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HUMAN CARDIODYNAMIC RESPONSES TO HEAT EXPOSURE: IMPEDANCE PLETHYSMOGRAPHIC MEASUREMENTS OF CARDIAC OUTPUT

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DECEMBER 1967

AEROSPACE MEDICAL RESEARCH LABORATORIES
AEROSPACE MEDICAL DIVISION
AIR FORCE SYSTEMS COMMAND
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The experiments reported herein were conducted according to the "Principles of Laboratory Animal Care" established by the National Society for Medical Research.

The voluntary informed consent of the subjects used in this research was obtained as required by Air Force Regulation 169–8.

700 - January 1968 - CO455 - 21-472

AMRL-TR-67-93

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FOREWORD

The experimental work reported herein was conducted in the Department of Physiology, St. Louis University School of Medicine, 1402 South Grand Blvd., St. Louis, Missouri 63104, in partial fulfillment of Contract AF 33 (615) - 3614, administered by the Aerospace Medical Research Laboratories, Aerospace Medical Division, Wright-Patterson Air Force Base, Ohio. Mr. John F. Hall, Jr., Chief, Biothermal Branch, Environmental Medicine Division, served as contract monitor for the Biomedical Laboratory. The work was performed under project 7222, "Biophysics of Flight," Task 722207, "Biophysics of Flight: Human Thermal Stress." Bernell Coleman, Assistant Professor of Physiology, was the Principal Investigator of this research project. The work described in this report was performed during the period March 1966 through December 1966.

Part of the cost of this research was covered by grant HE-07070 from the United States Public Health Service.

This report has been reviewed and is approved.

WAYNE H. McCANDLESS
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ABSTRACT

Cardiac outputs of seminude resting male subjects were estimated from measurements of transthoracic impedance pulses as recorded by the tetropolar impedance plethysmographic technic. Ambient temperature was increased from 28 to 43 C and maintained at this level for 1.5 - 2.0 hours. Increases in cardiac output were small, variable (ranging from 0.3 to 1.8 liters/min.), and were caused by small increases both in heart rate and stroke volume. The greater cardiac output was temporally related more closely to the augmented stroke volume which was, in turn, closely related to cutaneous vasodilatation as demonstrated by the cutaneous opacity pulses, the implication being that the augmented stroke volume probably results from the greater cutaneous venous return. The ratio of increase in cardiac output to sweating was about the same as previously shown; changes in oral temperature were small in all subjects, averaging about 0.5 F. Validation of the electrical impedance technic as a measure of right ventricular stroke output was accomplished in dogs through comparisons of pulmonary arterial blood flow (electromagnetic flowmeter) and dye dilution (cardiogreen) cardiac output with that obtained by the impedance method. The correlations between the three methods were 0.792 and 0.916, respectively.

SECTION I

INTRODUCTION

A heat load which is acceptable to the thermoregulatory mechanisms of the resting subject should have only a moderate effect on the cardiovascular system. Exposure to an ambient temperature of 43 C, and dry air moving slowly, is such a heat load as evidenced by the hydrated subject's ability to accept it indefinitely with only a slight rise in oral temperature and steady sweating rates (reference 1). Yet this heat load induces sweating over the body surface and modest cutaneous vasodilatation. Assuming that the increase in peripheral blood flow during heat exposure is entirely cutaneous, the increase in cardiac output should be no greater than the blood flow in the skin. This has been inferred on the basis of simultaneous measurements of blood flow in the forearm and cardiac output (reference 2). In the experiments cited, oral temperatures rose 2 F or more, and cardiac output increased about 3.3 liters/min. The increase in forearm blood flow paralleled that in cardiac output. There was no appreciable change in stroke volume, the increase in cardiac output being due almost entirely to an accelerated heart rate. Other reports indicate increases in cardiac output far greater than attainable levels in cutaneous blood flows (reference 3), the increased cardiac output in the latter experiments being due primarily to an augmented stroke volume. However, any increase in cardiac output in excess of that required to supply the increase in cutaneous blood flow would not be useful.

The experiments, which are described in the present report, were designed to examine more closely the temporal relation of cardiac output, heart rate, stroke volume, cutaneous blood flows, skin and oral temperatures, and evaporative cooling during exposure of the resting subject to a heat load which is acceptable to the thermoregulatory mechanisms. Therefore, this report will deal with an exercise in regulation. Attempts were also directed toward determining whether cutaneous vasodilatation directly elicits an increase in cardiac function, or whether the two circulatory events develop coincidently but independently. The experimental objectives necessarily demanded that all observational manipulations of the subject be nontraumatic, nonexcitatory and totally uninteresting to the resting subject. The results support our earlier suggestion that the increase in cardiac output, during moderate heat exposure, results from a greater cutaneous venous return (reference 4).

SECTION II

METHODS

Observations were made on male medical students who had participated in many experiments in the climate chamber. Therefore, the data were believed to be reasonably free from psychological influences. The subjects, clad only

in cotton briefs, lay in a semireclining position on a sheet of stainless steel screen supported in turn by the weighing device which has been previously described (reference 5). After the subjects were allowed to equilibrate with the chamber temperature of 28 C for 2 hours, the ambient temperature was rapidly elevated (1 C per min) to 43 C at which it was then maintained.

