Human Cardiovascular Adjustments to Rapid Changes in Skin Temperature during Exercise

By Loring B. Rowell, Ph.D., John A. Murray, M.D., George L. Brengelmann, Ph.D., and Kenneth K. Kraning, II, Sc.D.

ABSTRACT

In 11 normal men, central circulatory responses were measured while skin temperature was changed in a square-wave pattern during uninterrupted exercise (26% to 64% maximal oxygen consumption). Skin temperature was changed at 30-minute intervals, beginning at 32°C. On raising it to 38.2°C at low oxygen consumption (V\textsubscript{O\textsubscript{2}}), cardiac output increased 2.5 liters/min, and central blood volume, aortic mean pressure, and stroke volume fell (7%, 7%, and 11%, respectively). Right atrial mean pressure fell 2.2 and 2.3 mm Hg during control and heating periods, respectively. All variables returned to control levels when skin temperature was reduced toward 26.9°C. Raising it to 40°C reproduced these changes with a more clear-cut drop in right atrial mean pressure. Results indicated reduced peripheral venous tone and cutaneous pooling of blood during heating and rapid reversal on cooling. On raising skin temperature to 38.7°C at high V\textsubscript{O\textsubscript{2}}, cardiac output increased 19% (3.1 liters/min), stroke volume decreased 14%, and central blood volume rose slightly. Aortic mean pressure fell during the control period and was maintained or rose during heating periods. On cooling, central blood volume and stroke volume rose, cardiac output remained elevated, and aortic mean pressure fell. Increases in cardiac output during heating were related to skin temperature and not to V\textsubscript{O\textsubscript{2}} or body temperature. At high V\textsubscript{O\textsubscript{2}}, circulatory adjustments favor metabolic rather than thermoregulatory demands.

ADDITIONAL KEY WORDS: cardiac output, stroke volume, peripheral vascular resistance, heart rate, central blood volume, aortic and arterial blood pressure, circulatory regulation, peripheral circulation

Previous studies of human cardiovascular adjustment to superimposition of competitive thermal and metabolic stresses have depended upon manipulation of environmental temperature rather than skin temperature. But, where natural conditions are simulated, skin temperature may vary throughout the exposure (1). It will also vary over the body surface and may well be reduced, the more severe the exercise, due to an increased sweating response (1). In short, skin temperature is not tightly controlled by exposing subjects to controlled environments, but it is important in both direct local control and reflex control of the vasoactive state of the skin (2). Accordingly, it is important to control this variable when attempting to understand the mechanisms governing human cardiovascular responses to metabolic and thermal stresses.
TABLE 1
Characteristics of Subjects and Oxygen Uptake While Wearing Water-Perfused Suits

