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CUTANEOUS BLOOD FLOWS IN CALF, FOREARM, CHEEK AND EAR DURING CHANGING AMBIENT TEMPERATURE

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FOREWORD

The experimental work reported herein was conducted in the Department of Physiology, Saint Louis University School of Medicine, Saint Louis, Missouri, in fulfillment of Contract AF 33(616)-7077 administered by the Aerospace Medical Laboratory, Wright Air Development Division. Mr. John F. Hall, Jr., of the Biothermal Section, Biophysics Branch, Aerospace Medical Laboratory, served as the contract monitor. The work was performed under Project No. 7164, "Physiology of Flight," Task No. 71830, "Human Thermal Stress in Extended Environments." Dr. James W. Colbert, Jr., is Dean of the School of Medicine, and Dr. A. B. Hertzman, Director of the Department of Physiology, is the principal investigator of this research contract.

The excellent cooperation of a group of medical and undergraduate students who served as subjects and also as assistants in the research is acknowledged and commended. Mrs. Alice Johnson performed the stenography for this report.

ABSTRACT

When seminude subjects were exposed to heat, the onset of cutaneous vasodilatation occurred simultaneously in the calf, forearm, cheek and ear. Progress of vasodilatation in the calf and toe often differed from that in the forearm, cheek and ear. Vasodilatation in the calf was either small or stabilized early while the forearm vessels continued to dilate markedly. One subject, a "poor" sweater with unusually high skin temperatures, repeatedly presented an exception in that vasodilatation in the calf exceeded that in the forearm. The usual failure of skin temperature to rise as much in the calf as in the forearm appeared to be related to the lesser cutaneous vasodilatation. There was no evidence that local sweating elicited local vasodilatation.

PUBLICATION REVIEW

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INTRODUCTION

The cutaneous vasodilatations which were elicited in the finger and forearm by exposure of the resting subject to a slowly rising ambient temperature were described in a previous technical report from this laboratory [1]. A consistent correlation was not observed between the vascular responses in these two regions during rising body temperature and between the onset of sweating and cutaneous vasodilatation in the forearm. The local skin temperature seemed to exert a significant influence on the temporal course of the cutaneous vasodilatation in the forearm.

These observations were extended to other regions in the present study since the evidence for regional differences in the vasomotor innervation of the skin [2,3,4] and in the sweating response to heat [5] indicated that the relations of cutaneous blood flow, temperature and sweating should be examined in the same skin area and in several representative regions. The finger, toe, ear, calf, forearm and cheek were selected because the vasomotor innervation of the digits and ear is vasoconstrictor [4,11], that of the calf and forearm both vasoconstrictor and vasodilator [3] and that of the face largely vasodilator [4,11]. Vasoconstrictor reflexes are usually absent in facial skin even during application of cold which only gradually elicits vasoconstriction there [6].

It is possible that the estimation of the peripheral conductance obscures regional differences in cutaneous vascular behaviour as completely as the measurement of weight losses conceals regional differences in sweating rates [7]. If the local skin temperature determines the local vascular tone, the observational correlations of these two items should be made on a regional basis. The studies presented in the previous technical report [1] were limited to the forearm and finger. It was recommended that they should be extended to other regions. This has been done in the present series of experiments which included observations on the lower extremity and face as well as the forearm and finger.

Methods

The subjects were nude, except for brief shorts, and rested in a semi-reclining position on a specially designed chair with the back constructed of stainless steel screen. They were young medical or college students to whom the experimental procedure was explained in detail.

Two patterns of heat stress were imposed on the subjects as in the previous report. In the first, the ambient temperature was increased slowly from 25° to 45° C. over a period of 3 hours. In a second pattern the ambient temperature was cycled between 25° and 45° C. at a rate of about one cycle per hour.

Observations

The cutaneous volume pulses were recorded by means of specially designed photoelectric photometers which were mounted in plastic rings that permitted firm application to the skin with relative freedom from artifacts due to movements. Figure 1 illustrates the construction and mounting of these miniaturized units.

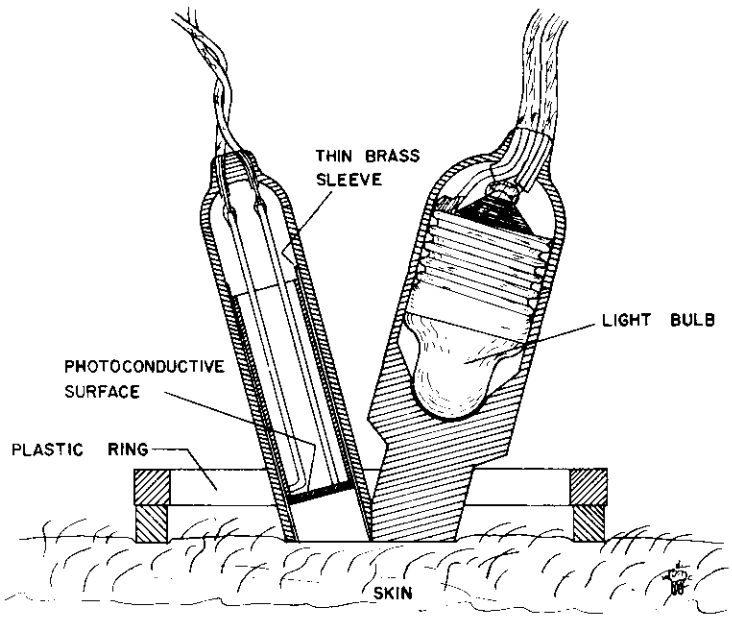


Figure 1. Photoelectric plethysmograph using a Clairex photoconductive cell. The plastic ring mount permits a firm anchoring to the skin, and avoids artifacts due to movements as well as changes in pressure of the unit against the skin.