The stroke volume of the right ventricle was calculated from the transthoracic impedance pulse as described in a previous report (reference 4). Pulmonary flow or right ventricular output was calculated as the product of stroke volume and heart rate. The advantages of the procedure are the possibilities of obtaining instantaneous and continuous recording of the impedance product, with minimum subject constraint and discomfort and without surgical or puncture procedures. Body temperatures were inscribed by thermocouples. Cutaneous opacity pulses were recorded by photoelectric plethysmographs (reference 6). The amplitudes of the opacity pulses were considered as an index of the level of arterial perfusion of the skin. Evaporation rate from the body surface was indicated continuously by the weighing device (reference 5).

Transthoracic impedance pulse estimation of cardiac output.

The tetrapolar impedance plethysmographic method used in this laboratory for predicting cardiac output has been described in a previous report (reference 4). Although those studies showed a high degree of correlation between the RISA method and the impedance technic in a limited number of human experiments, the selection of the latter technic for climate chamber experiments required further evaluation under various conditions of experimentally altered cardiac activity. Dogs were chosen for these experiments since some manipulations of cardiac activity could be attempted which would be undesirable in the human subject.

Two approaches were used to assess the value of the impedance method in estimating cardiac output. The first was to determine whether the impedance method would produce cardiac output results comparable with those obtained simultaneously by the dye dilution technic (cardiogreen). The second approach was to compare (both quantitatively and temporally) the impedance values with simultaneous pulmonary arterial flow as measured by electromagnetic flowmeter.* If the observation that variations in thoracic impedance during the cardiac cycle result from volume pulsation in the pulmonary circuit is correct (reference 7), then a close correlation could be predicted between these two approaches. This would be true even though the magnitude and duration of the impedance pulse will be determined by the inflow-outflow relations during a particular pulse cycle.

*Blood flow in the pulmonary artery was measured by a sine wave electromagnetic flowmeter (Medicon Multiflo Model M-4000) using a noncannulating probe. Pulmonary flow zero was considered to be that portion of the instantaneous pulmonary flow trace immediately preceding the sharp systolic upstroke. The baseline was frequently checked during each experiment. Calibration was accomplished by allowing saline to pass through the probe from a gravity feed into a graduated cylinder. In the calibration procedure, it is essential that the flow condition established produces a constant rate of flow.

Figure 1 shows simultaneously recorded impedance and pulmonary flow pulses along with the electrocardiogram. The upstroke of the impedance pulse represents a decrease in impedance coincident with forward flow in the pulmonary artery. As would be expected, the onset of flow in the pulmonary artery precedes the impedance change by a few milliseconds. Both peak flow and peak change in impedance occurs simultaneously, whereas the dicrotic notch in the impedance pulse precedes the backflow component of the flow pulse. These observations are consistent with those which indicate that the primary pathway for current flux in the thorax is through the lungs (reference 8).

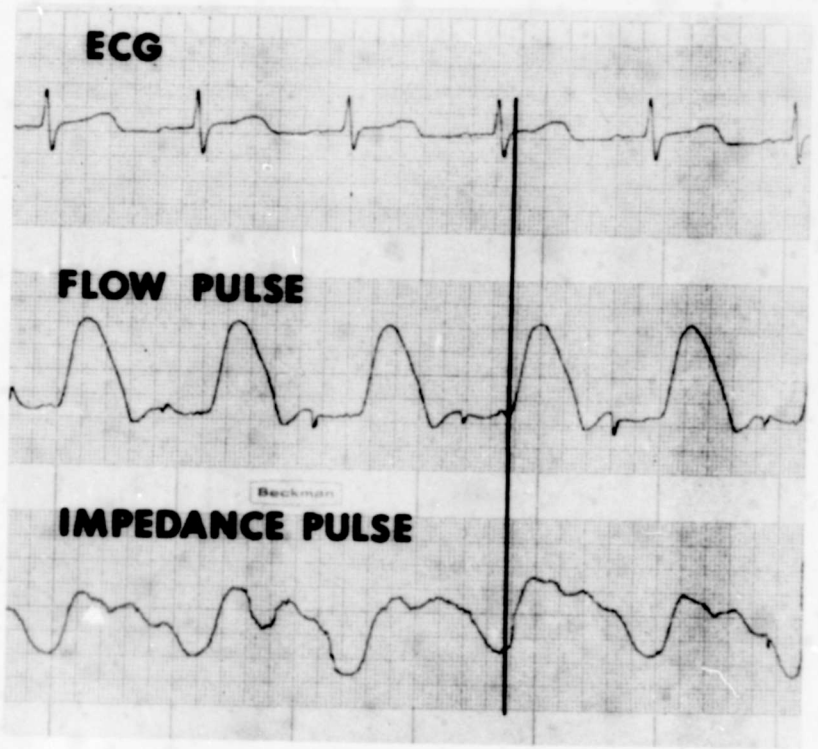

Figure 1. Simultaneous recordings of ECG, pulmonary arterial flow pulses and transthoracic impedance pulses. The upstroke of the impedance pulse represents a decrease in impedance.

SECTION III

RESULTS

Validation of the impedance cardiac output method.

Results showing the correlations of dye cardiac outputs and pulmonary arterial flows with values estimated by the impedance method are presented in figures 2 and 3. The plots represent individual determinations on 15 dogs during various states of cardiac function. Although the relations of the two sets of data tended to be linear, extrapolation of the line of least squares regression to zero indicated that the impedance method progressively underestimated cardiac output as the latter decreased below a liter per minute. One would predict this effect from the changing inflow-outflow relations during a pulse cycle. However, the high degree of correlation (0.792 and 0.916 for impedance cardiac output vs. pulmonary flow and dye dilution cardiac output, respectively) would suggest that the impedance pulse estimation of cardiac output is a fairly accurate estimate of pulmonary flow, and hence of stroke output by the right heart.

