Age alters the cardiovascular response to direct passive heating

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Minson, Christopher T., Stacey L. Wladkowski, Anthony F. Cardell, James A. Pawelczyk, and W. Larry Kenney. Age alters the cardiovascular response to direct passive heating. J. Appl. Physiol. 84(4): 1323–1332, 1998.—During direct passive heating in young men, a dramatic increase in skin blood flow is achieved by a rise in cardiac output (Qc) and redistribution of flow from the splanchnic and renal vascular beds. To examine the effect of age on these responses, seven young (Y; 23 ± 1 yr) and seven older (O; 70 ± 3 yr) men were passively heated with water-perfused suits to their individual limit of thermal tolerance. Measurements included heart rate (HR), Qc (by acetylene rebreathing), central venous pressure (via peripherally inserted central catheter), blood pressures (by brachial auscultation), skin blood flow (from increases in forearm blood flow by venous occlusion plethysmography), splanchnic blood flow (by indocyanine green clearance), renal blood flow (by p-aminohippurate clearance), and esophageal and mean skin temperatures. Qc was significantly lower in the older than in the young men (11.1 ± 0.7 and 7.4 ± 0.2 l/min in Y and O, respectively, at the limit of thermal tolerance; P< 0.05), despite similar increases in esophageal and mean skin temperatures and time to reach the limit of thermal tolerance. A lower stroke volume (99 ± 7 and 68 ± 4 ml/beat in Y and O, respectively, at P< 0.05), most likely due to an attenuated increase in inotropic function during heating, was the primary factor for the lower Qc observed in the older men. Increases in HR were similar in the young and older men; however, when expressed as a percentage of maximal HR, the older men relied on a greater proportion of their chronotropic reserve to obtain the same HR response (62 ± 3 and 75 ± 4% maximal HR in Y and O, respectively, P< 0.05). Furthermore, the older men redistributed less blood flow from the combined splanchnic and renal circulations at the limit of thermal tolerance (960 ± 80 and 720 ± 100 ml/min in Y and O, respectively, at P< 0.05). As a result of these combined attenuated responses, the older men had a significantly lower increase in total blood flow directed to the skin.

aging; hyperthermia; cardiac output; skin blood flow; cardiovascular responses

The cardiovascular adjustments of young men to direct passive heating were elegantly studied by Rowell and colleagues in the 1960s and early 1970s (24–29). These investigators used invasive measurements to investigate the cardiovascular responses to direct heating of the skin with water-perfused suits (26, 27, 29), heating subjects to the limits of thermal tolerance to elicit maximal hemodynamic responses to heat stress (27). Cardiac output (Qc) in some subjects more than doubled from baseline to the limit of thermal tolerance. This dramatic increase in Qc was accomplished in the young subjects by an increase in heart rate (HR) and an increase in, or maintenance of, stroke volume (SV), despite a fall in central venous pressure (CVP). The increase in SV was therefore attributed to an increase in sympathetic stimulation primarily affecting the β-receptors. In addition, the high skin and core temperatures resulted in a redistribution of blood flow from the splanchnic and renal vascular beds, independent of baroreceptor stimulation (29). Using this model to study thermoregulatory control, Rowell (24) determined that total skin blood flow (SkBF) in young men could increase up to 7.6 l/min during maximal passive heat stress.

