Radial Artery-to-Aorta Pressure Difference after Discontinuation of Cardiopulmonary Bypass


To test whether the radial artery-to-aorta pressure gradient seen in some patients after cardiopulmonary bypass (CPB) is due to reduction in hand vascular resistance, the authors compared pressures in the ascending aorta with pressures in the radial artery before and after CPB in 12 patients. They increased hand vascular resistance by briefly occluding the radial and ulnar arteries at the wrist and recorded that effect on the radial artery-to-aorta pressure relationship. They also recorded the effect of wrist compression on radial artery pressures before and after CPB in 38 patients not having aortic pressure measurements. Before CPB in the first 12 patients, the radial systolic arterial pressure (SAP) was significantly higher (P < 0.05) than the ascending aortic SAP, and wrist compression did not significantly affect that difference (P > 0.05). After CPB, the radial artery and aortic SAPs were not statistically different (P > 0.05), but wrist compression restored the higher radial artery SAP. The mean arterial pressure (MAP) was equal in four patients and 1–3 mmHg higher or lower in eight patients before CPB, and wrist compression did not alter those relationships. After CPB, MAP was equal in four patients; radial MAP was 1–3 mmHg higher or lower in six patients, and 7 and 10 mmHg lower in the last two patients. Wrist compression did not affect the dispersion across the zero mark in the first ten patients, but it increased radial MAP by 7 mmHg in the last two patients. In the final 38 patients, wrist compression did not affect radial MAP before CPB, but increased it by 7–12 mmHg in six patients after CPB. This increase was statistically significant (P < 0.05). These findings strongly suggest that a radial artery pressure lower than the aortic pressure after CPB is due mainly to a marked decrease in hand vascular resistance. (Key words: Blood pressure; diastolic waves; radial artery-aorta difference. Cardiopulmonary bypass.)

IN THE NORMAL HUMAN SUBJECT, the systolic arterial pressure (SAP) can be 30–40 mmHg higher in the radial artery than in the aorta, and the mean arterial pressure (MAP) is equal to or 2–3 mmHg lower than that in the aorta. At the end of cardiopulmonary bypass (CPB), however, both SAP and MAP are sometimes much lower in the radial artery than in the aorta or the femoral artery. Stern et al. studying the radial artery-to-aorta pressure gradient following CPB in human patients, found only a weak correlation between that gradient and a decrease in forearm vascular resistance, concluding rightly that “reduced forearm vascular resistance is an incomplete explanation for the change in the relationship between aortic and radial systolic pressures.” These authors did not measure hand blood flow and were not interested in the role of hand vascular resistance in the phenomenon they were studying.

Mohr et al., studying the radial artery-to-aorta pressure gradient and the radial artery-to-femoral artery pressure gradient in a similar group of patients, found that the systemic vascular resistance was significantly lower in patients whose radial artery pressures were lower than their aortic or femoral artery pressures. These authors concluded that the low radial artery pressures were due to “peripheral constriction, volume factors, and proximal shunting at the vascular beds of the forearm” and probably in the splanchic area. The report by Pauca and Meredith, which showed that the aorta-brachial artery pressure differences post-CPB and in a patient with a radial arterycephalic vein fistula could be abolished by occluding the brachial artery distal to the monitoring site, seemed to indicate that since the arteriovenous communication was the cause of the lower brachial pressure, a similar mechanism, that is, a decrease in vascular resistance distal to the monitoring site, was the cause of the lower brachial artery pressure seen post-CPB.

A similar mechanism could explain the lower radial than aortic pressure sometimes seen after CPB—that is, a decrease in vascular resistance in the hand. Among the reasons for this possibility are: 1) that hand vascular resistance has a tendency to change is well known; it can decrease, increasing the hand blood flow 5- to 10-fold in response to anesthesia, sympathetic blockade, or body heating. 2) Stern et al. found that the decrease in forearm vascular resistance was insufficient to explain the post-CPB radial artery hypotension, and 3) most anesthetics increase rather than decrease forearm vascular resistance in humans. A marked decrease in hand vascular resistance could increase flow velocity in the radial and ulnar arteries, thus decreasing the lateral pressure (Bureloullii principle). This decrease in lateral pressure would enhance the reduction in the diameter of the arteries already initiated by the low intravascular pressure maintained during CPB. In turn, a minimal decrease in the diameter of these vessels would increase the resistance.
to flow in proportion to the fourth power of the radius (Poisuille law). Thus, a marked decrease in hand vascular resistance has the possibility of decreasing the radial artery pressure and interfering with the local compensatory increase in blood flow.