The use of a solid photoconductive unit such as the Clairex photocell increases flexibility in mechanical design and eliminates circuit problems which result from the high impedance of phototubes but introduces a high temperature coefficient. Calibration was done by simply turning the light off and on. The amplitude of the skin pulse was then expressed as per cent of photoelectric current, I_p. Since the calibration was done at every interval of recording, small changes in the light emission from the tungsten filament and larger changes in the resistance of the photoconductive element due to change in its temperature were automatically corrected for in calculating the pulse

amplitude as per cent of I_p. Equivalent blood flow in the same skin area is approximated by using the expression,

$$\text{pulse} = 0.01\ I_p \doteq 0.1\ cm^3/cm^2 \text{ skin area/minute.}$$

Records were measured as in a preceding report.[1] Recordings of skin pulses were taken from the skin areas stated in the individual experiments.

Regional sweating was measured by means of dessiccating capsules. Skin and oral temperatures were recorded by thermocouples on a Brown 16 point recorder. Individual readings of skin temperature often seemed to be disturbed by beads of sweat which apparently altered local temperature. Subjects were weighed before taking their position in the chair and at the conclusion of the observations.

Results

Slowly rising ambient temperature.

Forearm and calf.

The responses of three subjects to the standard heat exposure have been selected to illustrate the diversity of the cutaneous vascular responses in the calves of subjects in whom the forearm's cutaneous vasodilatations were essentially similar.

A typical response was exhibited by subject F1 in that cutaneous vasodilatation in the calf was much less than that in the forearm (Figure 2). During the control period (ambient temperature 25° C.), the amplitudes of the skin pulses of the forearm and calf of subject F1 were essentially similar. At the end of the exposure to heat (25° to 45° C. in three hours), the skin pulses in the forearm were about four times as large as those in the calf. Possible regional differences in the rates of arterial perfusion of the skin, even in the hot subject, are well illustrated in this figure.

Although the cutaneous vasodilatation in the calf began at about the same time as that in the forearm, when ambient temperature was about 30.0° C., the vasodilatation attained its maximal value in the calf long before it had done so in the forearm, and remained at this level for two hours while chamber temperature was rising. The pulses in the toe pads were inscribed simultaneously and their amplitudes have been entered in this figure. The correspondence in time of the vasodilatations in the toe pad and calf skin might suggest a similarity in the vasomotor controls in these two regions.

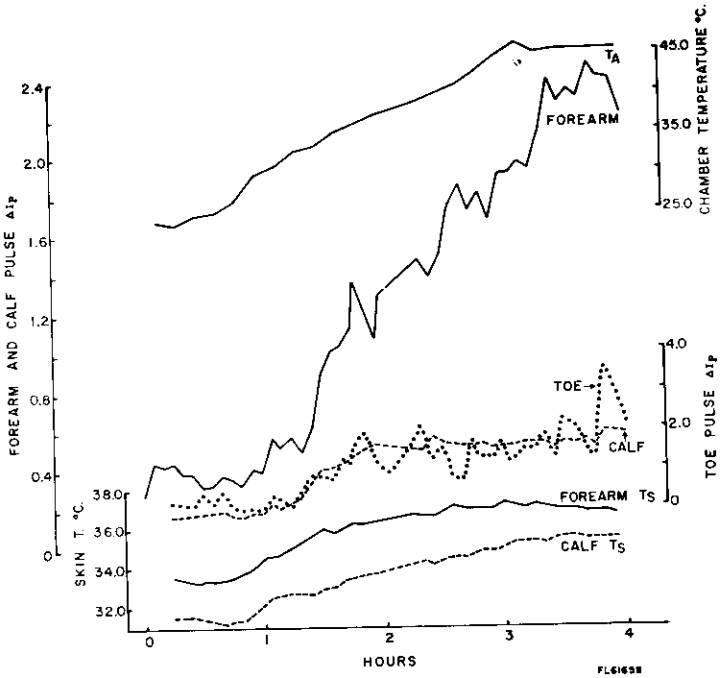


Figure 2. Changes in the cutaneous volume pulses of the calf, toe pad and forearm and in the skin temperatures (T_S) of calf and forearm during rising ambient temperature (T_A). Amplitudes of skin pulses, I_p, are charted as per cent of photoelectric current. Note different scale for the toe pulses. Experiment of FL61658.