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yrs.)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Max. ( \dot{V}_{O_2} ) (L/min)</th>
<th>% Workload</th>
<th>Workload (mph)</th>
<th>( \dot{O}_2 ) uptake* (liters/min)</th>
<th>% Max. ( \dot{V}_{O_2} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.S.</td>
<td>23</td>
<td>174</td>
<td>72.7</td>
<td>3.57</td>
<td>0</td>
<td>2.7</td>
<td>0.94</td>
<td>26.3</td>
</tr>
<tr>
<td>Ki.</td>
<td>21</td>
<td>185</td>
<td>83.6</td>
<td>4.16</td>
<td>0</td>
<td>2.7</td>
<td>1.26</td>
<td>30.3</td>
</tr>
<tr>
<td>F.W.</td>
<td>27</td>
<td>180</td>
<td>72.4</td>
<td>3.80</td>
<td>0</td>
<td>2.7</td>
<td>1.00</td>
<td>26.3</td>
</tr>
<tr>
<td>D.B.</td>
<td>22</td>
<td>175</td>
<td>64.5</td>
<td>3.05</td>
<td>0</td>
<td>2.7</td>
<td>0.54</td>
<td>30.8</td>
</tr>
<tr>
<td>G.Z.</td>
<td>23</td>
<td>178</td>
<td>71.8</td>
<td>3.61</td>
<td>0</td>
<td>3.5</td>
<td>1.32</td>
<td>36.6</td>
</tr>
<tr>
<td>B.B.</td>
<td>21</td>
<td>185</td>
<td>90.2</td>
<td>4.33</td>
<td>0</td>
<td>3.5</td>
<td>1.49</td>
<td>34.4</td>
</tr>
<tr>
<td>M.H.</td>
<td>28</td>
<td>183</td>
<td>78.6</td>
<td>3.58</td>
<td>7.5</td>
<td>3.5</td>
<td>2.29</td>
<td>64.0</td>
</tr>
<tr>
<td>G.M.</td>
<td>21</td>
<td>175</td>
<td>80.6</td>
<td>4.53</td>
<td>7.5</td>
<td>3.5</td>
<td>2.57</td>
<td>52.3</td>
</tr>
<tr>
<td>G.T.</td>
<td>22</td>
<td>182</td>
<td>76.7</td>
<td>5.15</td>
<td>7.5</td>
<td>3.5</td>
<td>2.29</td>
<td>44.5</td>
</tr>
<tr>
<td>P.H.</td>
<td>27</td>
<td>178</td>
<td>68.9</td>
<td>3.86</td>
<td>7.5</td>
<td>3.5</td>
<td>1.98</td>
<td>51.3</td>
</tr>
<tr>
<td>P.K.</td>
<td>22</td>
<td>177</td>
<td>72.4</td>
<td>3.73</td>
<td>7.5</td>
<td>3.5</td>
<td>2.28</td>
<td>61.1</td>
</tr>
</tbody>
</table>

*Oxygen uptake was determined under identical experimental conditions but without catheters on another occasion.

Our experimental design was based on controlling skin temperature as closely as possible and manipulating it in a square-wave pattern between extremes during uninterrupted exercise. It was hoped that this would enable (1) production of substantial changes of skin blood flow superimposed upon several levels of constant metabolic rate and, presumably, constant flow to exercising muscles and (2) possible clarification of the relative roles of central and skin temperatures in the cardiovascular response to thermal and exercise stresses. Further, we hypothesized that sudden, marked elevation of skin temperature after establishment of the normal circulatory response to exercise would elicit cardiovascular adjustments in both central and peripheral circulations which heretofore have been missed. This hypothesis was based on the finding of Bevegard and Shepherd (3, 4) that elevating skin temperature abolishes the normal increments in the tone of resistance and capacitance vessels of the limbs which develop in response to exercise.

**Procedures and Methods**

The subjects were 11 university students, 21 to 28 years old. None was acclimatized to heat nor had any been in active physical training. Only two men (B. B. and P. K.) were physically active. Their physical characteristics are described in Table 1. All volunteers were very thoroughly familiarized with all details of the experiment. They were given a thorough physical examination, including electrocardiogram, chest x-ray, and routine laboratory examination of blood and urine. All were in excellent health.

Studies were conducted during the winter months in a room maintained at 24° ± 2°C. Maximal oxygen uptake was determined in each subject, using either the method of Taylor et al. (5) or multistage exercise on a treadmill to exhaustion (6). The subjects were divided into two groups. Six subjects worked at low workloads which required 26.3% to 38.9% of maximal oxygen uptake (Table 1). The work consisted of walking on the treadmill at 0% grade at either 2.7 or 3.5 mph. The five others performed heavy exercise (7.5% grade, 3.5 mph) which required 51.3% to 64.0% of maximal oxygen uptake. The average energy requirements of these three levels of exercise were 14.5, 17.2, and 29.8 ml \( O_2/kg/ min. \), respectively.

Before the final experiments in which subjects were catheterized, all had performed the entire experimental procedure at least once. These preliminary studies served to standardize the subjects' thermal and metabolic responses and to familiarize both the subjects and investigators with the former's responses and capacities. The collection of expired air was omitted from the final experiment to eliminate any effect of the respiratory apparatus on right atrial pressure. Previous studies have shown oxygen uptake during exercise to be unaffected by the presence of intravascular catheters (7, 8). Briefly, 105 minutes of continuous exercise at a fixed load were divided into three 30-minute periods and a fourth and final 15-minute period. During these periods the temperature of water perfusing a special garment was 21°C (period 1); 45°C and...
CIRCULATORY RESPONSES TO HEATING AND COOLING

Skin temperatures ($T_s$) averaged for each subject from five points on the body surface. Data on the left are from six subjects at the low workloads (0% grade on the treadmill). F.W. and B.B. on the left had a lower water temperature (40°C) perfusing the suit from 30 to 60 minutes (broken lines). Data on the right are from five subjects at the high workload (7.5% grade).

