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REPORT No. J-292-1 (Final Report)

GRANT No. DA-CRD-AG-S92-544-66-G36

AD 663828

COLD ADAPTATION OF KOREAN WOMEN DIVERS

by

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ABSTRACT

The present investigation was undertaken to ascertain the occurrence of vascular adaptation in the Korean women divers who are daily exposed to severe cold water stress. For this purpose, the metabolic and thermal responses were studied in six women divers(ama) and six non-diving women (control) during two hour immersion to water of 30, 31 and 33C. In addition, the regional heat flux and the limb blood flow were also measured by using Hatfield gradient calorimeter discs and mercury-in-rubber strain gages, respectively. At the end of two hour immersion, the maximal tissue insulation, as calculated by(rectal temp.-water temp.)/skin heat loss, was greater in the ama than in the control in both seasons when a comparison was made at a given subcutaneous fat thickness. However, the lower arm and finger blood flow at a given water temperature was greater in the ama than in the control, indicating that the elevation of I in the ama is not due to a greater peripheral vasoconstriction. Although there were considerable individual variations, the skin heat flux from the limb at a given temperature was not much different between the two groups. In other words, the skin heat flux for a given blood flow was lower in the ama as compared to the control. Moreover, the occlusion of limb blood flow resulted in reductions of skin heat flux by the same magnitude in both groups. On the basis of these results, it is speculated that the ama do not develop vascular adaptation as such but seem to have anefficient counter-current heat exchanger.

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INTRODUCTION

In the course of studying the pattern of cold acclimatization as developed in the women divers of Korea(ama), it was noted that the peripheral tissue insulation for a given subcutaneous fat thickness was consistently greater in ama as compared to non-diving women(control) living in the same community(1-3). This suggested to us that the ama upon exposure to cold seem to develop a vascular adaptation, i.e., a greater vasoconstriction of peripheral vessels, thereby reducing the magnitude of heat loss. Previously, this hypothesis was tested in our laboratory by measuring the regional heat flux with a view that the heat flux from the peripheral region in a given cold water environment may be lower in the ama than in the control. However, contrary to this view, the actual magnitude of peripheral heat flux was found to be greater in the ama than in the control(4).

In the present investigation, the peripheral blood flow during a course of cold water exposure was additionally measured along with the regional heat flux, in order to further ascertain the occurrence of a vascular adaptation in the ama.

METHODS

Experiments were carried out in August, 1966(summer studies) and in January, 1967(winter studies) on six each of the ama and the control subjects who were selected at random from a community in Pusan area. Although the same control subjects were used for both summer and winter studies, two out of six ama used for summer studies were idle in winter and thus had to be replaced with active divers for winter studies. All of these subjects have been extensively used in many previous studies and thus are well acquainted with the experimental procedures. Their age and physical characteristics are tabulated in Table 1.

To insure a uniform degree of cold exposure in all experiments, the subjects wearing cotton swim suits were immersed in a constant-temperature water bath, supine on the plastic-mesh cot with only their faces above the water. The temperature of water bath was regulated to within 0.01C at 33, 31 and 30C. This water immersion method was used in previous studies conducted in our laboratory and is described in detail elsewhere(5). In the present investigation, the metabolic and the thermal responses during a two hour immersion at each water temperature were compared between the two groups of subjects. In addition, the regional heat flux and the limb blood flow were also determined during immersion. O_2 consumption was measured for 10 out of every 30 minutes with a Collins spirometer. Rectal temperatures were measured to within 0.01C with a thermistor calibrated over the range 35-40C. For the measurement of regional heat flux, calibrated gradient calorimeter discs of the type described by Hatfield(6) were glued and taped to the skin overlying the center of abdomen, the upper and lower arms, the thigh

Table 1. Physical characteristics of subjects

Subjects (Number)	Age (yrs)	Weight (kg)	Mean Fat* Thickness (mm)	Adiposity* (kg)	Lean Body Mass (kg)
			August, 1966 (Summer)		
Ama(6)	40 ± 2	49.4 ± 1.6	1.38 ± 0.30	7.5 ± 1.5	41.9 ± 1.[illegible]
Control(6)	36 ± 2	55.5 ± 2.4	2.39 ± 0.21	12.0 ± 0.9	43.5 ± 1.6
			January, 1967 (Winter)		
Ama(6)	41 ± 2	52.7 ± 2.4	1.57 ± 0.38	9.0 ± 1.4	43.9 ± 1.7
Control(6)	36 ± 2	55.5 ± 3.2	2.31 ± 0.38	11.8 ± 1.6	43.7 ± 1.8

(Mean ± SE)

*The measurement of mean fat thickness and the computation of adiposity were based on the method described by Allen et al. (7).

and calf, and the tip of middle finger. The electromotive force generated by the heat flow discs was continuously recorded by an Offner oscillograph operated at a sensitivity of 1-5 μV/mm pen deflection. Underwater calibration of these discs in our hands gave an actual calibration factor of 1.6 kcal/hr/m^2/μV. Blood flow to the forearm and the middle finger was measured by mercury-in-rubber strain gages(8). At the end of 2 hour immersion period, the blood flow to the arm was occluded for 10 minutes while the lower arm and the finger heat flux was recorded continuously, in order to estimate the contribution of limb metabolic heat to limb heat loss. Tissue insulation(I) was computed from the formula, I=(rectal temp.-bath temp.)/rate of skin heat loss, using measurements obtained during the last 30 minutes of immersion. As in earlier studies conducted in our laboratory(2, 5), the skin heat loss was assumed to equal metabolic rate minus respiratory heat loss(8% of metabolic rate). In those instances where rectal temperature continued to decline, the net loss of body heat(0.83 x Δ rectal temp. x 0.6 x body wt.) was added to metabolic rate in estimating the skin heat loss.

