. *Effect of chronic local cold exposure on finger temperature responses.* J. Appl. Physiol. 17(2): 317–322. 1962.—Caucasian subjects from a temperate climate immersed their right index fingers in a stirred ice-water bath for 20 min four times daily for 1 month. During subsequent test immersion, fingers exposed in this way showed an earlier initiation and a more rapid rate of spontaneous rewarming and a higher and more labile final temperature level than did either the same finger before the prolonged cold exposure or other digits not chronically cold exposed. The inference derived from these data that blood flow is elevated in chronically cold-exposed fingers was supported by digital calorimetric determinations. Since finger temperature responses were modified only in the cold-conditioned digit, the term "local cold conditioning" appears justified. Variation of the number of daily exposures made clear a progressive modification of skin temperature responses to cold. During the month of repeated cold exposures, pain associated with this type of cold exposure diminished and finally disappeared. These experimental data parallel most observations on peripheral vascular responses to natural cold exposures and may suggest a mechanism for the changes under both conditions.

IN MAN AND SMALL ANIMALS increased skin and extremity temperatures commonly result from prolonged experimental exposure to cold (1–5). Also, during acute cold stress both finger blood flow and extremity temperature increase more in people indigenous to arctic and subarctic areas than in persons unaccustomed to cold (6–10). Although cold-induced cyclic vasodilation in the finger (CIVD), first reported by Sir Thomas Lewis (11), has disclosed many variables in peripheral vascular responses, it has received only limited attention in studies of acclimatization to cold. Furthermore, these studies have not yielded consistent data or interpretations (6, 8, 9, 12, 13), although pain responses usually are reported to diminish progressively during successive exposures. The present study was designed to supplement available data which suggest changes in the pattern of CIVD or other vascular responses as a result of direct and local cold stress.

Received for publication 28 July 1961.
[1] This study was supported in part by grants 2G-260 and HTS5204 from the National Institutes of Health.

METHODS

In Caucasian residents of a temperate climate skin temperature and heat loss were measured in fingers during an ice-water immersion before, during, and after a program of prolonged local cold exposure. Since the position of the subject in relation to the exposure bath may be an important variable in such experiments (14), the subjects rested supine with the hand at heart level for a few minutes before and during all measurements. Also, exercise and thermal exposure were restricted for some time prior to each measurement to minimize variations in body thermal states. The room temperature was maintained at 22 ± 2 C during all measurements. Only one set of measurements on a given finger was made on any given day. The finger was immersed to the same depth in the stirred ice water in all tests.

In five subjects the temperature and heat loss of the right index finger exposed to stirred water at 0 C were measured before, during, and after the terminal two phalanges had been cold conditioned by immersion in a stirred ice-water bath for 20 min four times daily for 1 month. Similar values for the left index finger were obtained before, during, and after the chronic cold exposure of the right finger; other fingers on the right hand were also tested this period. In a second experiment five additional subjects underwent a similar regimen except that the number of 20-min exposures each day was varied from two to four. The response of the exposed finger was measured several times during the month. A different series of five subjects was tested to determine the effect on finger temperature responses of reduced central thermal states produced by an acute whole-body cold exposure. In one series of experiments the test finger was not protected during the whole-body exposure in a room at 7 C for as long as 1 hr; in another, the finger was immersed in water kept at 33 C throughout the period in the cold room. Immediately after the acute whole-body exposures, CIVD responses were recorded