For the final experiment, subjects reported to the laboratory at noon. All had eaten breakfast at least 3 hours earlier. A Teflon catheter 70 to 80 cm long (18-gauge, thin-wall) was introduced percutaneously into the left brachial artery using a modified Seldinger technique. This catheter was positioned under fluoroscopic guidance in the descending aorta just below the origin of the left subclavian artery. A no. 7 double-lumen catheter 100 cm long was introduced through a small incision into the left antecubital vein. This catheter was positioned in the middle of the right atrium.

The plan of the experiment was to measure aortic blood pressure and cardiac output every 5 minutes. These measurements were made every 2 minutes for 6 minutes after each change in suit temperature. Thereafter, the regular 5-minute schedule was followed. Heart rate, right atrial mean pressure, and blood, rectal, and skin temperatures were measured continuously. In five subjects (three at the low and two at the high workloads), 1 ml of arterial blood was withdrawn at the tenth and final minute of each thermal period for lactate determination. Forty-five milliliters of arterial blood were drawn at the twentieth minute of each period except the last for calibration of the densitometer. A total of from 500 to 900 ml of 0.5N heparinized saline was...
slowly infused into the aortic catheter between determination of cardiac output and blood pressure. At the end of the experiment, the suit was perfused with cold (10°C) water while the subject recovered.

Control of skin temperature was accomplished by means of a suit\(^1\) of form-fitting coarse nylon mesh lined with small vinyl tubes spaced 1 to 2 cm apart. The suit and tubing covered the entire body surface except the face. The weight of this suit filled with water plus the rubberized nylon coverall was 3 kg. The purpose of the coverall was to minimize evaporative cooling and uneven heating of the skin.

Skin temperature was measured from five thermocouples (26-gauge, insulated, copper-constantan wires) attached to three points on the subject's back and two on the front of the torso. Sites of placement were the same on all subjects. These thermocouple junctions were 2 cm long and placed on the skin at a right angle to the tubing in the suit. This meant that only a small fraction of the thermocouple junction was ever in direct contact with the water-filled tubing. The five junctions were connected in parallel to average their outputs (resistances of the five leads were equal). Temperatures were referenced to a cold junction maintained at 0.0°C in a stirred ice bath. Thermocouple voltages were electrically offset to 33°C by a precision voltage source and suitably amplified.\(^2\)

The temperature of right atrial blood was measured from an insulated thermocouple junction (40-gauge, teflon-insulated, copper-constantan wire) positioned at the tip of the longer lumen of the double-lumen catheter, but not occluding it. These wires were sealed into one arm of a Y connector at the hub of the lumen. The reference junction was held at a constant temperature of 38.5°C ± 0.01°C. Thermocouple output was amplified by a Medistor Model A-60 amplifier. The other arm of the Y connector was connected to a pressure transducer.

Water was perfused through the suits at a constant rate of 8 liters/min from one of two 40-liter, constant-temperature baths. Temperature