There is evidence that advanced aging may alter the cardiovascular mechanisms that underlie human thermoregulatory control. It has been well established that the SkBF response to increasing core temperature is reduced as a function of age (13, 17) and that the attenuated response is due to a reduced active vasodilator sensitivity (16) and a reduced maximal SkBF capacity (22) in aged skin. It is unknown, however, how control of central hemodynamic function and blood flow to visceral organs may be affected by aging during direct passive heating. The reduced β-adrenergic responsiveness of the heart in advanced age may attenuate the chronotropic and inotropic reflex responses to passive heating. During exercise, when the muscle pump aids venous return and, therefore, cardiac filling pressure, an age-related increased reliance on the Frank-Starling mechanism partially compensates for the reduced adrenergic responsiveness and helps maintain SV. However, during passive heat stress, blood is pooled in the cutaneous veins, CVP is reduced, and the increase in Qc is not explained by the Frank-Starling mechanism of the heart (23). Within this construct, it is reasonable to hypothesize that older individuals would respond to direct passive heating with an altered cardiac profile. Similarly, it is not known whether splanchnic (SBF) and renal blood flows (RBF) would be reduced to a greater extent during passive heating in older individuals to compensate for an attenuated Qc response, although evidence from studies during exercise suggests that such compensation is unlikely (10, 15).

Therefore, the purpose of this investigation was to compare the central and peripheral hemodynamic responses of young and older men during direct passive heating to the limit of thermal tolerance. Specifically, it was hypothesized that the older men would have a lower Qc at the limit of thermal tolerance, which would be associated with a lower SkBF. Furthermore, it was hypothesized that the older men would have an atten-
ated contribution of redistributed blood flow from the splanchnic and renal vascular circulations to the skin.

MATERIALS AND METHODS

Subjects

All procedures utilized in this investigation were approved in advance by the Committee for the Protection of Human Subjects of the Office of Regulatory Compliance, The Pennsylvania State University. After approved informed consent procedures, seven young (Y; 19- to 28-yr-old) and seven older (O; 64- to 81-yr-old) men were recruited to participate in the study.

Before participating in the experimental protocol, each subject underwent a screening procedure that included 1) a physical examination by a physician, 2) measurement of skinfolds as an estimate of adiposity (1), 3) a resting 12-lead electrocardiogram, 4) blood tests to establish that hepatic and renal function were normal, 5) measurement of supine, seated, and standing blood pressure (BP), and 6) a maximal graded exercise test on a treadmill. All subjects were healthy nonsmokers who were not currently taking any medication that had the potential to impact the cardiovascular or thermoregulatory variables of interest. Subject characteristics are presented in Table 1.

Experimental Procedures

On the day of an experiment, subjects reported to the laboratory in a fasted state at 0700. Subjects were weighed before and after the experimental protocol on a scale accurate to ±10 g, and weight loss was kept to within 0.5 kg during the experiment by infusion of normal saline. A 20-gauge peripherally inserted central catheter (PICC; SoloPICC 71956, SoloPak) was inserted in the basilic or cephalic vein of the right arm and advanced into the superior vena cava to the level of the third or fourth intercostal space. Placement of the PICC was verified by a chest X-ray, and adjustment to the placement was made if necessary, followed by a second X-ray. The PICC was connected to a pressure transducer (model 42647-05 Transpac IV, Abbott) and attached to the subject at the intersection of the catheter tip (as determined from the X-ray) and the midaxillary line. The transducer was calibrated before and after the experiment using a water manometer.

A second catheter was inserted into a forearm or hand vein of the right arm for infusion of a solution containing indocyanine green (ICG; Becton Dickinson) and p-aminohippurate (PAH; Merck) for the measurement of SBF and RBF, respectively. A third catheter was inserted into the antecubital vein of the left arm for venous sampling.

After instrumentation (described below) was completed, subjects were dressed in a water-perfused suit and a plastic coverall to inhibit evaporative cooling. Subjects wore only thin shorts under the water-perfused suit, which covered the entire surface of the body with the exception of the head, feet, and arms below the elbow. The feet were wrapped in plastic to minimize heat loss. The subjects were placed in the supine position, and thermoneutral water (~34°C) was circulated through the suit to keep the subject from becoming overheated during the remainder of the set-up procedure. The baseline period consisted of 50 min, during which thermoneutral water from a circulating water bath was circulated through the water-perfused suit. At the end of the baseline period, the water in the suit was switched to a second bath with 50°C water. The heating period was continued until the limit of thermal tolerance was reached. The limit of thermal tolerance was defined as the time at which 1) the subject expressed that he was unable to continue, 2) esophageal temperature (T es) reached 39.5°C, or 3) the subject was unable to control hyperventilation as determined by end-expiratory CO2 concentration. When one of these criteria was attained, the recovery period began and the subject was cooled with cold water (~15°C) from a third water bath for 20 min.

