Thermal regulation during water immersion[1]

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CRAIG, ALBERT B., JR., AND MARIA DVORAK. *Thermal regulation during water immersion.* J. Appl. Physiol. 21(5): 1577–1585. 1966.—Ten subjects were studied during head-out immersion in nine different water temperatures ranging from 24 C to 37 C. The period of immersion at each temperature was 1 hr, during which time various body temperatures, pulse rate, blood pressure, and $\dot{V}O_2$ were observed. In water temperatures less than 35.6 C there was a reduction in central body temperature despite the fact that vasomotor controls of heat loss were evident. Increased heat production was noted if the water temperature was 30 C or less. Water temperatures of 36 C or more imposed a heat stress on the subject causing an increase in the pulse rate and pulse pressure. It is suggested that there is a very narrow range of water temperature (35.0–35.5 C) which can be considered as "neutral."

blood pressure; circulatory responses; heart rate; heat exchanges; hyperthermia; hypothermia; oxygen consumption; temperature, auditory canal; temperature, rectal; temperature, skin; tissue insulation; thermal conductivity

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MAN'S ABILITY to adjust to a range of atmospheric conditions as measured by temperature, humidity, and air movement has been studied extensively. From this large volume of literature it is known that responses to thermal stresses involve adjustments of heat production, alterations of the circulation, and changes in water vaporization from the surface.

Except for decreased loss of heat by vaporization, it would be expected that similar responses would occur during water immersion. Several studies (4, 5, 10, 11, 14, 18, 20) during cold-water immersion indicate that increased heat production does occur at certain critical water temperatures, depending on the time of immersion, and that blood flow to the limbs changes quite dramatically (19). However, most of the studies were designed to study man's tolerance to cold, and there is a remarkable paucity of data to evaluate the heat stresses of warm-water immersion.

The present investigation was designed to study the responses of a group of subjects in a range of water temperatures which would include both cold and heat stresses. The purpose was to provide a description of man's responses in water of different temperatures. The results are compared to those from studies of man in the air environment.

In addition, these studies provide a working definition of a "neutral" range of water temperatures in which man's physiology can be studied. To date it has been difficult to know if the results of studies employing water immersion can be ascribed to the effects of immersion itself or to the thermal stresses imposed (3, 15).

METHODS

Ten male subjects were selected for these experiments. They were of similar build and none was considered to be obese. Their physical and physiological characteristics are listed in Table 1. Each subject was immersed for 1 hr at a selected temperature, and experiments in other water temperatures were repeated at approximately weekly intervals. All of the subjects finished the entire series. The experiments were done between December 28 and April 15.

No attempt was made to control the time of day at which the experiments were done. Each subject came to the laboratory at his own scheduled time which was generally either morning or afternoon for that individual. All subjects were at least 1 hr postprandial. They changed to swimming trunks, lay on a cot, and were covered with a light cotton blanket for at least 20 min before control measurements of $\dot{V}O_2$ and heart rate were made. The room temperature varied between 25 C and 28 C.

The subjects were then transferred to a stretcher which was made of aluminum mesh (60% open area) and which was suspended from a track anchored to the ceiling. A back- and headrest made of the same material enabled the subjects to be in a semirecumbent position. When they were in the water the only points of contact with the stretcher were the buttocks and the upper posterior surface of the back at about the scapulae.

The stretcher was raised to the level of the water bath and moved into position in preparation for immersion. Blood pressure and skin temperatures at six different sites (8) were measured immediately before immersion.

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TABLE 1. *Characteristics of subjects* ($n = 10$)

Physical		Physiological at Rest	
Age, year	26 ±5.6	Resting $\dot{V}O_2$, ml/min	279 ±26
Height, cm	177.1 ±3.2	Resting metabolic rate, kcal/m² per hr	43.1 ±4.0
Weight, kg	72.8 ±5.2	Temp, rectal, °C	36.95 ±.17
Surface area, m²	1.89 ±.09	Temp, ear, °C	37.04 ±.19
LBM, kg	61.4 ±6.4	Temp, finger, °C	34.2 ±1.1
% Fat	15.5 ±8.1	Temp, abdomen, °C	34.8 ±.7
Subcut fat thickness, mm	5.4 ±1.4	Temp, mean, skin, °C	34.2 ±.3
		Heart rate, beats/min	±4.4 63
		Blood pressure, mm Hg	126/81 ±5/±5

Values are means ± standard deviation. Lean body mass measure by whole body counter (13). Subcutaneous fat thickness measured with Gersmehl calipers, 2 mm allowed for skin thickness, arithmetic mean, 6 sites (7).

The subjects were then quickly immersed until the level of the water was just below the chin anteriorly and the occipital ridge posteriorly. During the immersion the subjects were not permitted to cross their legs or hold their arms against the body.

The water bath was 1.2 m wide, 2.5 m long, and 1.5 m deep. The two circulating pumps assured adequate mixing and circulation of the water, and the temperature was controlled within ± .05 C.

All temperature measurements were made with thermistors whose resistance was read by specially constructed Wheatstone bridge circuits. These probes were calibrated at the same time and in the same water baths. The rectal and ear temperature thermistors could be read to ± .01 C and did not vary more than ± .025 C between frequent recalibrations during the series of experiments. The skin and water temperature probes could be read to ± .05 C and rechecked to this range when recalibrated.