Circulatory and temperature data from subject P.S. during mild exercise at 2.7 mph (9% grade). The magnitude, direction, and variability of this subject's responses typify those of the five other subjects studied at the two lower workloads. The temperatures of water perfusing the suit are shown in °C at the top of the columns separating the 30-minute periods. \(T_r\) = rectal temperature in °C. \(T_B\) = temperature of right atrial blood in °C. \(CO\) = cardiac output in liters/min. \(HR\) = heart rate in beats/min. \(SV\) = stroke volume in ml. \(CBV\) = central blood volume in liters. \(AoMP\) = aortic mean pressure in mm Hg. \(RAMP\) = right atrial mean pressure in mm Hg. \(TPR\) = total peripheral resistance in arbitrary units, mm Hg/liter/min.
was changed almost instantaneously by switching to a second bath previously set at the desired temperature. Techniques for calibration of the densitometer (modified Gilford 103IR) and calculation of cardiac output, mean transit time, and central blood volume were identical to those described earlier (8). Calibrations for the concentration of indocyanine green in blood taken at different times during each experiment agreed within 1% to 3% within any subject. The densitometer had constant sensitivity to indocyanine green with background levels of dye up to 9 mg/liter. Background levels of dye did not exceed 5 mg/liter during the study. Five milligrams of dye were rapidly injected (in 0.5 seconds) from a calibrated 1-ml syringe electrically equipped to record the beginning and end of injection. Injection was by displacement from the side hole, located 2 cm behind the tip of the double-lumen catheter. This eliminated any prolonged cooling of the thermocouple junctions (located at the tip of the other lumen) by the cool dye. Aortic blood pressure was recorded with a Statham P23Gb pressure transducer. The entire manometric system (catheter, gauge, Honeywell 131-2C carrier amplifier, and 24-Hz galvonometer) had unity gain to 20 Hz with 64% of critical damping. Right atrial mean pressure was measured with a Statham P23Db transducer and Honeywell 131-2C carrier amplifier and a previously described (9) electrical averaging circuit (critically damped, 0.16 Hz, low pass active filter). Both manometric systems were statically calibrated with a mercury manometer (aortic pressure) or a water manometer (venous pressure) at multiple levels before and after each experiment. Zero pressure was referred to a leveling bottle set at the level of the subject's fourth interspace. Heart rate was continuously measured using a heart-rate meter (Quinton Instruments) which was calibrated at each experiment with a pulse generator and was accurate to within ±1 beat/min. All data were recorded at full scale on a Honeywell 1508 Visicorder and on a Sanborn 3905 FM tape recorder. Calibrations for all data channels were repeated immediately after the end of the experiment. Electrical drift in all channels was negligible. All data, except cardiac output, which was measured by rectangulation from the original records (10), was processed by hand from tape-recorded data replayed at very slow recorder paper speeds. Aortic mean pressure was measured from electrically averaged (as above [8]) pulsatile pressure replayed from magnetic tape.

Blood lactate was determined using the method of Ström (11) and previously described procedures (7).

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Results

RESPONSES AT LOW OXYGEN CONSUMPTIONS

Data from one of the six subjects studied at low oxygen consumption (0.9 to 1.5 liters/
The average responses for this group are shown in Figure 3. Qualitatively, the responses to heating or cooling for any subject were strikingly representative of the entire group.

Cardiac output, heart rate, stroke volume, central blood volume, and the temperature of right atrial blood were relatively constant during the first 30-minute period (referred to as the control period) during which skin temperature averaged 32.9°C (33.9°C to 32.3°C). Aortic mean pressure, right atrial mean pressure, and total peripheral resistance fell slightly during the control period. On heating the skin to 38.3°C (average at 60 minutes), cardiac output and heart rate rose, while stroke volume, central blood volume, aortic mean pressure, right atrial mean pressure, and total peripheral resistance all fell. The temperature of right atrial blood fell about 0.5°C due to an increase in the amount of blood returning to the right atrium from the skin which at that time was well below the rectal temperature (Fig. 1). The changes in each variable at the end of any 30-minute period are given in appropriate units and as percentages of that variable at the end of the previous period (or as percent of the entire control period at the end of the second 30-minute period) in Table 2.

When skin temperature was lowered at 60 minutes, there was a rapid fall in the heart rate and the temperature of right atrial blood. Cardiac output fell after a 5-minute delay and returned to control values at 90 minutes. Stroke volume, central blood volume, aortic mean pressure, right atrial mean pressure, and total peripheral resistance rose rapidly and overshot control values in four men (Fig. 2). Five to ten minutes after cooling began, the temperature of right atrial blood began to rise again. Undoubtedly, this was due to the accumulation of metabolic heat, undissipated because of cutaneous vasoconstriction. At 90 minutes, the rapid rise in skin temperature from 26.9°C toward 39.1°C brought a more rapid, and in three subjects a greater, response in cardiac output. For other variables, responses were similar to the first rise in skin temperature.