Measurements

T es, mean skin temperature (T sk), HR (from a 3-lead electrocardiogram), and CVP (via the PICC) were measured continuously throughout the entire protocol. Other data were collected at 10-min intervals during the baseline period and every 5 min during the heating and recovery periods. Venous blood (7 ml) was drawn at Qc and was measured simultaneously, then BP and forearm blood flow (FBF) were measured. Pilot work in our laboratory determined that this sequence of data collection allowed sufficient time after BP and FBF measurements for ICG and PAH concentrations at the sampling site to be in equilibrium with the rest of the circulation.

Temperatures. T es was calculated as the unweighted average of eight copper-constantan thermocouples placed on the upper and lower chest, upper and lower back, stomach, shoulder, thigh, and calf. T es was measured at the level of the right atrium from a thermistor located in the lumen of a sealed pediatric feeding tube. During insertion, subjects drank water (5 ml/kg body wt) to ensure that they were adequately hydrated before the experimental procedures.

FBF. Two BP cuffs and a mercury-in-Silastic strain gauge were placed on the left arm for venous occlusion plethysmography as described by Whitney (37). Each FBF determination comprised the average slope of three or more separate measurements. Changes in FBF were assumed to reflect changes in forearm SkBF, because underlying inactive muscle blood flow does not change under conditions of heating (6). The upper BP cuff was also used for the measurement of systolic (SBP) and diastolic BP (DBP) by brachial auscultation. Mean arterial pressure (MAP) was calculated as (0.33·SBP) + (0.67·DBP).

Qc, Qv was determined by an acetylene-rebreathing technique (30, 31, 33) utilizing a mass spectrometer to measure gas concentrations. SV was calculated as Qc ÷ HR. Total peripheral resistance (TPR) was calculated as [MAP – CVP]/Qc.

SBF and RBF. To measure SBF without catheterization of the hepatic vein, an estimate of the resting extraction ratio (ER) for ICG is needed. Studies using younger subjects have assumed a dye extraction of 0.85 (8); however, individual

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Older</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>23 ± 1</td>
<td>70 ± 3*</td>
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<tr>
<td>Weight, kg</td>
<td>75 ± 5</td>
<td>88 ± 4*</td>
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<td>Height, cm</td>
<td>175 ± 2</td>
<td>179 ± 2</td>
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<td>Ao, m²</td>
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<td>21.2 ± 0.2</td>
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<td>VO2max, ml·kg⁻¹·min⁻¹</td>
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<td>Body fat, %</td>
<td>13 ± 2</td>
<td>24 ± 2*</td>
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<td>PV, liters</td>
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<td>3.0 ± 0.1</td>
</tr>
<tr>
<td>ml/kg</td>
<td>48 ± 2</td>
<td>37 ± 2*</td>
</tr>
<tr>
<td>BV, liters</td>
<td>5.8 ± 0.3</td>
<td>3.1 ± 0.2*</td>
</tr>
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*Significantly different from young (P < 0.05).
hepatic extraction of dyes can vary among subjects and presents a potential source of error, particularly among subjects of different ages (4). We measured the ER in each subject by an intravenous bolus injection technique based on a two-compartment model of ICG removal from the plasma by the liver (9). This procedure was performed on a separate day after subject screening and at least 5 days before the experimental protocol. Subjects were supine for a minimum of 30 min before withdrawal of an aliquot of blood to serve as a spectrophotometer blank and the bolus injection of ICG (0.5 mg/kg body wt). Five minutes after injection, a 5-ml venous sample was collected in a lithium heparin tube, then venous samples were obtained every 3 min for 30 min. Samples were centrifuged at 3,000 rpm for 20 min, and the plasma concentration was measured by spectrophotometry (absorbency of 805 nm, and again at 910 nm to test for turbidity). A separate ER was calculated for each subject from the two slopes of the plasma disappearance curve of ICG by computer (Sigma Plot, San Rafael, CA) utilizing the Marquardt-Levenberg algorithm. In addition, plasma volume (PV) and blood volume (BV) were also measured in nine of the subjects (5 O and 4 Y) on a separate day by Evans blue dye. PV was determined in the remaining five subjects from the extrapolated time 0 concentration of the ICG disappearance curve. The correlation between PV measurements using these two methods has been estimated to be 0.85–0.93 (9). Subsequent changes in PV and BV were calculated from the changes in hematocrit (Hct) and hemoglobin concentration measured in triplicate in accordance with the procedure of Dill and Costill (7).