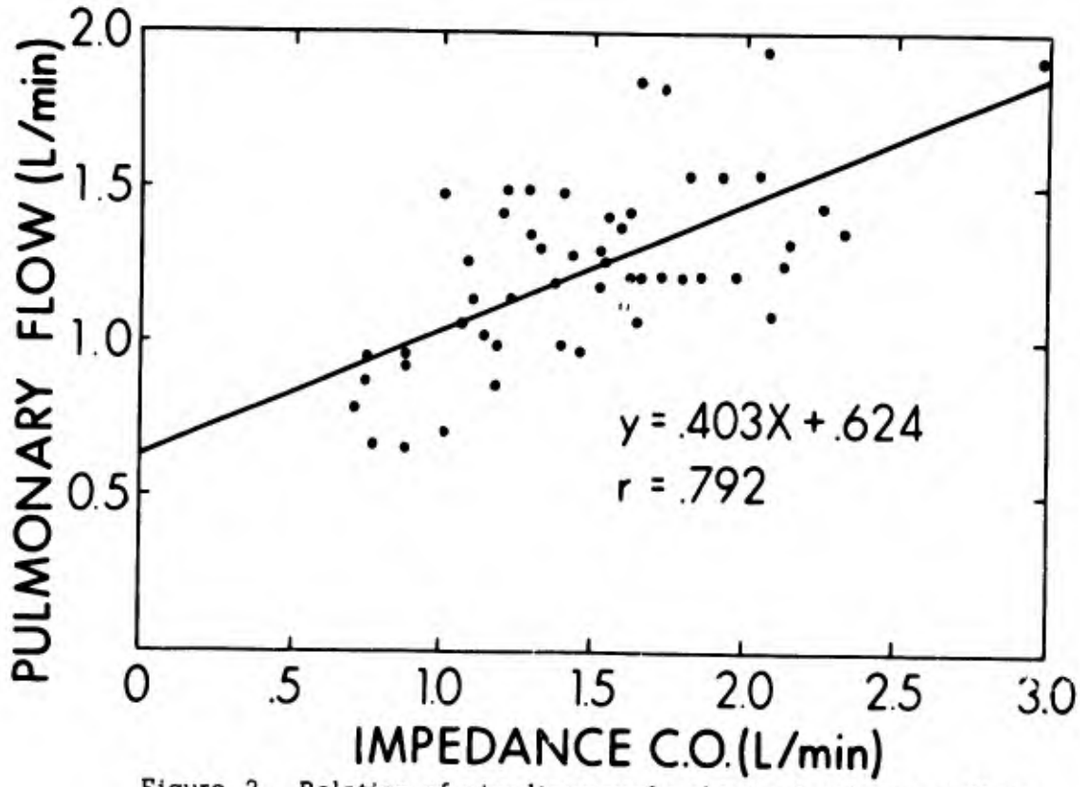

Figure 2. Relation of simultaneously obtained pulmonary flow pulses and transthoracic impedance pulses.

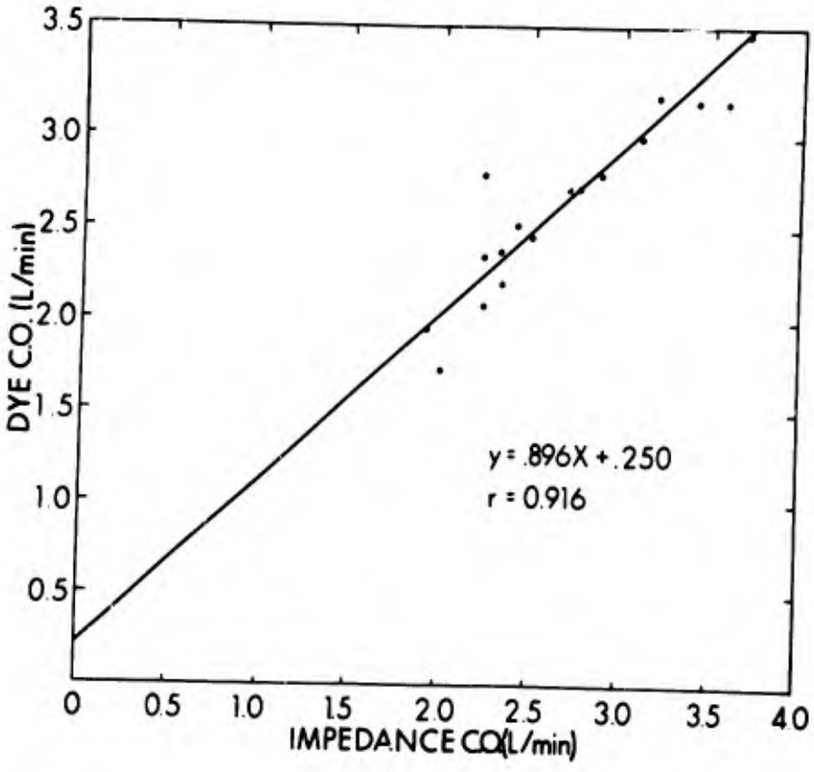

Figure 3. Relation of cardiac output obtained from simultaneously recorded dye dilution curves (cardiogreen) and impedance pulses.

