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Can We Trust the Direct Radial Artery Pressure Immediately Following Cardiopulmonary Bypass?

David H. Stern, M.D., John I. Gerson, M.D.,†* Forrest B. Allen, M.D.,‡ *Frederick B. Parker, M.D.§*

Reversal of the usual relationship between aortic and radial artery pressure can occur in patients following cardiopulmonary bypass. Radial systolic (and often radial mean) pressures were lower, relative to aortic pressure, after cardiopulmonary bypass than before bypass in all 18 patients studied. The systolic pressure difference (aortic minus radial) was large enough to be of clinical concern (12–32 mmHg) in 13 patients. The change persisted for 10–60 min, gradually returning toward normal. The change temporally was associated with warming at the end of cardiopulmonary bypass and lowered forearm vascular resistance. Relative forearm vascular resistance (x) predicted the systolic aortic minus radial pressure difference (y) by the equation $y = -0.34x + 17$ for all patients ($r = -0.49$, $P < 0.001$). The authors conclude that radial artery pressure does not accurately reflect central aortic pressure in the immediate postbypass period. (Key words: Anesthesia: cardiovascular. Blood pressure: measurement. Monitoring: Blood pressure.)

ARTERIAL BLOOD PRESSURE is an important measurement for patient management during open-heart surgery at the critical time of discontinuation of cardiopulmonary bypass (CPB). The radial artery commonly is chosen to measure blood pressure. Direct radial artery pressure (RP) generally is assumed to reflect blood pressure throughout the arterial tree. It has been known for at least 40 years that systolic arterial pressure increases, relative to central aortic pressure (AP), as the site of measurement is moved from proximal to distal in the arterial tree, although mean arterial pressure remains nearly constant throughout.[1] This usual relationship often is altered immediatedly following CPB. Incidence, magnitude, duration, and a possible cause of this discrepancy were studied.

Methods

Eighteen adult patients undergoing cardiac surgery were studied. Informed consent and approval by the Committee for the Protection of Human Subjects were obtained. Surgical procedures included coronary artery bypass grafting, aortic and mitral valve replacements, and combinations of these. The anesthetic consisted of fentanyl in a mean dose of 75 ± 26 (SD) μg/kg, with halothane added in five patients.

Measuring devices in all cases included an 18-gauge Teflon® left radial artery catheter and right forearm and forehead skin temperature probes. Right forearm blood flow was measured using a Whitney-type mercury-in-silastic strain gauge plethysmograph.[2-4] A wrist cuff was inflated to 250–300 mmHg in order to isolate a cylindric arm segment prior to rapid inflation of the upper arm cuff to 40 mmHg for measurement of forearm blood flow. The right arm was padded and placed inside a rigid plastic cylinder to minimize motion artifacts from surgical personnel leaning against it.

We measured AP immediately before and immediately after CPB using either a 20-gauge needle in some patients or a 14-gauge Teflon® cannula in other patients inserted into the ascending aorta; the same system was used for any given patient before and after CPB. The cannula routinely is inserted for infusion of cardioplegic solution; the needle was inserted at the discretion of the surgeon. Both radial and aortic pressure monitoring systems included 7 feet of standard pressure monitoring tubing, three stopcocks, and a continuous flush device. We measured the dynamic response of each system using the technique for transient testing described by Gabe.[5] To eliminate measurement system artifact from pre- and post-CPB comparisons, we did not alter AP or RP monitoring system components during an operation. Radial and aortic monitoring systems were leveled to the same height and calibrated against a mercury column for each operation.

In eight patients, we recorded indirect brachial artery pressure obtained from standard blood pressure cuff inflation pressure at reappearance of the radial pulse waveform or Korotkoff sounds, if these were present.

Systemic vascular resistance (SVR) data were obtained for all patients during CPB. SVR was estimated using CPB pump output for cardiac output and radial artery pressure.

We employed standard descriptive statistics and used linear regression with *t* testing of slopes for significance

* Research Fellow, Department of Anesthesiology.
† Associate Professor of Anesthesiology.
‡ Assistant Professor of Anesthesiology.
§ Professor of Surgery.

