Anesthesiology
81:1411–1421, 1994

Thermoregulatory and Anesthetic-induced Alterations in the Differences among Femoral, Radial, and Oscillometric Blood Pressures

James M. Hynson, M.D.,* Daniel I. Sessler, M.D.,† Azita Moayeri, B.S.,‡ Jeffrey A. Katz, M.D.§

Background: A decrease in radial artery blood pressure relative to central arterial blood pressure is commonly associated with the rewarming phase of cardiopulmonary bypass. Decreased hand vascular resistance has been suggested as a possible mechanism. Although decreased blood viscosity due to hemodilution may contribute to decreased hand vascular resistance, thermoregulatory vascular responses to core hyperthermia also may be important.

Methods: Seven healthy volunteers were studied. Volunteers first were cooled until thermoregulatory vasoconstriction was evident. Next, each was warmed until intense sweating developed. After a cool-down period, general anesthesia was induced with propofol and N_2O. Femoral artery pressure (a surrogate for central arterial pressure) and radial artery and oscillometric (brachial artery) pressures were compared during each of six defined thermoregulatory and anesthetic study conditions. To determine the effect of hand vascular resistance on blood pressure differences, measurements were compared before and after occlusion of hand blood flow. Upper-extremity blood flow was evaluated by forearm and fingertip plethysmography and laser Doppler flowmetry.

Results: Forearm, fingertip, and cutaneous blood flow increased significantly during warming and were maximal during intense sweating. During thermoregulatory vasoconstriction, femoral, radial, and oscillometric mean blood pressures were similar. In contrast, radial artery mean pressure was 5 ± 1 mmHg less than femoral artery mean pressure and 12 ± 8 mmHg less than oscillometric mean pressure during intense sweating. Hand compression reduced these differences. The contour of the radial artery pressure waveform was dramatically altered by thermoregulatory and anesthetic conditions. Radial artery systolic pressure exceeded both femoral artery and oscillometric systolic pressures during vasoconstriction but was less than these during intense sweating. Hand compression reestablished the exaggerated radial artery systolic pressure during all study conditions.

Conclusions: Thermoregulatory and anesthetic-induced alterations in upper-extremity blood flow substantially influence the relations among femoral artery, radial artery, and oscillometric blood pressure measurements. (Key words: Arterial blood pressure, measurement: direct; femoral; noninvasive; oscillometric; radial. Measurement techniques: fingertip blood flow; forearm blood flow; vasomotor index; volume plethysmography. Temperature: regulation. Thermoregulation: sweating; vasoconstriction; vasodilation.)

A DECREASE in radial artery blood pressure relative to central arterial blood pressure is commonly observed during rewarming and after discontinuation of cardiopulmonary bypass[1–7] and may in part result from decreased hand vascular resistance.[2,8] Hemodilution and decreased blood viscosity likely contribute to decreased hand vascular resistance[8]; however, two thermoregulatory, vascular phenomena also may be important.

First, hand blood flow is largely determined by the vasomotor status of thermoregulatory arteriovenous shunts located on the palmar surface of the hand and fingertips.[9] Depending on thermal conditions, these shunts can alter fingertip blood flow over a greater than tenfold range.[10] Second, changes in vascular resistance can result from active cutaneous vasodilation that occurs with thermoregulatory sweating.[11,12] Sweating is commonly, but not uniformly, observed during the rewarming phase of cardiopulmonary bypass and is the expected response to central nervous system hyperthermia.[13,14] Active cutaneous vasodilation in response to sweating, apparently is mediated by a yet-to-be-identified chemical released from stimulated sweat glands.[11,12,15] During vigorous sweating, total skin blood flow can exceed 7 l/min and equal cardiac output to the remainder of the body.[11] Because the upper extremities provide a large skin-surface area relative to

* Assistant Professor of Anesthesia.

† Associate Professor of Anesthesia.

‡ Research Associate.

§ Professor of Clinical Anesthesia.

Received from the Thermoregulation Research Laboratory, Department of Anesthesia, University of California, San Francisco. Accepted for publication August 25, 1994. Supported by National Institutes of Health grants GM39723 and GM49670 and Augustine Medical. Presented in part at the annual meeting of the American Society of Anesthesiologists, Washington, DC, October 1993.

Address correspondence to Dr. Hynson: Department of Anesthesia, University of California–San Francisco Mount Zion, 1600 Divisadero Street, San Francisco, California 94115.

tissue volume, sweating dramatically increases upper-extremity blood flow.[16]

We have previously demonstrated that thermoregulatory and anesthetic conditions alter the relation between oscillometric (brachial artery) and direct radial artery blood pressure measurements: oscillometric mean blood pressure exceeded direct radial artery mean blood pressure during thermoregulatory vasodilation and propofol–N_2O anesthesia but not during thermoregulatory vasoconstriction.[17] These results suggest that radial artery mean blood pressure is less than central mean blood pressure when upper-extremity blood flow is high (because of thermoregulatory or anesthetic-induced vasodilation). Because pressures were measured by different techniques, we were unable to determine whether higher oscillometric mean pressure was artifactual or reflected a true central-to-radial artery mean pressure difference.

To further evaluate the effect of thermoregulatory factors on blood pressure measurements, we studied femoral artery (assumed to represent central arterial pressure), radial artery, and oscillometric (brachial artery) blood pressures in volunteers exposed to environmental conditions sufficient to induce the full range of thermoregulatory vascular responses, from intense vasoconstriction to maximal sweating and active vasodilation. As in our previous study, volunteers were also studied during propofol–N_2O general anesthesia. Specifically, we tested the hypotheses that (1) thermoregulatory sweating decreases radial artery mean pressure relative to femoral artery and oscillometric mean pressures; (2) thermoregulatory sweating alters the relation of these pressures by increasing upper-extremity and hand blood flows; and (3) thermoregulatory and anesthetic-induced changes in upper-extremity blood flow (vascular resistance) alter the contour of the radial artery pressure waveform.

Materials and Methods

With approval from the University of California–San Francisco Committee on Human Research and informed consent from the volunteers, we studied seven young, healthy men. None of the volunteers was obese, was taking any medications, or had a history of cardiac or vascular disease. All refrained from food and beverage for at least 8 h before the start of the study.