The flexible rectal probe was inserted between 20 and 30 cm into the rectum. There was no control over whether it was against the anterior or posterior surface of the rectum. The ear probe, which was a small thermistor inside a polyethylene tubing (od .64 mm) was inserted into the external auditory canal until the end hit the tympanic membrane as indicated by pain or by an audible click. During preliminary experiments, the subjects complained of discomfort when the probe remained against the membrane, so in the present series the thermistor was withdrawn 1 to 2 mm. The tubing was then taped to the pinna and cotton was loosely packed into the canal. Another piece of cotton was placed in the outer folds of the pinna which was then covered with pieces of adhesive tape to keep out drops of water. This ear was then covered with a knitted headband of the type used by skiers to protect the ears from cold. Temperature at this site is referred to as ear temperature in the illustrations and tables.

Surface skin temperatures were measured with disk thermistors 9.9 mm in diameter and 2.4 mm thick. The mean skin temperature before immersion was calculated as the weighted average of observations at six different sites (8). Two other disk thermistors were used to measure surface temperatures during immersion. One was placed on the palmar surface of the distal phalanx of the right middle finger, and the other on the skin of the right anterior abdominal wall just above the iliac crest. Each thermistor was covered with a small wad of cotton. The finger was then covered with a latex finger cot, the proximal end of which was cut to assure that it did not restrict blood circulation. The thermistor on the abdomen was covered with adhesive tape. No attempt was made to keep these thermistors dry. The coverings were used to restrict the circulation of water in these areas with the expectation that the thermistors would simulate a probe just below the surface of the skin.

$\dot{V}O_2$ was measured with the closed circuit Benedict-Roth apparatus. Two control measurements were averaged, and during immersion the observations were timed so that the middle of the 6-min period occurred at 10, 20, 30, 40, 50, and 60 min of immersion.

Heart rate was counted during the measurement of $\dot{V}O_2$ in both the control and experimental periods. There was no evidence that breathing 100% O_2 influenced the subject's heart rate during these experiments.

Special methods were necessary to measure blood pressure by the auscultatory technique during immersion. A U tube filled with mercury replaced the usual anaeroid gauge. The end of the U tube, which is usually

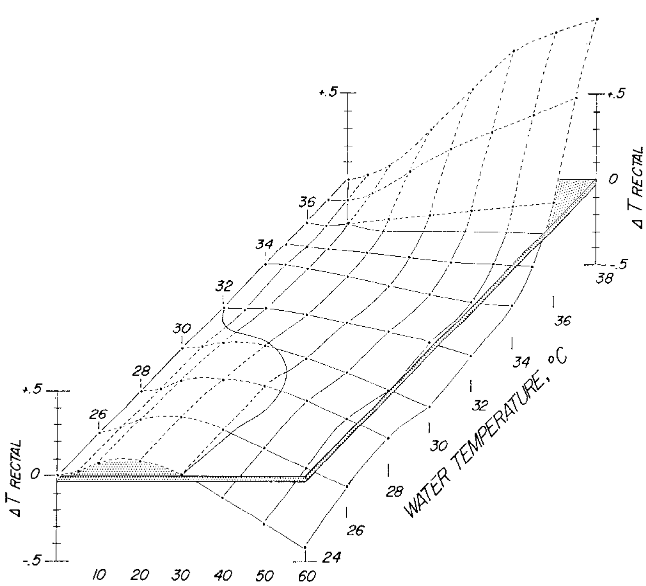

FIG. 1. Relationships between changes in rectal temperature, water temperature, and time of head-out immersion. Dotted lines indicate an increase and the solid lines a decrease in rectal temperature as compared to control readings before immersion.

open to air, was connected to tubing on the other end of which was attached a small latex rubber balloon. This compensating bulb was fixed to the blood pressure cuff. The cuff and the compensating bulb were kept at heart level when the subject was in air or when he was in water. Thus, the blood pressures were referred to ambient pressure at the level of the heart in air and in water, and were differential pressures in keeping with the usual convention.

The data from all 90 experiments were considered acceptable for evaluation and interpretation with the following exceptions. In two experiments the cotton surrounding the ear thermistor became damp and the temperature measurements were obviously low. In one experiment in 24 C water, the subject's $\dot{V}O_2$ was recorded as being between 1.0 and 2.5 liters/min. These values were more than twice as great as any other subject and were also out of line with this subject's previous measurements in 26 C water. These results were discarded even though the suspected leak in the apparatus could not be located.

Calculations of heat exchanges were made using the assumption that there was a peripheral mass equal to .4 body weight. During immersion the peripheral temperature was assumed to be water temperature. The core temperature as measured by the rectal temperature was assumed to be the temperature of the remaining .6 of the body's mass. The use of rectal temperatures rather than ear temperatures was preferred only because of the usual convention of using rectal measurements for such calculations (20).

In calculating thermoconductivity or its reciprocal, insulation index, it was assumed that 8% of the heat loss occurred through the respiratory tract and that all of the immersed surface was available for heat exchange (20). When the central temperature was not constant, the total change in heat stores was calculated as occurring through the skin.

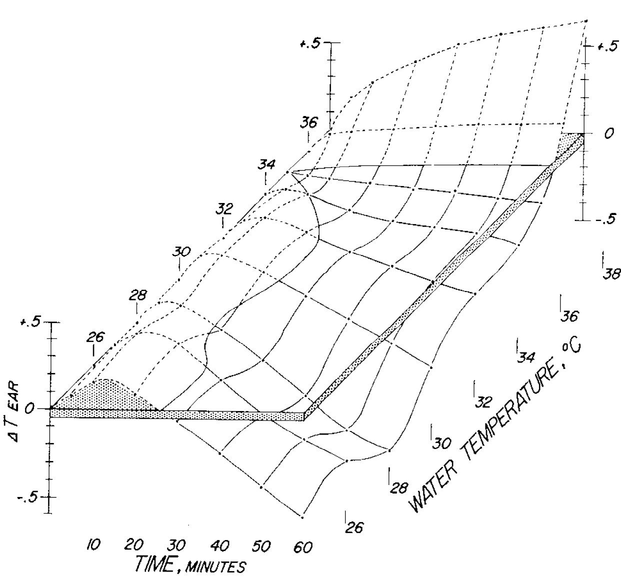

FIG. 2. Relationships between changes of temperature in the insulated external auditory canal (ear), water temperature, and time of immersion. Dotted lines indicate an increase and the solid lines a decrease in ear temperature as compared to control readings before immersion.