RESULTS

A. Overall Metabolic and Thermal Responses: In the present investigation, all subjects exhibited no sign of shivering at all bath temperatures. Since the results obtained at the lowest bath temperature(i.e., 30C) are most important, those are illustrated in Fig. 1 as representative metabolic and thermal responses to cold stress. In addition, rectal temperatures, skin heat loss and the I value for each subject at different bath temperatures are summarized in Tables 2 and 3.

At all bath temperatures, rectal temperature was reduced continuously during 2 hours in water although it tended to level off after 90 minutes in water of 33 and 31C. In general, rectal temperature of the ama tended to be somewhat lower than that of the control in summer while this trend was reversed in winter. Metabolic rate as reflected by the skin heat loss was maintained at a higher level in the ama as compared to that of the control in all experiments, although the magnitude of difference between the two groups was gtreater in winter than in summer. Both the limb blood flow and the regional heat flux decreased during the first hour of immersion but maintained a steady level during the second hour.

The values of tissue insulation, computed on the basis of rectal temperature and skin heat loss at the end of immersion period, was not significantly different between the two groups in both seasons. However, the mean skin fat thickness was considerably greater in the control as compared to the ama and hence the tissue insulation for a given skin fat thickness is considered to be greater in the ama than in the control.

B. Regional Skin Heat Flux: Although there were minor inconsistent

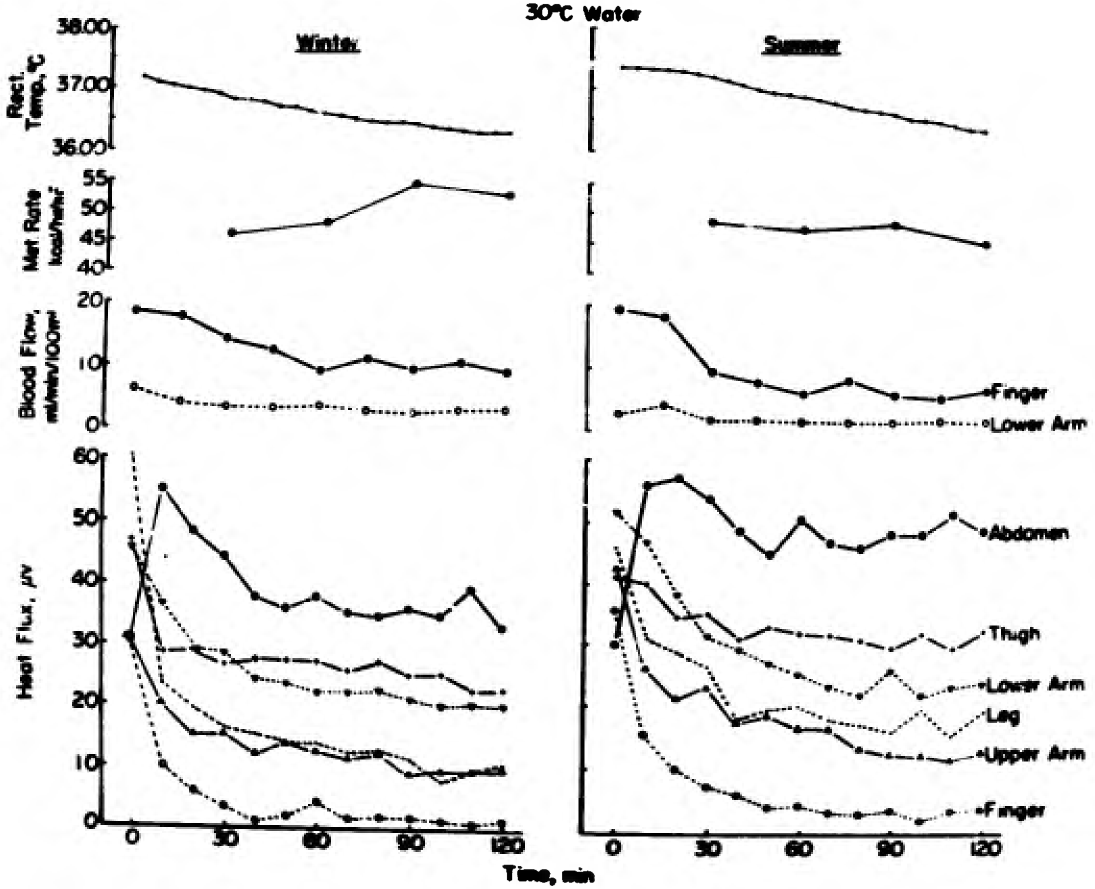

Figure 1. Metabolic and thermal responses of the man during 2 hour immersion in 30C water. Each point represents the average of data obtained from six subjects.

Table 2. Rectal temperature, skin heat loss and tissue insulation(I) of the Ama at the end of 2 hour immersion period.