In one experiment attempts were made to follow changes in cardiac output produced by opening and closing an A-V fistula (femoral artery to femoral vein). Table 1 was constructed from these data. The observed changes are similar to those reported in the literature (reference 9). Although there were small changes in heart rate, the predominant increase occurred in stroke volume. The stroke volume change is consistent with the changes in venous return and peripheral resistance that takes place when a large A-V fistula is opened or closed. With the fistula open there was a small decrease in the basic thoracic impedance, indicating a greater volume of blood between the electrodes. Closing the fistula produced the opposite impedance change. The directional changes in impedance between the electrodes are what would be expected from changing central blood volume.

TABLE I

EFFECT OF A-V FISTULA ON IMPEDANCE PRODUCT CARDIAC OUTPUT AND IMPEDANCE PULSE AMPLITUDE (ΔZ)

Procedure	ΔZ Ω	S.V. ml	H.R. Beats/min	Cardiac Output L/min
Fistula closed	0.18	13.91	188	2.61
Fistula open	0.24	21.15	191	4.03
Fistula closed	0.15	12.60	185	2.33
Fistula open	0.23	19.80	194	3.83
Fistula closed	0.18	15.41	178	2.73

Values for impedance pulse (ΔZ), stroke volume (S.V.), heart rate (H.R.), and cardiac output are averages of recordings made during each procedure. The chronological sequence is that given in the table. All measurements were made on the same dog.

Further validation of the directional correctness of the impedance method of estimating cardiac outputs is shown in table II. In this study the effects of exercise on cardiac output, stroke volume and heart rate were observed at various intervals following a series of exhausting procedures involving weight lifting and calisthenics. All measurements were made with the subject in the reclining position and while the breath was held in the normal expiratory position. Since with present instrumentation uniform cardiac impedance pulses can only be recorded during apnea, it was necessary to wait 4 minutes before taking the first postexercise measurement. Before this time the subject was unable to hold his breath at the end of expiration. In this experiment cardiac output was still almost double 4 minutes after cessation of exercise, while stroke volume and heart rate were elevated by 22.4 and 47.6%, respectively. There was a gradual decrease in stroke volume with time, while heart rate and cardiac output remained significantly elevated. These directional changes also agree with those in the literature where cardiac output was measured, following exercise, by the direct Fick (references 10, 11) and indicator dilution (reference 12) methods.

TABLE II

EFFECT OF EXERCISE ON IMPEDANCE PULSE CARDIAC OUTPUT

		S.V. ml	H.R. Beats/min	C.O. L/min
Control:		76	63	4.8
Time after cessation of exercise in minutes:	4	93	93	8.7
	8	87	90	7.8
	12	81	95	7.7
	16	68	95	6.5
	20	68	100	6.8

All measurements made with the subject in the reclining position following a series of exhausting exercises involving weight lifting and calisthenics.

The results from these studies indicated that the impedance method for estimating cardiac output should find considerable utility in those situations where relative changes in cardiac output are important measurements.

General features of the responses to heat stress

Changes in basic thoracic impedance (Z) were inconsistent. About half of the subjects tested demonstrated decreases, while in the other half basic thoracic impedance either increased or remained unchanged. Heart rates increased within 10 minutes after the beginning of the rise in ambient temperature and reached a peak shortly after the latter attained a level of 43 C, after which heart rates remained reasonably constant provided there was no further rise in chamber temperature. In most experiments heart rates became stabilized during the first 30 minutes of heat exposure. Estimated stroke volume, on the other hand, remained constant or decreased slightly during the initial exposure, but began to increase after ambient temperature reached 43 C and cutaneous blood flow in the forearm and calf had become significantly elevated. Therefore, the first 30 minutes and the terminal hour of a 1.5 - 2.0 hour exposure were selected for comparison of the several measurements made at these times with their values during the control period.

Illustrative data from eight different subjects are entered in table III. Calculated cardiac output increased only slightly (av. 0.43 liter/min) during the first 30 minutes of heat exposure. This increase reflects the small rise in heart rates. The major change in cardiac output (av. 1.13 liters/min) occurred during the terminal hour concomitant with the augmented stroke volume. Variations in these responses were noted in repeat experiments on the same subject. With the exception of subject F, heart rates increased moderately in all experiments. Similarly, only subject B demonstrated a decrease in the estimated stroke volume.