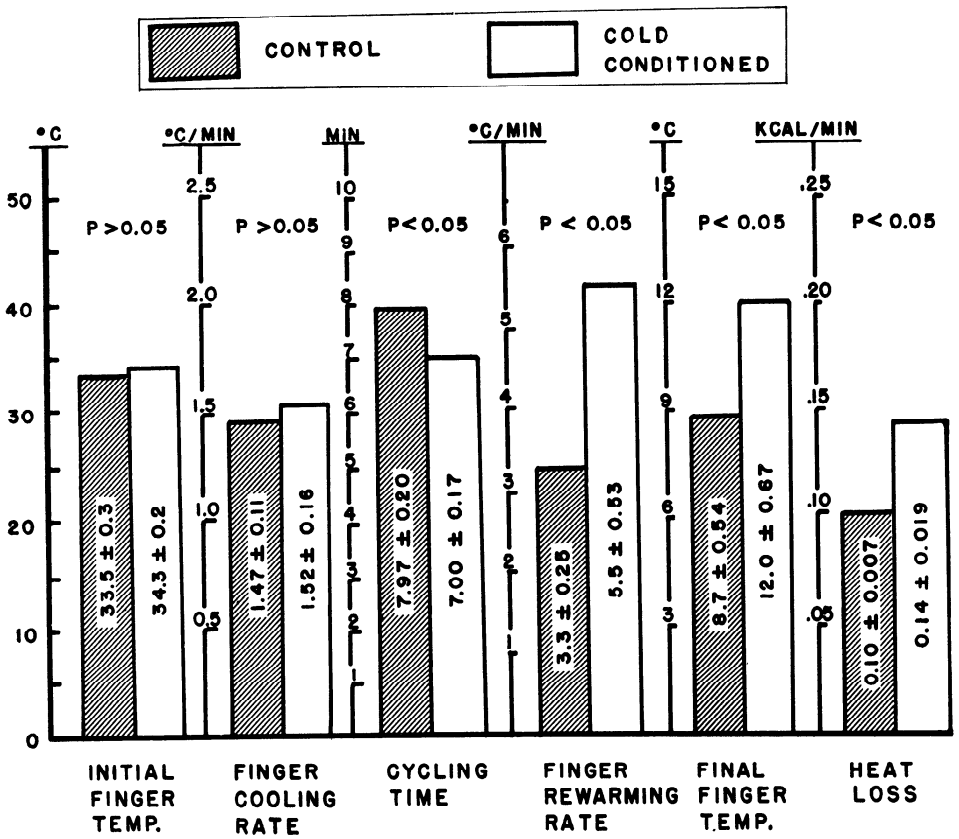

FIG. 1. Mean responses (with SD of mean) of control and cold-conditioned fingers to immersion in stirred ice water. Statistical significance of difference between control and experimental values is indicated above each pair of bars. Data for cold-conditioned fingers that did not initially cool to 0 C were omitted from calculation of finger cooling rate. Cycling time refers to period between immersion and onset of spontaneous rewarming. Heat loss was measured during plateau of finger temperature after spontaneous rewarming.

under the same testing conditions as in the other experiments.

The skin temperature of the finger was measured at the digital pad with a 36-gauge copper-constantan thermocouple attached with 1 cm^2 of plastic tape. The thermocouple was always located in the same relative position and the tape was applied so that, although secure, it did not occlude blood flow. The temperature was recorded on a single-point, constantly recording, calibrated Honeywell potentiometer. Heat loss was calculated from the temperature change of a calorimeter containing a known volume of water at a known initial temperature. These measurements are expressed only for the final plateau of temperature response at the end of the test exposure.

Since the responses of the right index finger prior to the month of cold conditioning and the responses of the matching contralateral left index finger throughout the experimental period did not differ significantly, these data have been accepted as comparable control values. The ice-water bath in the testing and cold-conditioning periods was agitated constantly by an electrically powered stirrer. Probability values were calculated using a *t* test on the grouped data from all participating subjects.

RESULTS

The initial response of a previously unexposed finger to immersion in stirred ice water was a rapid decline in skin temperature to approximately 0 C. After several minutes the temperature rose to a plateau at 6–10 C. This pattern and its time course (see Fig. 2) are comparable to those previously described for cold-induced vasodilatation in the finger (6, 8, 11, 13, 14). Responses of this type were recorded from the right index finger before the chronic cold exposure and at all times from fingers not chronically cold exposed.