Subj.	Mean Fat Thickness mm	Rectal Temp., C			Skin Heat Loss kcal/hr/m^2			I value C/kcal/hr/m^2		
		33C water	31C water	30C water	33C water	31C water	30C water	33C water	31C water	30C water
					August, 1966 (Summer)					
A-1	1.85	37.10	37.01	36.55	42.5	58.4	43.6	0.095	0.103	0.150
A-2	0.45	36.98	36.32	35.94	40.7	45.0	55.6	0.098	0.118	0.107
A-3	1.63	36.56	37.00	36.40	39.8	37.1	38.3	0.090	0.162	0.166
A-4	2.30	37.03	36.84	36.59	41.5	51.0	43.6	0.098	0.114	0.151
A-5	0.60	37.17	36.97	36.65	45.4	54.8	65.1	0.092	0.109	0.102
A-6	1.45	36.82	36.10	36.35	37.6	38.2	42.1	0.102	0.133	0.150
Mean	1.38	36.94	36.70	36.41	41.3	47.4	48.1	0.141 ± 0.009*		
					January, 1967 (Winter)					
A-1	2.62	37.44	36.70	36.49	47.8	55.8	50.0	0.093	0.102	0.130
A-2	0.67	36.99	36.58	36.44	44.5	40.3	47.7	0.090	0.103	0.135
A-4	2.15	36.91	36.25	36.28	38.4	44.7	46.8	0.102	0.117	0.134
A-5	0.90	37.08	36.57	36.51	53.6	48.8	61.6	0.076	0.114	0.106
A-7	0.65	36.70	36.17	35.82	55.31	54.2	57.4	0.067	0.095	0.101
A-8	2.45	36.73	36.17	36.42	46.4	54.1	51.0	0.080	0.096	0.126
Mean	1.57	36.98	36.41	36.33	47.6	49.7	52.4	0.124 ± 0.006*		

*Average(± 1SE) of maximal I values in each individual subject.

Table 3. Rectal temperature, skin heat loss and tissue insulation(I) of the control at the end of 2 hour immersion period.

Subj.	Mean Fat Thickness mm	Rectal Temp., C			Skin Heat Loss kcal/hr/m^2			I value C/kcal/hr/m^2		
		33C water	31C water	30C water	33C water	31C water	30C water	33C water	31C water	30C water
		August, 1966 (Summer)								
C-1	2.50	36.80	36.98	36.33	42.1	52.5	52.9	0.090	0.114	0.119
C-2	2.15	36.62	36.71	36.30	36.6	42.4	52.8	0.099	0.135	0.119
C-3	1.90	37.18	36.99	36.71	45.7	39.8	49.9	0.092	0.151	0.134
C-4	2.08	37.37	36.57	37.01	36.7	42.7	44.5	0.119	0.131	0.158
C-5	3.33	37.12	36.72	36.70	40.0	40.1	37.0	0.103	0.143	0.181
C-6	2.40	36.95	36.95	-	46.9	54.4	-	0.084	0.109	-
Mean	2.39	37.01	36.82	36.61	41.3	45.3	47.4	0.142 ± 0.010*		
		January, 1967 (Winter)								
C-1	2.95	36.83	36.70	36.56	43.8	47.7	46.5	0.087	0.119	0.141
C-2	1.68	36.62	36.02	36.40	45.8	48.4	48.9	0.079	0.100	0.131
C-3	1.23	36.57	35.97	36.27	-	50.7	59.1	-	0.098	0.106
C-4	1.88	36.67	35.82	35.70	42.5	46.2	48.5	0.086	0.104	0.118
C-5	3.78	35.90	36.16	36.17	45.1	45.5	40.3	0.064	0.113	0.153
C-6	2.33	37.08	36.50	35.97	39.2	39.4	42.1	0.104	0.139	0.142
Mean	2.31	36.61	36.20	36.18	43.3	46.3	47.6	0.132 ± 0.007*		

*average(± 1SE) of maximal I values in each individual.

variations in the values of regional skin heat flux as a function of the water temperature, the overall trend was very similar in all temperatures. Hence the skin heat flux from each region obtained during the last 30 minutes of immersion to water of 30, 31 and 33C is summarily shown in Fig. 2, in order to illustrate the regional distribution of heat loss. It is evident from this figure that while heat flux from the abdominal region is the highest the limbs also lose a considerable amount of heat even at these low water temperatures.

In order to estimate the total amount of heat lost from each region, the regional heat flux was multiplied by the weighting factor indicated in the top line of Table 4 adjusted to a total skin area of 1 square meter. In this calculation, 68% of the total skin area was represented, toes, head and chest being omitted from study. Although the heat flux from the finger was measured, the magnitude was so low that it was neglected in this consideration. In the right column of Table 4, 68% of skin heat loss estimated from the metabolic rate are also indicated for comparison. From this calculation, it is clear that the sum of regional heat loss as estimated from the value of directly measured heat flux is reasonably close to that of skin heat loss estimated indirectly from the value of metabolic rate, indicating that the present method is reliable in quantitating the regional heat loss.

Although there were considerable individual variations, the average skin heat flux from each of the six regions was not significantly different between the two groups of subjects.