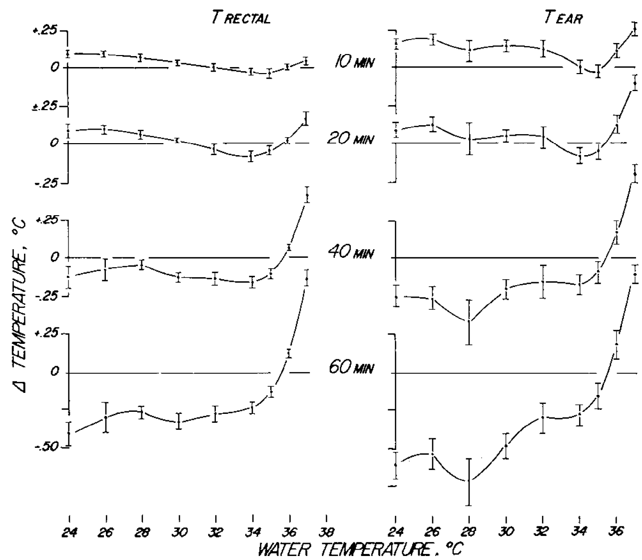

FIG. 3. Changes in rectal and ear temperature during the times indicated as functions of water temperature. Vertical bars indicate ±SE.

RESULTS[3]

Central temperatures. In Figs. 1 and 2 the rectal and ear temperature changes have been depicted as functions of time and water temperatures. When the water temperature was 36 or 37 C there was a continuous increase in the central temperatures. The changes in rectal temperature of three subjects during an hour's immersion in 38 C water are also shown in Fig. 1. Other subjects were not subjected to this water temperature because of discomfort. Although the three subjects were not unduly uncomfortable during the period of immersion at 38 C, each experienced a severe generalized throbbing headache several hours afterwards.

When the water temperature was 35 C or less, the central temperatures were below control values at the end of the hour's immersion. These decreases in temperature were preceded by an initial rise in the rectal temperature when the water was 30 C or less. The ear temperature showed an initial increase when the water was 34 C or less. These changes in temperature resulted in an apparent paradox which can best be seen in Fig. 3. After 20 min of immersion, it was evident that the

[3] Detailed tables of the results and statistical evaluations have been deposited as Document number 8967 with the ADI Auxiliary Publications Project, Photoduplication Service, Library of Congress, Washington, D. C. 20540. A copy may be secured by citing the Document number and by remitting $1.25 for photoprints, or $1.25 for 35-mm microfilm. Advance payment is required. Make checks or money orders payable to: Chief, Photoduplication Service, Library of Congress.

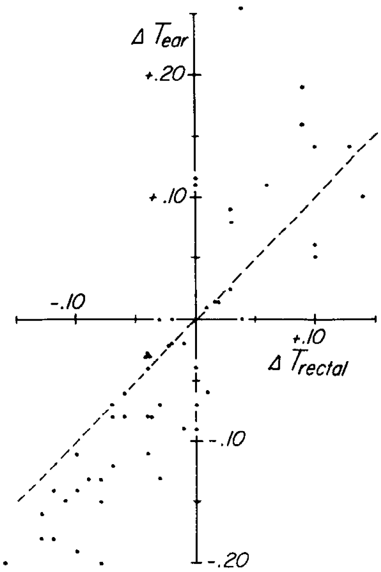

FIG. 4. Comparison of the changes in rectal temperature with changes in ear temperature during 10-min intervals. Each point represents the average changes for the group of 10 subjects during 10-min intervals of the hour immersion. Results from experiments in all nine water temperatures are plotted. The 45° diagonal indicates equal temperature change at each measuring site. When the decrease in the ear temperature is more than .05 C, changes at this site are of greater magnitude than changes observed in rectal temperature. Increases in these central temperatures cannot be quantitatively related in this study.

subject's central temperatures were higher in the cold water than in 34 C water. This has been noted before (10, 18).

Although the rectal and ear thermistors indicated temperature changes which were qualitatively similar, there were significant quantitative differences as shown in Fig. 4. The mean change in rectal temperature between successive 10-min observations has been plotted against change in ear temperatures at the same times. In general, the changes in ear temperature occurred faster and were greater than those noted in the rectum (6, 12). When the temperature reduction in the rectum during a 10-min interval was more than .05 C, the change of ear temperature was approximately 1.5 times greater than the rectal change.

When the central temperatures increased, the change in ear temperature was most often greater than the rectal, but in several intervals, rectal temperature increased more than the ear temperature. The latter results were noted in the last 20 min of immersion in 36 and 37 C when the ear temperature was nearing a steady value. The increase in rectal temperature lagged behind that in the ear, so in the 40- to 60-min period the rectal temperature was changed more rapidly than the ear. The same relationships were not noted in the cooler waters because, as mentioned before (19), the subjects' central temperatures were nowhere near attaining a steady value at the end of an hour.