The major differences in this pattern for a finger that had been repeatedly exposed to cold for 1 month were: a shortening of the lapsed time between the immersion and the onset of spontaneous rewarming (cycling time); a more rapid increase in temperature (finger rewarming rate); and a higher plateau (final finger temperature), which was more labile than in control responses. Figure 1 presents a composite of the control responses and those of the cold-conditioned fingers, expressed as means of the responses of all subjects. The difference in the rate of finger cooling seen in Fig. 2*A* was not consistently observed in all subjects. Since the rate of finger cooling was defined in terms of the time to reach 0 C, data for subjects showing cold-conditioned patterns like those seen in Fig. 2*B* and *C* were excluded from this calculation. Figure 1 also illustrates the significantly greater heat loss from cold-conditioned fingers during the ice-water exposure.

The pattern of the differences between the control and chronically cold-exposed fingers was the same whether the fingers were tested alternately on the same day or individually on successive days. If the two fingers

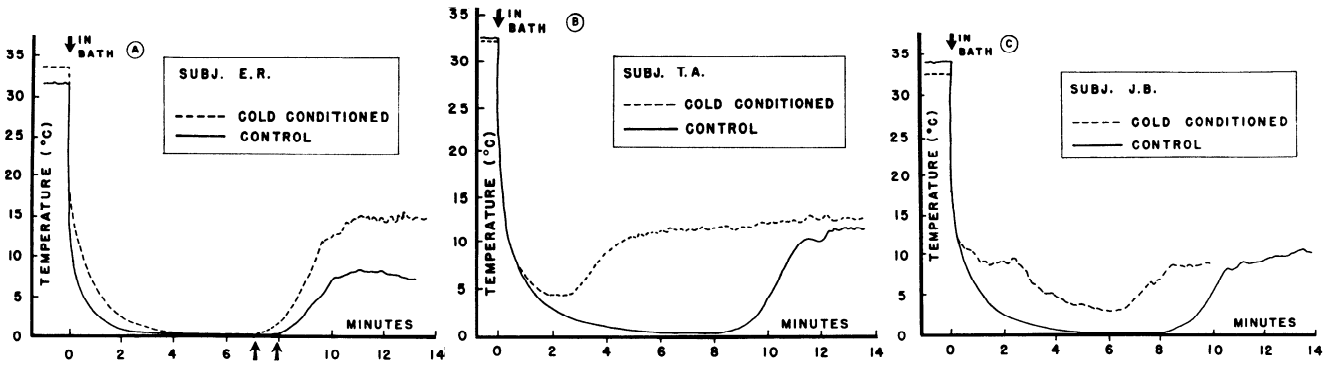

FIG. 2. Three patterns of temperature response to immersion in stirred ice water seen after exposure of right index finger to cold for 80 min/day for 1 month. *A: left arrow* on *abscissa* indicates onset of rewarming in cold-conditioned finger, *right arrow*, onset of rewarming in control finger. Differences in temperature of control and cold-conditioned fingers at beginning of immersions were not statistically significant.

were tested simultaneously, however, differences between the control and cold-conditioned digits were reduced, but the pattern of the contrasts remained qualitatively similar to that seen during alternate testing.

The changes in vascular response, reflected in digital skin temperature and heat loss, occurred only in the cold-conditioned right index finger. No adjustments of response were seen in any adjacent ipsilateral or contralateral digits as a result of the chronic cold exposure of the right index finger. Also, no significant difference in the temperatures of the control and the cold-conditioned fingers was recorded as long as the digital skin temperature was above that at which CIVD responses are triggered, i.e., above about 15 C.

Figure 3 shows the effect of various degrees of chronic cold exposure on CIVD patterns. Note that the changes in response developed progressively as the length of cold exposure increased and that the sequence is similar to the order in which Fig. 2 is arranged.

Finger temperature progressively decreased with increasing lengths of whole-body cold exposure to 7 C. It can be seen in Fig. 4 that subsequent CIVD testing at an ambient temperature of 22 C, with the lower initial finger temperatures, the cycling time, i.e., the time before spontaneous rewarming, lengthened progressively in an approximately linear manner. Spontaneous rewarming times became less predictable at initial finger temperatures below about 18 C. In addition to the effects on cycling time, a reduced rate of rewarming and a reduced final temperature level were recorded. Maintaining an elevated finger temperature during the whole-body cold exposure by keeping the finger in a 33 C water bath did not result in shorter cycling times, more rapid rewarming, or higher finger temperatures.

