原 著

Hemodilution Has an Important Role in Femoral-to-Radial Artery Pressure Gradient after Cardiopulmonary Bypass

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Abstract

We evaluated the relation between hematocrit values and femoral-to-radial artery pressure differences in patients undergoing coronary artery bypass graft surgery to confirm whether the reduction in hand vascular resistance is caused by hemodilution. Anesthesia management and monitoring followed the standard method for cardiac surgery. Radial and femoral artery pressures through catheters were measured using disposable transducers and a monitoring system. Hematocrit and blood viscosity from the femoral artery were measured before and after CPB, and radial and femoral artery pressures were recorded simultaneously. Spearman rank regression analysis was performed between changes of hematocrit from the baseline values (ΔHt) and systolic, mean and diastolic values of femoral-to-radial artery pressure differences (ΔP). ΔHt and ΔP of systolic arterial pressure showed a good correlation with a correlation coefficient (r) of 0.716. Although there was a correlation between ΔHt and ΔP of mean arterial pressure ($r=0.617$), no correlation was obtained between ΔHt and ΔP of diastolic arterial pressure. There was correlation between blood viscosity and either ΔP of systolic or mean arterial pressures ($r=-0.810$ and -0.713, respectively). These results indicate that hemodilution has an important role responsible for the femoral-to-radial artery pressure gradient after CPB.

Key words: Hemodilution, Hematocrit, Viscosity, Femoral-to-radial artery pressure gradient, Cardiopulmonary bypass

Introduction

Numerous investigators have reported discrepancies between radial and aortic blood pressure during rewarming and after cardiopulmonary bypass (CPB) (1-5). Although the radial artery is the most common site for intraarterial blood pressure monitoring, it may not reflect aortic pressure during and after CPB. Pauca et al. reported that following CPB, the lower radial SAP and MAP than aortic SAP and MAP seem to be related to a decrease in hand vascular resistance, because restoring that resistance by wrist compression increased the peripheral pressures (4). Peripheral vasoconstriction, low blood volume, and proximal shunting have also been suggested as a possible mechanism (2). Urzua proposed that purely passive hydraulic factors secondary to hemodilution would explain the decrease in peripheral resistance and the increase in proximal arterial resistance underlying the aortic-to-radial artery pressure drop (6). The aim of this study is to assess the role of hemodilution during CPB in radial arterial hypotension following CPB.

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Methods

The study protocol was approved by the institutional human investigation committee, and informed consent was obtained from 20 patients. All patients received morphine 7 to 10 mg and scopolamine 0.4 mg as a premedication. Anesthesia was induced and maintained with fentanyl 88－120 μg/kg in oxygen and air (FIO_2 of 0.4). Vecuronium was used for muscle relaxation. Throughout anesthesia, trinitroglycerin 0.2 μg/kg/min or prostaglandine E_1 0.02 μg/kg/min was used as vasodilators. Dopamine and/or dobutamine 3－5 μg/kg/min was used as needed after CPB.

Patients monitoring included pulmonary artery, central venous, radial and femoral artery pressures, electrocardiogram (lead II and V_5), temperatures (forehead, palm, esophageal and bladder), SPO_2 and $ETCO_2$. Radial cannulation was performed using a 3.2 cm 20 gauge teflon cannula in the either right or left hand under local anesthesia before induction of anesthesia. A disposable transducer (Transpac, Abott Critical Care Systems, U. S. A.) which has the mean natural frequency of 36.8 Hz, and a damping coefficient of 0.13, and a monitor (M1166 A, Hewlett Packard, U. S. A.) were used for measurement of radial artery pressure. After induction of anesthesia a 7.5 cm 19 gauge teflon cannula was inserted into the femoral artery, and connected to the same type of pressure transducer and the monitor for measurement of femoral artery pressure. Hematocrit and blood viscosity (OP-Reometer, Iwamoto Seisaku, Kyoto), and simultaneous femoral and radial blood pressures were measured before and 10, 30, 60 and 90 or 120 min after CPB. Palm (peripheral) and forehead (core) temperatures were compared to evaluate peripheral circulation.

Spearman rank regression analyses were performed between changes in hematocrit from the baseline (Δ Ht) and femoral-to-radial artery pressure differences (Δ P) for SAP, MAP and DAP. Regression analysis was also performed between blood viscosity values and Δ P changes in five patients. A p-value less than 0.05 considered significant.

Results

The 20 patients were ASA physical status 3 or 4 aged 61 ± 6 yr, weighing 61 ± 5 kg, with 2 to 4 coronary vessel disease undergoing coronary artery bypass surgery. The mean CPB time was 169 ± 45min. All patients were included in the evaluation of pressure gradients before and after CPB.

