Differences Between Aortic and Radial Artery Pressure Associated with Cardiopulmonary Bypass

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Previous investigators have identified an aortic-to-radial artery pressure gradient thought to develop during rewarming and discontinuation of cardiopulmonary bypass. The authors measured mean aortic and radial artery pressures before, during, and after cardiopulmonary bypass in 30 patients, to determine when the pressure gradient develops. The pressure gradient was also measured before and after intravenous injections of sodium nitroprusside (1 μg/kg) and phenylephrine (7 μg/kg) to determine the effect of changes in systemic vascular resistance. A significant (P < 0.05) pressure gradient (mean ± SEM = 4.9 ± 0.7 mmHg) developed upon initiation of cardiopulmonary bypass. This gradient did not change significantly during the middle of bypass (4.2 ± 0.5 mmHg), with rewarming (4.8 ± 0.7 mmHg), immediately prior to discontinuation of bypass (4.6 ± 0.7), or 5 and 10 min following bypass (4.9 ± 0.9 and 4.8 ± 0.7 mmHg). Sodium nitroprusside significantly decreased systemic vascular resistance, by 15 ± 2%, during the middle of bypass but did not affect the pressure gradient. Likewise, phenylephrine increased the systemic vascular resistance by 52 ± 6% and 34 ± 4% during the middle of bypass and rewarming, respectively, without affecting the pressure gradient. Although the exact mechanisms responsible for the pressure gradient remain unknown, these results suggest its etiology is associated with events occurring during initiation of cardiopulmonary bypass rather than with rewarming or discontinuation of cardiopulmonary bypass. (Key words: Surgery, cardiac: cardiopulmonary bypass. Monitoring, arterial pressure: aortic; radial.)

The radial artery, though the most common site for invasive blood pressure monitoring, may not reflect aortic pressure during or after cardiopulmonary bypass (CPB). Numerous investigators have reported discrepancies between radial and aortic blood pressure during rewarming and after the discontinuation of CPB.1-9 This aortic-to-radial artery pressure gradient, which has been reported to be as great as 52 mmHg,3 may be clinically important for patient management.

The etiology of the pressure gradient has been extensively investigated but remains controversial. Previous investigators have demonstrated that a significant portion of the discrepancy was due to decreased arm1 and hand8,9 vascular resistance. Peripheral vasoconstriction, low blood volume, and proximal shunting have also been suggested as a possible mechanism.9 Continuous infusion of vasodilators during CPB also appears to cause an increased femoral-to-radial artery pressure gradient in the postbypass period.9

Bazareel et al. compared radial to subclavian artery but not aortic pressures throughout CPB and found that the gradient was significantly increased during the rewarming period of CPB.8 Although many other investigators have also suggested that the aortic-to-radial artery gradient develops during the rewarming period of CPB, nobody has demonstrated this phenomenon. In this study, we compared mean aortic to mean radial artery pressure before, during, and after CPB, in order to identify when and how much of a pressure gradient develops. Second, to determine the influence of changes in systemic vascular resistance (SVR) on aortic-to-radial artery pressure gradients, we measured pressures before and after intravenous injection of sodium nitroprusside and phenylephrine.

Materials and Methods

This study was approved by the human investigation committee at the University of Virginia, and informed consent was obtained from 30 patients. All patients received morphine sulfate (0.1 mg/kg) and scopolamine (0.3 mg) as a premedication. Anesthetic induction consisted of sufentanil (12-17 μg/kg) and midazolam (6 mg). Metocurine (0.2 mg/kg) and pancuronium (0.05 mg/kg) were used for muscle relaxation. Dobutamine (5 μg·kg⁻¹·min⁻¹) was administered during separation from CPB.