Peripheral temperatures. The insulated surface thermistors indicated that under certain conditions a considerable temperature difference between the probe and the water existed (Fig. 5). The temperature of the finger probe was very near water temperature when the latter was between 24 and 32 C. The greatest differential was noted when the water was 34–36 C, but again at 37 C there was only a slight difference. The insulated thermistor located on the anterior abdominal wall was significantly warmer than the probe on the finger when the water was less than 34 C. The difference between the abdominal thermistor and the water increased as the water became colder.

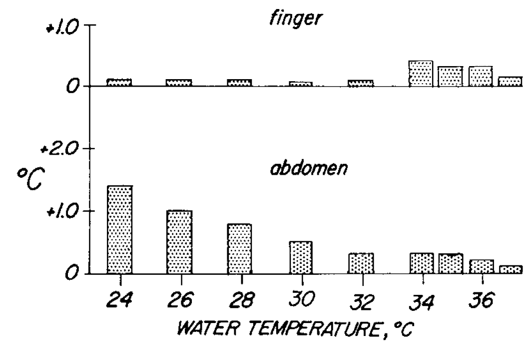

FIG. 5. Difference in the temperature of the covered skin site from the water temperature at the end of the hour's immersion.

If these thermistors reflect the subsurface skin temperature, it is evident that the periphery of the body is not at a uniform temperature at the end of an hour's immersion in water less than 34 C.

Oxygen consumption. Shivering and increased $\dot{V}O_2$ was observed throughout the hour in water of 28 C or colder, and was greater at the end of the period of immersion than at the beginning (Fig. 6). As has been observed many times by others (2), shivering occurred first in waves and then became continuous and uncontrollable. In 24 C shivering was so severe that the subjects were in continuous movement involving all muscles of the extremities and trunk. It was somewhat surprising that the $\dot{V}O_2$ was not increased more than 2.2 times, but it must be remembered that the subject in water is not working against gravity.

In Fig. 7, change in the $\dot{V}O_2$ at different times has been plotted as a function of water temperature. If one wishes to define the range of water temperatures in which the subjects would not experience shivering, it is necessary to consider the time of immersion. $\dot{V}O_2$ would be increased in water less than 30 C if the period of immersion were 40 min or less, but if the immersion at rest were for 1 hr, the water temperature would have to be at least 32 C to prevent shivering (20).

In water of 37 C there was a slight, but significant, increase in the $\dot{V}O_2$ at the beginning and at the end of the hour. This might be explained by the subjects' movements, for most people became slightly restless in this temperature. The increase in $\dot{V}O_2$ did not parallel the increase in central temperatures, so it is hard to attribute it to increased resting metabolic rate.

Cardiovascular responses. In the warm water (36 and 37 C) there was a decrease of the systolic and diastolic blood pressures, an increase in pulse pressure, and an increase in the heart rate (Figs. 7 and 8). The changes in the blood pressure were immediate and were constant throughout the hour, but the increase in heart rate was greater at the end than at the beginning of the period of immersion. These observations are probably the result of peripheral vasodilitation caused by the heat load in the warm water.

In water of 35 C or less the heart rate was significantly less than control, even when the subjects were shivering. In this cooler range the heart rate was independent of water temperature. In general, the systolic blood pressure was also slightly less than control, but the diastolic pressure was not different. However, at 24 and 26 C the blood pressure was not different from control values.

These alterations of cardiovascular function in the midrange of water temperatures suggest that the blood flow to the skin was minimal and that the demands for cardiac output were also minimal under these resting conditions. The relative increase in systolic blood pressure in the cold water may have been the result of the exercise of shivering. One would have also expected to have an increase in the heart rate with shivering. In air the heart rate has been observed to increase with shivering, but only in the upright subject. When the subjects were in the supine position, there was little or no increase in rate (2).

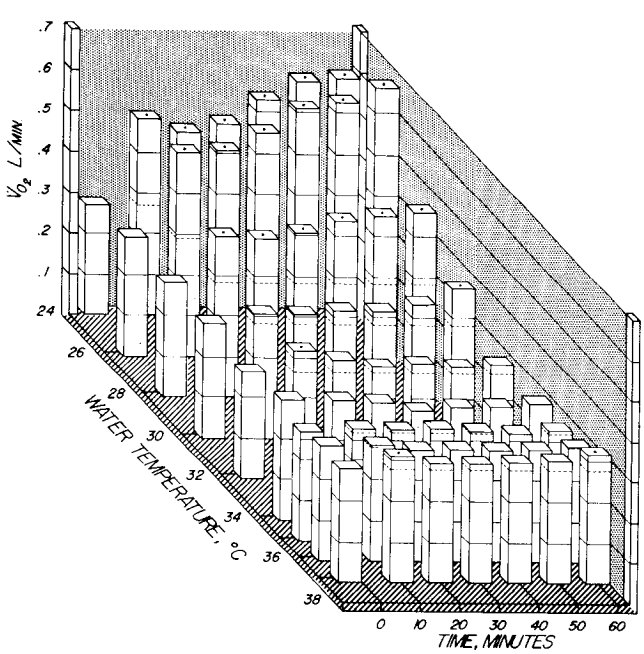

FIG. 6. $\dot{V}_{O_2}$ before and at different times of immersion as a function of water temperature. The columns to the left of zero time indicate the average of duplicate control observations. Dashed lines on the columns indicate the control value before immersion in the indicated water temperature. Dots on the tops of some columns signify that the $\dot{V}_{O_2}$ is significantly different from control ($P \leq .05$).