During the experimental trial, SBF and RBF were determined simultaneously from continuous infusion of ICG and PAH, respectively. These methods and the potential sources of error have been analyzed in detail previously (28, 34) and will only be discussed briefly. After 20 ml of blood were drawn to serve as a blank, intravenous injection of a priming dose of ICG (0.10 mg/kg body wt) and PAH (8.0 mg/kg body wt) was followed by a constant infusion of ICG (0.5 mg/ml) and PAH (12 mg/ml) at a rate of 1.0 ml/min. As described above, blood was drawn every 10 min after the start of infusion during the baseline period. To allow for dye equilibration, only the 40- and 50-min samples were used to calculate baseline values. Plasma concentrations of ICG were measured spectrophotometrically (as described above), and plasma concentrations of PAH were determined with the color reagent N-(1-naphthyl)-ethylenediammonium dichloride (2).

Although hepatic extraction of ICG remains constant during periods of heat stress (28), SBF changes during passive heating. Corrections for the resulting non-steady-state condition were necessary, since the dye removal rate no longer equaled the dye infusion rate. Therefore, splanchic plasma flow (SPF) was calculated from the rate of change of dye concentration as follows

$$\text{SPF} = \frac{l}{(C_{a2} - C_{a1})/dt} - \frac{\text{PV}}{I(\text{ER} - C_{a})}$$

where l is the infusion rate (0.5 mg/min), C_{a2} and C_{a1} are peripheral venous (which are equal to arterial) dye concentrations at times 2 and 1, respectively, dt is the time difference between samples (5 min during heating and recovery), and C_{a} is the arterial dye concentration. SPF was calculated as

$$\text{SPF} = \frac{l}{(1 - \text{Hct})}.$$  

Because of the short protocol and the rapid changes in RBF, urine collection of PAH (the preferred method to measure RBF) was not possible in this study. In the absence of urine collection, a potential source of error, namely, the extrarenal extraction of PAH, becomes a concern. Furthermore, the same constraints for the measurement of SBF exist for the measurement of RBF; specifically, the assumption of equality between

### RESULTS

#### Subject Characteristics

The physical characteristics of the subjects are presented in Table 1. The young and older men differed in age by ∼45 yr. The older men were significantly heavier and had a higher percent body fat and a lower maximal
O₂ uptake (P < 0.05). The two groups did not differ significantly in height or body surface area (A₀). Because we recruited only normally active subjects (i.e., non-sedentary and non-endurance-trained), all subjects were between the 25th and 75th percentile rankings for their respective age groups for anthropometric and maximal O₂ uptake values (14).

Although no significant difference was observed in the measured PV when expressed in liters, PV was 23% lower when expressed relative to body weight (ml/kg) in the older men. In addition, the older men had a lower calculated BV (P < 0.05), due in part to their lower measured Hct (42 ± 1 and 37 ± 2% in Y and O, respectively, P < 0.05). Both groups of men lost ~0.6 kg of body weight during the protocol due to sweating (0.7 ± 0.3 and 0.5 ± 0.4 kg in Y and O, respectively), and there was no difference between the groups of men (P > 0.05). Evaporative water loss was minimized by the plastic coverall and by the infusion of ~500 ml of normal saline solution to keep the sampling catheter patent during the entire protocol.