Why did the cutaneous vasodilatations in the calf and toe fail to increase while that in the forearm did? This regional difference in cutaneous vascular responses to heat was observed often in these studies. A possibly important influence in the failure of the calf vessels to dilate as greatly as those in the forearm may be inferred from Figure 3. This graph of the relation between the local skin temperatures and the local skin pulses indicates that the calf skin vessels failed to dilate markedly in the experiment of Figure 2 simply because its local skin temperature did not rise to high enough levels. Thus, the amplitudes of the skin pulses in the two regions were the same at similar levels of local temperature. The precipitous increase in the forearm pulses began when local T_S was 35.7° C. which was 0.2° C. greater than the maximum value attained in the calf. The onset of

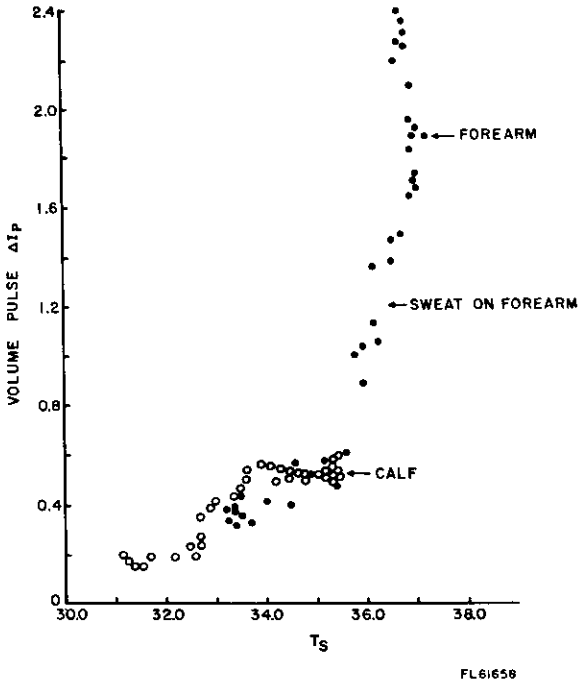


Figure 3. Relation of the skin pulses in the forearm and calf to the local skin temperature. Experiment of Figure 2.

vasodilatation in the two skin regions occurred at different levels of local T_S (forearm T_S = 33.7° and calf T_S = 31.4° C.). Sweating on the forearm appeared at the time that the calf vasodilatation stabilized.

When subject Fe was exposed to the standard heat stress, vasodilatation in the calf skin was slight, the maximal pulse amplitude was only 0.3% I_p (Figure 4). At the same time, the toe pulses (not shown) were also small while the finger vessels seemed to be widely if not maximally dilated. Perhaps the clue in this experiment was the early onset of sweating in this subject. Possibly as a result, skin temperatures stabilized early at relatively low levels. Forearm vasodilatation attained its maximal level early in the heat exposure and then tended to decrease somewhat while forearm sweating increased. A relatively high level of vascular tone continued in the lower extremity (calf and toe) while that in the upper extremity (forearm and finger) decreased early in the rise in ambient temperature. This same subject exhibited a similar behaviour in a preceding experiment.

An unusually large cutaneous vasodilatation occurred in the calf of subject K when he was exposed to the slowly rising ambient temperature (Figure 5). The dilatation in the calf skin began at about the same time as that in the forearm and proceeded more swiftly than that in the latter. Ambient temperature was about 30.0° C. at the time

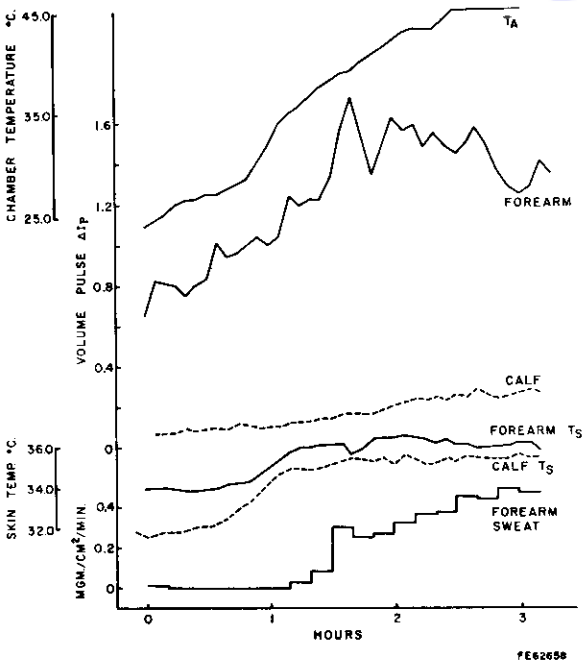


Figure 4. Changes in the cutaneous volume pulses and temperatures of the calf and forearm during rising ambient temperature. Experiment of FE62658.

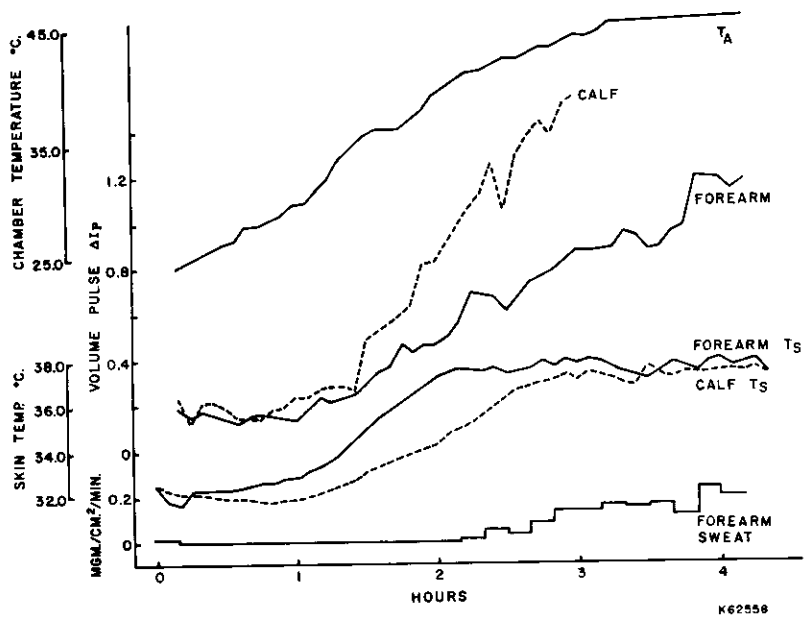


Figure 5. Changes in the cutaneous volume pulses and temperatures of the calf and forearm and in forearm sweating during slowly rising ambient temperature. Experiment of K62558.