Measurements of the central blood volume should not be construed to represent accurate estimates of intrathoracic blood volume but rather to reliably describe directions of change in this volume. Since the aortic sampling site was distal to the subclavian arteries, the increase in blood flow to the arms on heating and decrease on cooling caused a false increase and decrease, respectively, in true mean transit time to the sampling site. Since these changes were of opposite sign to the true changes, the decrease in central blood volume with heating and the increase with cooling were underestimated. This has been discussed previously (8).

Although right atrial mean pressure cannot be considered as effective right ventricular filling pressure because intrathoracic pressure was not subtracted, the changes in the former must have closely paralleled the changes in effective filling pressure. Ventilation rose only 15% above control values by the end of heating and was still elevated 10% between the seventieth and seventy-fifth minutes of cooling after right atrial mean pressure had risen. Normally, right atrial mean pressure remains almost constant up to maximal work despite marked rises in ventilation, while reductions in intraesophageal pressure are comparatively minor (12).

Quantitatively, the variation among these subjects appeared to be related primarily to the different levels of skin temperature reached during the first 30 minutes of heating. The smaller changes in all variables during heating in subjects F.W., D.B., and D.R. appear to result from their lower skin temperature. Raising perfusion temperature to 45°C at the low workload in the remaining subjects gave an almost equal skin temperature and equal increase in cardiac output at the two levels of oxygen consumption (Fig. 1).

*Raw data from the other subjects were deleted from this paper by the editors to conserve space. Copies of these data are available from the author on request.*
**TABLE 2**

Summary of Average Cardiovascular Changes in Six Subjects at Low Oxygen Uptakes

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Cardiac output (liters/min)</th>
<th>Heart rate (beats/min)</th>
<th>Stroke volume (ml)</th>
<th>Central blood volume (liters)</th>
<th>Aortic mean pressure (mm Hg)</th>
<th>Right atrial mean pressure (mm Hg)</th>
<th>Total peripheral resistance (mm Hg/liter/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>21°C</td>
<td>initial: 11.0</td>
<td>initial: 0°*</td>
<td>initial: 105</td>
<td>initial: 1.18</td>
<td>initial: 1.20</td>
<td>initial: 100</td>
<td>initial: 6.0</td>
</tr>
<tr>
<td>final: 11.0</td>
<td>final: 0°*</td>
<td>final: 0°*</td>
<td>final: 107</td>
<td>final: 1.20</td>
<td>final: 1.12</td>
<td>final: 100</td>
<td>final: 6.0</td>
</tr>
<tr>
<td>40°C-45°C</td>
<td>initial: 13.5</td>
<td>initial: 2.5</td>
<td>initial: 146</td>
<td>initial: 1.20</td>
<td>initial: 1.12</td>
<td>initial: 100</td>
<td>initial: 6.0</td>
</tr>
<tr>
<td>final: 13.5</td>
<td>final: 2.5</td>
<td>final: 2.5</td>
<td>final: 146</td>
<td>final: 1.20</td>
<td>final: 1.12</td>
<td>final: 100</td>
<td>final: 6.0</td>
</tr>
<tr>
<td>49°C</td>
<td>initial: 14.3</td>
<td>initial: 3.9</td>
<td>initial: 107</td>
<td>initial: 1.20</td>
<td>initial: 1.12</td>
<td>initial: 100</td>
<td>initial: 6.0</td>
</tr>
<tr>
<td>final: 14.3</td>
<td>initial: 3.9</td>
<td>initial: 3.9</td>
<td>initial: 107</td>
<td>initial: 1.20</td>
<td>initial: 1.12</td>
<td>initial: 100</td>
<td>initial: 6.0</td>
</tr>
</tbody>
</table>

Averages were taken at the beginning and end of each 30-minute period (indicated by the temperature of the water perfusing the suit which is given at the top of each column) and the final 15-minute period. Changes (Δ) are given in appropriate units (upper number) and in percent (lower number) which was calculated from the ratio of the change to the value at the beginning of that period. Initial and final notations do not apply to this column.
### TABLE 3

Summary of Average Cardiovascular Changes in Four Subjects at High Oxygen Uptakes