Protocol

Volunteers lay supine throughout the study. During placement of vascular catheters and other monitoring equipment, they were covered with a cotton blanket. Volunteers were first exposed to an ambient temperature near 21°C until vasoconstriction occurred (vasomotor index ≤0.2; see section "Thermoregulatory Responses" below), then warmed using forced air (anterior surface) (Bair Hugger model 200, cover 300, Augustine Medical, Eden Prairie, MN) and circulating water (posterior surface) (Blanketrol II, Maxi-Therm mattress 276, Cincinnati Sub-Zero, Cincinnati, OH). The forced-air warmer was set on "high," and the circulating water mattress to 42°C. Before warming, all but the face and right arm of each volunteer was wrapped in plastic film to prevent evaporative heat loss. To prevent locally mediated vasodilation, the right arm and hand were not directly heated.[18]

Warming continued until tympanic membrane temperatures had increased at least 0.25°C beyond that triggering maximal sweating. Warming was then discontinued, the plastic film removed and volunteers allowed to cool passively for 1 h ("cool-down"). After approximately 60 min of cooling and establishment of a vasomotor index less than 0.8, anesthesia was induced by intravenous administration of propofol, 2 mg/kg, and maintained using propofol 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and inhaled N_2O 60%. Volunteers breathed spontaneously through a face mask. After 30 min, propofol–N_2O was discontinued and volunteers were allowed to awaken.

Unwarmed intravenous fluids were administered at approximately 150 ml/h, but increased to approximately 350 ml/h during active warming to prevent dehydration due to sweating. Routine anesthetic monitoring included electrocardiography, pulse oximetry and capnography.

Measurements

Blood Pressure. Femoral artery blood pressure was measured using a 5.1-cm-long, 20-G catheter (Angiocath, Deseret, Sandy UT) inserted into the right femoral artery approximately 2 cm distal to the inguinal ligament. Radial artery blood pressure was measured using an identical catheter placed in the right radial artery approximately 1 cm proximal to the wrist. Forearm venous pressure was measured from an identical catheter inserted into a large forearm vein just proximal to the wrist; the catheter was inserted with the opening facing centrally, that is, in the direction of blood flow.

Each catheter was connected to arterial pressure extension tubing (213 cm long) (Abbott Laboratories, North Chicago, IL). The liquid-filled component of the monitoring system was attached to a Transpac dispos-

able transducer (Sorenson, Abbott Laboratories, North Chicago, IL). Visible bubbles were purged from the tubing and transducer housing. A single stopcock was positioned on the proximal end of the tubing to allow transducer calibration against atmospheric pressure.

Two dual-channel, cardiac monitors (0–20-Hz band width; Tektronix 414, SpaceLabs, Redmond, WA) were used to condition and display the transduced pressure signals. Two monitors were used to allow simultaneous display of the digital values for each arterial pressure. Monitors and transducers were calibrated by simultaneously applying a constant pressure to each transducer. Agreement to within 1 mmHg over the range of 0–200 mmHg was established before proceeding with each study.

All transducers were zeroed at the level assumed to be that of the right atrium while volunteers remained supine. Analog output for each arterial blood pressure measurement was recorded intermittently on 5-mmHg/mm chart paper using a physiologic recorder (0–100-Hz band width; model 220, Gould, Valley View, OH). The dynamic characteristics of the catheter-transducer system were determined by measuring the damping coefficient and undamped natural frequency *in situ* at 30- to 60-min intervals using the fast-flush technique.[19] Damping coefficients were 0.24 ± 0.03 (mean ± standard deviation) and undamped natural frequencies were 19 ± 4 Hz.

Mean femoral and radial artery pressures were recorded simultaneously at end-expiration from the digital displays of the Tektronix monitors, which determine mean pressures electronically *via* a signal averager. Because arterial pressure varies over the respiratory cycle, systolic and diastolic pressures were determined by averaging the highest and lowest values during one respiratory cycle on the recorded waveform (the same cycle during which mean pressures were recorded).

Oscillometric blood pressure was measured over the right brachial artery using an Ohmeda 2140 monitor (Madison, WI). This monitor is manufactured by Critikon (Tampa, FL) and is mechanically and functionally identical to a Dinamap 8100 monitor (Critikon). A standard adult arm cuff was used for all volunteers.

All blood pressure measurements were obtained at 20-min intervals, except during propofol–N_2O anesthesia when they were recorded at 10-min intervals. At each interval, all measurements were repeated after occlusion of blood flow to the hand by compression of the hand with a pressure-infusion bag inflated to 150 mmHg.

Blood Flow. Forearm blood flow was measured with a strain gauge (Extensometer, University of Melbourne, Melbourne, Australia) placed around the widest part of the forearm.[20] The Extensometer is similar to a system based on mercury in silicone elastomere, but it uses a capacitive (rather than resistive) effect to provide a signal proportional to change in forearm width.[20] To initiate forearm blood flow measurements, a pneumatic cuff placed just distal to the elbow was inflated to 50 mmHg to occlude venous return. Total forearm blood flow was determined from the rate at which forearm circumference increased and converted to milliliters per minute per 100 milliliters tissue.

Fingertip blood flow was measured from the right middle finger by venous occlusion plethysmography as previously described.[21] Skin capillary blood flow was measured on the medial surface of the right forearm using a laser Doppler flowmeter (Periflux 3, Perimed, Piscataway, NJ).

Flow measurements were obtained at the same intervals described for blood pressures. All flow measurements were obtained from the right upper extremity while the arm and hand remained horizontal (at the level of the right atrium). Forearm flows were obtained with and without hand compression. Fingertip blood flows were not measured during hand compression.

Sequence of Measurements. Because all hemodynamic measurements were obtained in the same right upper extremity, we minimized the impact of one measurement technique on another by sequencing measurements as follows: fingertip blood flow, forearm blood flow, femoral and radial artery blood pressures, oscillometric pressure. This sequence, excluding fingertip flow, was then repeated following hand compression. At least 1 min was allowed to elapse between flow and pressure measurements to permit venous decompression.