of onset of the vasodilatation but the local skin temperature was nearly 1.0° C. higher in the forearm at this moment. The parallel character of the two cutaneous vasodilatations is shown best by charting them in relation to each other as was done in Figure 6 which was derived from another similar experiment on the same subject. At the moment of onset

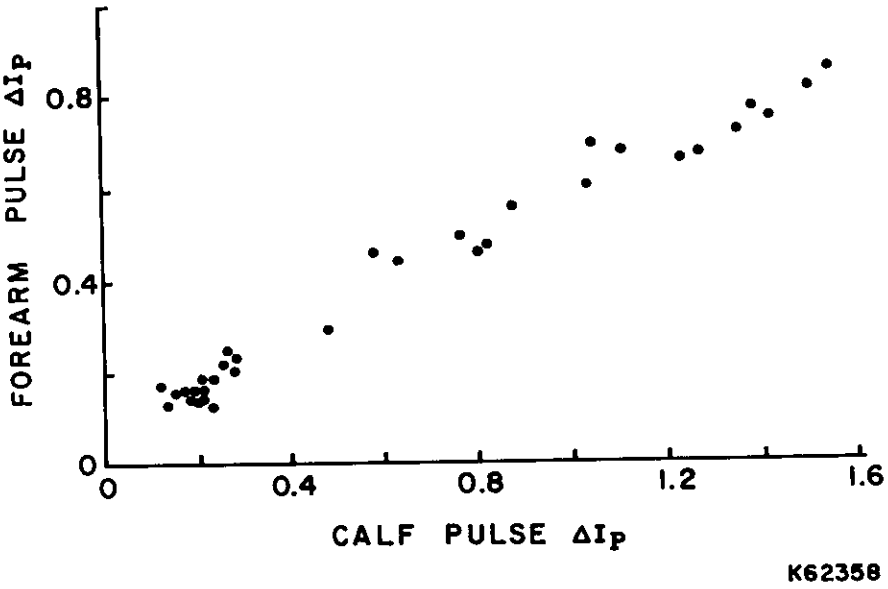


Figure 6. Relation of cutaneous volume pulses in forearm to those in calf during rising ambient temperature. Experiment similar to Figure 5.

of the vasodilatation the pulse amplitudes were equal in the two skin areas. The slope of the straight line relation was such that at the end of the recording of the skin pulses in the calf, the absolute vasodilatation was nearly twice as great there as in the forearm. The relation of the progress of the cutaneous vasodilatation to the local skin temperature differed markedly in the two regions. Figure 7 shows that the skin pulses in the calf increased linearly with the rise in calf T_S while those in the forearm showed the usual precipitous increase when T_S rose above 37.0° C. This inflection in the forearm pulse - T_S graph occurred long after the appearance of sweating on the forearm. The amplitude of the skin pulse in the calf at the end of the observations was near the maximal values which have been observed in this region while that in the forearm was less.

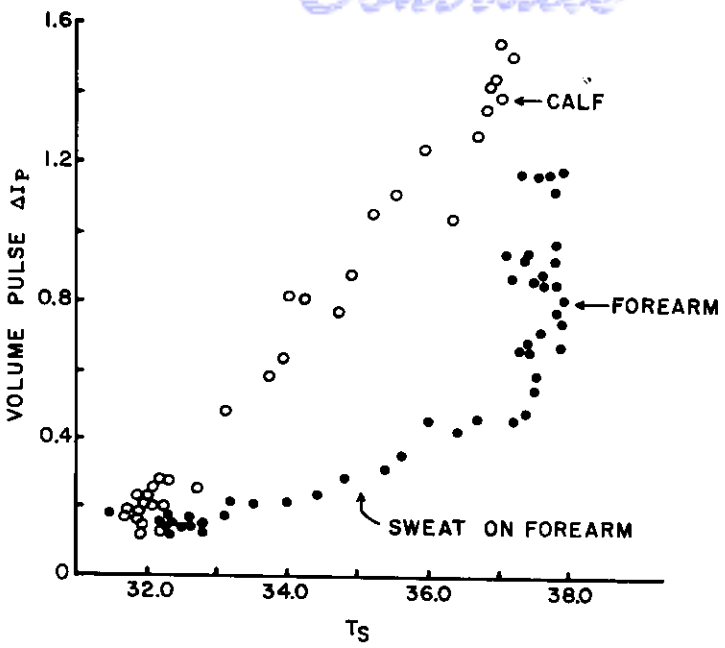


Figure 7. Relation of cutaneous volume pulses in forearm and calf to the local skin temperature. Experiment of figure 5.