C. <u>Relation Between Skin Heat Flux and Blood Flow</u>: The limb blood flow was measured while the subjects were immersed in water(Table 5). As stated earlier, the lower arm and finger blood flow decreased during the first hour of immersion but levelled off during the second hour (Fig. 1). Individual values of this steady state blood flow at various water temperatures are shown in Fig. 3 along with the corresponding heat flux. As expected, the finger blood flow decreased along with the heat flux as the water temperature was lowered. Although there were considerable individual variations in the values of both blood flow and heat flux, the average figures tended to be somewhat greater in the ama than in the control at all temperatures in both seasons. Interestingly enough, the finger blood flow at a given temperature was not different between summer and winter in both groups of subjects while the heat flux at 30 and 31C water tended to be lower in winter than in summer. However, the possible significance of this finding is not clear at present.

In contrast to this behavior of the finger, changes in the blood flow and the heat flux of lower arms were not related. In other words, as the water temperature was lowered, the blood flow was progressively lowered while the heat flux tended to increase(Fig. 3). As in the case of fingers, considerable individual variations were also noted. However, the average blood flow at a given temperature tended to be higher while the average heat flux tended be lower in the ama than in the control in

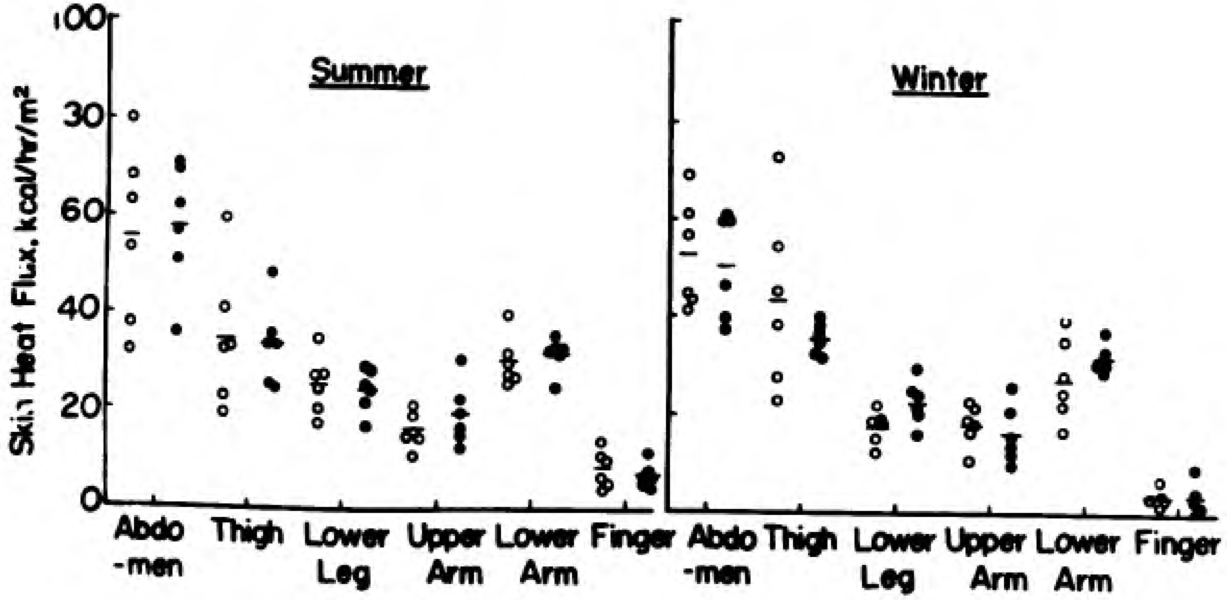

Figure 2. Average regional skin heat flux during the last 30 minutes of immersion in 30, 31 and 33C water. Each point represents the average of values obtained in 30, 31 and 33C water for each subject. Open and solid circles represent the ama and the control, respectively.

Table 4. Average regional heat flux during the last 30 minutes of 2 hour immersion in 30, 31 and 33C water.

	Abdomen	Upper Arms	Lower Arms	Thighs	Lower Legs	Total	Skin Heat* Loss X .68
Area, m^2	.18	.08	.08	.18	.16	.68	
Skin Heat Flux (kcal/hr/area)							
August, 1966 (Summer)							
Ama							
30C water	14.5	1.7	3.0	9.2	4.9	33.3	32.7
31C water	12.1	1.2	2.9	5.1	2.9	24.2	32.2
33C water	10.5	1.0	1.1	4.4	4.4	21.4	28.1
Mean	12.4	1.3	2.3	6.2	4.1	26.3	31.0
Control							
30C water	12.5	1.7	3.0	6.9	4.5	23.6	32.2
31C water	9.7	1.5	2.6	5.1	3.8	22.7	30.8
33C water	9.2	1.6	2.1	6.1	3.6	22.6	28.1
Mean	10.5	1.6	2.6	6.0	4.0	24.6	30.4
January, 1967 (Winter)							
Ama							
30C water	10.2	1.1	2.6	6.6	2.4	22.9	35.6
31C water	9.8	1.5	2.0	7.1	2.8	23.2	33.8
33C water	8.5	1.9	2.1	9.8	3.1	25.4	32.4
Mean	9.5	1.5	2.2	7.8	2.8	23.8	33.9
Control							
30C water	8.7	1.2	2.6	5.9	3.5	21.9	32.4
31C water	8.1	1.2	2.5	5.9	3.5	21.2	31.5
33C water	10.4	1.6	2.7	7.8	3.9	26.4	29.4
Mean	9.1	1.3	2.6	6.5	3.6	23.2	31.1
Grand Mean	10.3	1.4	2.4	6.7	3.6	24.5	31.6
± 1SE	0.5	0.1	0.2	0.5	0.2	1.0	0.6

* Values of skin heat loss were taken from Tables 2 and 3.