Patient monitoring included a triple lumen pulmonary artery catheter (7.5-Fr, Baxter®), electrocardiogram, and a 1.75-inch 20-G catheter (Arrow®) inserted in the left radial artery. Reported temperatures were monitored via an esophageal probe (Mallinkrodt®). Cardiac output was determined using triplicate room temperature thermodilution, computed through a Marquette 500 Tram monitoring system. After sternotomy, the tip of a 20-G catheter, identical to that used in the radial artery, was placed into the aorta proximal and away from the CPB flow stream but distal to the site of cross clamping, to allow continuous aortic pressure monitoring. The accuracy of

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Received from the Department of Anesthesiology, University of Virginia Health Sciences Center, Charlottesville, Virginia. Accepted for publication March 24, 1992. Presented in part at the 13th Annual Meeting of the Society of Cardiovascular Anesthesiologists.

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this technique was validated by common iliac artery pressure monitoring via a 6-inch 20-G catheter (Argon®) in 10 patients. Aortic root, common iliac, and radial artery pressures were transduced identically through 60-inch high-pressure tubing attached to two or three transducers (Viggo-Spectramed T-4812ADR®, mean resonant frequency 38 Hz, mean damping coefficient 0.14). The Tram System was calibrated using a simulator (Data Sim 6000®). The transducers were zeroed to air at the beginning of each case.

Esophageal temperature, cardiac output or pump flow, and mean radial artery (MAPr) and mean aortic (MAPa) pressures were recorded at the following intervals:

1. Five minutes before the initiation of CPB.
2. In early CPB, after cooling (esophageal temperature = 28.1 ± 0.1°C).
3. During the middle of CPB, 15 min after interval 2.
4. During rewarming, when esophageal temperature increased to 33°C.
5. Immediately prior to discontinuation of CPB.
6. Five minutes after discontinuation of CPB.
7. Ten minutes after discontinuation of CPB.

The data are expressed as the mean ± standard error of the mean. The MAPr and MAPa are compared statistically by repeated-measures analysis of variance.

The effects of phenylephrine and sodium nitroprusside were evaluated during midbypass. Phenylephrine was also evaluated during rewarming. If the MAPr decreased to below 55 mmHg, phenylephrine (7 µg/kg intravenously) was administered. If MAPa increased to above 85 mmHg, sodium nitroprusside (1 µg/kg intravenously) was administered. The MAPr and MAPa were recorded immediately before drug administration and 3 min after injection. Pump flow was held constant during the individual drug study periods. The change in SVR was based on the change in MAPa, since CVP and pump flow remained constant. Radial artery pressure and the pressure gradient before and after sodium nitroprusside and phenylephrine were compared using analysis of variance. Significance was achieved at P < 0.05.

### Results

The 30 patients were ASA physical status 3 or 4, aged 63 ± 4 yr, weighing 83 ± 5 kg, with 2-4 coronary vessel disease, undergoing coronary artery bypass grafting. All patients were included in the evaluation of pressure gradients throughout CPB. The hematocrit, esophageal temperature, and cardiac output or pump flow are shown in Table 1. There was no recordable difference between mean central aortic and common iliac artery pressure during CPB, thus validating the central aortic pressure measurement technique.

Prior to CPB the systolic, diastolic, and mean gradients were insignificant. Five minutes after CPB there was a significant (P < 0.05) systolic (12.4 ± 1.4 mmHg), diastolic (2.4 ± 0.5 mmHg), and mean (4.9 ± 0.9 mmHg) pressure gradient between aorta and radial artery (table 2). There was no change in the pressure gradient at 5 and 10 min post-CPB. A clinically significant systolic gradient (≥ 10 mmHg) post-CBP occurred in 19 of 30 patients and was as great as 34 mmHg. Likewise, a clinically significant MAP gradient (≥ 5 mmHg) occurred in 18 of 30 patients and was as great as 15 mmHg.

There was a statistically insignificant gradient between MAPa and MAPa pre-CBP (0.8 ± 0.2 mmHg). During early CPB, the MAPr and MAPa became significantly different (4.9 ± 0.7 mmHg) and remained significantly different throughout the study (fig. 1). Furthermore, the difference between MAPr and MAPa did not change significantly during midbypass (4.2 ± 0.5 mmHg), rewarming (4.8 ± 0.7 mmHg), immediately before discontinuation of CPB (4.6 ± 0.7 mmHg), or 5 and 10 min after discontinuation of CPB (4.9 ± 0.9 and 4.8 ± 0.7 mmHg). A clinically significant MAP gradient (≥ 5 mmHg) during CPB occurred in 23 of 30 patients and was as great as 16 mmHg.

