Possibility of A-V Shunting Upon Cardiopulmonary Bypass Discontinuation

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Measurements of arterial blood pressure during anesthesia are most commonly obtained from the radial or brachial artery; however, on discontinuation of cardiopulmonary bypass (CPB), the blood pressure measured at either of these sites is often considerably lower than that measured either in the ascending aorta or the femoral artery; that discrepancy may persist for 10 min to 1 h. Although the most popularly accepted, albeit unproven, cause of this temporary phenomenon is vascular spasm, the unexpected availability of a patient with bilateral arteriovenous (A-V) fistulae, who was in need of standard invasive monitoring during cardiopulmonary bypass (CPB), allowed us to recognize an alternative mechanism—reduction in vascular resistance. This mechanism had been suggested by Stern et al., who, however, came to the conclusion that "reduced forearm vascular resistance is an incomplete explanation for the change in the relationship between aortic and radial systolic pressure."

To study this problem further, we compared brachial arterial and aortic blood pressure in the patient with the A-V fistulae with brachial arterial and aortic blood pressures in two other patients without a fistula immediately after removal of the CPB.

METHODS AND RESULTS

All data from these three patients were recorded on a thermal recorder. The same transducer and an equivalent length of tubing was used for all sets of comparison.

The blood pressure monitoring system was balanced and calibrated statically to a mercury standard before and after each observation. Its natural frequency and damping coefficient were determined at the end of the recording by the flush method, as described by Gardner. The mean natural frequency of the system when used in the radial, brachial, or aortic sites was 19 (range 14–25) Hz; the mean damping coefficient was 0.22 (range 0.12–0.28). Occlusion of the distal radial or brachial arteries did not change the frequency response to a measurable extent. The mean arterial blood pressure was obtained by electronic integration.

Case 1 (A-V fistula patient). This 57-yr-old man had been receiving alternate-day dialysis for 2 yr because of renal failure. He had radial artery-caphalic vein shunts in both forearms. In preparation for a 3-vessel coronary artery bypass graft, his left brachial artery was cannulated for temporary monitoring of arterial blood pressure; insertion of a femoral line after completion of the surgical preparation was planned. Ten minutes after all preanesthetic preparations had been completed and before induction of anesthesia, the brachial artery blood pressures, unoccluded and occluded (distal to the monitoring cannula, proximal to the shunt), were obtained within a 50-s interval (table 1, column 1). As can be seen in figure 1 and table 1, occlusion of the brachial artery increased the systolic, diastolic, and mean blood pressures. A second set of arterial blood pressures was obtained after induction of anesthesia (table 1, column 2).

After cannulation of the aorta, blood pressures were recorded directly from the ascending aorta through the side-port of the aortic cannula and from the brachial artery without and with occlusion (table 1, column 3). These blood pressures were taken consecutively with the same transducer within a total period of 45 s.

Case 2. A 55-yr-old man underwent four-vessel coronary artery bypass grafting. At the end of CPB, his brachial systolic arterial blood pressure fluctuated between 95 and 100 mmHg; his blood gas levels and acid-base balance were normal. Digital compression to occlusion of the brachial artery distal to the cannulated site caused the change shown in figure 2. All readings were taken within periods of 5 s each, except where the mean blood pressures were recorded, and those were obtained within periods of 30 s each. Thirty minutes later (table 1, column 5), the discrepancy in arterial blood pressure readings with occlusion was still present, the mean discrepancy being 9 mmHg. In the intensive care unit, 90 min after discontinuation of CPB, compression of this patient's forearm caused only minimal changes in arterial blood pressure.

Case 3. A 66-yr-old woman underwent four-vessel coronary artery bypass grafting. Her arterial blood pressures are shown in table 1; each comparison was made over a period of 5 s, and included blood pressures taken from the ascending aorta through the side-port of the aortic cannula. The first comparison (column 1) was made 4 min after CPB was discontinued and the last (column 5) was made 12 min later. Twenty-six minutes after discontinuation of CPB, there were no differences in arterial blood pressure when the distal brachial artery was occluded.