Thermoregulatory Responses. Sweating was measured on the upper chest or abdomen (in volunteers with abundant chest hair) by passing 2 l/min of anhydrous oxygen across a skin surface area 6 cm in diameter that was covered with an air-tight, adhesive ostomy appliance (3706 and 3806, Hollister Products, Libertyville, IL).[22] Cutaneous water loss (in grams per meters squared per hour) was calculated from the gas flow rate (model FMA-5000, Omega Engineering), gas temperature and relative humidity (model HX93, Omega Engineering, Stamford, CT).[22]

Core temperature was measured using Mon-a-Therm tympanic membrane thermocouple probes (Mallinck-

Table 1. Minutes at Each Treatment Stage

Treatment	Minutes
Cooling	60 ± 29
Warming	150 ± 36
Uncovered	57 ± 8
General anesthesia	42 ± 7

rodt Anesthesiology Products, St. Louis, MO) connected to an Iso-Thermex electronic thermometer (Columbus Instruments International, Columbus, OH). The thermocouple was placed at the tympanic membrane.

Peripheral thermoregulatory vasoconstriction was assessed using core temperature, fingertip temperature, and ambient temperature to compute the vasomotor index.[23] The vasomotor index was calculated as $(T_{finger} - T_{ambient})/(T_{tympanic} - T_{ambient})$, where T = temperature. Values less than 0.2 indicate significant vasoconstriction; values exceeding 0.8 were assumed to indicate marked vasodilation.[24]

Data Analysis

Data were analyzed by grouping and averaging measurements according to treatment, sweating rate and vasomotor index. We defined six conditions: vasoconstriction, vasodilation (without sweating), mild sweating, intense sweating, cool-down, and general anesthesia. Vasoconstriction was defined by a vasomotor index less than or equal to 0.2 during passive cooling. Vasodilation was defined as a vasomotor index greater than 0.5 during warming. Mild sweating was defined as a sweating rate between 10% and 75% of the maximum rate observed and intense sweating as a rate exceeding 75% of maximum. The time interval from cessation of warming (and sweating) until induction of general anesthesia defined the cool-down period.

Differences between radial and femoral artery pressures were calculated at each 10- or 20-min interval as the radial artery pressure minus the femoral artery pressure. Differences between radial artery and oscillometric (brachial artery) pressures were calculated similarly at each interval. In each case, systolic, diastolic and mean pressure differences, with and without hand compression, were determined.

Results are reported as means ± standard deviation. Differences in blood pressures, were compared across study conditions using a nonparametric method for multiple comparisons.[25] Pressure differences before and after hand compression were compared using the Wilcoxon signed-rank test. Changes in forearm, fingertip and laser Doppler blood flows were evaluated by repeated-measures analysis of variance and Dunnett's tests using values obtained during vasoconstriction as references for comparisons. $P < 0.05$ indicated statistical significance.

Results

Volunteers weighed 72 ± 11 kg, were 178 ± 7 cm tall, and were 20–40 yr old. Cooling lasted 60 ± 29 min, and heating lasted 150 ± 36 min. The duration of each study phase is given in table 1. Maximum sweating rates were 307 ± 181 $g \cdot m^{-2} \cdot h^{-1}$ and were observed at tympanic membrane temperatures of 37.7 ± 0.4°C. These values did not differ significantly from those reported previously under similar conditions.[22]

Blood pressure data are reported for six of seven volunteers, because in one volunteer the femoral artery catheter became dislodged during the cool-down phase. Forearm flow data are reported for five of seven volunteers: one (above) was excluded because of catheter dislodgment and the second because of plethysmograph malfunction. At least one complete set of blood pressure and flow measurements was obtained from all volunteers for each study condition. Typically, three measurements were available for averaging.

Blood Pressure Measurements

Femoral artery pressure and heart rate for each study condition are given in table 2. Differences between radial and femoral artery mean pressures varied with thermoregulatory and anesthetic conditions (table 3). Vasoconstriction was associated with nearly identical radial and femoral artery mean pressures. In contrast, during intense sweating, radial artery mean pressure

Table 2. Heart Rate and Femoral Artery Blood Pressures during Each Study Condition

Condition	Heart Rate (beats/min)	Femoral Artery Blood Pressure (mmHg)		
		Systolic	Mean	Diastolic
Vasoconstriction	64 ± 4	127 ± 13	90 ± 9	69 ± 8
Vasodilation	66 ± 5	114 ± 13	80 ± 8	60 ± 8
Mild sweating	82 ± 8	113 ± 13	79 ± 10	60 ± 8
Intense sweating	93 ± 9	110 ± 12	76 ± 9	57 ± 8
Cool-down	67 ± 6	118 ± 13	83 ± 11	65 ± 8
Propofol/N_2O	65 ± 8	88 ± 8	64 ± 6	49 ± 5

Table 3. Differences between Direct Radial and Direct Femoral Artery Blood Pressures

	Radial – Femoral					
	Systolic		Mean		Diastolic	
Condition	Before HC	During HC	Before HC	During HC	Before HC	During HC
Vasoconstriction (VC)	16 ± 9	20 ± 7*	2 ± 1	2 ± 1	2 ± 5	1 ± 4
Vasodilation (VD)	8 ± 4	21 ± 10*	−1 ± 1	2 ± 2*	−1 ± 4	1 ± 6
Mild sweating (MS)	4 ± 7	20 ± 6*	−3 ± 2	1 ± 1*	−2 ± 4	1 ± 4*
Intense sweating (IS)	−1 ± 4	17 ± 7*	−5 ± 1	−1 ± 1*	−2 ± 3	−1 ± 4
Cool-down (CD)	14 ± 9	18 ± 8	0 ± 1	2 ± 1*	1 ± 5	3 ± 4*
Propofol/N_2O (GA)	10 ± 7	19 ± 7*	−1 ± 1	2 ± 1*	0 ± 4	1 ± 5
$P < 0.05$†	b, c, f, g, j, k, l, m, n, o		a, b, c, d, e, g, j, k, m, n	c, g, i, j, k		

Values are mean ± SD.

HC = hand compression.

* Statistically significant differences ($P < 0.05$, Wilcoxon signed-rank test) between values recorded during hand compression *versus* before hand compression.