Twenty of 30 patients met the criteria required to receive sodium nitroprusside and 18 of 30 to receive phenylephrine during mid-CPB. Twenty patients received phenylephrine during rewarming and were evaluated separately. Sodium nitroprusside decreased MAP, from 88.2 ± 1.6 to 75.5 ± 1.9 mmHg, resulting in an 15 ± 2%

### Table 1. Temperature, Hematocrit, and Cardiac Output Associated with Cardiopulmonary Bypass

<table>
<thead>
<tr>
<th>Period</th>
<th>Temperature (°C)</th>
<th>Hematocrit (%)</th>
<th>Cardiac Output/Pump Flow (l/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-CBP</td>
<td>35.4 ± 0.1</td>
<td>40 ± 2</td>
<td>4.1 ± 0.4</td>
</tr>
<tr>
<td>Early CPB</td>
<td>28.1 ± 0.1</td>
<td>24 ± 1</td>
<td>4.7 ± 0.2</td>
</tr>
<tr>
<td>Mid-CBP</td>
<td>28.5 ± 0.2</td>
<td>24 ± 1</td>
<td>4.9 ± 0.2</td>
</tr>
<tr>
<td>Rewarm</td>
<td>33.9 ± 0.2</td>
<td>24 ± 1</td>
<td>5.5 ± 0.2</td>
</tr>
<tr>
<td>Before separation</td>
<td>36.9 ± 0.1</td>
<td>24 ± 1</td>
<td>5.5 ± 0.2</td>
</tr>
<tr>
<td>5 min post-CPB</td>
<td>37.0 ± 0.2</td>
<td>24 ± 1</td>
<td>5.6 ± 0.4</td>
</tr>
<tr>
<td>10 min post-CPB</td>
<td>36.9 ± 0.2</td>
<td>24 ± 1</td>
<td>5.5 ± 0.3</td>
</tr>
</tbody>
</table>

CPB = cardiopulmonary bypass.
Table 2. Aortic–Radial Artery Pressure Gradient Before, During, and After Cardiopulmonary Bypass

<table>
<thead>
<tr>
<th>Period</th>
<th>Mean (mmHg)</th>
<th>Systolic (mmHg)</th>
<th>Diastolic (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-CBP</td>
<td>0.81 ± 0.16</td>
<td>−2.25 ± 0.6</td>
<td>1.67 ± 0.7</td>
</tr>
<tr>
<td>Early CPB</td>
<td>4.92 ± 0.68*</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Mid-CPB</td>
<td>4.19 ± 0.51*</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Rewarm</td>
<td>4.77 ± 0.68*</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Before separation</td>
<td>4.62 ± 0.72*</td>
<td>12.4 ± 1.4*</td>
<td>2.4 ± 0.5*</td>
</tr>
<tr>
<td>5 min post-CBP</td>
<td>4.88 ± 0.91*</td>
<td>11.8 ± 1.4*</td>
<td>2.4 ± 0.5*</td>
</tr>
<tr>
<td>10 min post-CBP</td>
<td>4.61 ± 0.71*</td>
<td>−</td>
<td>−</td>
</tr>
</tbody>
</table>

Mean ± SEM.

* Significantly (P < 0.05) different than pre-CBP but not from each other.

decrease in SVR. During midbypass, phenylephrine increased MAP, from 50.0 ± 2.4 mmHg to 74.4 ± 2.2 mmHg, resulting in a 52 ± 6 increase in SVR. Likewise during rewarming, administration of phenylephrine increased MAP, from 48.1 ± 1.1 mmHg to 63.8 ± 2.3 mmHg, resulting in a 34 ± 4% increase in SVR. Despite these changes in vascular resistance, there was no change in the pressure gradient resulting from intravenous injection of sodium nitroprusside or phenylephrine (fig. 2). Statistically, there was a 95% probability of detecting a 1.4–1.5-mmHg change in the pressure gradient after phenylephrine and sodium nitroprusside and a 75% probability of detecting a 1.0–1.2-mmHg change.