Evidence of psychological effects on temperature responses was seen in records for both cold-conditioned and control fingers. Figure 5 shows the effect of emotional stress induced by verbal suggestion on the response of a chronically cold-exposed finger. The early part of the tracing is very similar to the usual cold-conditioned finger temperature response for this subject (Fig. 2*B*). The curve of cooling after the induction of the emotional stress, however, is essentially like the initial cooling curves in Fig. 2*A* and for control responses in general. A similar effect was seen in the absence of local cold conditioning in another emotionally stressed subject whose finger remained at the temperature of the ice bath for 25 min. In other tests without emotional stress the same finger showed the usual control response, beginning to rewarm 7 min after immersion.

During the repeated immersions in the cold-conditioning period, the intense pain associated with this type of exposure to cold diminished and finally disappeared completely.

One of the most important prerequisites (6) for obtaining consistent and predictable CIVD responses is to expose the digit to a constant and a duplicable cold stress which may be readily obtained by rapid and uniform stirring of the ice-water bath. The importance of stirring the ice-water bath should be noted for cold conditioning as well as test exposures.

DISCUSSION

The results affirm the view that a change in cold-induced vasodilatation (CIVD) develops during prolonged exposure of a finger to cold as a local phenomenon. The disparity between the data presented here and those from studies in which no change in CIVD followed experimental cold exposure of an extremity (12, 13) is probably attributable to differences in the amount of the chronic (conditioning) cold exposure. In the investigation of Glaser et al. (12) the period of chronic cold exposure was 1 min at 4 C six times daily for 9 days and in Eagan's (13) it was 5 min at 0 C six times daily for 17 days. These exposures are considerably less than those used here, 20 min at 0 C four times daily for 1 month, and those in the study of Yoshimura and Iida (6), 30 min daily for 1 month, which yielded evidence of peripheral vascular cold adaptation. In the experimental series illustrated in Fig. 3, the changes in the temperature patterns had not yet begun at the times corresponding to the total chronic cold exposure in the experiment of either Glaser et al. or Eagan. Also, they