Representative recordings of radial, femoral arterial pressures along with ECG and pulmonary artery pressure from one patient are shown in Figure 1. Although radial SAP was slightly higher than femoral SAP before CPB, femoral SAP was higher than radial SAP after CPB. When hematocrit values returned near to the baseline value, radial SAP became close to femoral SAP.

Regression analyses between Δ Ht and Δ P in 78 paired data for SAP and MAP are show in figure 2. Although there were significant correlations for SAP (Spearman rank correlation coefficient (r) of 0.716; $P < 0.01$) and for MAP ($r = 0.617$; $P < 0.01$) ,there was no significant correlation for DAP. There were also significant correlations between blood viscosity and SAP ($r = -0.810$, $P < 0.01$), and MAP ($r = -0.713$) as show in figure 3. There was only a small difference between forehead and palm temperatures after CPB even when radial artery pressure was low as shown in figure 4.

Discussion

It is well documented that there can be a clinically significant difference between aortic and radial artery pressure after CPB. Most investigators have reported that this gradient develops during rewarming from CPB. We demonstrated that there were significant differences between radial and femoral artery pressure for SAP and MAP after CPB. SAP and MAP gradients were as great as 50 and 15mmHg after CPB. These differences may be clinically significant and may affect decisions for patients management. Gravlee et al. recommended that aortic or femoral artery pressure should be checked before administering vasopressors or in-

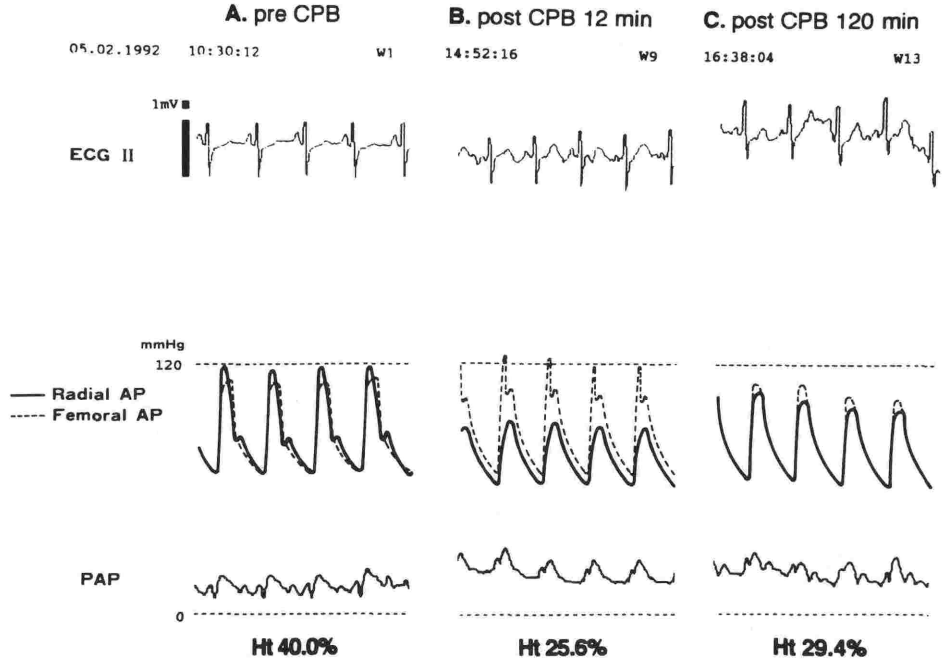

Figure 1. Representative tracings of radial and femoral artery pressures together with electrocardiogram and pulmonary artery pressure before and after cardiopulmonary bypass.