Temperature and Hemodynamic Variables

The average group responses for the variables measured during the experimental protocol are displayed vs. time in Figs. 1–4. To allow for temperature and hemodynamic stabilization and for ICG and PAH equilibration in the blood after the start of infusion, only the last two baseline measurements (i.e., at 40 and 50 min of baseline) are presented.

Although there were no age differences in time to maximal tolerance (68 ± 6 and 64 ± 7 min in Y and O, respectively, P > 0.10), the individual tolerance time to direct passive heating varied among subjects (60–80 and 55–85 min in Y and O, respectively). After baseline, responses during the first 60 min of heating are plotted for each variable, followed by the averaged values during the last 20 min of each subject’s heating period to account for the differences in tolerance time.

Temperature responses. Because the water-perfused suit is designed to tightly control skin temperature and the subjects were unable to dissipate much heat through sweating due to the plastic coverall, Tₚₖ was not different between the groups of men at baseline or throughout the heating protocol (Fig. 1). No differences were observed in Tₑₛ at any time period. Therefore, the calculated mean body temperature likewise did not differ between the two groups at baseline (36.3 ± 0.4 and 36.0 ± 0.6°C in Y and O, respectively) or at the limit of thermal tolerance (38.8 ± 1.0 and 38.7 ± 0.8°C in Y and O, respectively).

Cardiac responses. Baseline values of Qₑ, HR, and SV did not differ between the young and older men (Fig. 2). During heat stress, Qₑ was significantly lower in the older men at all time points during the first 60 min of heating (age × time interaction, P < 0.05). Qₑ remained significantly lower in the older men during the final 20 min of heating (P < 0.05). No differences in HR when expressed as beats per minute were observed between the groups. When expressed as a percentage of maximal HR, the HR was significantly higher after 35 min of heating in the older men (62 ± 3 vs. 75 ± 4%). Although the pattern of SV response was different between the groups, no significant differences in SV were observed during the first 60 min of heat stress. However, SV was significantly lower during the last 20 min of heating in the older than in the young men (68.3 ± 6.3 vs. 89.8 ± 6.5 ml/beat for final value, P < 0.05), and in the older men the final SV was significantly lower than their
baseline SV (68.3 ± 4.3 vs. 91.7 ± 5.5 ml/beat, P < 0.05). There were no differences in baseline CVP or ΔCVP during heat stress between the groups at any time.

Other cardiovascular responses. Baseline FBF and SBF were similar in the older and young men (Fig. 3). FBF was lower in the older than in the young men at all time points after 15 min of heating (P < 0.05) as well as during the final 20 min of heating (8.7 ± 1.4 vs. 14.4 ± 1.6 ml·100 ml⁻¹·min⁻¹ at 20 min of heating and 18.3 ± 1.8 vs. 28.1 ± 2.0 ml·100 ml⁻¹·min⁻¹ for final value). There were no differences in SBF between the two groups of men during the heating protocol. There was a significant age difference in RBF, with a lower flow at baseline in the older men (873 ± 64 vs. 1,137 ± 70 ml/min, P < 0.05) and at all time points during heating (674 ± 26 vs. 847 ± 69 ml/min for final value, P < 0.05); however, there was no difference in the pattern of responses. When the redistribution of blood flow from the splanchnic and renal vascular beds is combined (i.e., the combined decrease in SBF and RBF from baseline to maximal heating), the older men redistributed significantly less blood flow away from these two circulations than did the young men (0.96 ± 0.08 vs. 0.72 ± 0.10 l/min, P < 0.05). Similar to the differences observed for FBF, the predicted change in flow directed
to the skin (ΔSkBF) from baseline was significantly lower in the older men at all time points after the first 10 min of heating (P < 0.05).