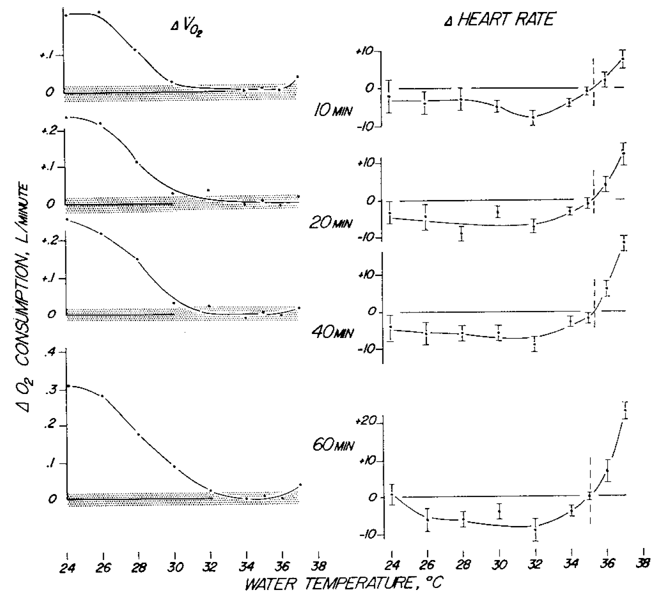

FIG. 7. Changes in $\dot{V}_{O_2}$ and heart rate at the times indicated as a function of water temperature. The shaded area on the left indicates ±SE of the average of 10 control duplicate observations of $\dot{V}_{O_2}$. Vertical bars in the right-hand graph show the SE of the changes in heart rate from control observations. Vertical dotted lines indicate the water temperature in which no change of heart rate would occur at rest.

Subjective observations. Most of the subjects enjoyed immersion in the temperature range of 34–36 C. After being in 37 C water, they reported that they were tired, restless, and had difficulty studying.

When immersed in 30 and 32 C they felt cold initially, but these sensations passed within 3–5 min, and they were comfortable for the rest of the hour. Water of 28 C seemed very cold to everyone and to most was the coldest. In fact, several wondered how they could complete the series of experiments by going in 24 and 26 C. However, the latter temperatures did not seem so difficult once the initial response to immersion had passed.

After being in cold water for the hour, the subjects felt quite comfortable when they had dried and dressed. They said that leaving the cold water was better than leaving water of 30 to 34 C, for they felt warmer. Three of the subjects felt tired the evening after being in water of 24 C, but the rest felt well and could work effectively.

There was no increased incidence of upper respiratory infections in this group of subjects during the series of experiments. Two subjects missed their weekly appointments because of pharyngitis, the beginning of which was not related in time to the previous period of immersion. None of the subjects attributed a "cold" to these experiments.

Calculated heat exchanges. In Table 2 we have listed the heat production for the first 30 min of immersion and for the last 30 min. These data were derived from the graphical integration of a plot of $\dot{V}_{O_2}$ versus time by use of the assumption that 1 liter O_2 is equivalent to 4.825 kcal. In calculating changes in heat stores, it was assumed that heat exchanges in the periphery were complete after 30 min of immersion and that the entire layer of the periphery would be at water temperature. Temperature of the covered thermistor placed on the abdominal skin indicates that this latter assumption may cause an overestimation of the peripheral heat losses.

In calculating the changes in the central heat stores, we have used the rectal temperatures because measurements at this site are the most commonly reported. However, we have shown that rectal temperature changes are slower than those measured in the external auditory canal and might underestimate central heat changes.

The results of such indirect calculations indicate that the major heat exchanges are due to changes in the heat storage of the periphery. During the last half-hour the increased heat production in the cold water limits, but does not prevent, a loss of heat from the core.

The results of computed thermoconductivities or reciprocals, insulation indices, are also shown in Table 2. Theoretically such calculations are possible only in steady states where the central temperature is not changing. When the temperature was changing at the end of the hour, the heat exchange was assumed to occur across the skin (20).

Thermal conductivities calculated for 30, 40, and 50 minutes were not different from those noted at 60 min. It is apparent that the thermoconductivity decreased with the decreasing water temperature. In the lower water temperatures, 24–30 C, the thermoconductivity was minimal and was independent of water temperature.

DISCUSSION

The thermoregulatory mechanisms of these subjects immersed in water for an hour may be assessed in terms of the changes in core temperature. In Fig. 9 the changes in ear temperature between 10 and 60 min have been plotted as a function of the temperature differential between the ear temperature and the water. The use of the 10-min ear temperature rather than the control temperature is preferred because in the first few minutes of immersion in water 34 C or less there is an initial increase of core temperature. During this time there is a rapid heat loss from the periphery and a change in conductivity. The paradoxical increase in the central temperature noted in this study and by others (10, 18) may represent a transient decrease in the loss of heat which is generated in the core. Burton and Bazett's (10) mathematical and physical models suggested that when the peripheral temperature is changed suddenly, unusual thermal gradients may exist and that changes in conductivity could be such as to account for transient retention of heat.