† a = VC *vs.* VD, b = VC *vs.* MS, c = VC *vs.* IS, d = VC *vs.* CD, e = VC *vs.* GA, f = VD *vs.* MS, g = VD *vs.* IS, h = VD *vs.* CD, i = VD *vs.* GA, j = MS *vs.* IS, k = MS *vs.* CD, l = MS *vs.* GA, m = IS *vs.* CD, n = IS *vs.* GA, o = CD *vs.* GA; nonparametric multiple comparisons.[25]

was 5 ± 1 mmHg less than femoral artery mean pressure. During propofol–N_2O anesthesia, the differences between radial and femoral artery mean pressures were minimal (table 3).

The difference between radial and femoral systolic pressures also varied significantly across study conditions (table 3). During vasoconstriction, radial artery systolic pressure was 16 ± 9 mmHg greater than femoral artery systolic pressure, whereas during intense sweating, these pressures were nearly identical. During the cool-down period and also during propofol–N_2O anesthesia, differences between radial and femoral systolic pressures were similar to those observed during vasoconstriction.

Differences between radial and femoral diastolic pressures were small and did not vary significantly across study conditions (table 3).

Differences between radial artery and oscillometric (brachial artery) blood pressures are provided in table 4. Across thermoregulatory study conditions, the relations between radial artery and oscillometric systolic blood pressures showed a pattern similar to that ob-

Table 4. Differences between Direct Radial Artery and Oscillometric Brachial Artery Blood Pressures

	Radial – Oscillometric					
	Systolic		Mean		Diastolic	
Condition	Before HC	During HC	Before HC	During HC	Before HC	During HC
Vasoconstriction (VC)	15 ± 9	22 ± 8	−3 ± 6	−4 ± 2	−8 ± 9	−6 ± 10
Vasodilation (VD)	6 ± 7	21 ± 11*	−5 ± 10	−1 ± 7	−6 ± 9	−2 ± 10
Mild sweating (MS)	−5 ± 17	13 ± 10*	−11 ± 8	−8 ± 8	−8 ± 10	−3 ± 9
Intense sweating (IS)	−14 ± 10	5 ± 11*	−12 ± 7	−9 ± 8	−6 ± 10	−6 ± 10
Cool-down (CD)	10 ± 11	15 ± 15	−7 ± 7	−1 ± 6*	−3 ± 11	0 ± 10*
Propofol/N_2O (GA)	−6 ± 6	6 ± 13*	−8 ± 8	−5 ± 4*	−1 ± 8	2 ± 9
$P < 0.05$†	b, c, e, f, g, i, k, m, n, o	d, e, f, g, i, k, l		a	h, i	o

Values are mean ± SD.

HC = hand compression.

* Statistically significant differences ($P < 0.05$, Wilcoxon signed-rank test) between values recorded during hand compression *versus* before hand compression.

† a = VC *vs.* VD, b = VC *vs.* MS, c = VC *vs.* IS, d = VC *vs.* CD, e = VC *vs.* GA, f = VD *vs.* MS, g = VD *vs.* IS, h = VD *vs.* CD, i = VD *vs.* GA, j = MS *vs.* IS, k = MS *vs.* CD, l = MS *vs.* GA, m = IS *vs.* CD, n = IS *vs.* GA, o = CD *vs.* GA; nonparametric multiple comparisons.[25]

served between radial and femoral artery pressures. Radial artery systolic pressure was 15 ± 9 mmHg greater than oscillometric systolic pressures during vasoconstriction but was 14 ± 10 mmHg less than oscillometric pressures during intense sweating. Radial artery mean pressure was similar to oscillometric mean pressures during vasoconstriction but less than oscillometric mean pressures during intense sweating. Nevertheless, the difference between radial artery and oscillometric mean pressures did not vary significantly across study conditions (table 4).

Hand Compression. Hand compression data are also presented in tables 3 and 4. Hand compression significantly changed the differences between radial and femoral artery mean pressures under each study condition except vasoconstriction (table 3). During intense sweating, hand compression nearly eliminated the difference between radial and femoral artery mean pressures (from −5 to −1 mmHg). Hand compression during vasoconstriction slightly increased the difference between radial and femoral systolic pressures (from 16 ± 9 to 20 ± 7 mmHg). However, with hand compression, the difference in systolic pressures did not vary across study conditions (table 3).

Waveform. The contour of the radial artery pressure waveforms was greatly altered by thermoregulatory and anesthetic conditions (fig. 1A). During vasoconstriction, pressure waveforms were sharply peaked; during intense sweating they appeared much smoother. The effects of hand compression on the contour of the radial artery pressure waveforms are shown in figure 1B. The contour of the femoral artery pressure waveforms during vasoconstriction was less sharp than that of the radial artery waveforms and, consequently, less affected by thermoregulatory and anesthetic conditions (fig. 1C).

Venous Blood Pressure. Forearm venous pressure varied with study conditions (fig. 2). Forearm venous pressure measured during intense sweating was significantly greater than that measured during vasodilation and cool-down (10 ± 3 mmHg *vs.* 6 ± 3 mmHg). Hand compression significantly decreased forearm venous pressure during mild sweating, intense sweating, and propofol–N_2O (fig. 2).

Blood Flow Measurements

Plethysmographic and laser Doppler measures of upper-extremity blood flow demonstrated significant changes with thermoregulatory and anesthetic conditions (fig. 3). Fingertip blood flow increased dramatically during mild and intense sweating compared with vasoconstriction. Propofol–N_2O anesthesia induced high fingertip blood flow nearly identical to that observed during sweating and intense sweating.

Forearm blood flow also varied with study conditions and was approximately twice as great during intense sweating as during vasoconstriction (fig. 3). Forearm cutaneous flow, as determined by laser Doppler, was dramatically increased during mild and intense sweating compared with vasoconstriction (fig. 3). In contrast to fingertip blood flow, laser Doppler flow during propofol–N_2O anesthesia was less than that observed during mild or intense sweating, and did not differ significantly from laser Doppler flow during vasoconstriction.