<table>
<thead>
<tr>
<th></th>
<th>$21^\circ\text{C}$</th>
<th>$40^\circ\text{C}$</th>
<th>$10^\circ\text{C}$</th>
<th>$50^\circ\text{C}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output (liters/min)</td>
<td>initial 16.3</td>
<td>final 16.3</td>
<td>+3.1</td>
<td>19.4</td>
</tr>
<tr>
<td></td>
<td>final 19.4</td>
<td>17.9</td>
<td>+19%</td>
<td>20.1</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>initial 127</td>
<td>final 140</td>
<td>+13</td>
<td>140</td>
</tr>
<tr>
<td></td>
<td>final 140</td>
<td>140</td>
<td>+11%</td>
<td>181</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>initial 123</td>
<td>final 123</td>
<td>-17</td>
<td>123</td>
</tr>
<tr>
<td></td>
<td>final 109</td>
<td>109</td>
<td>-13.8%</td>
<td>122</td>
</tr>
<tr>
<td>Central blood volume (liters)</td>
<td>initial 1.28</td>
<td>final 1.28</td>
<td>+0.05</td>
<td>1.28</td>
</tr>
<tr>
<td></td>
<td>final 1.33</td>
<td>1.33</td>
<td>+3.9%</td>
<td>1.41</td>
</tr>
<tr>
<td>Aortic mean pressure (mm Hg)</td>
<td>initial 104</td>
<td>final 89</td>
<td>-15</td>
<td>89</td>
</tr>
<tr>
<td></td>
<td>final 89</td>
<td>89</td>
<td>-14.4%</td>
<td>92</td>
</tr>
<tr>
<td>Total peripheral resistance (mm Hg/liter/min)</td>
<td>initial 6.6</td>
<td>final 5.8</td>
<td>-0.8</td>
<td>5.8</td>
</tr>
<tr>
<td></td>
<td>final 5.8</td>
<td>5.8</td>
<td>-12%</td>
<td>4.9</td>
</tr>
</tbody>
</table>

The results from subject P.K. were omitted. Presentation of data is as described in Table 2 except for cardiac output, stroke volume, and central blood volume where averages from the initial period ($21^\circ\text{C}$) were used for comparison with the end of the second period ($40^\circ\text{C}$).
CIRCULATORY RESPONSES TO HEATING AND COOLING

FIGURE 4
Average circulatory and temperature data from four subjects during heavy exercise (7.5% grade, 3.5 mph). Data from P.K. were omitted (see text and Fig. 6). Abbreviations as in Figure 2.

RESPONSES AT HIGH OXYGEN CONSUMPTIONS
Average responses of four subjects at the higher oxygen consumptions (2 to 2.4 liters/min) are shown in Figure 4 and Table 3. Responses of a representative subject are shown in Figure 5. The data from subject P.K. were omitted from this summary because his responses differed so from those of the other four men. Despite a 1.5 to 2 times greater rate of endogenous heat production and higher temperatures of right atrial blood and rectum in these four men, the increase of 3.1 liters/min in cardiac output (range 2.9 to 3.4 liters/min) during heating was similar to that seen at the lower levels of work (range 1.5 to 4.4 liters/min). The correlation between the increase in cardiac output and the increase in skin temperature had a coefficient of 0.74 for all measurements between 30 and 60 minutes (except for subject P.K.) at all levels of work. The major differences in the circulatory responses at the two levels of oxygen uptake during heating were the tendency for aortic mean pressure...
and central blood volume to increase along with constant total peripheral resistance at the higher oxygen uptake; the rise in heart rate and fall in stroke volume were only slightly greater at this level. On sudden cooling, there was always a marked drop in aortic mean pressure in contrast to the rise always seen at low oxygen uptake (except subject G.Z. who also showed an initial fall).