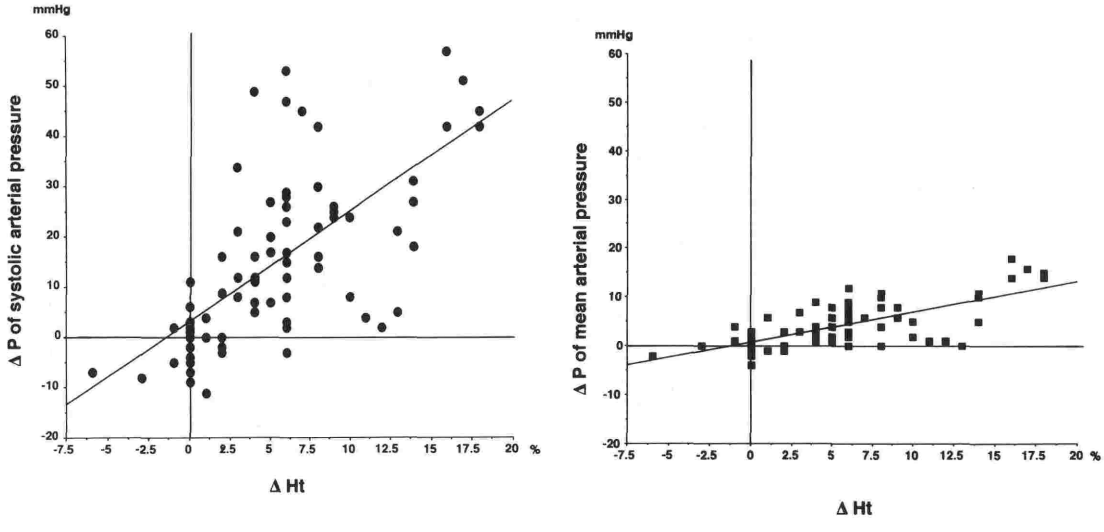

Figure 2. Relashionship between changes of hematocrit (Δ Ht) and femoral-to-radial artery pressure differences (Δ P) for systolic and mean arterial ppressures. There were positive correlations between Ht and femoral-to-radial pressure gradients for systolic and mean pressures.

otropes to treat hypotension observed in the radial artery, because radial artery pressure underestimated aortic SAP and MAP by 10min after CPB (8,9). In our institute cardiac surgeons routinely insert a cannula into the femoral artery on our request, and this is used for pressure measurement and as a route for an intra-aortic balloon pump if needed.

It is still controversial on the etiology of the aortic-to-radial or femoral-to-radial pressure gradient following CPB. Stern et al. found only a weak correlation between the gradient and a decrease in forearm vascular resistance (1). Pauca and Meredith reported that a decrease in vascular resistance distal to the monitoring site was the cause of lower brachial artery pressure after CPB, because the arteriovenous communication had a role of lower brachial artery pressure (3). Pauca et al. showed

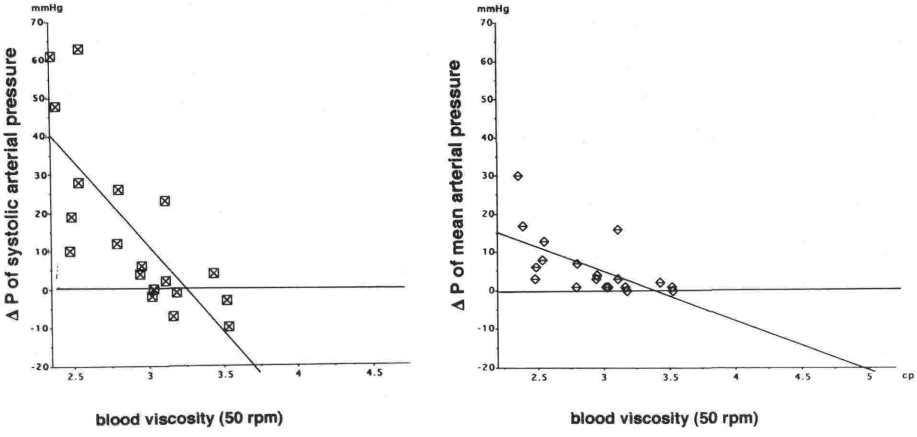

Figure 3. Relashionship between viscosity changes and femoral-to-radial artery pressure differences for systolic and mean arterial pressures. There were negative correlations between blood viscosity and femoral-to-radial pressure gradients for systolic and mean pressures.