Other Cases. We have observed the effect of arterial occlusion distal to the cannulated site of brachial or radial arteries in 15 additional

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patients who showed higher aortic than peripheral arterial blood pressures at the termination of CPB. The diastolic arterial blood pressure increased 2 mmHg in two of the 15 patients; the systolic arterial blood pressure increased 2–25 mmHg in all patients; and the mean blood pressure measured in the radial artery increased 3–5 mmHg in three of seven patients. The difference between the mean blood pressure measured in the aorta and brachial artery decreased from 8 to 3 mmHg in four of the eight patients as the blood pressure in the brachial artery increased. Occlusion distal to the radial artery cannulation was accomplished by manual circumferential compression of the wrist over the hub of the cannulating catheter. These cases were selected from a large number of cases in which aorta-peripheral artery blood pressure differences were present, and were the only ones with records complete enough to show the differences clearly.

**DISCUSSION**

Although arterial blood pressure is not commonly measured in the brachial artery during CPB, our clinical impression that a brachial artery-aorta pressure difference at discontinuation of CPB is less common than a radial artery-aorta pressure difference has led us, over the last 7 yr, to use this monitoring site in approximately 20% of our CPB patients. This local practice permitted us to compare the effect of compression of the brachial artery distal to the cannulated site in patients after discontinuation of CPB with that effect in a patient with end-stage renal failure when his brachial artery was compressed distal to the monitoring site and proximal to his arteriovenous shunt.

Our findings suggest that the discrepancy between the aortic blood pressure and the blood pressure measured in the radial or brachial arteries at the discontinuation of CPB is due to a regional decrease in vascular resistance. However, the changes produced in the arterial blood pressure tracing when distal compression was applied to the cannulated artery could also have been produced by the summation of the pulse waves generated by the systolic contractions of the heart, and those that result from their reflection at the end of the occluded artery.

Wave summation is a term introduced by Wiggers in order to explain why the systolic blood pressure found in the femoral and dorsalis pedis arteries is sometimes higher than that at the root of the aorta, and why the peak systolic blood pressure in the radial artery is restored to a level equal to that measured at the root of the aorta: the pulse pressure introduced in the aorta during its transmission to the branching arteries loses its finer oscillations, its rate of pressure elevation is retarded, and its final peak pressure is reduced by damping of the fundamental wave. At the capillary level, however, there is a sudden increase in the resistance to flow, and a reflected pressure wave is produced, one that combines with the fundamental wave, producing, at some distance from the aortic root, a systolic arterial blood pressure peak equal to or higher than that origi-

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**TABLE 1. Comparison of Perioperative Aortic and Artery Blood Pressures in Three Patients Undergoing Cardiopulmonary Bypass**

<table>
<thead>
<tr>
<th>Artery</th>
<th>Column 1</th>
<th>Column 2</th>
<th>Column 3</th>
<th>Column 4</th>
<th>Column 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td></td>
<td>127/72 (94)</td>
<td>97/55 (72)</td>
<td>110/65 (80)</td>
<td></td>
</tr>
<tr>
<td>Free brachial</td>
<td></td>
<td>162/82 (114)</td>
<td>125/60 (80)†</td>
<td>150/75 (105)†</td>
<td></td>
</tr>
<tr>
<td>Occluded brachial</td>
<td></td>
<td>152/75 (107)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascending aortic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 2</td>
<td></td>
<td>99/62</td>
<td>95/61</td>
<td>96/60</td>
<td>100/62</td>
</tr>
<tr>
<td>Free brachial</td>
<td></td>
<td>116/64‡</td>
<td>120/67‡</td>
<td>115/65§</td>
<td>124/69§</td>
</tr>
<tr>
<td>Occluded brachial</td>
<td>90/49</td>
<td>110/49§</td>
<td>110/50§</td>
<td>115/61§</td>
<td>115/52§</td>
</tr>
<tr>
<td>Ascending aortic</td>
<td>110/49</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Numbers in parentheses are mean blood pressures; see case reports for the exact time at which a measurement was made.
† Digital compression proximal to shunt.
‡ Digital compression of brachial artery.
§ Manual circumferential compression of forearm.