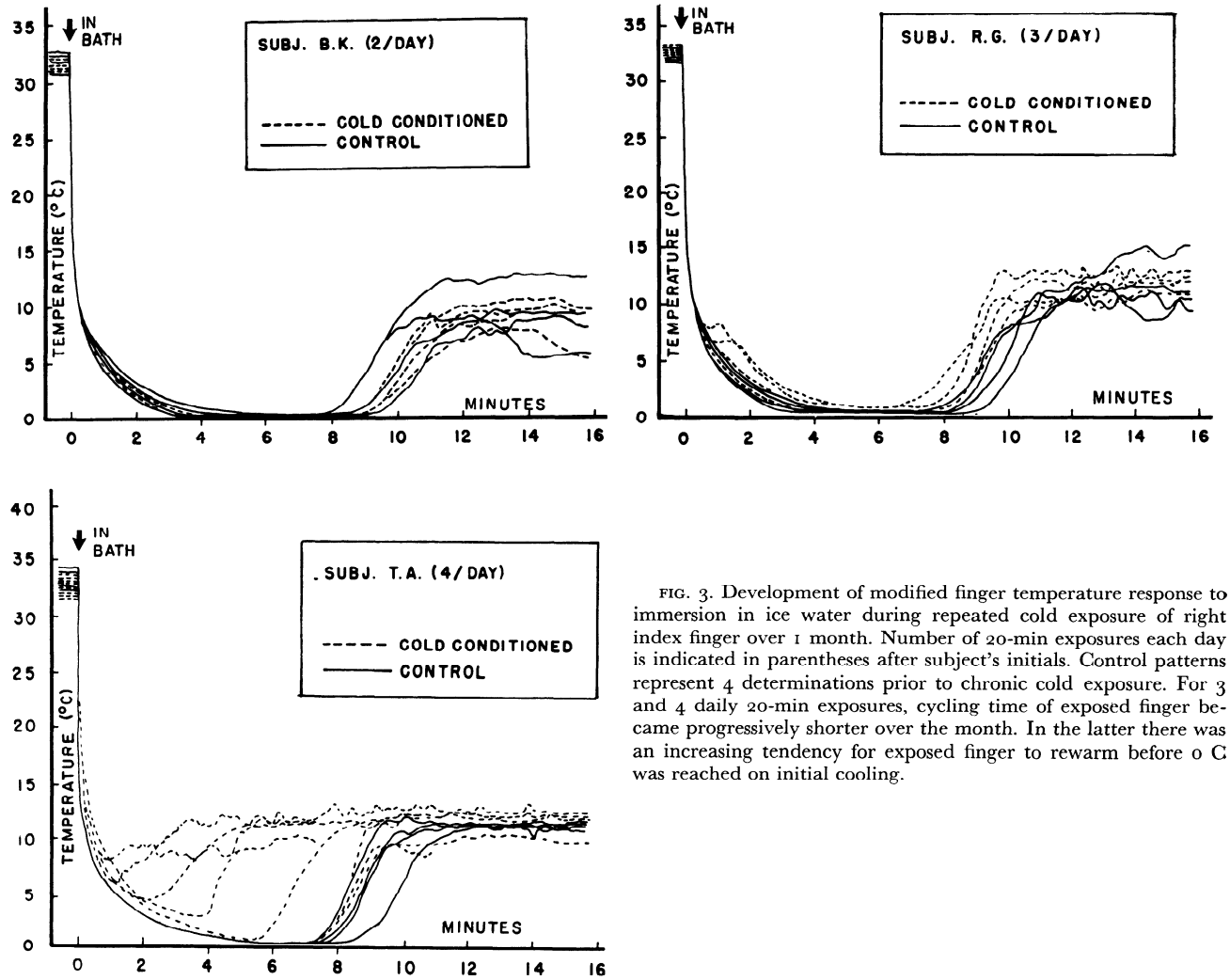

FIG. 3. Development of modified finger temperature response to immersion in ice water during repeated cold exposure of right index finger over 1 month. Number of 20-min exposures each day is indicated in parentheses after subject's initials. Control patterns represent 4 determinations prior to chronic cold exposure. For 3 and 4 daily 20-min exposures, cycling time of exposed finger became progressively shorter over the month. In the latter there was an increasing tendency for exposed finger to rewarm before 0 C was reached on initial cooling.

reported diminution rather than disappearance of pain; diminution occurred at corresponding times in our experiments but was followed by the disappearance of pain with additional cold exposure.

The local nature of the cold-conditioned changes is evidenced by the similarity of control responses in both index fingers before the chronic cold-exposure period and the responses in the left index finger after prolonged cold exposure of the right index finger. Equally important, the lack of changes in CIVD in any digit other than the chronically exposed right index finger seems to preclude involvements of neural or circulatory mechanisms subserving diffuse vasomotor control.

The central thermal state of the body is a potentially important variable in measurements of CIVD (15). Acute whole-body exposure to 7 C modified the thermal responses to subsequent ice-water immersion in previously unexposed fingers (Fig. 4), a phenomenon also reported by Blaisdell (15). This change was not obliterated by maintenance of the finger temperature at 33 C during the whole-body cold exposure. Without such protection, however, the initial skin temperature of the finger would appear to be a reasonable index of central thermal state in the absence of emotional stress. Under such conditions finger temperature was generally related to the length of time spent in the cold room. These data suggest, therefore, that the developing CIVD patterns are more related to central thermal states than to initial finger temperature, per se.