TABLE III

CARDIAC, CIRCULATORY, TEMPERATURE AND EVAPORATIVE DATA DURING HEAT STRESS

Subject	Period	Z Ω	ΔZ Ω	H.R. Beats/min	S.V. cc	C.O. L/min	Skin Pulse ΔIP Forearm	Skin Pulse ΔIP Calf	Skin Temp C Forearm	Skin Temp C Calf	T_a C	T_o F	Evap. Rate g/m^2 per hr
	1	23.78	0.40	49	89.02	4.32	0.09	0.08	34.30	34.40	28.73	98.10	
A	2	26.30	0.48	53	87.96	4.60	0.14	0.15	37.17	35.82	42.54	98.20	
	3	30.05	0.64	55	89.14	4.89	0.21	0.19	37.61	36.52	44.43	98.42	
	1	26.53	0.42	64	75.82	4.82	0.06	0.17	34.26	35.26	29.90		26.30
B	2	26.80	0.37	77	64.93	5.00	0.09	0.19	36.75	36.32	43.81		89.70
	3	28.75	0.35	79	65.40	5.17	0.16	0.20	37.32	36.39	45.00		122.50
	1	26.11	0.42	72	82.87	5.92	0.10	0.12	35.50	35.70	31.06		
C	2	26.42	0.39	80	76.55	6.10	0.12	0.17	37.42	36.42	42.92		
	3	26.47	0.43	79	84.20	6.62	0.18	0.23	37.14	36.69	45.91		
	1	17.16	0.30	58	103.03	6.03	0.09	0.17	35.30	35.50	27.86	97.60	
D	2	20.38	0.40	66	100.19	6.56	0.25	0.18	37.50	36.37	42.01	98.20	
	3	21.41	0.51	67	112.95	7.58	0.34	0.18	37.20	36.48	43.66	98.60	
	1	23.25	0.53	65	98.65	6.34	0.29	0.10	35.60	35.30	29.05	98.05	30.50
D	2	22.91	0.49	72	94.24	6.74	0.37	0.09	37.70	36.22	41.34	98.46	126.93
	3	21.63	0.47	76	102.41	7.75	0.39	0.12	37.40	36.05	43.24	98.54	152.30
	1	24.85	0.59	56	95.53	5.33	0.28	0.22	35.20	35.26	31.76		
D	2	23.65	0.56	61	102.13	6.20	0.41	0.29	36.80	35.98	42.65		
	3	21.23	0.53	58	118.84	6.92	0.49	0.32	36.10	36.30	42.90		
	1	13.06	0.21	54	107.13	5.76	0.07	0.09	35.36	33.70	29.23		35.20
E	2	13.70	0.22	60	102.56	6.15	0.10	0.18	37.21	35.24	43.04		89.92
	3	14.58	0.28	63	115.43	7.22	0.19	0.21	37.23	35.75	46.24		177.44
	1	25.36	0.44	54	76.82	4.15	0.13	0.10	35.20	33.80	29.23		
F	2	25.95	0.43	55	72.69	3.97	0.19	0.20	36.97	35.28	43.27		
	3	25.67	0.48	54	82.76	4.44	0.30	0.24	37.28	36.21	46.84		
	1	17.40	0.20	60	89.51	5.32	0.14		33.90		30.36		
G	2	15.46	0.21	61	115.66	7.11	0.36		36.50		42.12		
	3	14.52	0.32	64	111.25	7.09	0.45		36.30		44.03		
	1	18.26	0.22	56	90.40	5.03	0.17		34.50		30.40		
G	2	17.08	0.19	60	86.35	5.18	0.28		36.80		42.30		
	3	16.04	0.19	63	99.18	6.21	0.44		36.50		43.40		
	1	22.82	0.57	52	109.65	5.66	0.29		34.35		28.32	98.00	25.50
H	2	22.22	0.58	49	117.75	5.77	0.61		36.64		43.62	98.10	107.20
	3	21.95	0.58	60	120.69	7.22	0.78		36.75		46.77	99.10	195.00

Period 1 - Control.
2 - Average values during first 30 minutes of heat exposure (recordings taken at 5 minute intervals).
3 - Average values for final 60 minutes of a 90 minute exposure.

Cutaneous vasodilatation in the forearm and calf occurred in all experiments. This change in arterial perfusion of the skin followed the rise in skin temperature, an observation which has been previously reported from this laboratory (reference 13). However, there was no consistent quantitative relationship between the level of arterial perfusion of the skin and cardiac output. This finding is compatible with the observation of regional differences in cutaneous vascular responses to rising ambient temperature (reference 6).

Evaporative rates increased early during the rising phase of ambient temperature and reached a plateau when the latter was stabilized. The rates during the terminal hour of exposure averaged 162 g/m^2 per hr., with a range from 122.5 to 195.0 g/m^2 per hr. At these levels of evaporative cooling, sweating usually occurs over the entire body surface. Assuming metabolic heat production of 50 $kcal/m^2$ per hr., the evaporative rates indicated an environmental constant of 7.0 $kcal/m^2$ per hr. C.

Temporal relationships of the various cardiovascular and cutaneous responses.

The sequence of changes in cardiac dynamics, skin pulses (ΔIP), evaporative rates and temperatures are shown in figures 4, 5, 6 and 7. As illustrated, the increases in skin pulses and evaporative rates occurred early in the exposure to heat and the major fraction of these increases took place during the first 30 minutes, after which, both of these parameters remained relatively constant. The changes in skin pulses and evaporative rates were closely related to changes in skin temperature.