Since this article deals with changes in vascular resistance, a reminder of well-known facts seems in order. First, the terms peripheral vascular resistance and systemic vascular resistance only relate MAP measured peripherally or centrally to cardiac output. Second, the MAP is directly related to steady blood flow through arteriolar and capillary vessels, while SAP and diastolic arterial pressure (DAP) are pressure oscillations in arteries of more than 1000 μ in diameter. Thus, changes in SAP are related to changes in peripheral vascular resistance only insofar as the changes in peripheral vascular resistance can modify the pressure oscillations in the arterial system. Third, the ability of variations in vascular resistance to elicit changes in SAP and DAP cannot be quantitated by the classic determination of systemic vascular resistance, but this effect can be estimated by a study of reflected waves. And, fourth, quantitative evaluation of vascular resistance in the hand requires measurement of hand blood flow as well as radial or ulnar artery pressure.

The objective of this study was to assess the effect of excluding hand circulation on the radial artery pressure recorded before and after CPB, and thus to assess the role of hand vascular resistance in radial arterial hypotension following CPB. Because we could not measure hand blood flow, our assessment of changes in hand vascular resistance was only qualitative.

Materials and Methods

Twelve patients being operated on for coronary artery bypass grafting or mitral valve replacement were studied. None of the patients required aortic balloon counterpulsation, epinephrine infusion, dopamine at doses larger than 3 μg·kg⁻¹·min⁻¹, or vasodilators other than 1–2 μg/kg nitroglycerin. The patients received preanesthetic medication consisting of lorazepam (0.05 mg/kg) and morphine (0.1 mg/kg). Fentanyl (50–70 μg/kg) was administered as the primary anesthetic, with pancuronium being given for muscle relaxation. In all patients, monitoring included electrocardiography, radial and pulmonary artery pressures, and finger pulse oximetry (Nellcor pulse oximeter). Additionally, a skin temperature sensor (Mallinckrodt) was placed on the dorsum of the hand where the radial artery pressure was being monitored. There were no intravenous infusion catheters in this hand. However, in six of the patients, one 22-gauge catheter was inserted in the dorsum of the hand for the withdrawal of venous blood for oxygen saturation determinations before and after CPB.

Radial artery and aortic pressures were measured through 2-inch 20-gauge Teflon catheters, one inserted into the radial artery and one into the ascending aorta. For the aortic measurements, 1 inch of the catheter was inserted into the lumen of the ascending aorta at approximately a 30° angle, its tip pointing distally, and the remaining length was secured to the outside of the vessel with silk sutures and retaining ties. This catheter was in place only during complete anticoagulation with heparin, as confirmed by an activated clotting time greater than 400 s. Radial and ulnar artery occlusion was accomplished by circumferential compression of the wrist over the cannulating catheter, the operator’s thumb being placed on the dorsal part of the wrist while the middle finger compressed the arteries. The degree of occlusion by manual compression at the wrist was confirmed in the thumb by the pulse oximeter.

The blood pressures at the aortic and radial artery sites were measured with the same transducer connected by equivalent lengths of tubing; a third pressure, obtained at the side-port of the aortic cannula, was measured with a different transducer, and its output served to ensure that the pressure comparisons were made only when this pressure had remained unchanged while the radial artery and aortic pressures were being recorded. The system used to measure the radial artery and aortic pressures was balanced and calibrated statically to a mercury standard before and after each observation. The natural frequency and damping coefficient of this system were determined by the flush method at the beginning and end of each recording session, as described by Gardner.

The recordings of aortic and radial artery pressures (with and without wrist compression) were obtained consecutively within a period of 20–30 s; at least three sets of recordings for each comparison were made. The total periods of observation were 5 min before CPB and 10 min after CPB was discontinued. The surgical procedure was stopped briefly, or more commonly restricted, during recording to avoid any displacement of the heart, the great vessels, or the monitoring catheters and connecting tubing.