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**FIG. 1. Brachial artery blood pressure recording from case 1. Occlusion of the brachial artery distal to the intraarterial cannula and proximal to the arteriovenous fistula produced the changes seen in the blood pressure recording. Paper speed 5 mm/s.**
nally introduced into the aorta. Augmentation of the systolic blood pressure by wave summation has been confirmed by other investigators at the levels of the abdominal aorta and the iliac arteries.\(^5\) Relevant to this presentation is the finding by Van Bergen et al.\(^5\) that distal occlusion of the brachial artery increases the systolic arterial blood pressure measured proximally by wave summation. In contrast to that finding, distal compression of the monitored artery increased the diastolic and mean arterial blood pressure, as well as the systolic blood pressure, in our patient with an A-V fistula and in the patient in case 2. Van Bergen et al. did not measure the aortic pressure, and their patients did not have a downstream shunt, but their results are the only other explanation for our finding of an increase in brachial arterial blood pressure by distal occlusion.

The similarity of changes in arterial blood pressure seen during occlusion of the A-V fistula, and those seen at discontinuation of CPB during distal occlusion of the cannulated artery, suggests that the changes produced by distal compression of the brachial artery in the patient in case 2, in seven of the 15 additional patients, and in the patient with the A-V fistula could be of the same nature. The changes seen in the patient in case 3 and the other eight of the 15 additional patients, where distal compression of the cannulated artery increased only the systolic arterial blood pressure without altering the mean arterial blood pressure, are most likely a wave summation phenomenon.

Although the relationship between blood pressure and blood flow in the arterial system is not linear, at the capillary level, where pressure waves can be ignored, Ohm’s law can be followed, and mean arterial blood pressure is proportional to the product of blood flow times the peripheral vascular resistance. Peripheral vascular resistance at the capillary level separates the arterial and venous blood pressure systems by restricting blood flow, just as the floodgate of a water dam restricts the flow of water and maintains a water level, or pressure.\(^6\) We can see that, if pressure equals resistance times flow, pressure must fall when resistance falls. Although this simplistic assumption ignores compensations, such as the marked increase in blood flow that occurs when resistance decreases and the increased role of reactive impedance that occurs proximal to the cannulated site when the distal peripheral vascular resistance (resistive impedance) is markedly reduced, it does help us to understand the low arterial blood pressure that is seen proximal to an A-V fistula. The high resistance to blood flow that normally separates arteries and veins has been removed, and the normally higher arterial blood pressure will tend to approximate the blood pressure found in the veins. Occlusion of the shunt will restore peripheral vascular resistance, separating the arterial blood pressure from the venous blood pressure. Severe arteriolar dilation in the hand and forearm regions could remove, to varying degrees, the resistance that separates the arterial circulation from the venous circulation, similar to the effect of an A-V fistula.

The study by Stern et al.\(^1\) on post-CPB aorta-radial artery blood pressure differences showed those differences to correlate inversely with the forearm vascular resistance. However, statistically, the correlation coefficient was only 0.49, or a coefficient of determination of 0.24, implying that only 24% of the central-to-peripheral blood pressure difference could be explained by a decrease in forearm vascular resistance. We speculate that, had those authors measured the hand arterial blood flow rather than the forearm arterial blood flow, they would have found a larger correlation between the blood pressure gradient and the reduction in hand vascular resistance.