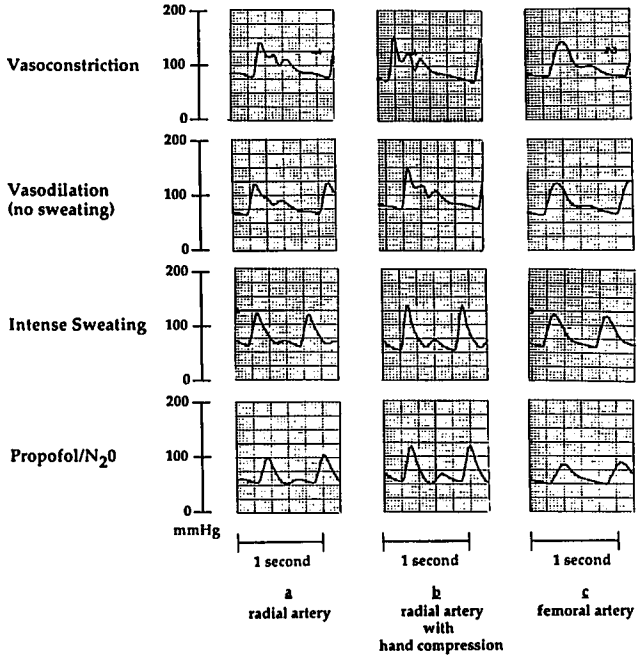

Fig. 1. Radial artery pressure waveforms (*A*) before and (*B*) during hand compression and (*C*) femoral artery waveforms are shown for one volunteer during four of the six study conditions. During vasoconstriction, the radial artery pressure waveform showed a sharp systolic peak followed by two secondary peaks. Progressing through vasodilation and then intense sweating, the contour of the waveform became smoother and the secondary peaks disappeared. The waveform contour during propofol–N_2O was similarly very smooth. Hand compression during intense sweating or propofol–N_2O increased the peak (systolic pressure) of the radial artery pressure waveform. The femoral artery pressure waveform became slightly smoother during warming and more noticeably so during propofol–N_2O. (Of the six volunteers, this subject exhibited the smallest difference between radial and femoral artery systolic pressures during vasoconstriction.)

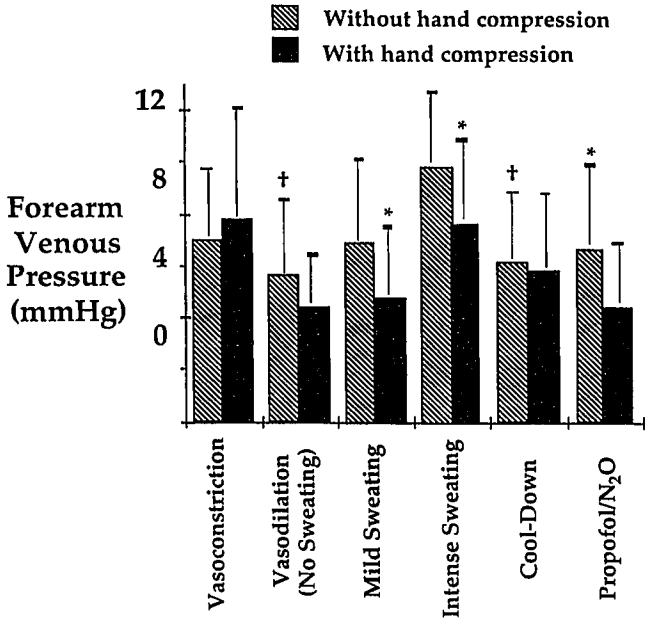

Fig. 2. Venous pressures, measured without hand compression, during vasodilation and during cool-down were significantly less than those measured during intense sweating. With hand compression, venous pressures did not vary across study conditions. Hand compression significantly decreased forearm venous pressure during mild sweating, intense sweating, and propofol–N_2O anesthesia. *Statistically significant differences ($P < 0.05$) between values measured with and those measured without hand compression; †statistically significant differences compared with intense sweating.

Discussion

Our results indicate that thermoregulatory and anesthetic-induced changes in upper-extremity blood flow have pronounced effects on blood pressure measurements. Sweating and active vasodilation increase upper-extremity and hand blood flow sufficiently to produce consistent femoral-to-radial artery and oscillometric-to-radial artery mean pressure differences. In addition, thermoregulatory changes in vascular tone alter the character (contour) of the radial artery pressure waveform; the exaggerated pulse pressure and systolic blood pressure typical of peripheral measurement sites is prominent during thermoregulatory vasoconstriction but disappears during sweating.

Central-to-radial Artery Mean Pressure Differences

Active cutaneous vasodilation associated with sweating may be a factor contributing to the central-to-radial artery blood pressure difference commonly observed[1–7] during the rewarming phase of cardiopulmonary bypass. Stern *et al.* reported aorta-to-radial artery mean pressure differences ranging from 0–8 mmHg. The observed differences were associated with increased forearm blood flow during rewarming and the authors concluded that decreased forearm vascular resistance contributed to the difference.[1]

Pauca *et al.* reported similar mean pressure differences after cardiopulmonary bypass and found that wrist compression distal to the radial artery catheter (to increase hand vascular resistance) substantially reduced the difference in some patients.[2] In another report, Pauca and Meredith demonstrated that the prebypass brachial artery pressure proximal to a dialysis