In these three experiments, the initial calf pulses, as also the initial calf skin temperatures, were less than the corresponding values in the forearm skin (average of pulses 0.15% and 0.25% of I_p and average of local T_S 32.1° and 33.8° C. in calf and forearm respectively). The preliminary 1 hour control period (T_A = 25° C.) during which the subject cooled, decreased skin temperatures more in the lower extremity. Possibly, this may have prevented as great an increase in these temperatures during the rising phase of ambient temperatures. Correspondingly, the skin pulses in the calf were smaller. The marked exception to this statement is presented in Figure 5. The subject of this experiment was a "poor" sweater and his skin temperatures (calf and forearm) attained somewhat higher values than the corresponding ones in other subjects. Even so, the sharp vasodilatation in the calf began while local skin temperature was still below 33.0° C. The vasodilatation in the toe (not shown in Figure 5) was similar to that in the calf.

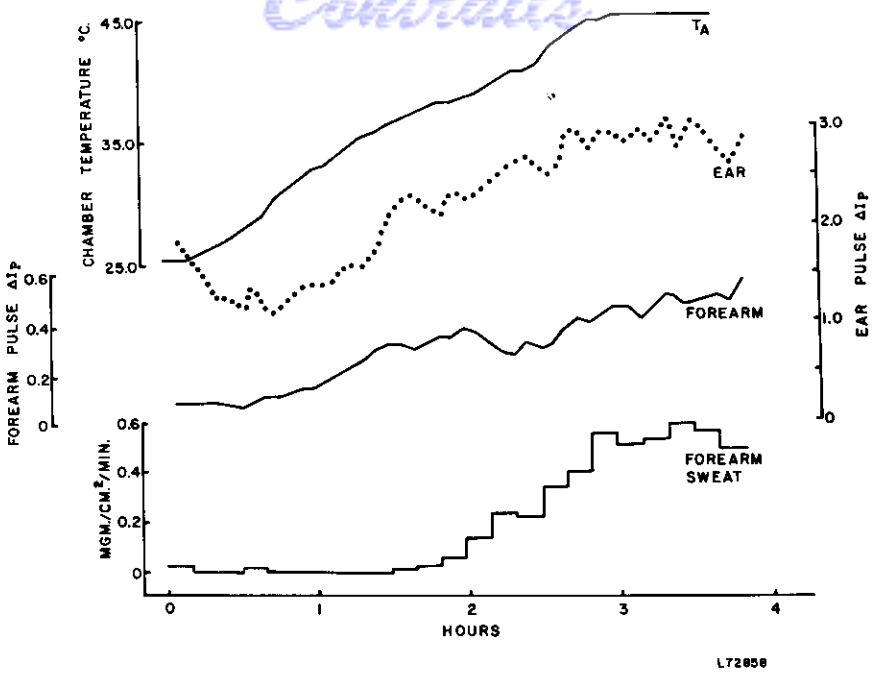


Figure 8. Changes in the cutaneous volume pulses of the ear and forearm during rising ambient temperature. Experiment of L72858.

Forearm and ear.

A preliminary report of Fox, Goldsmith, and Kidd[4] stated that the vasodilatation in the ear during indirect body heating proceeded in a manner which was analogous to that in the forearm. This finding was based on the use of a specially designed surface calorimeter. The results of six experiments, one of which is charted in Figure 8 tend to confirm their report. The onset of vasodilatation in the ear occurred very nearly at the same time as that in the forearm. The per cent increase in the ear pulses was somewhat less than that in the forearm pulses. The reverse was true in the experiment charted in Figure 9. The relations of the two pulses in this experiment as vasodilatation developed are illustrated in Figure 10. Similar straight line graphs were obtained in all experiments, but the slopes of the lines differed in the several experiments. The increases in the amplitudes of the ear pulses were usually greater than the increases in the forearm pulses. However, the greater magnitude of the vasodilatation in the ear does not necessarily represent a relatively greater relaxation of vascular tone but may instead be due to the greater number of blood vessels per unit volume of tissue.

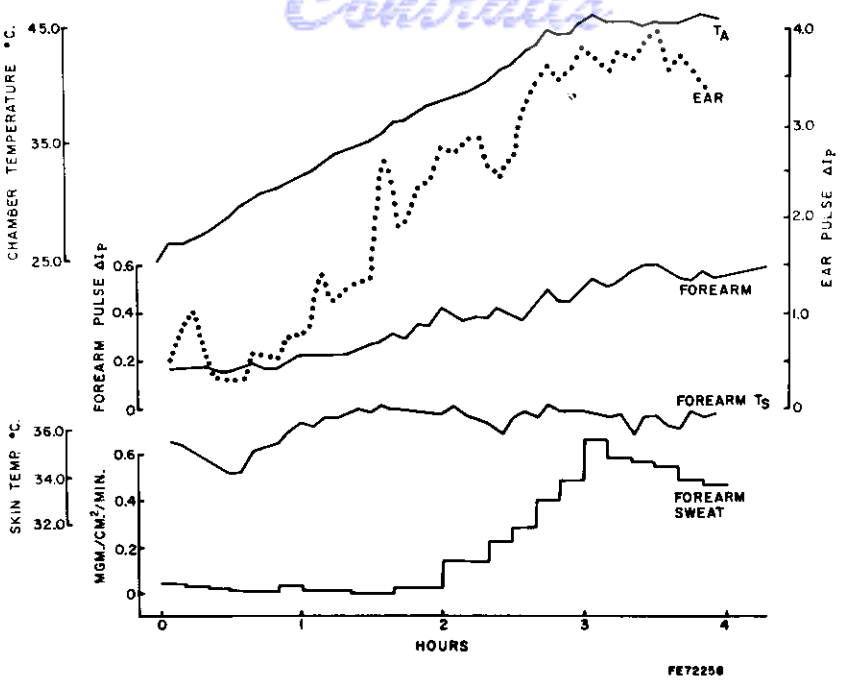
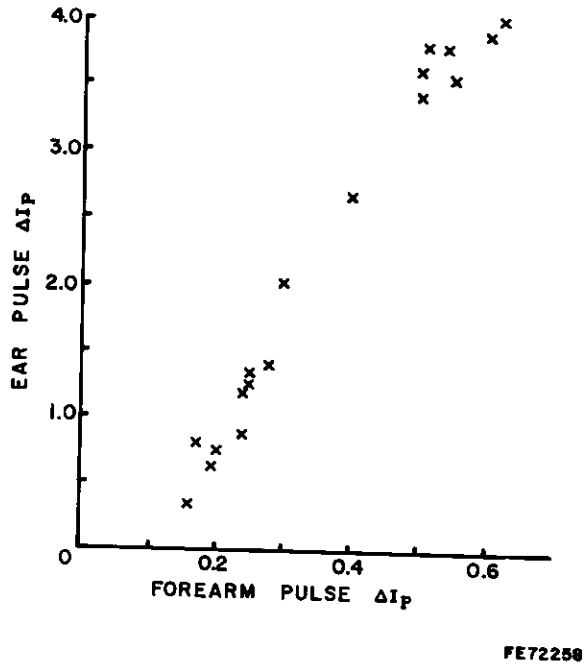