All subjects, except P.K., experienced very rapid and dramatic amelioration of symptoms of exhaustion after 10 minutes of cooling. Subject P.K. (Fig. 6) was unable to meet the circulatory demands added by increased heat load. He showed an almost continuous fall in aortic mean pressure which was reversed only slightly by cooling. His drop in stroke volume from 120 to 83 ml/beat was the largest in the study. We do not know the reason for his inability to maintain aortic mean pressure and total peripheral resistance. Our expectation was that his performance would improve, as it did in the other men at the high workload, when suit-perfusion temperature was reduced from 45°C to 40°C after the initial trial run. At the fifty-fourth minute of exercise P.K. experienced lightheadedness, and suit temperature was immediately reduced to 10°C. After a period of temporary symptomatic relief, he again experienced lightheadedness, along with slight visual blurring at the sixty-fourth minute. As we were about to end the experiment, aortic mean pressure rose and the subject experienced relief. Because of these problems, the final heating period was reduced to 8 minutes. His recovery from
extreme fatigue after the experiment followed an entirely normal course over the next 24 hours. Subject P.K. was apparently overdriven by thermal stress. Because of the exhaustion of chronotropic reserve and depressed stroke volume, cardiac output could be neither increased nor maintained. Similar responses were observed in a previous study (8).

All subjects, including P.K., were well hydrated during the experiment. The administration of 0.5N saline and the suppression of sweating caused by the impermeable outer suit kept net water loss in these men down to 0.5% (0.1% to 1.2%) of body weight at the lower oxygen uptakes and to 0.9% (0.6% to 1.5%) at the higher levels.

Arterial blood lactate concentration showed no marked elevation suggestive of reduced muscle blood flow during heating (Fig. 7).

**Discussion**

During moderate exercise, cardiac output and heart rate increased with heating and decreased with cooling of the entire body surface. Stroke volume and aortic pressure decreased with heating and increased with cooling. During more severe exercise, although similar changes occurred in cardiac output, heart rate, and stroke volume, aortic mean pressure did not change during heating. When the skin was cooled, aortic pressure fell transiently, followed by a return to control levels, compared with a sustained increase during milder exercise.

There is strong evidence that heating and cooling the skin caused marked changes in the peripheral circulation. Clearly, the rapid fall in temperature of right atrial blood before cardiac output rose at the lower workloads indicated a shift in blood flow from deep to superficial veins (in still relatively cool skin), which were dilated by heating. There are several reasons for assuming that the subsequent increase in cardiac output was directed primarily to skin. The progressive decrease in the difference between temperatures of the skin and right atrial blood to an average of 0.2°C at 60 minutes indicates an increase in skin blood flow. At rest, muscular flow in the limbs is not increased with heating (10), and the rise in cardiac output of 3 to 4 liters/min parallels the rise in skin blood flow (13). There are data suggesting that muscle blood flow may even be reduced during exercise in a hot environment since arterial lactate concentrations tended to be higher than normal (7, 14) while oxygen uptake was maintained at (7, 8) or slightly below control values (14, 15). However, our subjects showed no rise in blood lactate concentration.

Visceral organs are the only others likely to receive any substantial fraction of an increment in cardiac output. During heating, renal (16) and splanchnic blood flow (7) fall below levels normally found at the same intensity of exercise in cool environments. Thus, total cutaneous blood flow may exceed the increment in cardiac output by quantities equal to or greater than those redistributed from visceral organs.

The observed responses of cardiac output, stroke volume, and heart rate may be the resultant of many factors, including reflexes from the skin (17) and humoral responses elicited by changes in temperature of the skin and right atrial blood per se. However, focusing on the periods immediately following changes in skin temperature reveals that the central responses were at least partly due to alterations in the peripheral circulation. Since the magnitude and direction of some changes depended upon the level of metabolism, results from the high and low levels are discussed separately.

**RESPONSES AT LOW OXYGEN UPTAKE**

We interpret the changes we observed as follows. Raising skin temperature abolished the level of peripheral vasoconstriction which is normally increased in proportion to the severity of exercise (3, 4). Recent evidence supports such a response to heating the skin in man (3, 4) and blood in dogs (18). Along with dilatation of the resistance vessels to the skin, the volume of the more compliant cutaneous venules and veins presumably increased suddenly. This caused a transient lag in venous return so that the output of the left ventricle exceeded that of the right, causing a
sudden reduction in central blood volume. Right atrial mean pressure either continued to fall or fell further as heating began, with further gradual pooling of blood in the cutaneous venous system. The increase in cardiac output was not adequate to compensate for the fall in total peripheral resistance, and aortic mean pressure fell further. Of course, other interpretations for these changes can be offered, but ours is strengthened by the responses to cooling as discussed below.