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Address reprint requests to Dr. Gerson.

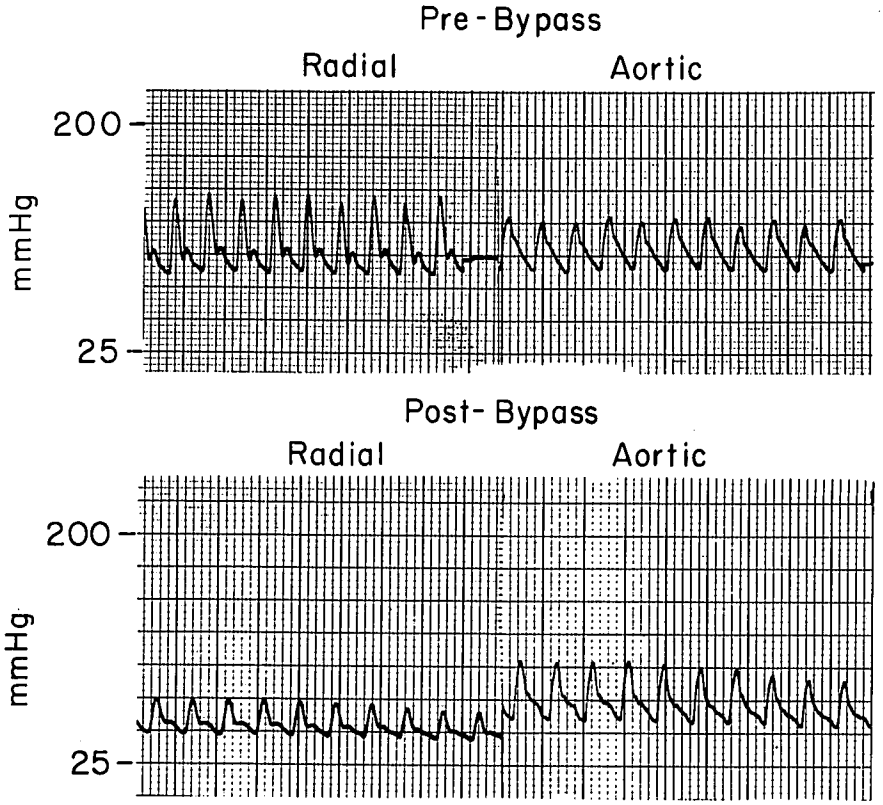

FIG. 1. Photograph of data showing the reversal of usual relationships between simultaneous AORTIC and RADIAL pressures after CPB in one patient.

of predictive statistics. Paired SVR data were analyzed by paired *t* testing, while analysis of variance (ANOVA) and Student-Newman-Keuls (SNK) procedure were used to analyze forearm vascular resistance data.

Results

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 of 0.1 to 0.2.

We defined ΔP as the systolic pressure difference between AP and RP. (ΔP = AP − RP). In all cases, prior to the start of CPB, aortic systolic pressure was less than or equal to radial, with ΔP ranging from 0 to −30 mmHg (mean = −19 mmHg, median = −20 mmHg). After CPB, in 13 of 18 patients (72%), aortic pressure was greater than radial, with ΔP > 12 mmHg (range = 12–32 mmHg, mean = 20 mmHg, median = 18 mmHg). In most cases the largest ΔP occurred when CPB first was discontinued and gradually returned to nearly the prebypass relationship over 10–60 min (mean = 20 min). In five of 18 patients (28%), the post-CPB ΔP ranged from −13 to +2 mmHg (mean = −4 mmHg, median = −3 mmHg). Thus, although ΔP increased from its pre-CPB value in all patients in this group as well, the magnitude of the change was not felt to be of clinical concern. Figure 1 shows the change in the relation between AP and RP for a single patient. That a ΔP of 12 mmHg or greater is of clinical concern is shown by the low mean value of systolic AP for the entire group at the time of maximum ΔP: 86 ± 18 mmHg.