Pressure responses. MAP was well maintained throughout the protocol in both groups of men, with no significant differences observed between the groups (Fig. 4). The older men had a higher DBP at baseline and throughout the heating period (P < 0.05), but there were no differences in SBP. There was also a significant effect of age on TPR at all time points (P < 0.05).

The present study is the first to quantify the difference in SkBF between older and young men by comparing the increase in blood flow to the skin by measuring Qc and its distribution to the major vascular beds. Figure 5 displays the separate contributions of Qc, SBF, and RBF to the total increase in ΔSkBF from baseline to the end of heating. The young men increased SkBF by an average of 5.8 l/min compared with an average increase of only 2.7 l/min by the older men. Although the calculated increase in SkBF in our young subjects is lower than the 7.6 l/min average increase reported by Rowell (24) and Detry and colleagues (6), all young subjects were well within their range of 3–11 l/min.

Repeatability. Repeatability tests were performed by two of the young men who volunteered to repeat the entire protocol and in one older man whose first test was stopped after 35 min of heating because of logistical problems. In the two young men, reliability coefficients of determination (r²) for the measurements of Qc, SBF, RBF, and FBF at 60 min of heating were 0.92, 0.86, 0.84, and 0.96, respectively. In the older man, r² for the variables at 30 min of heating were 0.85, 0.88, 0.79, and 0.94, respectively. Paired t-tests on these variables at baseline, every 15 min of heating, and at maximal tolerance did not reveal any significant intra-subject differences between the first and second tests.
DISCUSSION

The major finding of the present investigation was that the central cardiovascular responses to direct passive heating were altered in older compared with young men. Despite this, the tolerance time to maximal heating was similar. Specifically, $Q_c$ was significantly lower in the older than in the young men, despite similar increases in skin and core temperatures. A lower SV, most likely due to an attenuated increase in inotropic function during the heat stress, was the primary factor for the lower $Q_c$ observed in the older men. Increases in HR during the heating protocol were similar in the young and older groups; however, when expressed as a percentage of maximal HR, the older men relied on a greater proportion of their chronotropic reserve to obtain the same HR response. Furthermore, the older men redistributed less blood flow from the splanchnic and renal circulations. As a result of these combined attenuated cardiovascular responses, total blood flow directed to the skin was significantly lower in the older men.

Because it has been well established that the SkBF response to increasing core temperature is decreased as an effect of age (13, 17), it was not surprising that the increase in $Q_c$ directed to the skin was lower in the older men during passive heating. For total SkBF to increase, $Q_c$ must increase or a greater proportion of $Q_c$ must be diverted from other regions to the cutaneous circulation. During cycle exercise in the heat, a reduced ability to increase $Q_c$ and to redistribute blood flow from the splanchnic and renal vascular circulations has been reported in older subjects (10, 15). It is plausible that the reduced ability of older men to increase $Q_c$ and redistribute blood flow from the visceral circulations (particularly the renal bed) may not allow SkBF to be increased in older men to the same extent as in young men during passive heating. However, an alternate explanation might be that the lower SkBF was consistent with a matching of $Q_c$ to the attenuated vasodilator capacity in the aged skin and, therefore, not limited to any extent by the altered central cardiovascular function. Indeed, the differences observed in the hemodynamic profiles of the young and older men are very intriguing. Older individuals typically respond to an increased cardiac demand, such as during exercise, with an increased reliance on the Frank-Starling mech-
condition of passive heat stress, the heart is working in the low range of the Frank-Starling curve, where small decreases in filling pressure can induce a large decrement in \( Q_c \). Therefore, in young men the increased sympathetic drive to the heart seems more important to maintaining \( Q_c \) and arterial pressure (23). In older men the increased sympathetic drive to the heart may not be sufficient to increase \( Q_c \) because of decreased \( \beta \)-adrenergic receptor responsiveness. Therefore, intrinsic mechanisms of the heart may play a more important role during heat stress as individuals age. However, the pressure gradient between the cutaneous veins and the right atrium presumably was less in the older men (although this was not measured) because of a lower SkBF and slightly smaller fall in CVP during heating (\( P = 0.05 \)). This may have resulted in a functional reduction in venous return, leading to a lower filling pressure and contributing to the inability of the older men to maintain SV, particularly in light of their increased reliance on the Frank-Starling mechanism. In addition, as a large proportion of total BV is sequestered in the cutaneous veins when SkBF is high, the smaller BV of the older men may have compounded the problem of a reduced venous return.