The variation of the control and cold-conditioned finger temperatures at the beginning of some tests is not statistically significant (Fig. 1). Apparently slight differences similar to those seen in Figs. 2 and 3 are a random variation rather than an alteration of the response of the chronically cold-exposed finger to moderate ambient temperatures, or a significant variation in the central thermal state of the subject from test to test. The similarity of initial digital temperatures in cold-conditioned and control digits, as well as the similarity in control measurements before and after the month of local cold exposure, suggests that no consistent differences in central thermal state underlie the changes in CIVD patterns after cold conditioning.

Another variable of considerable significance involved

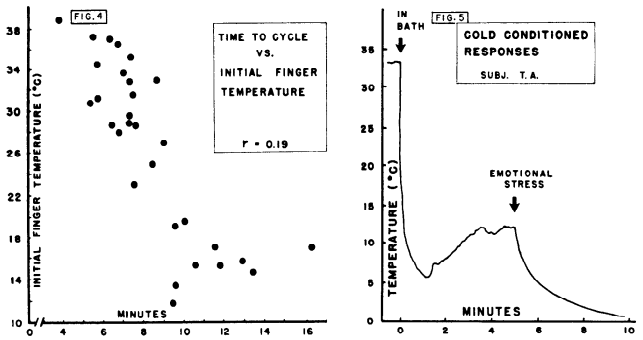

FIG. 4. Increase in time to onset of spontaneous rewarming of previously unexposed finger immersed in ice water when digital temperature had decreased during acute whole-body exposure to 7 C just before test. Note approximately linear relation between level of initial finger temperature and cycling time when initial temperature was above level at which CIVD is induced, ca. 15–18 C.

FIG. 5. Effect of induced emotional stress on temperature pattern of cold-conditioned finger immersed in stirred ice water. Note that first half of curve is similar to usual cold-conditioned response in this finger (Fig. 2*B*) and that second half is similar to initial cooling in a non-cold-conditioned finger.

in CIVD testing is the emotional state of the subject (oral report of J. P. Meehan, Jr., Symposium on Functioning and Protection of the Hands in a Cold Climate, Quartermaster Research and Development Center, Natick, Mass., 1956). Twice in the present experiments psychic states were shown to modify test responses. The emotional stress attending the pain in the control finger may have caused the deviant response patterns of the cold-conditioned finger when the two digits were immersed in the stirred ice water simultaneously. Although the differences in response between the two fingers (non-cold-conditioned left index and chronically cold-exposed right index) were not masked completely, simultaneous testing of cold-conditioned and non-cold-conditioned digits is obviously less satisfactory than alternate testing due to the introduction of the extraneous factor of pain responses.

A major feature of the CIVD responses in the cold-conditioned fingers was that they rewarmed more rapidly than did the control digits (Fig. 1). By inference the local blood flow during rewarming was greater in the chronically cold-exposed than in control fingers at the time of this measurement. Greenfield et al. (16) have suggested that the vasodilatation subsequent to this type of cooling allows a blood flow as great as that following other vasodilating procedures. With this suggestion in mind, it may be argued that bood flow was greater in the cold-conditioned than in the control finger during the latter stages of immersion as well. This argument is also supported by the calorimetric determinations (Fig. 1).

Since, as shown in Fig. 2*B* and *C* and Fig. 3, the tendency with repeated ice-water immersion was toward cessation of initial cooling before 0 C was reached, chronic exposure to cold might alter CIVD by destroying the ability of the vessels to constrict maximally. The emotion-induced decrease to 0 C (Fig. 5), however, indicates that the capacity for maximal vasoconstriction persists in the chronically cold-exposed finger. It is more reasonable to believe that chronic cold exposure induces a readjustment, but not a destruction, of vasomotor control mechanisms.

The disappearance of pain during the series of repeated cold exposures tends to support the statement by Greenfield et al. (17) that CIVD (in the finger not chronically exposed to cold) is accompanied by but does not result from pain. In other words, there appears to be no necessary parallel between the pattern of vasodilatation and the sensation of pain. Experience in our study is not consistent with Wolf and Hardy's suggestion (18) that the intensity of pain depends directly on the amount of cooling.