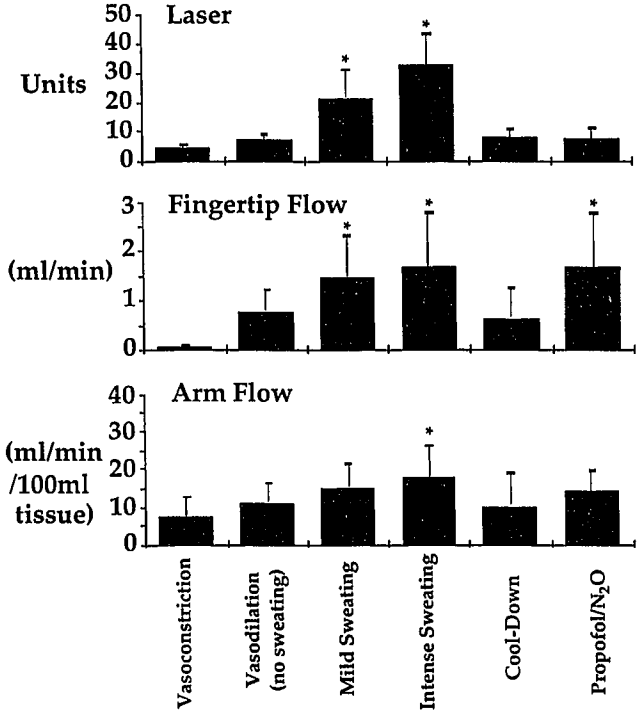

Fig. 3. Forearm, fingertip, and laser Doppler flows increased during warming. Fingertip blood flow increased approximately 40-fold from vasoconstriction to intense sweating. Forearm blood flow was approximately twice as great during intense sweating compared with that during vasoconstriction. Laser Doppler flow also was much greater during intense sweating than during vasoconstriction. Anesthesia with propofol–N_2O produced fingertip and forearm blood flows similar to those observed during intense sweating; however, laser Doppler flow was much less during propofol–N_2O anesthesia compared with intense sweating and did not differ significantly from that observed during vasoconstriction. *Statistically significant differences compared with vasoconstriction ($P < 0.05$; repeated-measures analysis of variance, and Dunnett's tests).

arteriovenous shunt increased markedly after compression of the artery distal to the catheter, and hypothesized that arteriovenous shunting through the hand might explain the effect observed in the radial artery during rewarming and postbypass.[26] Urzua proposed that decreased forearm and hand vascular resistance at the end of bypass could be explained purely by the hydraulic effects of hemodilution (decreased viscosity).[8]

Numerous additional studies have examined the relations between direct arterial blood pressures measured at central *versus* peripheral sites in patients undergoing cardiopulmonary bypass.[3–7] Common to all these studies is the inconsistency in the occurrence of the central-to-peripheral pressure difference. That is, some patients exhibit a difference, whereas others do not.

Although the central-to-radial artery mean pressure difference is typically observed near the end of the rewarming phase of cardiopulmonary bypass or in the immediate postbypass period, the roles of thermoregulatory responses—arteriovenous shunt dilation and sweating—in the development of this difference have not been examined. Specifically, none of the aforementioned studies reported whether sweating was present or absent.

In the current study, awake volunteers were aggressively warmed to provoke intense sweating. Sweating was associated with increased upper-extremity and hand blood flows relative to vasoconstriction, and consistently produced a 5-mmHg difference between femoral and radial artery mean pressures (and an $\approx$12-mmHg difference between oscillometric and radial artery mean pressures). Although the magnitude of the femoral-to-radial artery mean pressure difference was small, these findings suggest that central-to-radial artery pressure differences in patients during and after cardiopulmonary bypass may be influenced by the presence or absence of core hyperthermia and sweating.

Many studies of the central-to-radial artery pressure difference in cardiac surgical patients report that the difference disappears shortly after rewarming and termination of bypass.[1,6,7] Because patients typically exhibit a decrease in core temperature (*i.e.*, "afterdrop" resulting from redistribution of heat to peripheral tissues) once bypass is discontinued,[13] thermoregulatory responses to core hyperthermia are likely to be transitory. Therefore, the profound effect of sweating on upper-extremity vascular resistance would be expected to be brief. In our volunteers, the femoral-to-radial mean pressure difference disappeared almost immediately after discontinuation of warming and cessation of sweating (cool-down, table 3).

The contribution of sweating to the development of the central-to-radial artery pressure difference in cardiac surgery patients remains to be determined. In the presence of thermoregulatory arteriovenous shunt dilation, hemodilution and decreased blood viscosity would likely also contribute to the central-to-radial artery pressure difference. An increase in proximal resistance due to severe peripheral arteriosclerotic disease or from increased catecholamine concentrations, whether endogenous or exogenous, could also increase the difference. However, Rich *et al.* reported no change in the magnitude of the aorta-to-radial artery mean pressure difference before, compared with during, phenylephrine infusion.[4] Further study in cardiac patients correlating temperature, thermoregulatory responses (particularly sweating) and central-to-radial artery pressure differences is indicated.

In the current study, external hand compression, which essentially occluded hand blood flow, consistently decreased the femoral-to-radial artery mean pressure difference to 1 mmHg. That is, increasing hand vascular resistance *via* external compression essentially eliminated the femoral-to-radial mean pressure difference. Accordingly, in cases where substantial central-to-radial artery pressure differences are suspected, we recommend the simple maneuver, proposed by Pauca *et al.*, of compressing the radial and ulnar arteries at the wrist.[2,26,27] The effect of wrist or hand compression on the radial artery mean pressure will indicate if increased hand blood flow is influencing pressure measurements. Because patients' arms are commonly tucked at their sides during cardiac surgery, a remote compression system similar to that used in the current study may be helpful.

During most conditions associated with arteriovenous shunt dilation (mild sweating, intense sweating, and propofol–N_2O anesthesia), forearm venous pressure decreased significantly with hand compression (and a similar trend was observed during vasodilation but without statistical significance) (fig. 2). In contrast, hand compression had no effect on forearm venous pressure during vasoconstriction and cool-down (fig. 2). These results suggest that arterial pressure is partially transmitted through open arteriovenous shunts.

Oscillometric mean blood pressure exceeded radial artery mean pressure by approximately 12 mmHg during mild and intense sweating. Thus, oscillometric

mean blood pressure measured over the upper arm likely will be greater than radial artery mean pressure when a central-to-radial artery mean pressure difference exists. Oscillometric mean blood pressure also exceeded femoral artery mean pressure during mild and intense sweating (by 8 and 6 mmHg, respectively). In contrast, all three measures of mean blood pressure were similar during vasoconstriction. These results suggest that oscillometric blood pressure may overestimate aortic mean blood pressure when upper-extremity blood flow is high. Nonetheless, we recommend at least occasional oscillometric blood pressure measurement in all patients having radial artery catheters. Significantly greater oscillometric values may suggest that radial artery pressure does not accurately reflect central pressure.