We previously showed that redistributed blood flow from the splanchnic and renal circulations is less in older men exercising at 60% of peak \( \dot{O}_2 \) uptake in the heat (10, 15). In the present study the combined flows from these two circulations, i.e., the total redistributed flow from the visceral circulation, were significantly less in the older men (Fig. 5; \( P < 0.05 \)). Rowell and colleagues (29) previously showed that the increase in splanchnic and renal vascular resistance during heat stress was not due to a reduced BP and postulated that vasoconstriction of the splanchnic and renal vascular beds arises from a reflex response to the increase in skin and core temperatures. It is therefore unlikely that the difference observed in redistributed flow was due to a greater vasoconstrictor stimulus in the young men and probably reflects a greater end-organ response to sympathetic outflow. However, the extent to which differences in visceral vasoconstriction contribute to the total increase in SkBF is small (~0.25 l/min) compared with the much greater impact of a lower \( Q_c \) (Fig. 6).

MAP was maintained fairly well in both groups of subjects, and the transient, small changes in arterial pressure to passive heating were similar to those in the study by Rowell (24). There was a significant age effect on DBP at rest and throughout the heating protocol, although the pattern of response to heat stress was similar. This higher DBP and slightly elevated SBP resulted in a slightly higher MAP. MAP is known to increase mildly with age and is usually attributed to an increase in SBP. The reason for the elevated DBP in the present study is unknown and is not a consistent finding in healthy older normotensive patients, al-
Tsk and Tes. The site used for the measurement of Tsk is in line with this latter hypothesis. It is doubtful that the younger subjects had a higher rate of evaporative heat loss, since the two groups of men were covered, with the exception of the head, forearms, and hands, with a plastic coverall that had increased by 0.5°C. These data, combined with the high correlation between ΔFBF and ΔSkBF in the present study, provide support for the accuracy and reliability of FBF as an index of changes in SkBF.

It is interesting to note that the more rapid initial increase in ΔSkBF than in ΔFBF in the young men discussed above was not observed in the older men, despite the fact that the rise in Tsk and the rise in Tes were similar between the two groups. There are a few plausible explanations for this difference. It is possible that aged skin does not respond as rapidly to direct heating because of a higher resting vascular tone or reduced responsiveness to direct heating. Although less likely, it is also possible that the baroreceptors in the older men buffered the increase in SkBF to minimize the fall in MAP at the initiation of heating, suggesting that Qc in the older men could not increase sufficiently to match the fall in peripheral vascular resistance. The lack of a reflex inotropic response in the older men to the initial increase in Tsk is in line with this latter theory. Furthermore, there is evidence in young men that the baroreceptors limit heat-induced active vasodilation when combined with an orthostatic stress, such as lower body negative pressure (12).

The lack of a difference in Tes between the groups, despite much higher SkBFs in the younger men, is more difficult to reconcile. One might suspect that the higher blood flow to the skin during direct passive heating in the young men would bring more heat from the skin to the core, resulting in a more rapid increase in core temperature. It is possible that the ~10% larger A0 of the older men allowed for a greater total area of heat exchange, equalizing the rate of heat gain. It is also possible that there was a significant amount of heat directly conducted (vs. convected) through the tissues, accounting for the similar increases in Tsk, although there are no data to support this latter hypothesis. It is doubtful that the younger subjects had a higher rate of evaporative heat loss, since the two groups of men were covered, with the exception of the head, forearms, and hands, with a plastic coverall that minimized evaporative heat loss. This is confirmed by the similar loss of body weight during the protocol in the two groups of men.