Discussion

It is well accepted that there can be a clinically significant difference between aortic and radial artery pressure upon discontinuation of CPB. Previously, most investigators have believed that this gradient develops during rewarming from CPB. We demonstrated that a significant MAP gradient develops in early CPB and does not change throughout the bypass course, including during rewarming or discontinuation of bypass. Furthermore, the pressure gradient was unaffected by changes in SVR secondary to intravenous boluses of phenylephrine or sodium nitroprusside.

Stern et al. demonstrated a systolic aortic-to-radial pressure gradient that could be partially explained by decreased forearm vascular resistance post-CPB.1 Pauca et al.2 and Pauca and Meredith3 suggested that arterial venous shunting in the hand may also contribute to the gradient. These studies suggested that upper-extremity arteriolar vasodilation upon rewarming was responsible for the aortic-to-radial artery pressure gradient. In contrast, we found that a MAP gradient developed in early CPB well before rewarming and the associated decrease in arm and hand vascular resistance. Furthermore, phenylephrine increased SVR during the middle of CPB and rewarming, which should increase arm and hand vascular resistance, but did not affect the MAP gradient.

Maryama et al. suggested that infusion of vasodilators (nitroglycerin and nicardipine) during cardiac surgery may be responsible for the pressure gradient in the immediate post-bypass period.5 In contrast, we demonstrated that the pressure gradient developed well before infusion of vasodilators. Second, vasodilation, induced by sodium nitroprusside during CPB, decreased SVR but did not affect the pressure gradient.

Stern et al.1 and Gravlee et al.7 indicated that arm and hand vasodilatation, by itself, could not account entirely for the pressure gradient. Mohr et al. attributed the pressure gradient, in part, to peripheral vasoconstriction.4 A decrease in artery size or vasoconstriction may greatly increase

* MAP difference significant to p < 0.05
arm vascular resistance and thus increase the pressure gradient. Initiation of CPB, which is associated with cooling, nonpulsatile flow, and catecholamine release may cause vasospasm or decreased artery size. Because the MAP gradient developed in early CPB, our study suggests that this mechanism may be largely responsible for the pressure gradient. However, we cannot conclude from this study that this is the etiology of the larger systolic pressure gradient. Furthermore, it is unclear if hemodilution associated with the onset of CPB contributes to the pressure gradient.

The aortic-to-radial artery pressure gradient may be clinically significant. Stern et al. reported post-CPB systolic pressure gradients as great as 32 mmHg. In our study the mean and systolic pressure gradients were as great as 15 and 34 mmHg, respectively, post-CPB. The mean gradient was also significant during CPB and therefore may affect clinical decisions not only upon discontinuation of bypass but also during CPB. Other investigators have demonstrated that the pressure gradient may be partially eliminated by monitoring arterial pressure more centrally in the femoral or brachial artery.\(^7\)\(^5\) We found central aortic pressure monitoring to be simple, safe, and identical to the common iliac artery pressure.

In conclusion, we demonstrated that a mean aortic-to-radial artery pressure gradient develops in early CPB rather than after rewarming or upon discontinuation of CPB. Furthermore, the pressure gradient is not affected by SVR changes induced by phenylephrine or sodium nitroprusside. Although the mechanism remains unknown, this study suggests the etiology of the mean pressure gradient may involve events associated with initiation of CPB.

The authors thank Carl Lynch, III, M.D., Ph.D., Carol L. Lake, M.D., C. Michael Hahn, M.D., Mark Uggeri, M.D., D.D.S., Steve Roberts, M.D., Irving Kron, M.D., J. Milton Adams, Ph.D., and Curtis Tribble, M.D. for their assistance and input.

References