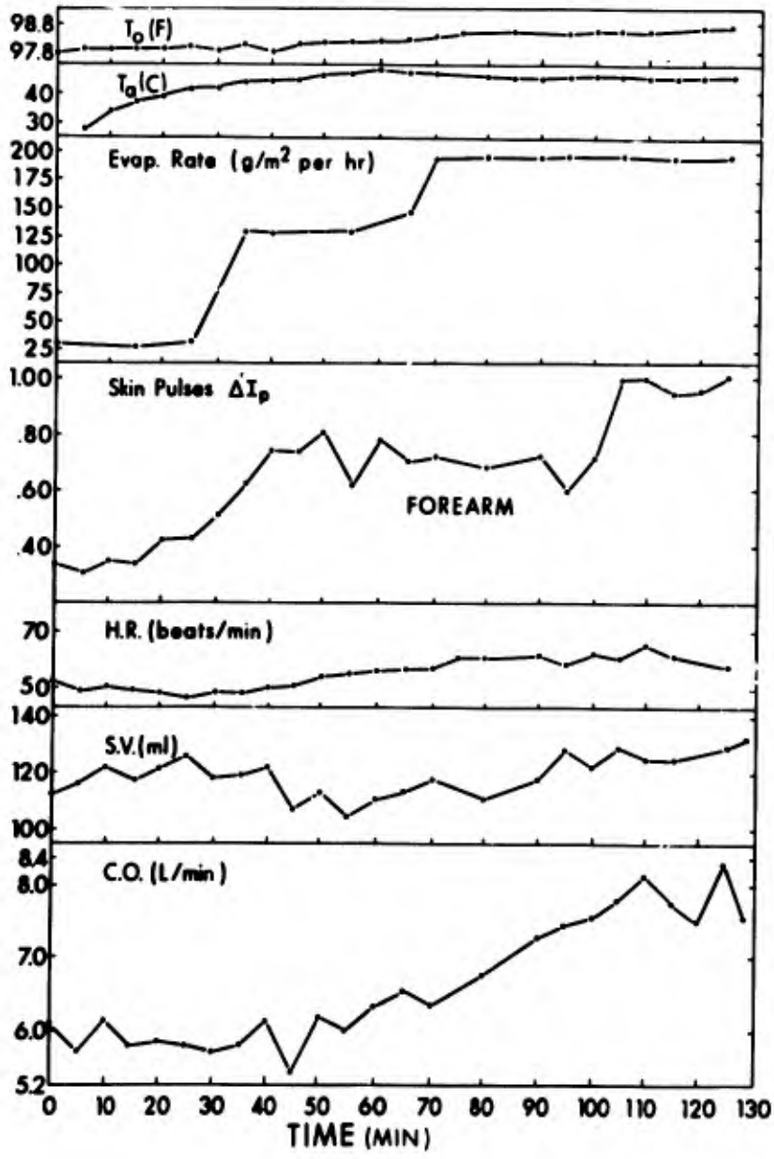

Figure 4. Changes in oral temperature (T_O), evaporative rate, cutaneous opacity pulses (ΔIP), heart rate (H.R.), stroke volume (S.V.) and cardiac output (C.O.) during a rapid rise in chamber temperature.

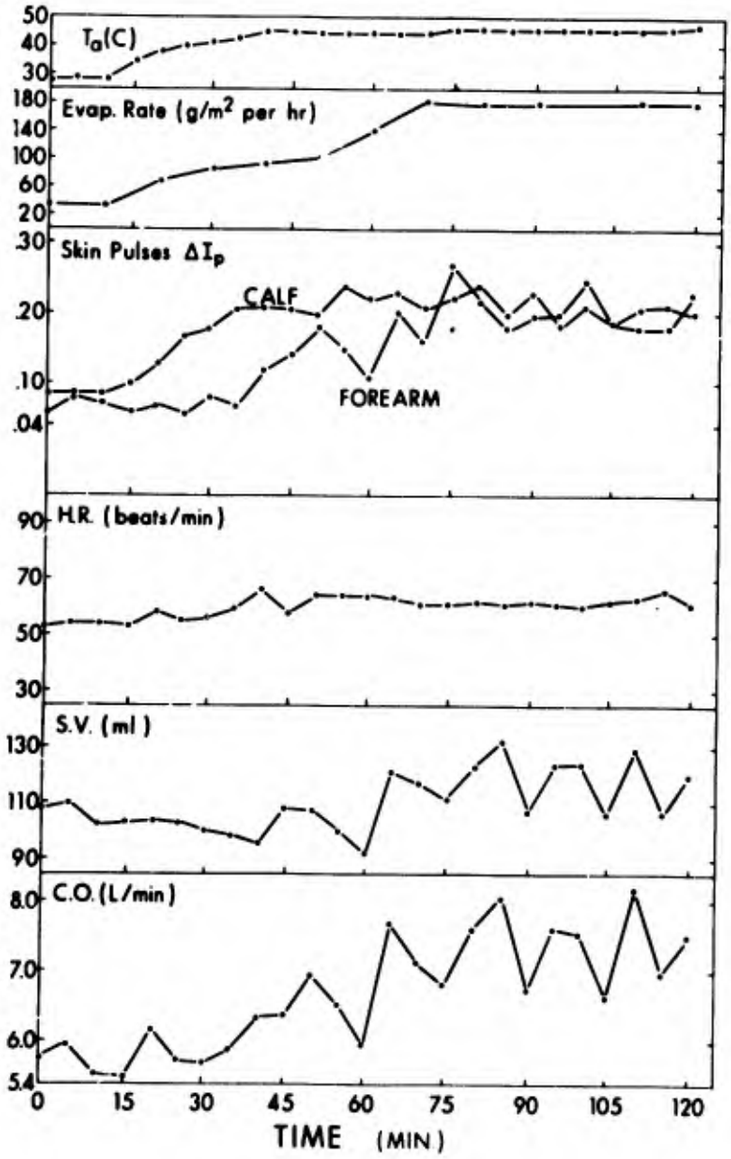

Figure 5. Changes in evaporative rate, cutaneous opacity pulses (ΔIP), heart rate (H.R.), stroke volume (S.V.), and cardiac output (C.O.) during a rapid rise in chamber temperature.