In effect the physiological responses can be evaluated over this 50-min interval. The data form a curve which appears to have three different slopes. When the temperature differential between the core is 0–1.2, the central temperature increases as indicated by line AB in Fig. 9. If the body had cooled in the same manner that it had been heated, the temperature changes indicated by the dashed line BC would have been observed. The slope of line AB was .67 C/hr per ΔC and that of BC, .12 C/hr per ΔC. A comparison of these two slopes measures the effectiveness of the physiological mechanisms which limit the rate of central cooling. The third

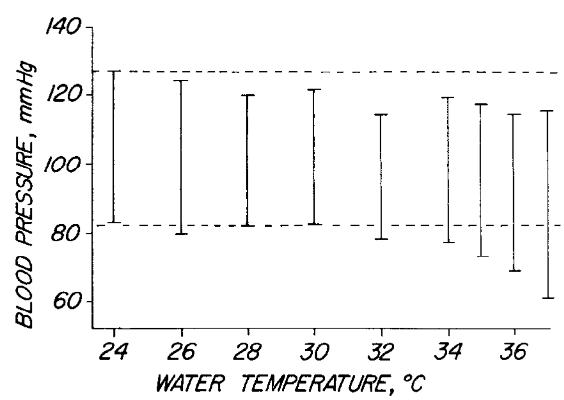

FIG. 8. Systolic and diastolic blood pressures at the end of the hour's immersion are shown as a function of the water temperature. Dotted lines indicate the mean control readings before immersion. Systolic and diastolic pressures were significantly less than control when the subjects were in water of 35 to 37 C. In water of 28 to 34 C, the systolic pressure was slightly, but probably significantly, reduced (P = .05).

TABLE 2. *Calculated heat exchanges during water immersion*

Water Temp, °C	0–30 min				30–60 min		At 60 min	
	Heat production, kcal	Heat stores, kcal		Total heat stores, kcal	Heat production, kcal	Heat stores, kcal	Therm cond, kcal/m² per hr per °C	Insul index, °C/kcal per m² per hr
		Peripheral	Central			Central		
37	42.7	+71.6	+11.1	+82.7	42.8	+10.7	55.2	.018
36	40.5	+33.4	+1.8	+35.2	40.5	+3.5	33.9	.030
35	40.2	+14.3	−2.1	+12.2	40.2	−1.0	22.8	.044
34	40.5	−12.0	−4.7	−16.7	40.5	−3.9	18.3	.055
32	43.2	−59.7	−2.9	−62.6	41.2	−7.2	11.7	.085
30	44.1	−101	−2.2	−103	47.0	−9.7	9.5	.105
28	56.7	−142	+1.1	−143	63.4	−10.7	10.3	.098
26	59.1	−192	+.7	−193	73.6	−11.5	9.8	.102
24	68.0	−247	+.4	−247	79.4	−15.0	9.0	.111

slope of the curve DF was .012 C/hr per ΔC, and indicates that as the thermal stress becomes greater, the decline in the central temperature is again limited by some other factor or factors. The effectiveness of these mechanisms is indicated by the difference between the slope BD and the slope of DF.

It is well known that vasoconstriction is the first major defense against heat loss. Burton and Bazett (10) indicated that vasomotor alterations were able to maintain a balance between heat loss and normal heat production when the rectal to water temperature difference did not exceed 4 C. Furthermore, they indicated that temperature differences exceeding 4 C lead to an increased heat loss which exceeds normal heat production. Their criteria of heat balance were derived from observations of direct calorimetry and are somewhat different than those derived from the central core temperature as in the present study. However, if the core temperature is decreasing and peripheral temperature is not increasing, that heat loss must exceed heat production. The measurement of core temperature affords a more sensitive indicator than would data from their experiments. It is also difficult to see how they achieved "steady states" in the short periods of study used. None of our central temperature measurements indicated a steady state, and Rennie et al. (20) have shown that in water as warm as 33 C, at least 2.5 hr are necessary to stabilize the rectal temperature.

The line BD in Fig. 9 indicates that vasomotor responses are helpful in limiting heat loss, but they alone are not completely effective during an hour's immersion.

Rennie (19) has measured forearm blood flow during long periods of immersion in water of different temperatures. In water of 35 C, there was no appreciable decline in forearm blood flow, but in all cooler baths, forearm flow decreased promptly and dramatically being minimal in water somewhere between 33 and 29 C. This range of water temperatures agrees very closely with the range included between points B and D in Fig. 9.

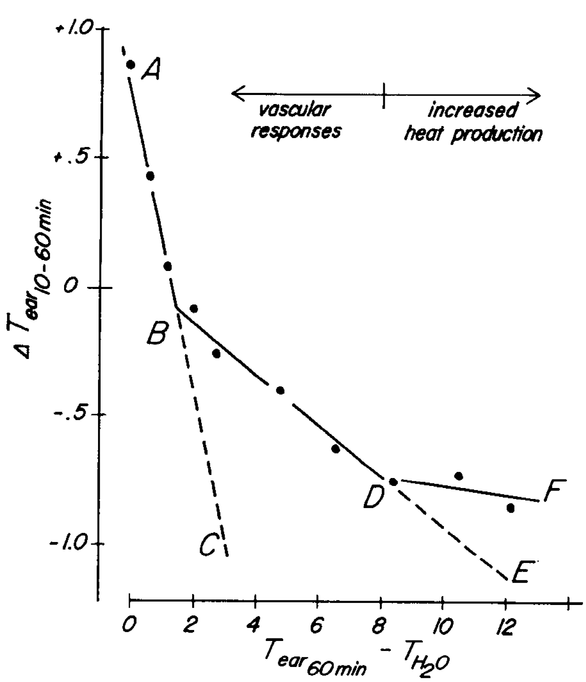

FIG. 9. Changes in ear temperature during the last 50 min of immersion are shown as a function of the temperature differential between the ear temperature and the water at the end of the hour.

Below point D, shivering and increased heat production were observed and were effective in limiting the rate of decrease in central temperature. In one way, this inflection point D might be considered to be a "shivering threshold" or "critical water temperature," as it has been called by others (20). It should be emphasized that this point would vary with different periods of exposure. In a 3-hr period of immersion, 33 C water has been found to be the lowest water temperature American male subjects could tolerate without shivering (20).