At both the low and high levels of oxygen uptake, interpretation of changes in response to the initial elevation in skin temperature was complicated by the presence of drift in several variables (Fig. 3). Ekelund and Holmgren (19) found that if work was prolonged in a cool environment, systemic and pulmonary arterial mean pressures, right ventricular end-diastolic pressure, and stroke volume gradually declined. The authors suggested that these changes might be due to gradual displacement of blood into peripheral veins consequent to peripheral vascular adjustments for thermoregulation (19). In our study, similar changes could be rapidly reversed by cooling the skin. Also, when skin temperature was maintained at low levels during the sixtieth to ninetieth minute, this downward drift was essentially eliminated. Thus, these changes do appear to be associated with thermoregulatory adjustments in the peripheral circulation.

Elimination of this drift during the cooling period (60 to 90 minutes) established a more stable baseline for measuring the responses to the second elevation in skin temperature (at 90 minutes). Here, reductions in aortic mean pressure, right atrial mean pressure, and total peripheral resistance were more obvious. After a 5-minute delay, stroke volume fell as before but somewhat faster.

We interpret the hemodynamic response to lowering skin temperature as follows. The fall in skin temperature caused cutaneous vasoconstriction. There are also previous reports of vasoconstriction with lowering of skin temperature (18, 20). Blood pooled in dilated cutaneous venules was rapidly displaced centrally. Right ventricular output momentar-
CIRCULATORY RESPONSES TO HEATING AND COOLING

of right atrial blood and central blood volume showed a small transient dip and rose thereafter—central blood volume only slightly. This suggests that there was no sudden, major redistribution of blood flow or volume to the skin. Judging from the magnitude of drift in aortic mean pressure and stroke volume during the control period, the major adjustments in the cutaneous and possibly other peripheral vascular beds may have already occurred during that period. The striking stability in temperature of right atrial blood after 15 minutes supports this reasoning.

Although 30 minutes of heating appeared to have altered flow through the skin, much the same as at the lower workloads (assuming the increment in cardiac output goes to the skin), this may have been accomplished without further significant displacement of blood volume peripherally. At least, behavior of aortic mean pressure and central blood volume during that interval suggested this. Unfortunately, reliable measurements of right atrial mean pressure could not be made at higher workloads because of large artifacts induced by walking up a grade. The fourfold increase in the difference between temperature of right atrial blood and skin temperature, despite an only 1.5- to 2-fold increase in heat production, suggests skin blood flow may have been even less at higher than at lower workloads. A thermally induced increase in muscle blood flow cannot be ruled out.

The dramatic drop in aortic mean pressure at high levels of work (and in subject G.Z., at a lower level) on cooling raises some perplexing questions. Where has vasodilatation occurred, and why have baroreceptor reflexes not buffered the changes more effectively—particularly in subject P.K.?

Possible explanations of the reversal of aortic mean pressure responses at the high oxygen uptake, that is, rise with heating and initial fall with cooling, are as follows: (1) The increase in cardiac output was unaccompanied by any further net vasodilatation so that total peripheral resistance remained constant from 45 to 60 minutes as aortic mean pressure and flow rose proportionally. This indicates that either there was not further vasodilatation of skin once it was heated or any which did occur was compensated for by vasoconstriction elsewhere (7, 16). (2) On lowering skin temperature, the drop in aortic mean pressure indicates a sudden reversal of compensatory vasoconstriction. The fall in aortic mean pressure is clearly not attributable in any subject to a major fall in cardiac output during a time when skin was still vasodilated (rapidly falling temperature of right atrial blood indicated skin vasodilatation at that time). Cardiac output was still close to peak values at that time.

A major feature of the cardiovascular response to competitive thermal and metabolic stress is the maintenance of aortic mean pressure during severe thermal stress when metabolic demands are very high in contrast to the fall seen at lower metabolic rates. Our data imply that when demands for oxygen transport are very high they will be maintained at the expense of thermoregulation.

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