A second group of 38 CPB patients was then studied by a simplified protocol. These patients had clinical characteristics similar to those of the study patients and, with the exceptions noted for the study patients, were also studied consecutively. In these 38 patients, we noted the increases in radial MAP of greater than 4 mmHg in response to the distal compression. Adding these results to those obtained from the 12 study patients, we determined the incidence of this response and its statistical significance before and after CPB. Aortic pressure was not measured, but in 11 patients the femoral artery pressure was measured through a 5-inch, 18-gauge Teflon catheter attached to a 48-inch extension tube and a transducer stat-
Table 1. Effect of Wrist Compression on the Radial Artery-to-Aorta Pressure Differences Before and After CPB (n = 12 Patients, Mean ± Standard Error)

<table>
<thead>
<tr>
<th></th>
<th>Radial Artery-to-Aorta Pressure Difference</th>
<th>Before Wrist Compression</th>
<th>During Wrist Compression</th>
<th>Before Wrist Compression</th>
<th>During Wrist Compression</th>
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</thead>
<tbody>
<tr>
<td>SAP (mmHg)</td>
<td>Before CPB</td>
<td>14.4 ± 3.0</td>
<td>17.3 ± 3.5</td>
<td>3.8 ± 3.8</td>
<td>15.6 ± 4.5*</td>
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<tr>
<td></td>
<td>After CPB</td>
<td>-0.3 ± 0.7</td>
<td>-0.6 ± 1.1</td>
<td>-2.1 ± 0.7</td>
<td>-2.5 ± 0.8</td>
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<tr>
<td>DAP (mmHg)</td>
<td>Before CPB</td>
<td>-1.3 ± 0.8</td>
<td>-0.2 ± 0.3</td>
<td>0.9 ± 1.1</td>
<td>-0.3 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>After CPB</td>
<td>14.8 ± 3.2</td>
<td>17.9 ± 5.4</td>
<td>5.9 ± 5.5</td>
<td>18.1 ± 4.0*</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>Before CPB</td>
<td>40.0 ± 8.6</td>
<td>48.4 ± 9.7</td>
<td>14.5 ± 6.7</td>
<td>49.4 ± 11.6*</td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td>Before CPB</td>
<td>1409.6 ± 129.6</td>
<td>1440.8 ± 131.2</td>
<td>857.6 ± 55.2</td>
<td>878.4 ± 56.8</td>
</tr>
<tr>
<td>SVR (dynes·sec·cm⁻²)</td>
<td>Before CPB</td>
<td>† Amplification = (radial pulse pressure - aortic pulse pressure) / aortic pulse pressure \times 100.</td>
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</table>

CPB = cardiopulmonary bypass; SAP = systolic arterial blood pressure; DAP = diastolic arterial blood pressure; MAP = mean arterial blood pressure; SVR = systemic vascular resistance.  
* P < 0.05 for corresponding value before wrist compression.

Data Collection and Assessment

Pressure data from both groups were recorded on a thermal recorder. MAP was obtained by electronic integration. Differences in radial artery and aortic pressures before and after CPB and with and without distal compression of the radial artery were calculated for SAP, DAP, and MAP. Values for pulse pressure (SAP−DAP) before and after CPB were obtained from the radial artery and aortic pressure tracings. Systemic and peripheral vascular resistances were calculated, relating the aortic and radial artery MAP−CVP, respectively, to the cardiac output and expressed as dynes·sec·cm⁻² after introducing the factor of 80. Additional variables recorded concomitantly with the compared pressures included skin temperature, rectal temperature, pulmonary artery blood temperature, hematocrit, cardiac output, central venous pressure, pulse rate, pump time, and oxygen saturation of venous blood from the hand in which the radial artery blood pressure had been measured. Only increases in MAP equal to or greater than 4 mmHg in response to wrist compression were considered clinically significant.

Repeated measures analysis of variance was used to assess the effects of bypass, occlusion, and the combination of these factors on blood pressure, pulse pressure, radial amplification of the pulse pressure, and systemic vascular resistance. A paired t test was used to compare differences in hand dorsal skin temperature, blood temperature, hematocrit, cardiac output, and heart rate. Correlation coefficients were calculated between the pressure differences and those compared variables plus age, oxygen saturation of venous blood obtained from the dorsum of the hand (the first six of the 12 study patients only), and pump time.

This study was approved by the Institutional Clinical Research Practices Committee, and patients gave their consent during the preanesthetic visit for all monitoring procedures. Normal anesthetic and surgical practices were unaltered during the study.

Results

The mean age of the ten male and two female study patients was 62 yr, ranging from 48–77 yr. The mean age of the additional 30 male and eight female patients was 65 yr (range 45–77 yr). All patients had equal pressures in both arms, as determined by the auscultatory method. The mean frequency response of the pressure monitoring system at the aortic and radial artery measuring sites was 25 Hz (range, 20–30 Hz); the mean damping coefficient was 0.26 (range, 0.20–0.39). Rectal temperature at discontinuation of CPB was 36.0–37.2° C.