Our observations in these case reports suggest that the aorta-brachial artery and aorta-radial artery blood pressure differences seen at the conclusion of CPB could be due to a decrease in peripheral vascular resistance of varying degrees. In mild cases, only the generation of the wave summation phenomenon would be impaired, yielding brachial systolic blood pressures lower than the blood pressure seen in the ascending aorta; additionally, since the systolic blood pressure in the radial artery is normally dependent on the wave summation phenomenon, a minor decrease in peripheral resistance would unmask the lower systolic blood pressure at the radial artery. In more severe cases, the production of an A-V fistula-type phenomenon would be reflected by the lower systolic and mean radial and brachial arterial blood pressures.

Further investigations are needed to better define and explain the apparent A-V shunting seen after CPB. A related question is whether vasodilators produce an aorta-peripheral artery blood pressure gradient. Besides finding the cause of the aorta-peripheral pressure gradient on discontinuation of CPB, we hope that the...
following related question can be answered: Is there also an aorta-peripheral blood pressure gradient when hypotensive anesthesia is produced by vasodilators?

REFERENCES


Impaired Myocardial Conduction in Patients Receiving Dilution Therapy during Enflurane Anesthesia


Drug interactions continue to be a potential cause of anesthetic-related morbidity and mortality, particularly when they involve the cardiovascular system. The potential for adverse effects on myocardial conduction in patients who are anesthetized with inhaled anesthetics while taking calcium channel blockers has been addressed previously. In animals, both groups of drugs depress SA node function and prolong atrioventricular conduction. There have been no clinical reports, however, of adverse effects on myocardial conduction in patients taking calcium channel blockers while undergoing anesthesia with inhaled anesthetics. This report describes two cases in which atrioventricular conduction and sinus node automaticity were altered in patients receiving diluzem while undergoing enflurane anesthesia. The first patient demonstrated impaired atrioventricular and sinus node function prior to anesthesia. Enflurane further impaired atrioventricular nodal conduction, and this effect was readily reversible both by discontinuing the anesthetic and by initiating surgical stimulation. The second patient demonstrated severe sinus bradycardia progressing to asystole following administration of enflurane.

REPORT OF CASE 1

A 69-yr-old man with a history of angina and shortness of breath on exertion was scheduled for mitral valve replacement and aortocoronary vein bypass grafting. He had angiographic evidence of stenosis of the left anterior descending and right coronary arteries, and moderate mitral regurgitation. His ejection fraction was 55%, and the ventricular wall motion was normal. Preoperative medications included diluzem 120 mg t.i.d., atenolol 50 mg q.d., and hydrocortisone 50 mg q.d. The admission electrocardiogram (ECG) revealed a sinus bradycardia of 47 bpm, PR interval of 0.24 s, and QRS of 0.07 s duration. The usual morning dose of diluzem and atenolol were given to the patient, who was then given lorazepam 2 mg and meperidine 75 mg im 1 h preoperatively. During preparation for anesthesia and surgery, ECG leads were applied, two peripheral intravenous lines were inserted, and the radial artery was cannulated. Arterial blood pressure (BP) was 120/70 mmHg and the heart rate (HR) was 35 bpm. The ECG showed sinus bradycardia. A pacing thermocellulation balloon-tipped pulmonary artery catheter was inserted via the right internal jugular vein, and the pulmonary capillary wedge pressure (PCWP) was 20 mmHg. Atrial and ventricular pacing was achieved with a diastolic threshold between 5-10 mamps. The pacing rate was increased by 5 bpm every 10 beats until Wenckebach periodicity occurred (i.e., the heart rate at which heart block of the Mobitz type 1, first occurred) (fig. 1). This happened at 90 bpm (BP during this maneuver was 140/70 mmHg). Atrial pacing was then set at 50 bpm and the BP stabilized at 120/60 mmHg. Anesthesia was induced with fentanyl 25 µg/kg iv, lorazepam 2 mg iv, and neuromuscular blockade was achieved with pancuronium .15 mg/kg iv. Three minutes later, the

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