Table 5. Average limb blood flow(ml/min/100ml) during the last 30 minutes of 2 hour immersion in 30, 31 and 33C water.

Region	Water Temp. (C)	Ama	Control	P value
		August, 1966 (Summer)		
Lower Arms	30	1.68 ± 0.98	2.04 ± 0.93	NS*
	31	2.13 ± 0.72	1.78 ± 0.61	NS
	33	2.13 ± 0.36	3.11 ± 0.77	NS
Finger	30	6.48 ± 0.81	3.40 ± 0.57	$P < 0.025$
	31	7.20 ± 1.88	3.72 ± 0.83	$P < 0.1$
	33	19.02 ± 3.10	11.82 ± 2.32	$P < 0.1$
		January, 1967 (winter)		
Lower Arms	30	3.00 ± 0.50	2.19 ± 0.40	NS
	31	4.09 ± 0.24	2.89 ± 0.85	NS
	33	5.10 ± 0.91	4.16 ± 0.61	NS
Finger	30	9.91 ± 2.11	5.69 ± 0.61	$P < 0.1$
	31	10.23 ± 1.76	5.43 ± 0.83	$P < 0.05$
	33	11.50 ± 2.17	8.19 ± 1.57	NS

(Mean ± SE)

*NS indicates the non-significant difference between the two groups($P > 0.05$).

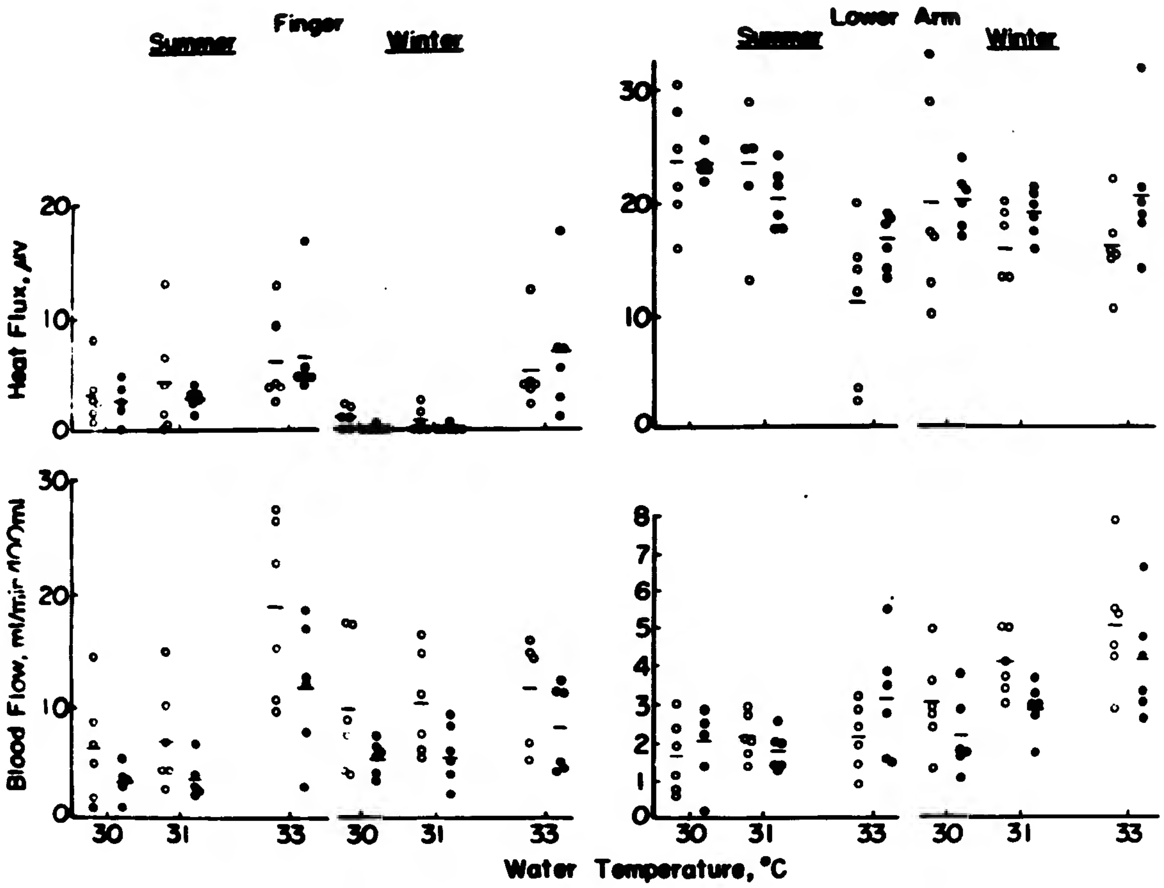

Figure 3. The skin heat flux and the blood flow of the lower arm and the finger measured during the last 30 minutes of immersion in 30, 31 and 33C water. Each point represents the individual subject while the horizontal bar indicates the average. Open and solid circles represent the arm and the control, respectively.

winter. No consistent trend in both measurements between the two groups was noted in summer.