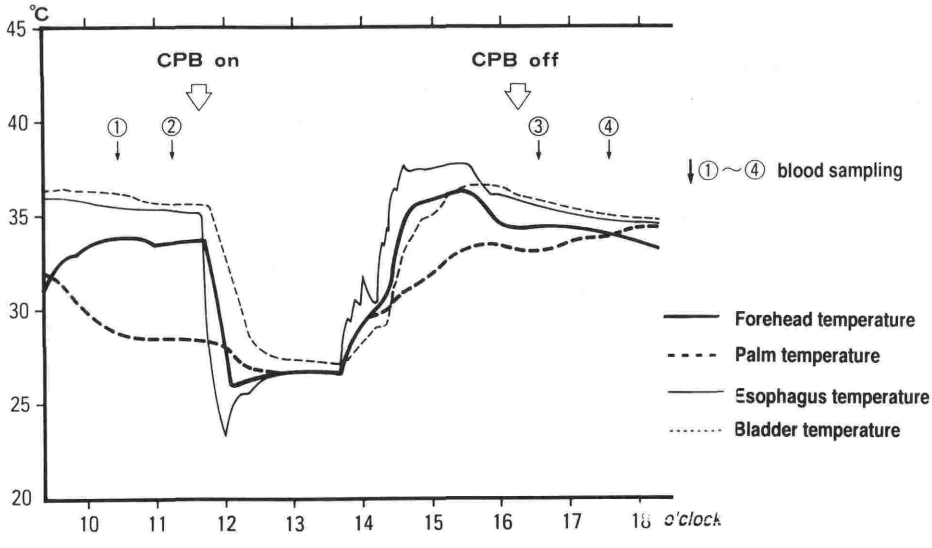

Figure 4. Temperature changes before, during and after cardiopulmonary bypass. Points of measurements are also shown.

that the radial-to-aortic arterial pressure gradient following CPB was restored by wrist compression, and they concluded that a decrease in hand vascular resistance seemed to explain the mechanism of the gradient (4).

We should answer why hand vascular resistance was deceased after CPB. Gordon et al. reported that there was a good correlation between the extent of pressure reduction in the radial artery and the decrease in blood viscosity by hemodilution, though they measured arterial pressure only at the radial artery (7). Urzua stated that passive hydraulic factors secondary to hemodilution would explain the decrease in peripheral vascular resistance (6). Hemodilution during CPB to a hematocrit around 20% reduces blood viscosity, and such viscosity reduction should decrease arteriolar resistance to approximately one half of normal, according to the Poiseuille equation. Due to the peculiar characteristics of blood, such reduction could be more marked at the microcirculation level. Total arterial resistance to follow results from the sum of a proximal resistance related to the size of the tube, and a peripheral resistance determined by arterioles, capillaries, and blood viscosity. Peripheral resistance and distal pressure decrease markedly in the presence of hemodilution. Further, because the arterial diameter is consequently reduced, proximal resistance increases, and a pressure gradient originates between the aortic and the distal pressures under hemodilution.

We clearly demonstrated that there were good correlations between the reduction of hematocrit and femoral-to-radial pressure differences for SAP and MAP. Further, there were negative correlations between blood viscosity and the femoral-to-radial pressure difference for SAP and MAP. Our results proved Urzua's hypothesis in clinical settings. Decreases in hand and forearm vascular resistance due to hemodilution may cause low radial artery pressure and lead to the femoral-to-radial pressure gradient after CPB.

Gordon et al. also reported that there was no apparent correlation between the extent of radial arterial hypotension and the presence or absence of postoperative cerebral or cardiac complications (7). Our results were in accordance with their conclusions. One reason why little or no postoperative cerebral or cardiac dysfunction occurred instead of prolonged radial arterial hypotension is that proximal arterial pressure in the aorta or femoral artery is much higher than radial artery pressure under hemodilution. Even if the peripheral pressure is low, it is conceivable that tissue perfusion is preserved during hemodilution, and evidently cerebral and coronary perfusions are well maintained during hemodiltion.

It has been suggested that the difference between femoral and radial pressures would invalidate all previous studies describing hemodynamics immediately after CPB, because these studies relied on radial artery pressure rather than femoral or aortic pressure (5,8). The proximal arterial pressure should be extrapolated for calculation of afterload or ventricular work. Some investigators demonstrated that the pressure gradient may be partially eliminated by monitoring arterial pressure more centrally in the femoral or brachial artery (5,8,9).

Mohr et al. attributed the pressure gradient during CPB, in part, to peripheral vasoconstriction (2). In our study, however, palm temperature was well maintained which showed vasodilation after CPB. A decrease in artery size or vasospasm associated with cooling, nonpulsatile flow, and catecholamine release after initiation of CPB may greatly increase hand vascular resistance and thus lead to the pressure gradient.

Recently, Rich et al. reported that the aortic-to-radial artery pressure difference is associated with events occurring during initiation of cardiopulmonary bypass rather than with rewarming or discontinuation of CPB (10). They stated that the exact mechanisms responsible for the pressure gradient remain unknown, and that it is unclear if hemodilution associated with the onset of CPB contributes to the pressure gradient. Our results could suggest that hemodilution, in part, after the initiation of

CPB may contribute to the pressure gradient.

In conclusion, we demonstrated that the femoral-to-radial artery pressure gradient after CPB correlates well with hemodilution. Accordingly, hemodilution has an important role for the femoral-to-radial artery pressure gradient after CPB.

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