Fig. 6. Increase in ΔSkBF (calculated from increase in Qc and redistributed flow from splanchnic and renal circulations from baseline to limits of subject’s thermal tolerance) vs. increase in forearm blood flow (FBF, measured by venous occlusion plethysmography) during direct passive heating with water-perfused suits in young (●) and older men (○). Values are means ± SE. Increase in ΔSkBF was highly correlated with measurement of FBF in our subjects (r2 = 0.93). On the basis of our findings, ΔSkBF can be predicted from measurement of ΔFBF in this group of subjects as follows: ΔSkBF (l/min) = [0.19 · ΔFBF (ml · 100 ml−1 · min−1)] + 0.19.

Though a great deal of heterogeneity exists (21), TPR fell in the young men by 38% on average, similar to the 29–58% decrease in TPR in the subjects studied by Rowell et al. (29). In the older men, however, TPR decreased only 23% during heating, which is not surprising in light of the lower cutaneous vascular conductance.

Because it was first reported that blood flow to the underlying musculature does not change during whole body passive heat stress or local heating of the forearm (6, 11), many investigators have used venous occlusion plethysmography during heat stress, i.e., changes in FBF, to represent changes in SkBF. However, questions exist as to whether this change in SkBF at the forearm is representative of changes in SkBF in other areas of nonacral skin and whether this method is equally valid for older individuals. In the present study, ΔFBF was highly correlated with ΔSkBF calculated from the combined changes in Qc, SBF, and RBF (r2 = 0.93; Fig. 6). Although these data do not address whether SkBF measured at different sites, such as in the calf, in an individual may vary, they do support the theory that changes in FBF can be used to represent changes in flow to the skin as a whole, irrespective of age. On the basis of our findings, the increase in ΔSkBF can be predicted from the measurement of ΔFBF in this group of subjects by the following formula: ΔSkBF (l/min) = [0.19 · ΔFBF (ml · 100 ml−1 · min−1)] + 0.19.

The slight deviation from a linear relationship between ΔFBF and ΔSkBF in the young men in Fig. 6 at the lower flow rates may be explained by a difference in the initial responsiveness of the skin to local and reflex increases in Tsk and Tes. The site used for the measurement of FBF by venous occlusion plethysmography was not directly heated by the water-perfused suit; therefore, an increase in FBF reflects a purely reflex response to passive heating. ΔSkBF, on the other hand, reflects an increase in blood flow to the skin due to the combination of local and reflex responses to the high skin temperature. A similar relationship between direct and reflex changes in FBF was observed in young men by Taylor et al. (32), when the authors simultaneously compared FBF responses to directly heating one forearm to ~42°C with the reflex increase in FBF in the contralateral, nonheated arm during whole body heat stress. A more rapid initial increase in FBF in the directly heated arm was noted; however, in most of the subjects FBF values in the two arms converged once Tes had increased by 0.5°C. These data, combined with the high correlation between ΔFBF and ΔSkBF in the present study, provide support for the accuracy and reliability of FBF as an index of changes in SkBF.

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In summary, the purpose of this investigation was to compare the cardiac and hemodynamic responses during direct passive heating to limits of thermal tolerance in young and older men wearing water-perfused suits. The lower SkBF in older men during heating was associated with a lower Q, and a reduced ability to redistribute blood from the visceral circulation. The cardiac responses to passive heat stress were also altered as an effect of chronological age, with the older men relying on a greater proportion of their chronotropic reserve to compensate for a reduced inotropic response.

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REFERENCES