To estimate the relative importance of convective heat transfer by blood flow and conduction of heat from limb interior to limb surface the skin heat flux from the lower arm and the finger was measured in winter during the occlusion of blood flow to the arm. Following immersion for 2 hours in water of various temperatures, the arm circulation was arrested by inflating an upper arm tourinquet for 10 min. At all temperatures, the finger heat flux was promptly reduced practically to zero following occlusion(Fig. 4). However, in the case of lower arms, the reduction in heat flux following occlusion was rather slight and thus a considerable amount of heat loss was observed even during the period of occlusion. This decrement, designated $\dot{H}_Q$, represents the circulatory component of total steady state heat loss from the respective region, while the heat flux from the non-perfused region, designated as $\dot{H}_m$, represents direct loss of metabolic heat from underlying tissue. As summarized in Fig. 5, the average magnitude of $\dot{H}_Q$ in the finger was proportional to water temperature while this trend was not clear in the lower arm. Moreover, the magnitude of $\dot{H}_m$ was practically zero in the finger at all temperatures, indicating that steady state heat flux from this region is entirely dependent upon the circulation. On the other hand, the majority of steady state heat flux from the lower arm was independent of circulation, as indicated by the greater $\dot{H}_m$(Fig. 5). On the average, only 30% of total heat flux from the lower arm are attributable to the circulatory component, and 70% to local metabolic heat. It is also important to note that the magnitude of both $\dot{H}_Q$ and $\dot{H}_m$ was not significantly different between the two groups of subjects.

DISCUSSION AND SUMMARY

The purpose of this investigation was to ascertain the occurrence of vascular adaptation in the ama who are daily exposed to severe cold water stress the year around(9-11). On the basis of observations that the maximal tissue insulation for a given subcutaneous fat thickness was significantly greater in the ama as compared to the control, we have suggested earlier that the ama may develop a greater vasoconstriction upon exposure to a given cold stress(1, 2). However, subsequent studies on the regional heat flux indicated that the magnitude of heat flux was uniformly greater in the ama in all regions as compared to the control, suggesting that there is no region where the heat loss is preferentially prevented(4). Although these findings seem to be inconsistent with the occurrence of vascular adaptation in the ama, there still exists a possibility that the regional heat flux could be greater in ama even in presence of a greater vasoconstriction by virtue of a higher metabolic rate(1, 2, 9, 11, 12) coupled with a lower resistance to heat flow due to a smaller subcutaneous fat thickness in the ama. Hence it was felt that the actual measurement of peripheral blood flow is necessary in order to further explore the occurrence of vascular adaptation.

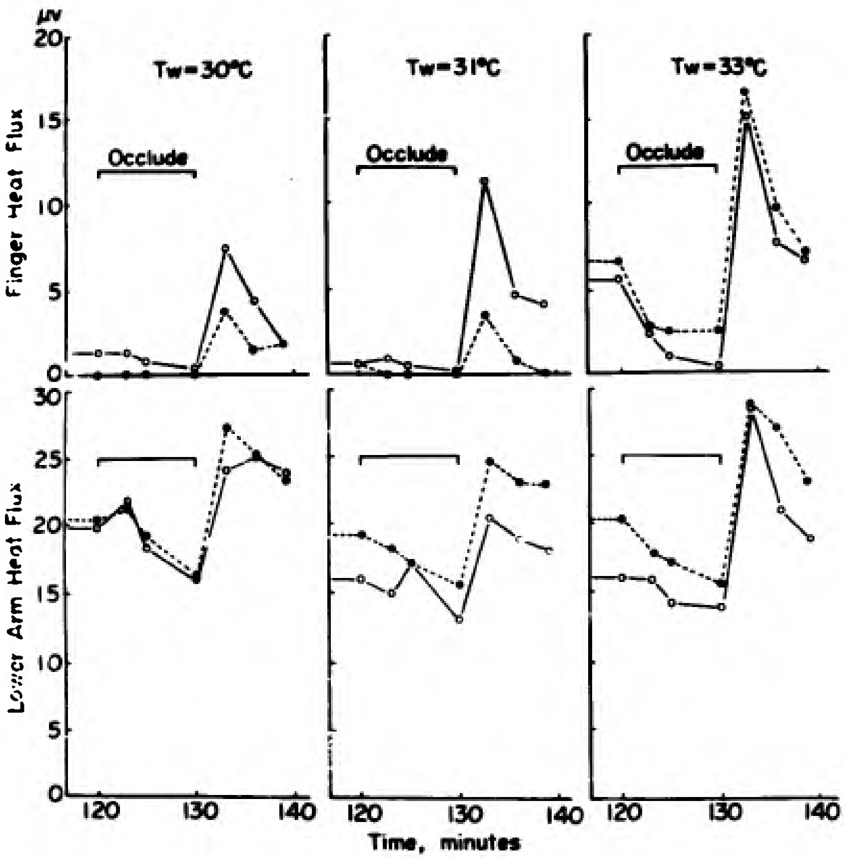

Figure 4. The effect of an upper arm tourniquet on skin heat flux from the lower arm and the finger. Each point represents the average of data obtained from six subjects. Open and solid circles represent the ama and the control, respectively. The upper arm occlusion was applied at the end of 120 minute immersion for a period of 10 minutes.

