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Differences Between Aortic and Radial Artery Pressure Associated with Cardiopulmonary Bypass

George F. Rich, M.D., Ph.D., Robert E. Lubanski, Jr., M.D.,† Thomas M. McLoughlin, M.D.†*

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 32 mmHg,[1] 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 arm[1] and hand[2,3] vascular resistance. Peripheral vasoconstriction, low blood volume, and proximal shunting have also been suggested as a possible mechanism.[4] Continuous infusion of vasodilators during CPB also appears to cause an increased femoral-to-radial artery pressure gradient in the postbypass period.[5]

Bazaral *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.[6] 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 $\mu g \cdot kg^{-1} \cdot min^{-1}$) 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

* Assistant Professor of Anesthesiology and Biomedical Engineering.
† Resident in Anesthesiology.

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Address reprint requests to Dr. Rich: University of Virginia Health Sciences Center, Department of Anesthesiology, Box 238, Charlottesville, Virginia 22908.

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 T4812ADR®; 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 (MAP_r) and mean aortic (MAP_a) 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 MAP_r and MAP_a 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 MAP_r decreased to below 55 mmHg, phenylephrine (7 μg/kg intravenously) was administered. If MAP_r increased to above 85 mmHg, sodium nitroprusside (1 μg/kg intravenously) was administered. The MAP_r and MAP_a 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 MAP_r, 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-CPB 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 MAP_r and MAP_a pre-CPB (0.8 ± 0.2 mmHg). During early CPB, the MAP_r and MAP_a became significantly different (4.9 ± 0.7 mmHg) and remained significantly different throughout the study (fig. 1). Furthermore, the difference between MAP_r and MAP_a 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_r from 88.2 ± 1.6 to 75.5 ± 1.9 mmHg, resulting in an 15 ± 2%

TABLE 1. Temperature, Hematocrit, and Cardiac Output or Pump Flow Associated with Cardiopulmonary Bypass

Period	Temperature (°C)	Hematocrit (%)	Cardiac Output/Pump Flow (l/min)
Pre-CPB	35.4 ± 0.1	40 ± 2	4.1 ± 0.4
Early CPB	28.1 ± 0.1	24 ± 1	4.7 ± 0.2
Mid-CPB	28.5 ± 0.2	24 ± 1	4.9 ± 0.2
Rewarm	33.2 ± 0.1	24 ± 1	5.5 ± 0.2
Before separation	36.9 ± 0.1	24 ± 1	5.5 ± 0.2
5 min post-CPB	37.0 ± 0.2	24 ± 1	5.6 ± 0.4
10 min post-CPB	36.9 ± 0.2	24 ± 1	5.5 ± 0.3

CPB = cardiopulmonary bypass.

TABLE 2. Aortic–Radial Artery Pressure Gradient Before, During, and After Cardiopulmonary Bypass

Period	Mean (mmHg)	Systolic (mmHg)	Diastolic (mmHg)
Pre-CPB	0.81 ± 0.16	−2.25 ± 0.6	1.67 ± .7
Early CPB	4.92 ± 0.68*	—	—
Mid-CPB	4.19 ± 0.51*	—	—
Rewarm	4.77 ± 0.68*	—	—
Before separation	4.62 ± 0.72*	—	—
5 min post-CPB	4.88 ± 0.91*	12.4 ± 1.4*	2.4 ± 0.5*
10 min post-CPB	4.81 ± 0.71*	11.8 ± 1.4*	2.4 ± 0.5*

Mean ± SEM.

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

decrease in SVR. During midbypass, phenylephrine increased MAP_r 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_r 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 Meredith[3] 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.

Maruyama *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 vasodilation, 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 vasospasm may greatly increase

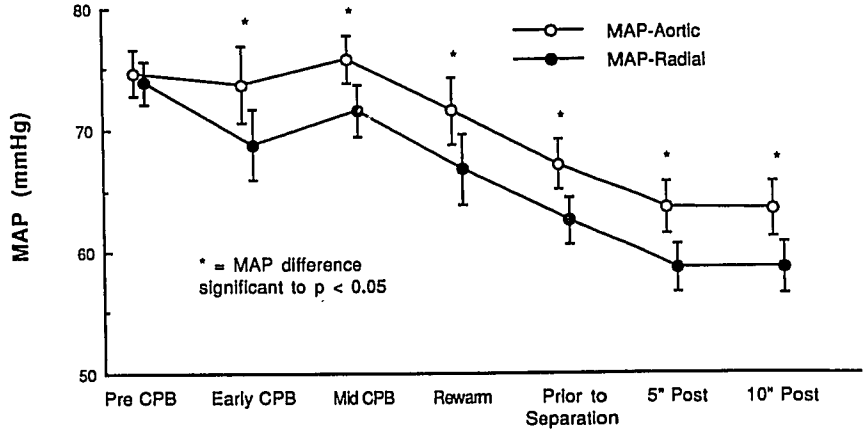

FIG. 1. Comparison of mean aortic and mean radial artery pressures associated with cardiopulmonary bypass (CPB) (mean ± SEM).

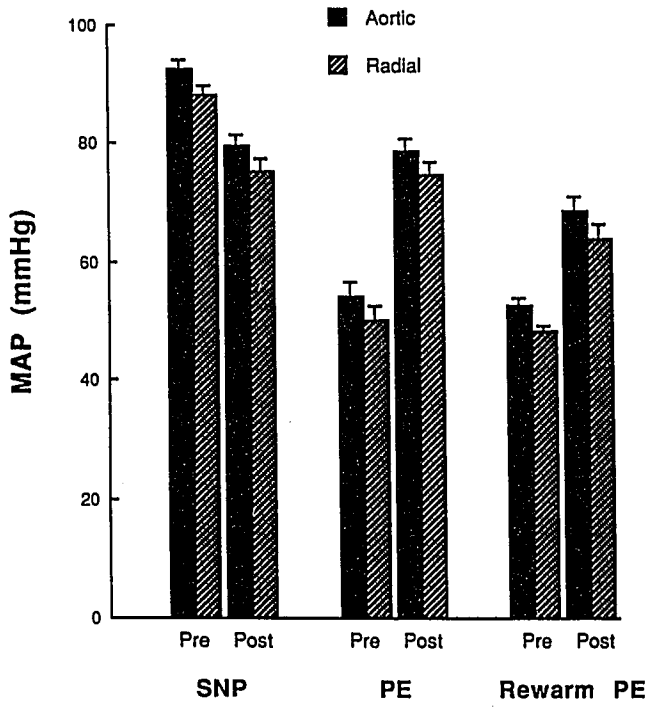

FIG. 2. Comparison of mean arterial pressures (MAP) before and after drug administration during cardiopulmonary bypass (mean ± SEM). SNP = sodium nitroprusside; PE = phenylephrine.

arm vascular resistance and thus increase the pressure gradient. Initiation of CPB, which is associated with cooling, nonpulsatile flow, and catecholamine release[10] 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.*[1] 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.[7–9] 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.

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