Figure 9. Changes in the cutaneous volume pulses of the ear and forearm during rising ambient temperature. Experiment of FE72258.

Figure 10. Relation of cutaneous volume pulses in the forearm to those in the ear in the experiment of Figure 9.



Forearm and cheek.

The vascular responses in the cheek were less regular in character than those in the forearm and ear.

Vasodilatation was negligible in the cheek in three of six experiments. In two, it appeared at the same time as in the forearm and progressed parallel to that in the forearm. In one experiment, vasodilatation occurred earlier in the cheek than in the forearm. The course of events in one experiment is illustrated in Figure 11.

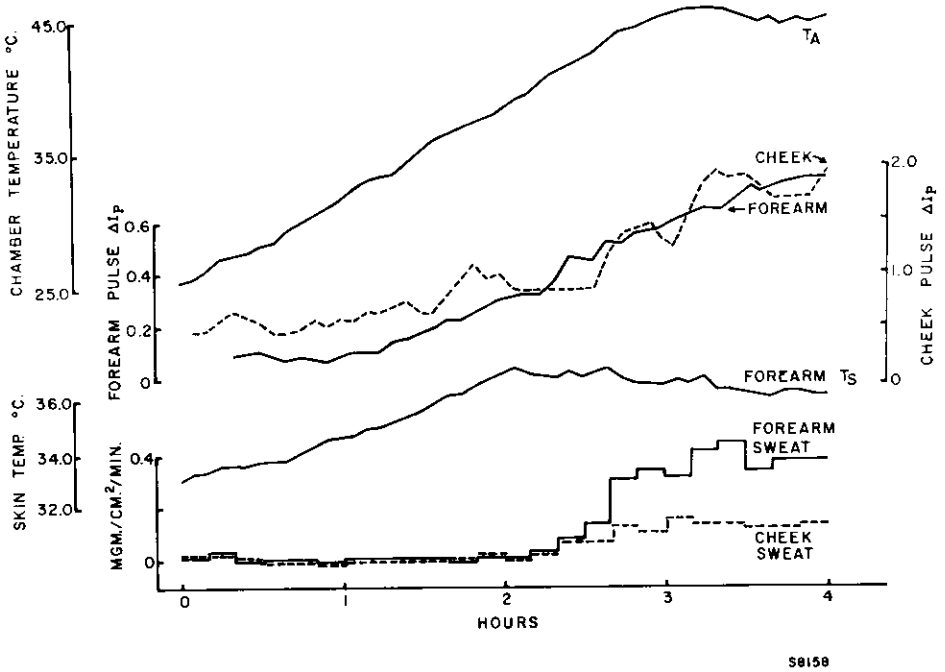


Figure 11. Changes in the cutaneous volume pulses and sweating in the forearm and cheek during rising ambient temperature. Experiment of S8158.

The onset of sweating occurred simultaneously in the forearm and cheek but showed no consistent relation to the onset of vasodilatation in the cheek. Considerable vasodilatation could occur in the cheek before sweating was detectable there.

Cycles in ambient temperature.

Calf and forearm. Six experiments were done on four subjects, to whom three complete cycles in ambient temperature (25° - 45° - 25° C.) were presented in each experiment. One such experiment is charted in Figure 12. The skin temperatures of the calf and forearm followed the

ambient temperature, but that of the calf was consistently lower than that of the forearm by as much as 4° C. When the amplitudes of the skin pulses were charted as per cent of the maximum pulse attained during the vasodilatation, the cycles of vasodilatation and of vasoconstriction in these two vascular beds seemed to develop concurrently.