Table 1 and figure 1 show that, for the 12 study patients, the SAP was significantly higher (P < 0.05) in the radial artery than in the ascending aorta before CPB, and that pulse pressure was 40% higher in the radial artery than in the aorta. Distal compression of the cannulated radial artery increased the difference in SAP from 14.0 to 17.3 mmHg and the difference in pulse pressure from 40 to 48%. Neither of these increases was significantly greater (P > 0.05) than the value obtained without wrist compression. When the values for each patient were considered, the radial SAP was 6–32 mmHg higher than the aortic SAP in ten of the 12 study patients and equal to the aortic SAP in two patients. Wrist compression increased the radial SAP by 3–12 mmHg in seven of the 12 patients.

After CPB, there was no statistical difference (P > 0.05) between the radial artery and aortic SAPs for the group, but analysis of individual values showed that the radial SAP was 4–25 mmHg higher than the aortic SAP in seven
of the 12 patients and 3–23 mmHg lower in the five remaining patients. Wrist compression restored the pre-CPB radial artery-to-aorta pressure relationship—that is, the radial SAP again became significantly higher than that measured in the aorta (P < 0.05). At this time, the increase in radial SAP in response to compression ranged from 3–34 mmHg in 11 of the 12 patients and was unchanged in one patient. The group pulse pressure was 14.5% higher in the radial artery than in the aorta, and it increased to 49% higher with wrist occlusion.

Before CPB, DAP at the aortic and radial artery sites was not statistically different, with and without wrist compression. However, after CPB, aortic DAP was statistically greater (P < 0.05) than radial DAP, whether or not distal compression was applied, although the radial artery-to-aorta pressure differences were only −2.5 ± 0.7 and −2.1 ± 0.7 with and without compression, respectively.

Although the difference in all 12 patients between the MAP measured in the aorta and that measured in the radial artery did not reach statistical significance (P > 0.05) either before or after CPB and either with or without wrist compression, analysis of individual values obtained before CPB showed that MAP was equal at both sites in four patients and 1–3 mmHg higher or lower in the radial artery than in the aorta in the remaining patients. Wrist compression did not alter this relationship. After CPB, radial MAP was equal to aortic MAP in four patients, 1–3 mmHg higher or lower than aortic MAP in six patients, and 7 and 10 mmHg lower than aortic MAP in the remaining two patients. Wrist compression did not affect dispersion around the zero mark in the first ten patients, but it increased radial MAP by 7 mmHg in the two patients with the highest radial artery-to-aorta differences. In the 38 additionally observed patients, wrist compression had no effect on radial MAP before bypass, but increased it by 7–12 mmHg in six patients after bypass. Adding these results to those observed in the first 12 patients permitted us to calculate the incidence of clearly measurable radial MAP increase (>4 mmHg) in response to wrist compression in a total of 50 patients; it proved to be 16%. Repeated measures analysis of variance showed that the increase was significant (P < 0.05) after CPB, but not before. Femoral and radial artery MAPs were within a 3-mmHg difference in patients who did not show a measurable response to wrist compression, but in patients whose radial MAP increased ≥7 mmHg after CPB, the latter pressure was similar to that measured in the femoral artery.

The vascular resistance was significantly higher (P < 0.05) before than after bypass. There was no difference between the vascular resistance calculated in the radial artery and that calculated in the ascending aorta except in the two patients who had radial MAPs lower than the aortic MAP by 7 and 10 mmHg, and those differences did not reach statistical significance (P > 0.05).

Diastolic waves in the radial artery pressure tracings were present in ten of the 12 patients before CPB. In all instances, the SAP was higher in the radial artery than in the aorta. Wrist compression at this stage increased the definition of the diastolic waves and SAP in two patients. Following CPB, diastolic waves were present in seven patients, all of whom also had higher radial artery than aortic SAPs. Wrist compression at this stage produced diastolic waves in four additional patients and increased the definition of the waves in the seven patients who had already shown them. The changes produced by wrist compression are illustrated in figure 2. Among the 38 patients whose aortic pressure was not measured, none of the six patients whose radial MAP increased ≥7 mmHg in response to wrist compression after CPB had diastolic waves before compression.

Post-CPB hematocrit, blood and skin temperature, cardiac output, systemic vascular resistance, and pulse rate changed significantly (P < 0.05) from pre-CPB values (table 2). None of these variables (or the changes they underwent during CPB) and none of the CPB durations correlated with the radial artery-to-aorta differences in SAP. Hand venous oxygen saturation was greater than 96% and did not correlate with the pressure differences in the six patients in whom it was measured.