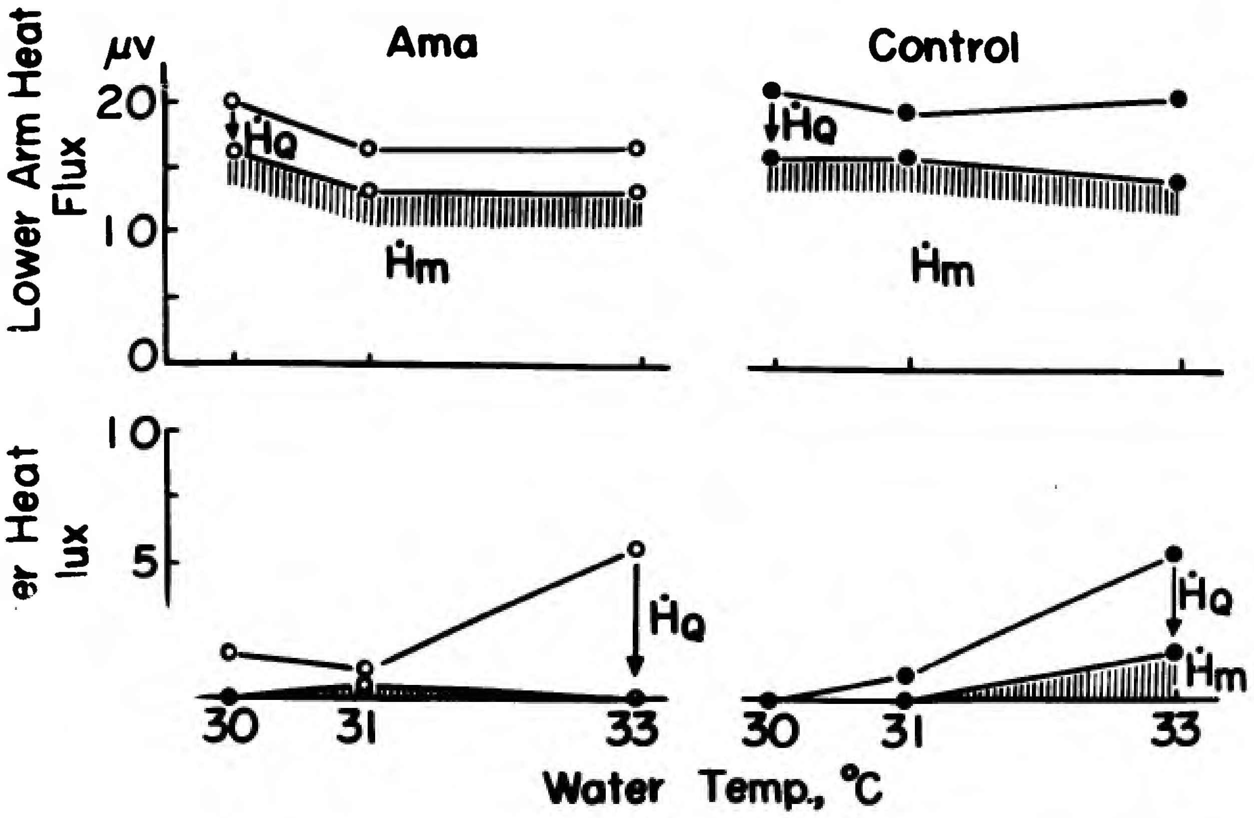

Figure 5. Heat flux from the lower arm and the finger under conditions of unrestricted blood flow(upper curves) and during occlusion of arm blood flow by a tourniquet(lower curves). Each point represents the average of data obtained from 6 subjects.

Preliminary experiments indicated that both the regional heat flux and the limb blood flow are continuously reduced during the first hour of immersion, after which they maintained a steady state level up to 3 hours(3, 4). Hence the immersion period was shortened to 2 hours in the present investigation because of our primary interest in the level of heat flux and blood flow during immersion. In previous studies, we noted that at least 50% of control subjects shiver within 3 hours of immersion to water of 30C(1). However, as a result of shortened immersion period, we have been able to lower the water temperature to 30C in all subjects without invoking any visible shivering. By the same token, we could have lowered the water temperature to below 30C for the ama in the present investigation, although this step was not taken.

The values of maximal tissue insulation, I, were similar in both groups despite a significant difference in the mean subcutaneous fat thickness between the two groups(see Tables 2 and 3). In other words, I values for a given subcutaneous fat thickness were greater in both seasons in the ama than in the control, in agreement with earlier findings (1-3). Moreover, the values of I obtained in the present investigation are not significantly different from the previously reported values, indicating that the extent of vasoconstriction as developed under the present experimental condition is comparable to that with the earlier method(2, 3). As these observations may seem satisfactory, we were surprised not to find any seasonal difference in the I value of ama. This may be attributed to the following three factors: 1) the insulation was measured at the end of 2 hour immersion by which thermal steady state was not reached, 2) the water temperature was not low enough in the ama, and 3) 2 out of 6 ama employed in summer were replaced with others in winter. However, no further attempt was made to resolve this matter since the confirmation of our earlier findings on elevation of maximal insulation in the ama in both seasons was sufficient for our present purpose.

