Title : CAN WE TRUST THE DIRECT RADIAL ARTERY PRESSURE IMMEDIATELY FOLLOWING CARDIOPULMONARY BYPASS?

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INTRODUCTION. Arterial blood pressure is an important parameter for patient management following cardiopulmonary bypass (CPB). Direct radial artery pressure (RP) is generally assumed to reflect systemic pressure. This assumption may be incorrect. We noticed a change in the usual relation between RP and central aortic pressure (AP) following CPB. Systolic RP is normally higher than systolic AP[1], but this relationship is altered following CPB. The incidence, magnitude, duration and possible causes of this discrepancy were studied.

METHODS. Twenty-five adult patients undergoing a variety of heart operations under different anesthetics were studied. Informed consent and approval by the Committee for the Protection of Human Subjects were obtained. Monitoring included an 18 gauge teflon left radial artery catheter and a right forearm skin temperature probe. Right forearm blood flow was measured using a Whitney-type mercury-in-silastic strain gauge plethysmograph. We measured AP immediately before and immediately after CPB, using either a 20 gauge needle or a 14 gauge teflon cannula inserted into the ascending aorta. Both radial and aortic pressure monitoring systems included 7 feet of pressure monitoring tubing, 3 stopcocks and a continuous flush device. Frequency response of all aortic and radial pressure monitoring systems was found to be limited by the connecting tubing to 23-25 hz regardless of cannula or needle, with a damping factor less than 0.16. To eliminate measurement artifact from pre- and post-CPB comparisons, we did not change AP and RP monitoring system components during an operation.

RESULTS. We defined ΔP as the systolic pressure difference between AP and RP (ΔP=AP-RP). Seven of 25 cases were discarded because we could not measure AP prior to CPB. In all remaining 18 cases, prior to the start of CPB, radial pressure was greater than aortic with ΔP ranging from 0 to -30 mmHg (mean= -19, median= -20). After CPB, in 13/18 patients (72%), radial pressure was less than aortic, with ΔP>5 mmHg (range= 12 to 32, mean=20, median=18). In most cases the largest discrepancy occurred when CPB support was first discontinued and gradually returned toward the pre-bypass relationship over 10-60 minutes (mean=20). The difference between mean AP and mean RP showed a similar discrepancy; it was always positive, increasing as much as 20 mmHg after CPB.

In 5/18 patients (28%), the post-CPB ΔP had a range of -13 to +2 (mean= -4,median= -3). ΔP increased from its pre-CPB value in all patients, but its change was of clinical concern in only 72%.

Relative forearm vascular resistance (x), calculated from mean AP÷forearm flow, predicted ΔP (y) by the relation y= -0.35x - 19 (r= -0.50, p<0.001) for the 13 patients showing a ΔP > 5 mmHg. An analogous regression line was not different (p>0.5) for the 5 patients with ΔP < 5 mmHg. Combining all patients into one group, the resulting regression line was y= -0.34x + 17 (r= -0.49, p<0.001). Forearm skin temperature always fell slowly during cooling (to a mean of 29.3° C, range=26.6 to 32.3) and rose rapidly (mean rate of 0.1° C/min) after a lag from the start of rewarming; these changes were associated with low and high arm flows, respectively.

DISCUSSION. Systolic RP is normally higher than systolic AP[1]; finding the opposite after CPB requires explanation. Our data is consistent with the hypothesis that lowered RP at the wrist results from diversion of flow to a vasodilated forearm vascular bed. We cannot rule out concurrent vasoconstriction of the radial artery. But hand temperature (measured in 8 patients) rose more rapidly than forearm temperature during rewarming, suggesting high radial artery flow. The observed forearm vasodilation is related temporally to the temperature increase during rewarming; a causal relation is unproven, but suspected.

We recommend direct measurement of AP following CPB whenever RP is low enough that treatment is contemplated, since AP may in fact be adequate. The unnecessary use of inotropic drugs may thus be avoided.

REFERENCE.

1. Berne RM, Levy MN: Cardiovascular Physiology. St. Louis, Mosby, 1967, pp 97-98