**Discussion**

Many investigators have found that the peripheral SAP is 30–40 mmHg greater than aortic SAP in awake humans.
and in animals. Specifically, Kroeker and Wood found that the radial SAP in 12 healthy awake physicians was 2-30 mmHg greater than SAP measured in the aortic arch and that pulse pressure was 46% greater in the radial artery than in the aorta.

Our findings in 12 patients anesthetized with fentanyl before undergoing CPB are similar to those above. Additionally, before CPB, compression of the wrist distal to the monitoring site had an insignificant effect on the radial SAP and the pulse pressure difference, and it had no effect on the vascular resistance as calculated from the values of radial MAP and cardiac output. However, at the conclusion of CPB, the mean radial SAP was not different from the aortic SAP. Wrist compression restored the higher radial SAP in the group as a whole, increasing the radial artery-to-aorta pulse pressure difference to the pre-CPB level.

Our findings agree with those of Stern et al. and Mohr et al. in relation to the reversed relationship between aortic and radial SAP at the end of CPB, as well as in relation to the radial MAP being lower than the aortic MAP. However, the radial artery-to-aorta pressure differences in their series were larger than ours, both before and after CPB. Anesthesia for the patients studied by Stern et al. had been supplemented with halothane, an agent that markedly increases hand blood flow, and core and skin temperatures in patients studied by Mohr et al. had been much lower than those temperatures in our patients.

The effect of wrist compression on radial SAP has not been reported previously in humans, but some authors have shown that occlusion of any distal artery causes little change in the pressure wave upstream unless that artery is severed downstream or the capillary bed downstream is dilated. The work of Hamilton in dogs supports our explanation of why radial SAP decreases after CPB. Hamilton found that the pulse waves, including the diastolic waves, in both femoral arteries were identical, and that the systolic peak in the femoral arteries was 30-40 mmHg higher than the systolic peak measured simultaneously in the carotid artery. At this stage, a small dose of acetylcholine injected into the right femoral artery decreased the SAP by 30-40 mmHg below that in the left femoral artery. After a few seconds, the pressures in both femoral arteries became equal and the diastolic waves disappeared, but they could be restored by a tight grasp at the knee, making a pseudovasconstriction, or by intrarterial injections of small doses of epinephrine. Hamilton theorized that the originally higher femoral SAP was due to summation of reflected waves and the fundamental wave, and that vasodilatation abolished the generation of such waves.

Hamilton’s concept of wave reflection and wave summation has been studied extensively by several investigators, including O’Rourke, who concluded that wave summation due to wave reflection is the most important explanation of the shape of the pulse wave—mainly of the increase in the systolic peak and the appearance of the diastolic wave. Briefly, O’Rourke’s explanation is that the pulse pressure introduced in the aorta is exposed at the capillary level to a sudden change in impedance, which greatly increases the resistance to flow, thereby producing reflected pressure waves. The reflected waves sum with the fundamental wave, producing, at varying distances from the aortic root, a systolic peak, which is sometimes higher than that of the wave originally introduced in the aorta. Systolic amplification and diastolic waves are always present when peripheral vascular resistance is high. All our patients whose radial SAPs were higher than their aortic SAPs showed diastolic waves.

DAP was lower in the radial artery than in the aorta, as would be expected, since DAP is highly dependent on

| TABLE 2. Measured Variables Evaluated for Correlation with Changes in Systolic Arterial Pressure (n = 12 Patients, Mean ± Standard Error) |
|---|---|---|
| Before CPB | After CPB |
| Hematocrit | 35.3 ± 0.9 | 25.1 ± 0.9* |
| Blood temperature (°C) | 34.6 ± 0.13 | 36.6 ± 0.2* |
| Skin temperature (°C) | 29.4 ± 0.74 | 34.5 ± 0.59* |
| Cardiac output (/min) | 4.1 ± 0.29 | 6.0 ± 0.38* |
| SVR (dynes · sec · cm⁻⁵) | 1409.6 ± 129.6 | 857.6 ± 55.2* |
| Heart rate (bpm) | 88.5 ± 0.91 | 97.3 ± 2.67* |
| Bypass time (min) | 94.3 ± 3.5 |

CPB = cardiopulmonary bypass; SVR = systemic vascular resistance. * P < 0.05.
wave reflection, and wave reflection is more likely to have an effect at the radial artery site. Nevertheless, the numerical value of the radial artery-to-aorta DAP difference reached only $-2.4 \pm 0.7$ SE mmHg, which justifies our assumption that variations in the pulse pressure were dependent on variations in SAP.