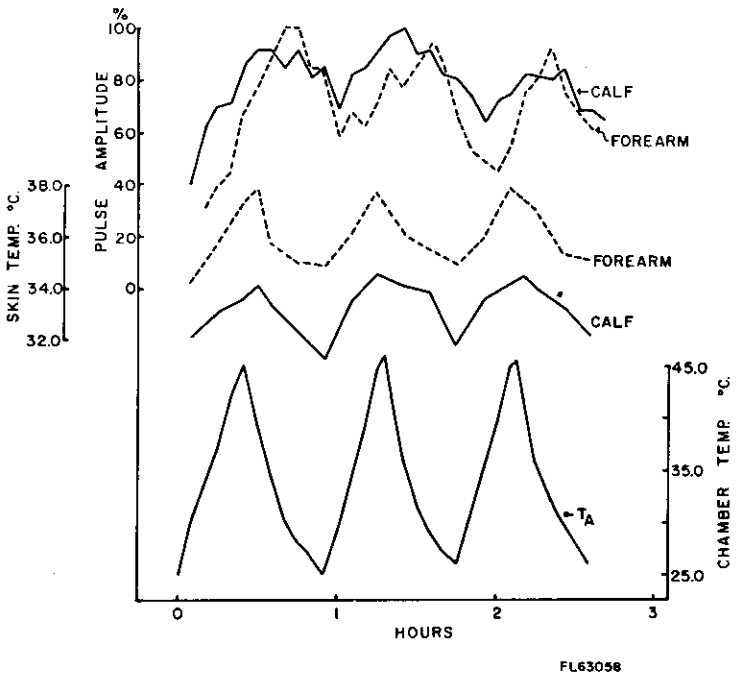


Figure 12. Changes in the cutaneous volume pulses and temperatures of the calf and forearm during cycles in ambient temperature. Amplitude of the skin pulse is charted as per cent of the maximum attained in the experiment. Experiment of FL63058.

Forearm, cheek and ear. Twelve experiments were done on five subjects. An illustrative experiment is charted in Figure 13. The tendency shown here of the cycles of vasoconstriction and of vasodilatation to proceed synchronously in these three areas was exhibited definitely in eight experiments. In two other experiments, the cheek vessels dilated during the first rise in ambient temperature and remained dilated during the remainder of the run. The cycles in the forearm and ear were essentially synchronous in ten experiments. In two others, the temporal relations of the cycles in ambient temperature and in the ear vessels were out of phase, probably due to technical errors in recording.

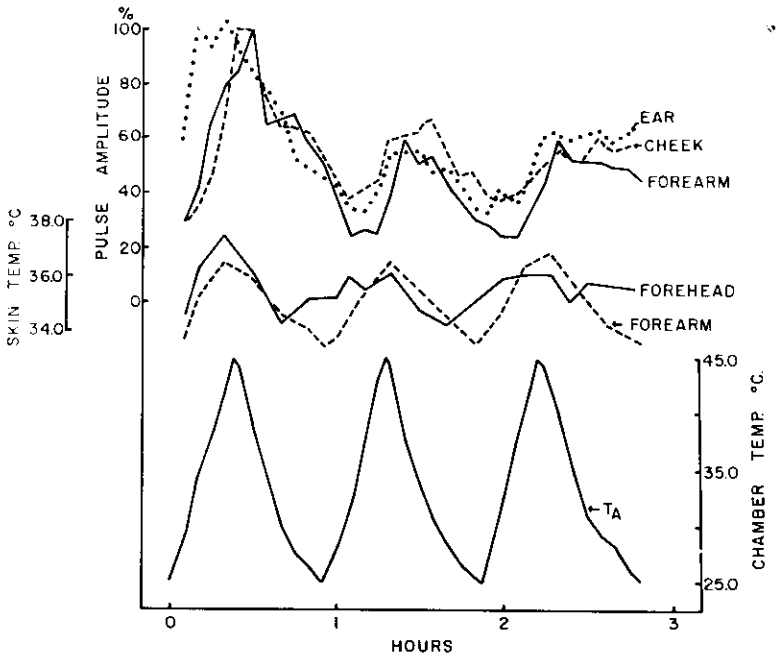


Figure 13. Changes in the cutaneous volume pulses of the ear, cheek and forearm and in the skin temperatures of the forehead and forearm during cycles in ambient temperature. Experiment of FE71158.

DISCUSSION

Benzinger considered the cutaneous vasomotor innervation to be the principal determinant of the level of cutaneous vascular tone and assigned a secondary influence to the local skin temperature [8]. Since he used the calculations of total conductance as an index of cutaneous blood flow, he failed to distinguish between possible regional differences in the cutaneous vascular responses which have been demonstrated [1,4,6,9]. Just as regional differences in the onset and progress of sweating are pertinent to the study of the nervous controls, so also are regional differences in the cutaneous vascular responses essential data in the description and analysis of the mechanisms which regulate the cutaneous blood flow.

The end ambient temperature of the heat exposure was high enough to elicit sweating in all regions of the body. Although these rates were submaximal this was an advantage in studying regional differences in the onset and progress of sweating [5] and likewise in the cutaneous vasodilatation.

The most striking regional differences in the cutaneous vasodilatations during the heat exposures were noted in the forearm and calf. Failure of cutaneous vasodilatation to occur in the calf or its progress to a relatively low level was observed in all but two experiments on one subject, although marked cutaneous vasodilatation appeared in the forearm in all experiments. Correspondingly, skin temperatures were higher in the forearm. In the exception, illustrated by Figure 5, skin temperature in the calf rose to approximately the same level as that in the forearm. Cutaneous vasodilatation in the ear and cheeks proceeded in a manner quite analogous to that in the forearm.