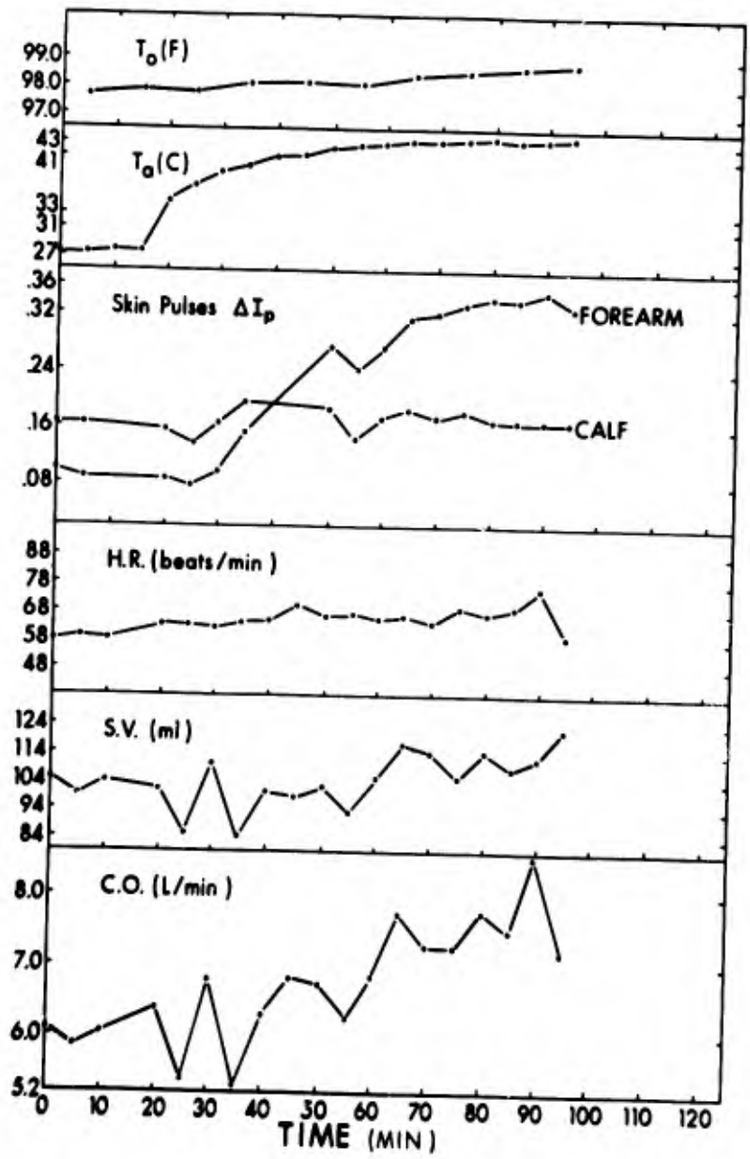

Figure 6. Changes in oral temperature (T_O), cutaneous opacity pulses (ΔIP), heart rate (H.R.), stroke volume (S.V.) and cardiac output (C.O.) during a rapid rise in chamber temperature.

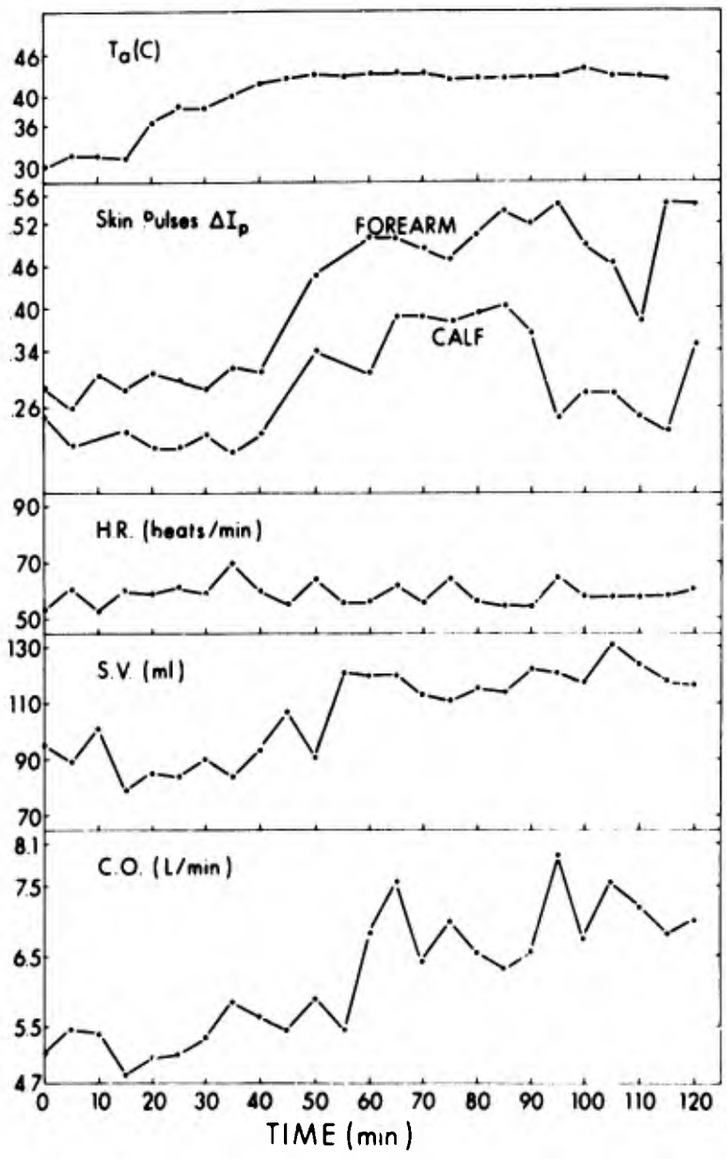

Figure 7. Changes in cutaneous opacity pulses (ΔIP), heart rate (H.R.), stroke volume (S.V.) and cardiac output (C.O.) during a rapid rise in chamber temperature.