Evaluation of the results of water immersion as shown in Fig. 9 might provide a framework in which cold adaptation in different ethnic and adapted groups could be assessed. The Korean Ama is said to require a lower bath temperature to produce shivering than nondiving women (20). Her effective insulation against heat loss appears to be greater than nondivers when allowances are made for the differences in subcutaneous fat.

The difference between the slope of lines AB and BD provides a measure of the effectiveness of the physiological mechanisms which increase insulation. It would not be influenced by fat thickness. The fat layer is a constant factor and provides an equal protection against heat gains, as well as losses, except by control of the blood flow in tissues including fat. In the cold-adapted diver, one would expect that the slope BD would be significantly less and that the inflection point D would occur at a point indicating a greater temperature differential between the core and the water.

The point D would represent a shivering threshold, and the slope of line DF would be a measure of the effectiveness of increased heat production in limiting the heat loss from the body core. A decreased slope of line DF would indicate that an adapted subject is able to maintain his insulation better or in some way is able to make better use of the increased heat production than a nonadapted subject.

The data derived from the present study can also be used to test a present-day concept of thermoregulation. Benzinger (5) has presented the hypothesis that vasomotor responses and increased heat production are controlled by the posterior hypothalamic center. Although peripheral stimuli are said to act directly upon the posterior center, it is inhibited by an anterior center which supposedly senses the central body temperature. His graph plotting $\dot{V}O_2$ as a function of ear temperature is presented in Fig. 10. The dotted lines represent data from his study of one man. The solid lines indicate the results from the present study of 10 subjects.

If the lines on this graph were horizontal, $\dot{V}O_2$ would be independent of central temperature and shivering

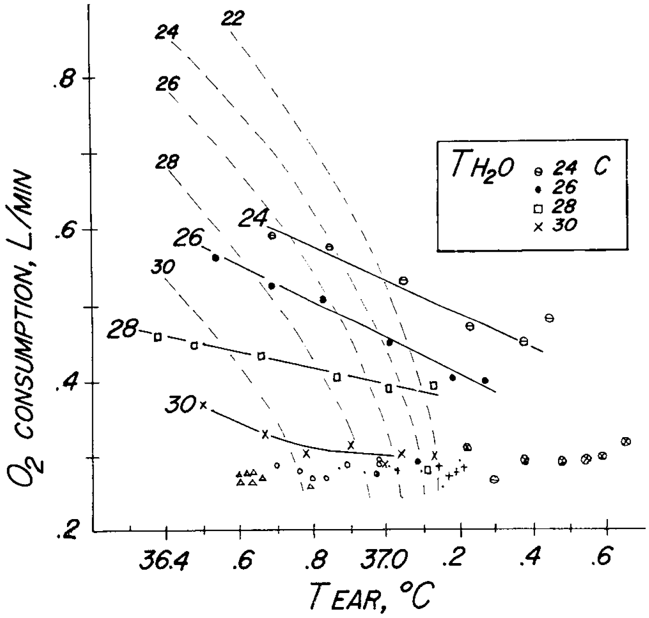

FIG. 10. $\dot{V}O_2$ is shown as a function of ear temperature during immersion in the different water temperatures indicated. Dotted lines and small numbers indicating water temperature are reproduced from Benzinger's article (5). Solid lines and the larger numbers indicate the data from the present study. Symbols which are not connected by lines and are not identified in the legend represent results in water temperatures which did not cause a significant increase of the $\dot{V}O_2$.

would be a function only of the peripheral stimulus. By contrast, if all the data for the different water temperatures fitted on one line, the slope of which was neither zero nor infinity, increased heat production would be a function only of some central temperature. As is evident in Fig. 10, the results support the idea that there is an interaction.

Although the data from our experiments suggest that increased heat production is less dependent upon the central temperature than Benzinger's results, both support the hypothesis. The major difficulty with our series is that we do not have enough information to indicate the central temperature which would be necessary to provide complete inhibition for the supposed posterior center at a time it was receiving peripheral impulses from the cold water. The conditions cannot be derived from simple extrapolations of the lines drawn through the data. This information could be obtained from experiments in which the subjects were preheated and then immersed in cold water.

One of the original purposes of these experiments was to describe a range of water temperatures, the responses to which could be compared to man's reaction in an air environment. To some extent this has been achieved as indicated in Fig. 9. It is apparent that there would be increased heat production at the end of an hour immersion if the temperature differential between the subject's core and water were more than 8 C. Adolph and Molnar (2), who studied the tolerance to cold of men exposed to outdoor weather, indicated that at the end of an hour increased heat production would occur if the air temperature was less than 18 C in shade or less than 10 C on sunny days. These data would imply a temperature difference between rectal core and the air of 19 and 27 C as compared to 8 C which we noted during immersion.

Comparison of the vasomotor responses in air and in water is less direct than the appraisal of heat production. Vasomotor responses appear to influence the rate of change of central temperature when the water bath temperature is between 28 and 35 C (Fig. 9). If changes in thermal conductivity are used as a tool to assess total blood flow in the skin (9), it would seem that flow is minimal below 32 C.

Hardy and Soderstrom (17) emphasized that air temperatures below 28 C represent a cold zone in which there is no further regulation of heat loss by changes in vasomotor activity. Between 28 and 30 C vasomotor regulation alone adjusts heat loss, and in this range their subjects were most comfortable. Above 30 C, blood flow to the skin increased with temperature and vaporization increased.

Greenfield (16) indicated that blood flow in the hand shows a moderate increase when the water temperature around the hand is between 15 and 29 C and from 29 to 35 C there is a faster rise. At temperatures between 25 and 35 C the blood flow is influenced by the over-all heat-regulating mechanisms of the body.