An explanation of these observations must pay particular attention to the usually slight cutaneous vasodilatation in the lower extremity at the time when the vessels in the forearm skin were widely dilated. This difference was not due to a lesser vascularity of calf skin; previous measurements [9] demonstrated that as high rates of blood flow existed in calf skin as in forearm skin when the blood vessels were maximally dilated. Our preceding study [1] suggested that the cutaneous vasodilatation which was elicited by exposure of the nude subject to a rising ambient temperature resulted from the operation of multiple factors, prominent among which was the local skin temperature. On applying this thought to the lower extremity, four factors required particular attention.

First, sweating which presumably liberates the vasodilator substance bradykinin [10] was always present on the calf when observed on the forearm. Although the reverse may be true in some subjects at low levels of sweating, we have never noted the absence of calf sweating at the time of forearm sweating in normal subjects. That bradykinin is a major factor in the cutaneous vasodilatation elicited by general body heating is contradicted by the experiments illustrated in Figures 2, 3 and 4, in which cutaneous vasodilatation in the calf was slight or almost absent despite the presence of sweating. This failure of sweating to induce vasodilatation in the same area had been noted previously in this laboratory [1]. It may be especially significant that the poorest sweater in the present series of experiments exhibited the greatest cutaneous vasodilatation in the calf (Figure 5).

Second, the failure of as marked cutaneous vasodilatation to occur in the calf as in the forearm appeared to be related in most of the experiments to lower skin temperatures in the calf. In the two experiments on the same subject (Figure 5) in which the calf vasodilatation exceeded that in the forearm skin, the calf skin temperature

Approximated the forearm skin temperature. An earlier onset of sweating at lower skin temperatures (Figure 4) prevented as great a rise in skin temperatures as occurred in subjects in whom sweating was delayed. Thus, in subject Fe of Figure 4, forearm sweating began at the end of the first hour of rising chamber temperature when it was 34° C. and in subject K of Figure 5 at the end of the second hour and when chamber temperature was 38° C. Sweating rates were correspondingly less and skin temperatures higher in subject K.

Third, if the cutaneous vasodilatation in the calf were due only to reduction in vasoconstrictor tone, one might expect the vasodilatation in the toe and calf to proceed in a parallel manner, as was true in these experiments. However, it is doubtful that the calf vasodilatation of Figure 5 can be accounted for only by release of vasoconstrictor control since neither nerve nor adrenergic block resulted in a great increase in blood flow in calf skin [3]. Some unpublished observations in this laboratory made during injection anesthesia of lumbar sympathetic ganglia demonstrated a small increase in calf skin pulses when the simultaneous increase in the toe pulses was very large. With the sole exception of subject K of Figure 5, the cutaneous vasodilatations in the calf elicited by the heat exposures were not distinguishable from those resulting from nerve block.

Fourth, there was no indication that the vasodilator supply to calf skin [3] was used in eliciting vasodilatation in these heat exposures, excepting in subject K of Figure 5. The striking vasodilatations in his calf skin and the unusual relation of the vasodilatation to the local skin temperature (Figure 6) suggest that vasodilator impulses were responsible for the vascular relaxation.

The behavior of the skin circulation in the thigh and calf should be of massive importance to thermal kinetics because of the large surface area of the lower extremity. However, one may predict from the equation for convective heat transfer [1] that if an earlier onset of sweating in the cooler lower extremity prevented as large an increase in local skin temperature as that occurring in the forearm, the required heat transfer could be effected without as large an increase in blood flow as that which would be required in the hotter forearm since the temperature of the entering arterial blood would be nearly the same in the two regions. This teleological argument agrees with our measurements of the skin pulses in the calf and forearm. The relation of the local skin temperature to the local vascular tone seems to be more than coincidental but the mechanisms of the relation are not clear.

BIBLIOGRAPHY

1. Senay, L.C.,Jr., M.L. Christensen and A.B. Hertzman. Finger and forearm cutaneous blood flows during changing ambient temperature. WADD, TR 60-15, March 1960, 46 pp.

2. Hertzman, A.B. "Vasomotor regulation of cutaneous circulation." Physiol. Rev. 39: 280-306, 1959.

3. Blair, D.A., W.E. Glover and I.C. Roddie. "Vasomotor fibres to skin in the upper arm, calf, and thigh." J. Physiol. 153: 232-238, 1960.

4. Fox, R.H., R. Goldsmith and D.J. Kidd. "Cutaneous vasomotor nerves in the human ear and forehead." J. Physiol. 150: 12, 1960.

5. Hertzman, A.B. "Individual differences in regional sweating." J. Appl. Physiol. 10: 242-248, 1957.

6. Hertzman, A.B. and L.W. Roth. "Absence of vasoconstrictor reflexes in the forehead circulation. Effects of cold." Am. J. Physiol. 136: 692-697, 1942.

7. Hertzman, A.B. "Individual differences in regional sweating." J. Appl. Physiol. 10: 242-248, 1957.

8. Benzinger, T.H. "On physical heat regulation and the sense of temperature in man." Proc. Nat. Acad. Sci. 45: 645-659, 1959.

9. Hertzman, A.B. and W.C. Randall. "Regional differences in the basal and maximal rates of blood flow in the skin." J. Appl. Physiol. 1: 234-241, 1948.

10. Fox, R.H. and S.M. Hilton. "Bradykinin formation in human skin as a factor in heat vasodilatation." J. Physiol. 142: 219-232, 1958.

11. Blair, D.A., W.E. Glover and I.C. Roddie. "Cutaneous vasomotor nerves to the head and trunk." J. Appl. Physiol. 16: 119-122, 1961.