We hypothesized that propofol-induced vasodilation would result in femoral-to-radial artery mean pressure differences similar to those observed during sweating and similar to the oscillometric-to-radial artery pressure differences observed in our previous study.[17] Surprisingly, femoral and radial artery mean pressures were nearly identical (and radial artery systolic pressure was actually approximately 10-mmHg greater than femoral systolic pressure; see discussion below). However, consistent with our previous results, oscillometric mean arterial pressure exceeded both radial and femoral artery mean pressures during propofol–N_2O anesthesia.[17]

Waveform Characteristics

The contour, or waveform, of the arterial blood pressure is determined by complex and incompletely understood interactions among the driving pressure (generated by the left ventricle), elastic characteristics of the arteries, and inertial and resistive properties of blood flowing in arteries.[28] Wave reflections from the peripheral circulation contribute substantially to the waveform's contour.[28] Wave reflections originate from branch points and, more importantly, peripheral arterioles (resistance vessels).[28] In general, the waveform contour is relatively peaked and narrow when pressure is measured at distal sites. The peak (systolic pressure) and trough (diastolic pressure) are therefore exaggerated relative to centrally measured pressures.[28]

In our volunteers, radial artery systolic pressure exceeded femoral systolic pressure during vasoconstriction. These results are consistent with those of Urzua *et al.*[29] The difference in systolic pressures progressively decreased with warming and increasing upper-extremity blood flow, indicating that substantially less wave reflection occurs when arteriovenous shunts and cutaneous capillaries are dilated. Hand compression during all study conditions (thermoregulatory and anesthetic) consistently reestablished the exaggerated radial artery systolic pressure.

Radial artery systolic pressure exceeded oscillometric systolic pressure during vasoconstriction (by 15 ± 9 mmHg) but was much lower than oscillometric systolic pressures during intense sweating (−14 ± 10 mmHg) and slightly lower (−6 ± 6 mmHg) during propofol–N_2O anesthesia. Interestingly, femoral artery systolic pressure was substantially less than oscillometric systolic pressures (−16 ± 5 mmHg) during anesthesia, suggesting that propofol–N_2O anesthesia, by producing vasodilation of the entire vascular tree, increases arterial compliance sufficiently to absorb (dampen) the arterial pressure pulse during transmission to the femoral artery.

Limitations

Numerous studies have demonstrated the importance of the dynamic characteristics of measurement systems, particularly the fluid dynamics of the catheter and tubing. Systolic pressures determined from transduced blood pressure signals may be grossly erroneous when frequency response characteristics of the catheter and tubing are inadequate.[19] The somewhat low, undamped natural frequency of our system likely exaggerated high frequency components of the pressure waveform and the radial artery systolic pressure during vasoconstriction. However, our catheter–tubing systems are typical of those clinically available, and had dynamic characteristics comparable to those reported elsewhere.[1–3]

Our femoral artery catheters were the same length as the radial artery catheters (5.1 cm); consequently, the tips of these catheters, at best, were positioned in the distal portion of the iliac arteries. Typically, longer catheters (10–15 cm) are used for femoral artery catheterization. We did not use longer catheters because they have poorer frequency (dynamic) responses. In addition, it is unlikely that mean pressures recorded 5–10 cm more proximal in the iliac artery would be appreciably different than those measured in the proximal femoral artery.

Although femoral and radial artery pressures were measured simultaneously, oscillometric pressure was not. Inflation of the upper arm cuff would have interfered with pressures measured in the distal radial artery. We measured oscillometric and radial artery pressures

in the same arm because significant differences in blood pressure can occur between the arms. Had we obtained oscillometric measurements from the left arm and recorded direct radial artery pressure from the right arm during cuff deflation, these measurements still could not be considered simultaneous because the oscillometric method does not determine systolic, mean and diastolic pressures from a single cardiac cycle. Nonsimultaneous measurements likely contributed to the large variability in the differences between oscillometric and direct arterial measurements.

Direct arterial mean pressures were taken at end-expiration. Oscillometric pressures were obtained during quiet breathing. Respiratory variations in blood pressure may have introduced some small bias between oscillometric and direct arterial pressures. In general, variations in direct arterial pressures with respiration were small.

Because systolic and diastolic pressures were measured from analog waveforms recorded on chart paper with 5-mmHg/mm gradations, these values could only be determined with an accuracy of ±2.5 mmHg. Errors due to lack of precision in the systolic and diastolic measurements would likely be small because we averaged measurements from each volunteer during most study conditions.

The oscillometric blood pressure device used in our study was manufactured by Critikon and is functionally identical to the Dinamap 8100. Different results may have been obtained had we used another manufacturer's unit. Most oscillometric blood pressure monitors determine mean arterial pressure by noting the cuff pressure at which oscillations are maximal.[30] However, various proprietary algorithms are used by different manufacturers to determine the systolic and diastolic pressures.[30]

We designed the study and data analysis primarily to detect significant alterations in the differences among blood pressure measurements across different thermoregulatory and anesthetic conditions rather than to evaluate the precision and bias of blood pressure measurement techniques. The latter goal would be more meaningful if true central blood pressure measurements were also available.

The femoral artery is generally considered a central site for blood pressure measurement[3–5,7]; we therefore assumed femoral artery pressure would reflect central aortic pressure. Nevertheless, every major artery exits the aorta proximal to the iliac and femoral arteries Interestingly, femoral artery systolic and mean pressures were substantially lower than those measured oscillometrically (at the brachial artery) during both propofol–N_2O and intense sweating. These differences may represent a bias in the oscillometric method (as noted above) during conditions of increased upper-extremity flow. Alternatively, they may indicate that femoral artery pressure does not reflect central pressure during conditions associated with intense vasodilation. If the latter is true, differences between central aortic and radial artery pressures will be even greater than those that we observed between the femoral and radial arteries during intense sweating.

In conclusion, thermoregulatory and anesthetic-induced changes in upper-extremity vascular tone and blood flow have important effects on blood pressure measurements. Increased forearm and fingertip blood flow during intense sweating are associated with a consistent femoral-to-radial artery mean pressure difference of 5 mmHg in healthy volunteers. This difference is nearly eliminated when hand blood flow is occluded by external compression. Oscillometric systolic and mean blood pressures measured over the upper arm exceed both radial and femoral artery pressures when upper-extremity and hand blood flows are high (during sweating and propofol–N_2O anesthesia) but not when flows are low (during thermoregulatory vasoconstriction).

These results suggest that radial artery pressure should be interpreted in the context of underlying thermoregulatory conditions. Over varying conditions affecting upper-extremity blood flow, oscillometric blood pressure may more consistently reflect central arterial blood pressure than does that measured at the radial artery.