In general, vasomotor responses of the subject immersed in water seem to occur in the same range as when the subject is in air. However, in water of even 35 C, there is a decrease in the central temperature. In air of this temperature the subject would experience a significant heat stress.

The comparison of the heat stresses in water and in air indicate dramatic differences. The immersed subject is greatly handicapped by an inability to increase heat loss by vaporization from the skin. A change in water temperature from 36 to 37 C makes the difference between a comfortable hour of immersion and an unpleasant hour. In air such temperature changes are not so critical (1).

Another goal of this study was to find a water temperature or a range of water temperatures which could be considered neutral. If by neutral one means that the mean body temperature would be the same at the end of an hour's immersion as in air before immersion, the water temperature would have to be 34.6 C. Under these conditions, though, there would be a decrease in central temperatures. In order to avoid a decrease in central temperature, a water temperature of 35.6 would be necessary, but under these conditions the mean body temperature would be increased. Using the heart rate as a criterion, one again finds only one water temperature, 35 C, in which the pulse rate would be the same as when the subject was in air. Probably the best compromise would be to choose a water temperature in the range between 35 and 35.5 C.

It should be emphasized that such conditions of neutrality obtain only for the subject at rest. In general, water of a given temperature imposes thermal stresses of considerably greater magnitude than does the air. It will be necessary to define another range of temperatures for the exercising subject.

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REFERENCES

1. ADOLPH, E. F. *Physiology of Man in the Desert.* New York: Interscience, 1947.
2. ADOLPH, E. F., AND G. W. MOLNAR. Exchanges of heat and tolerances to cold in men exposed to outdoor air. *Am. J. Physiol.* 146: 507–537, 1946.
3. BECKMAN, E. L., K. R. COBURN, R. M. CHAMBERS, R. E. DEFOREST, W. S. AUGERSON, AND V. G. BENSEN. Physiologic changes observed in human subjects during zero-G stimulation by immersion in water up to neck level. *Aerospace Med.* 32: 1031–1041, 1961.
4. BEHNKE, A. R., AND C. P. YAGLOU. Physiological responses of men to chilling in ice water and to slow and fast rewarming. *J. Appl. Physiol.* 3: 591–602, 1951.
5. BENZINGER, T. H., C. KITZINGER, AND A. W. PRATT. The human thermostat. In: *Temperature—Its Measurement and Control in Science and Industry.* New York: Reinhold, 1963, vol. 3, part 3, p. 637–665.
6. BENZINGER, T. H., AND G. W. TAYLOR. Cranial measurement of internal temperature in man. In: *Temperature—Its Measurement and Control in Science and Industry.* New York: Reinhold, 1963, vol. 3, part 3, p. 111–120.
7. BEST, W. R. An improved caliper for measurement of skinfold thickness. *J. Lab. Clin. Med.* 43: 967–970, 1954.
8. BURTON, A. C. Human calorimetry: II. The average temperature of the tissues of the body. *J. Nutr.* 9: 261–280, 1935.
9. BURTON, A. C. The thermal insulation of the tissues of the body. In: *Man in a Cold Environment,* edited by A. C. Burton and O. G. Edholm. London: Arnold, 1955, p. 73.
10. BURTON, A. C., AND H. C. BAZETT. A study of the average temperature of the tissues, of the exchanges of heat and vasomotor responses in man by means of a bath calorimeter. *Am. J. Physiol.* 117: 36–54, 1936.
11. CARLSON, L. D., A. C. L. HSIEH, F. FULLINGTON, AND F. W. ELSNER. Immersion in cold water and body tissue insulation. *J. Aviation Med.* 29: 145–152, 1958.
12. COOPER, K. E., W. I. CRANSTON, AND E. S. SNELL. Temperature in the external auditory meatus as an index of central temperature changes. *J. Appl. Physiol.* 19: 1032–1035, 1964.
13. FORBES, G. B., J. GALLUP, AND J. B. HURSH. Estimation of total body fat from potassium-40 content. *Science* 133: 101–102, 1961.
14. GOFF, L. G., H. F. BRUBACH, H. SPECHT, AND N. SMITH. Effect of total immersion at various temperatures on O_2 uptake at rest and during exercise. *J. Appl. Physiol.* 9: 59–61, 1956.
15. GRAVELINE, D. E. *Maintenance of Cardiovascular Adaptability During Prolonged Weightlessness.* U.S. Wright-Patterson Air Force Base: Aeronautical Systems Div., Tech. Rept. 61-707, 1961.
16. GREENFIELD, A. D. M. The circulation through the skin. In: *Handbook of Physiology. Circulation.* Washington, D.C.: Am. Physiol. Soc., 1963, sect. 2, vol. II, chapt. 39, p. 1325–1351.
17. HARDY, J. D., AND G. F. SODERSTROM. Heat loss from the nude body and peripheral blood flow at temperatures of 22° to 35° C. *J. Nutr.* 16: 492–510, 1938.
18. KEATINGE, W. R., AND M. EVANS. The respiratory and cardiovascular responses to immersion in cold and warm water. *Quart. J. Exptl. Physiol.* 46: 83–94, 1961.
19. RENNIE, D. W. Thermal insulation of Korean diving women and non-divers in water. In: *Physiology of Breath-Hold Diving and the Ama of Japan,* edited by H. Rahn and T. Yokoyama. Washington, D.C.: Natl. Acad. Sci.—Natl. Res. Council, Publ. No. 1341, 1965.
20. RENNIE, D. W., B. G. COVINO, B. J. HOWELL, S. H. SONG, B. S. KANG, AND S. K. HONG. Physical insulation of Korean diving women. *J. Appl. Physiol.* 17: 961–966, 1963.