Our theory that a shunt-like phenomenon had occurred at the level of the hand after CPB is based on: 1) the post-CPB appearance of radial artery-to-aorta MAP differences of 7 and 10 mmHg in two of 12 patients, which were reduced to 0 and 3 mmHg, respectively, in response to distal compression of the cannulated radial artery; 2) the post-CPB increase of 7–12 mmHg in response to wrist compression in six of the 38 patients whose aortas were not cannulated; and 3) the similarity of the radial and femoral MAPs ($\pm 3$ mmHg) before and after wrist compression in nonresponders and only after wrist compression in responders. Compression at the wrist would exclude a well-dilated, distal vascular bed, making the radial artery an extension of the catheter measuring the MAP present at the origin of the radial artery from the brachial artery. Because there is no flow in the occluded radial artery, the measured MAP would be unaffected by friction or by kinetic energy, and could not be higher in this artery than in the brachial artery. This presumed decrease in hand vascular resistance does not exclude a minor decrease in forearm vascular resistance, which could explain the incomplete disappearance of the radial artery-to-aorta MAP difference seen in one of the two study patients mentioned above.

We considered radial artery-to-aorta MAP differences to be of clinical importance only when equal to or greater than 4 mmHg because, in the ascending aorta, flow velocity is sufficiently high for kinetic energy to be seen as pressure and for up to 3.8 mmHg to be added to or subtracted from the lateral MAP. Whether there is an increase or decrease in MAP depends on the direction of the tip of the measuring catheter and the location of the tip within the lumen of the aorta. We tried to locate the tip close to the aortic wall and thus to avoid this uncertainty, but have no proof that we succeeded. This factor could explain the variation of 1–3 mmHg in the radial artery-to-aorta MAP seen in some of our patients.

We did not extend the comparison of the radial and aortic MAPs to more than 12 patients, despite evident post-CPB differences in only two of them, because this higher central than radial MAP has already been documented. However, we did try to determine the incidence of a clearly measurable increase in radial MAP in response to wrist compression in a total of 50 patients. The radial MAP increased clearly (7–12 mmHg) in 16% of these patients only after CPB. This finding suggests that wrist compression could be a useful maneuver to differentiate post-CPB radial artery hypotension from a more severe form of hypotension.

There are several speculative explanations for the lack of correlation between the peripheral-to-central pressure difference and some of the other variables monitored. Although hand skin temperature and hand venous oxygen saturation should be good indices of hand blood flow, it is quite possible that although the systemic vascular resistance appeared to have decreased considerably in these patients after bypass, the regional blood flow did not increase due to passive decrease in diameter in the brachial, radial, and ulnar arteries. It is also possible that there was no real decrease in systemic (as opposed to hand) vascular resistance after CPB; this variable was 41% lower after than before bypass, but could be explained by a concomitant 46% higher cardiac output, which could have been the effect of a pulse rate 43% higher than before bypass. Additionally, the sensitivity of skin temperature and hand venous oxygen saturation as indicators of changes in blood flow was blunted in this study, because, when the comparisons were made, skin temperature was no lower than $39^\circ$ C and hand venous oxygen saturation was no lower than 96%. It is also possible that the varying degrees of vascular disease in these patients had distorted the relationship we sought, since we excluded only those patients who required balloon counterpulsation, ephinephrine, more than 5 mg/kg of dopamine, or vasodilators other than prophylactic nitroglycerin. Since we did not assess sympathetic tone, we do not know whether this factor, which is normally dominant in the behavior of the hand circulation, played a role in the radial artery-to-aorta pressure difference seen.

In conclusion, following CPB, the radial SAP and MAP, which were lower than aortic SAP and MAP in some of our patients, seemed to be related to a decrease in hand vascular resistance, since restoring that resistance by wrist compression increased the peripheral pressures. The radial MAP should be the main variable to monitor clinically, because, except in a few instances following CPB, it is equal to or only a few mmHg lower than that measured in the aorta, whereas the radial SAP can be much higher or lower than that measured in the aorta. The wrist occlusion maneuver seems harmless, and its effect could be reassuring when judgment derived from observation of other hemodynamic variables needs reinforcement. So far, we have not seen increases in MAP in response to wrist compression in clinical situations other than at the end of CPB in patients similar to those reported here.

References

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