The authors thank Mallinckrodt Anesthesiology Products, Ohmeda, Cincinnati Sub-Zero, and Respiratory Support Products for providing equipment or supplies used in this study. They also appreciate the technical assistance provided by Michael Lopez and the editorial assistance of Winnifred von Ehrenberg.

References

1. Stern DH, Gerson JI, Allen FB, Parker FB: Can we trust the direct radial artery pressure immediately following cardiopulmonary bypass? ANESTHESIOLOGY 62:557–561, 1985

2. Pauca AL, Hudspeth AS, Wallenhaupt SL, Tucker WY, Kon ND, Mills SA, Cordell AR: Radial artery-to-aorta pressure difference after discontinuation of cardiopulmonary bypass. ANESTHESIOLOGY 70:935–941, 1989

3. Bazaral MG, Welch M, Golding LAR, Badhwar K: Comparison of brachial and radial arterial pressure monitoring in patients un-

dergoing coronary artery bypass surgery. ANESTHESIOLOGY 73:38–45, 1990

4. Rich GF, Lubanski RE, McLoughlin TM: Differences between aortic and radial artery pressure associated with cardiopulmonary bypass. ANESTHESIOLOGY 77:63–66, 1992

5. Maruyama K, Horiguchi R, Hashimoto H, Ohi Y, Okuda M, Kurioka T, Konishi K, Muneyuki M, Kusagawa M. Effect of combined infusion of nitroglycerin and nicardipine on femoral-to-radial arterial pressure gradient after cardiopulmonary bypass. Anesth Analg 70: 428–432, 1990

6. Gravlee GP, Brauer SD, O'Rourke MF, Avolio AP: A comparison of brachial, femoral and aortic intra-arterial pressures before and after cardiopulmonary bypass. Anaesth Intensive Care 17:305–311, 1989

7. Mohr R, Lavee J, Goor DA: Inaccuracy of radial artery pressure measurement after cardiac operations. J Thorac Cardiovasc Surg 94: 286–290

8. Urzua J: Aortic-to-radial arterial pressure gradient after bypass (letter). ANESTHESIOLOGY 73:191, 1990

9. Hales JRS: Skin arteriovenous anastomoses: Their control and role in thermoregulation, Cardiovascular Shunts: Phylogenetic, Ontogenetic and Clinical Aspects. Edited by Johansen K, Burggren W. Copenhagen, Munksgaard, 1984, pp 433–448

10. Sessler DI, Moayeri A, Støen R, Glosten B, Hynson J, McGuire J: Thermoregulatory vasoconstriction decreases cutaneous heat loss. ANESTHESIOLOGY 73:656–660, 1990

11. Rowell LB: Cardiovascular aspects of human thermoregulation. Circ Res 52:367–379, 1983

12. Brengelmann GL: Circulatory adjustments to exercise and heat stress. Ann Rev Physiol 45:191–212, 1983

13. Tinker JH, Roberts SL: Management of cardiopulmonary bypass, Cardiac Anesthesia. 3rd edition. Edited by Kaplan JA. Philadelphia, WB Saunders, 1993, pp 938–939

14. Sladen R, Sessler DI: Elevated thermoregulatory thresholds for sweating during rewarming from cardiopulmonary bypass (abstract). Anesth Analg 72:S258, 1991

15. Brengelmann GL, Freund PR, Rowell LB, Olerud JE, Kraning KK: Absence of active cutaneous vasodilation associated with congenital absence of sweat glands in humans. Am J Physiol 240:H571–H575, 1981

16. Edholm OG, Fox RH, MacPherson RK: Vasomotor control of the cutaneous blood vessels in the human forearm. J Physiol (Lond) 139:455–465, 1957

17. Hynson JM, Sessler DI, Moayeri A, McGuire J, Schroeder M: The effects of preinduction warming on temperature and blood pressure during propofol/nitrous oxide anesthesia. ANESTHESIOLOGY 79: 219–228, 1993

18. Hellon RF: Local effects of temperature. Br Med Bull 19:141–144, 1963

19. Gardner RM: Direct blood pressure measurement: Dynamic response requirements. ANESTHESIOLOGY 54:227–236, 1981

20. Brimacombe JR, Macfie AG, McCrirrick A: The Extensometer: Potential applications in anaesthesia and intensive care. Anaesthesia 46:756–761, 1991

21. Rubenstein EH, Sessler DI: Skin-surface temperature gradients correlate with fingertip blood flow in humans. ANESTHESIOLOGY 73: 541–545, 1990

22. Washington DE, Sessler DI, Moayeri A, Merrifield B, McGuire J, Prager M, Belani K, Hudson S, Schroeder M: Thermoregulatory responses to hyperthermia during isoflurane anesthesia in humans. J Appl Physiol 74:82–87, 1993

23. Szelényi Z, Hinckel P: Changes in cold- and heat-defense following electrolytic lesions of raphe nuclei in the guinea-pig. Pflugers Arch 409:175–181, 1987

24. Schroeder M, Sessler DI, Belani K, McGuire J, Moayeri A, Merrifield B, Washington D: The vasomotor index correlates well with volume plethysmography as a measure of absolute finger flood flow (abstract). ANESTHESIOLOGY 77:A589, 1992

25. Zar JH: Biostatistical Analysis. Englewood Cliffs, Prentice-Hall, 1974, pp 156–157

26. Pauca AL, Meredith JW: Possibility of A-V shunting upon cardiopulmonary bypass discontinuation. ANESTHESIOLOGY 67:91–94, 1987

27. Pauca AL, Wallenhaupt SL, Kon ND: Reliability of the radial arterial pressure during anesthesia. Chest 105:69–75, 1994

28. O'Rourke MF, Kelly RP, Avolio AP: The Arterial Pulse. Philadelphia, Lea & Febiger, 1992, pp 35–43

29. Urzua J, Sessler DI, Meneses G, Sacco CM, Canessa R, Lema G: Thermoregulatory vasoconstriction increases the difference between femoral and radial arterial pressure. J Clin Monit 10:229–236, 1994

30. Geddes LA: Cardiovascular Devices and their Applications. New York, Wiley, 1984, pp 67–72