The initial cardiac response was an elevated heart rate, which occurred simultaneously with the cutaneous vascular and sweating responses, reaching a plateau concurrently with ambient temperature. Although cardiac output increased slightly during the early part of the heat exposure, the principal increase occurred after ambient temperature was stabilized and the index of arterial perfusion (ΔIP) of the skin had reached a steady state. The increased cardiac output, and especially the plateau, was temporally related more closely to the augmented stroke volume than to heart rate changes. This latter observation contrasts with that of Karexinidis et al (reference 2) who heated their subjects by immersing the legs in hot water.

Changes in oral temperature (T_O) were small and showed no consistent quantitative relationship either to cutaneous vasodilatation or skin temperature. In most experiments, oral temperature increased about 0.5 F during the first hour of a heat exposure with an additional increase of 0.5 F during the terminal hour of a 1.5 - 2.0 hour experiment. It may be significant, however, that the slight rise in oral temperature was closely related temporally to the augmented stroke volume (figures 4 and 6), the implication being that the elevated core temperature may activate sympathetic augmentor fibers in the hypothalamus. If this proves to be true in subsequent experiments, the increased stroke volume observed in these studies may have resulted from an increased sympathetic activity.

SECTION IV

DISCUSSION

Transthoracic impedance pulses recorded during heat exposure demonstrated increased stroke volume and therefore force of cardiac contraction synchronously with cutaneous vasodilatation, particularly with that in the forearm. In most experiments, the correlation was quite good; in others the estimated stroke volume was relatively less than the vasodilatation in the forearm. But most, if not all, of the increase in cardiac output appeared to be accounted for by the cutaneous vasodilatation. In repeat experiments on the same subject, the greater cutaneous blood flows correlated with the greater thoracic impedance pulses.

These experiments have not been carried far enough to answer decisively the question, does the increase in cutaneous blood flow lead to an equal increase in cardiac output? However, it is clear that the load on the heart is closely related to the progress of the cutaneous vasodilatation. Since the latter is related, directly or indirectly, to the level of skin temperature, it would be expected that the requirement for cardiac output should rise with skin temperature. This, of course, would not be a simple linear relationship. Cutaneous blood flow increases greatly at skin temperatures above 36 C. Therefore, excessive cardiac loads might be predicted to develop as the skin temperature rises above this critical level.

The directional correctness of the last statement was demonstrated in experiments involving the work capacity of normal subjects in a hot environment (reference 14). In addition, measurements made both in normal subjects and cardiac patients during exposure to a hot and humid environment gave evidence of excessively large increases in cardiac outputs (reference 3). However, it is not clear from the data whether or not their skin temperatures were also higher.

An interesting feature of the cardiac responses to heat stress was the consistent indication of an increased stroke volume. The greater amplitude of the impedance pulses indicated that cardiac output compensated for the decrease in peripheral resistance. The increase in stroke volume during exposure of the resting subject to heat contrasts with the absence of change in stroke volume of sedentary subjects taking moderate exercises (reference 15). The regulatory mechanisms acting on the heart appear to operate differently in the two stresses. Heat exposure of the resting seminude subject seems to invoke Starling's law of the heart, i.e., an increased cutaneous venous return apparently leads to an increased stroke volume. Although it is not clear whether or not cutaneous vasodilatation determines the level of the cardiac response, both appear to be related to the level of body temperatures. If this proves to be true, a practical application would be directed at improvement in evaporative cooling. The sweating response of the subject would be a principal determinant of the level of circulatory strain elicited by heat.

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Security Classification

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)	2a. REPORT SECURITY CLASSIFICATION
St. Louis University School of Medicine 1402 South Grand Blvd. St. Louis, Missouri 63104	UNCLASSIFIED 2b. GROUP N/A

3. REPORT TITLE

HUMAN CARDIODYNAMIC RESPONSES TO HEAT EXPOSURE: IMPEDANCE PLETHYSMOGRAPHIC MEASUREMENTS OF CARDIAC OUTPUT

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)

Final Report, March 1966 - December 1966

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

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6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
December 1967	17	15

8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S)
AF 33(615)-3614 b. PROJECT NO. 7222 c. Task No. 722207 d.	9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report) AMRL-TR-67-93

10. DISTRIBUTION STATEMENT

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11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	Aerospace Medical Research Laboratories Aerospace Medical Div., Air Force Systems Command, Wright-Patterson AFB, OH 45433

13. ABSTRACT

Cardiac outputs of seminude resting male subjects were estimated from measurements of transthoracic impedance pulses as recorded by the tetropolar impedance plethysmographic technic. Ambient temperature was increased from 28 to 43 C and maintained at this level for 1.5-2.0 hours. Increases in cardiac output were small, variable (ranging from 0.3 to 1.8 liters/min.), and were caused by small increases both in heart rate and stroke volume. The greater cardiac output was temporally related more closely to the augmented stroke volume which was, in turn, closely related to cutaneous vadodilatation as demonstrated by the cutaneous opacity pulses, the implication being that the augmented stroke volume probably results from the greater cutaneous venous return. The ratio of increase in cardiac output to sweating was about the same as previously shown; changes in oral temperature were small in all subjects, averaging about 0.5 F. Validation of the electrical impedance technic as a measure of right ventricular stroke output was accomplished in dogs through comparisons of pulmonary arterial blood flow (electromagnetic flowmeter) and dye dilution (cardiogreen) cardiac output with that obtained by the impedance method. The correlations between the three methods were 0.792 and 0.916, respectively.

DD FORM 1 NOV 65 1473

Security Classification

Security Classification

14 KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Cardiac output						
Impedance plethysmograph						
Heat exposure						
Stroke volume						
Skin blood flow						
Sweating						
Body temperature						

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