Despite these differences in the value of I for a given subcutaneous fat thickness between the two groups of subjects, the overall regional heat flux was not significantly different(see Fig. 2) whereas the limb blood flow at a given temperature was clearly greater in the ama than in the control, with the exception of lower arm in summer(see Fig. 3). Actually, the average skin heat flux from lower arms, fingers and lower legs was only slightly greater in the ama than in the control in summer while it tended to be similar or even lower in the ama than in the control in winter(see Fig. 3). In other words, there seems to be a general tendency for the ama to lose a lesser amount of heat for a given blood flow through limbs in both seasons. Considering the fact that the subcutaneous fat thickness is considerably lower while the total skin heat loss(or metabolic rate) is greater in the ama than in the control, these findings are of great importance and suggest that the ama may have an efficient counter-current heat exchanger. However, the individual variations are so great that the above suggestion has to tested in future by measuring the "core-to-periphery" thermal gradient.

At any rate it is apparent that the elevation of I in the ama is not due to the greater peripheral vasoconstriction. Contrary to the expectation, the limb blood flow was actually greater in the ama in both seasons(see Fig. 3). However, this greater limb blood flow in the ama was not accompanied by the corresponding increase in the respective skin heat flux. Moreover, the occlusion of blood flow resulted in reductions of skin heat flux by the same magnitude in both groups(see Fig. 5). This again indicates that the greater blood flow as observed in the ama is not necessarily accompanied by the corresponding convective heat transfer.

When the limb was exposed for 2 hours to 10C and 20C water baths, the rate of fall of the hand blood flow was reported to be always slower in the Eskimos than in the controls, but the rate of fall of the muscle temperature was faster in the Eskimos(13). LeBlanc also reported that Gaspé fishermen who showed lower pressor responses to immersion of the hands and feet into ice water maintained the lower finger skin temperature(14). These findings are in agreement with the results obtained in the present investigation and strongly favor the existence of an efficient counter-current heat exchange which was originally postulated by Bazett et al. in 1948(15). Through this mechanism the arterial blood is somehow precooled before it reaches the peripheral zone, whereby reducing the thermal gradient. As a result of this reduction in thermal gradient, the peripheral heat loss is economized in the face of greater peripheral blood flow.

In summary, it is concluded that the elevation of maximal tissue insulation in the ama is not due to a greater reduction in the peripheral blood flow(i.e., vascular adaptation) but seems to be due to an efficient counter-current heat exchanger. Further investigations along this line are currently under progress in our laboratory.

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DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author)
Department of Physiology, Yonsei University
College of Medicine, Seoul, Korea

2a. REPORT SECURITY CLASSIFICATION
UNCLASSIFIED

2b. GROUP

3. REPORT TITLE

COLD ADAPTATION OF KOREAN WOMEN DIVERS (U)

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)
FINAL REPORT NO. 1, September 1966 - September 1967

5. AUTHOR(S) (First name, middle initial, last name)

SUK KI HONG

6. REPORT DATE
DECEMBER 1967

7a. TOTAL NO. OF PAGES
18

7b. NO. OF REFS
15

8a. CONTRACT OR GRANT NO.
DA-CRD-AG-S92-544-66-G36

b. PROJECT NO.
DF9777

c. TASK
d. 01 003FE

9a. ORIGINATOR'S REPORT NUMBER(S)
J-292-1

9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)

10. DISTRIBUTION STATEMENT
THIS DOCUMENT HAS BEEN APPROVED FOR PUBLIC RELEASE AND SALE; ITS DISTRIBUTION IS UNLIMITED

11. SUPPLEMENTARY NOTES

12. SPONSORING MILITARY ACTIVITY
AF Office of Scientific Research, Director of Life Sciences, 1400 Wilson Blvd., Arlington, Va. 22209 thru U. S. Army R&D Group (Far East), APO San Francisco 96343

13. ABSTRACT

The metabolic and thermal responses were studied in six women divers (ama) and six non-diving women (control) during two hour immersion to water of 30, 31 and 33C. In addition, the regional heat flux and the limb blood flow were also measured by using Hatfield gradient calorimeter discs and mercury-in-rubber strain gages, respectively. At the end of two hour immersion, the maximal tissue insulation, as calculated by (rectal temp.-water temp.)/skin heat loss, was greater in the ama than in the control in both seasons when a comparison was made at a given subcutaneous fat thickness However, the lower arm and finger blood flow at a given water temperature was greater in the ama than in the control, indicating that the elevation of tissue insulation in the ama is not due to a greater peripheral vasoconstriction. Although there were considerable individual variations, the skin heat flux from the limb at a given temperature was not much different between the two groups. In other words, the skin heat flux for a given blood flow was lower in the ama as compared to the control. Moreover, the occlusion of limb blood flow resulted in reductions of skin heat flux by the same magnitude in both groups. On the basis of these results, it is speculated that the ama do not develop vascular adaptation as such but seem to have anefficient counter-current heat exchanger. (Author)

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Adaptation						
Cold						
Metabolism						
Korean women						
Physiology						
Temperature						
Body						
Heat loss						
Korea						