If the disappearance of pain from the finger with a surface temperature close to 0 C is caused by cold anesthetization of nerve fibers, only afferent fibers can be blocked. In both control and cold-conditioned fingers the vasoconstriction triggered by emotional stress and maintained as long as 25 min at a surface temperature of 0 C demonstrates the functional integrity of efferent vasomotor fibers so that neither disappearance of pain nor the CIVD phenomenon results solely from cold blocking of nerve fibers.

Peripheral changes related to the temperature responses in the cold-conditioned fingers should occur in persons normally exposed to cold. Although some questions remain concerning the identification and influence of whole-body cold acclimatization in these people (19, 20), their hands, feet, and face are undoubtedly exposed to cold regardless of the amount and efficiency of the thermal protection provided by their clothing. Groups indigenous to cold regions have shown increased finger and hand blood flow, more rapid cycling time during exposure of individual digits, more rapid rewarming of the cooled finger, and maintenance of warmer extremities in the cold (6–8, 10). Further, LeBlanc et al. (9), in describing the vascular responses of Gaspé fishermen to an acute cold-water exposure, reported a decrease in pain and increase in finger temperature and heat flow from extremities repeatedly exposed to cold water.

The nature of the neural or vascular changes accompanying modification of CIVD in an extremity repeatedly exposed to cold remains to be identified. A considerable amount of evidence, including the results of the present study, indicates that these physiological adjustments may result from direct local exposure in both experimental and natural cold stress.

REFERENCES

1. HORVATH, S. M., A. FREEDMAN, AND H. GOLDEN. *Am. J. Physiol.* 150: 99, 1947.
2. CARLSON, L. D., A. C. YOUNG, H. L. BURNS, AND W. F. QUINTON. Wright-Patterson Air Force Base, Tech. Rept. no. 6247, USAF AMC, 1951.

3. CARLSON, L. D., H. L. BURNS, T. H. HOLMES, AND P. P. WEBB. *J. Appl. Physiol.* 5: 672, 1953.
4. ELSNER, R. W. Arctic Acromedical Laboratory, Ladd Air Force Base, Proj. no. 8-7951, Rept. no. 1, 1955.
5. RENNIE, D. W., AND T. ADAMS. Arctic Aeromedical Laboratory, Ladd Air Force Base, TR 57-37, 1958.
6. YOSHIMURA, H., AND T. IIDA. *Jap. J. Physiol.* 2: 177, 1952.
7. BROWN, G. M., AND J. PAGE. *J. Appl. Physiol.* 5: 221, 1952.
8. MEEHAN, J. P., JR. *Military Med.* 116: 330, 1955.
9. LEBLANC, J., J. A. HILDES, AND O. HEROUX. *J. Appl. Physiol.* 15: 1031, 1960.
10. ELSNER, R. W., J. D. NELMS, AND L. IRVING. *J. Appl. Physiol.* 15: 662, 1960.
11. LEWIS, T. *Heart* 15: 177, 1930.
12. GLASER, E. M., M. S. HALL, AND G. C. WHITTOW. *J. Physiol., London* 146: 152, 1959.
13. EAGAN, C. J. *Physiologist* 3 (No. 3): 51, 1960.
14. DICKSON, J. A. *J. Physiol., London* 135: 93, 1957.
15. BLAISDELL, R. K. Environmental Protection Section, Office of the Quartermaster General, Washington, D. C., Rept. No. 177, 1951.
16. GREENFIELD, A. D. M., G. A. KERNOHAN, R. J. MARSHALL, J. T. SHEPHERD, AND R. F. WHELAN. *J. Appl. Physiol.* 4: 37, 1951.
17. GREENFIELD, A. D. M., J. T. SHEPHERD, AND R. F. WHELAN. *J. Physiol., London* 113: 6P, 1951.
18. WOLF, S., AND J. D. HARDY. *J. Clin. Invest.* 20: 521, 1941.
19. ADAMS, T., AND E. J. HEBERLING. *J. Appl. Physiol.* 13: 226, 1958.
20. HEBERLING, E. J., AND T. ADAMS. *J. Appl. Physiol.* 16: 226, 1961.