For all 18 patients, the difference of the mean pressures (mean AP less mean RP) showed a discrepancy smaller than but similar to that for ΔP. Prior to CPB, it ranged from 0 to 8 mmHg (mean = 3 mmHg, median = 2.5 mmHg), while following CPB, its maximum ranged from 1 to 22 mmHg (mean = 9 mmHg, median = 8 mmHg). This difference was significant by paired *t* test ($P < 0.001$).

Relative forearm vascular resistance (x), which was expressed as a per cent of maximum during CPB, was 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 > 12 mmHg. An analogous regression line was not different ($P > 0.5$) for the five patients with ΔP < 12 mmHg. Combining all patients into one group, the resulting regression line was $y = -0.34x + 17$ ($r = -0.49$; $P < 0.001$) (fig. 2).

Forearm temperature decreased during CPB to a nadir of 26 to 33° C and then generally, but not always, increased during warming about 2–4° C (fig. 3).

SVR maxima during CPB averaged 2,506 ± 642 (SD)

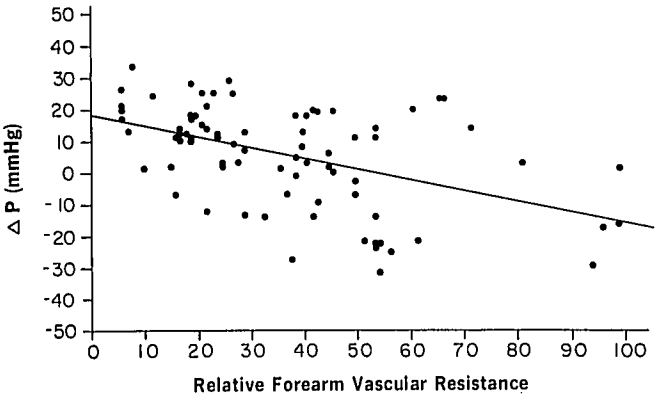

FIG. 2. Scatter plot and regression line of ΔP (mmHg systolic aortic minus radial) *versus* relative forearm vascular resistance per cent of maximum during CPB) for all patients y = 0.34x + 17 (r = −0.49, $P < 0.001$).

dyn • s • cm^{-5} and occurred within 14 ± 22 (SD) min of the lowest forearm temperature achieved. Individual patient data are presented in table 1. SVR fell dramatically during warming to a mean of 958 ± 392 (SD) dyn • s • cm^{-5}. This nadir occurred within 16 ± 8 (SD) min prior to discontinuation of CPB. The difference between minimum and maximum SVR was highly statistically significant ($P < 0.001$ by paired t test).

Relative forearm vascular resistance (FVR) values are presented in table 2. Each patient's maximum FVR was set to 100%. Relative FVR averaged 63 ± 22 (SD) % at the start of CPB; this was within 5 min of the prebypass ΔP. It was 52 ± 23% at the time of the lowest forearm temperature. At the end of CPB, FVR decreased to 31 ± 27%, and remained low at the time of maximum ΔP (34 ± 26%). These values are highly statistically significantly different ($P < 0.001$ by ANOVA). Both start-of-bypass and lowest-forearm-temperature FVR are statis-

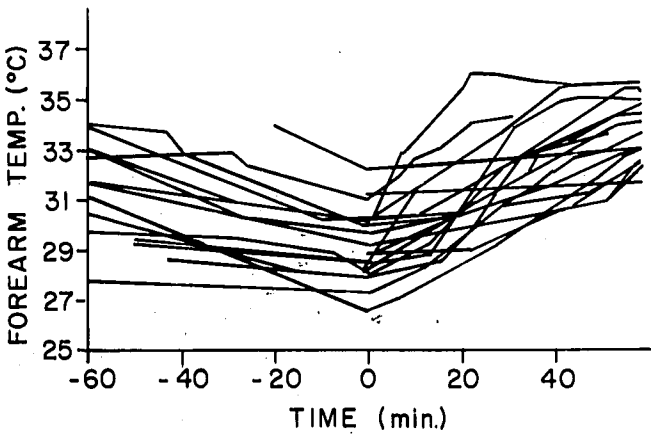

FIG. 3. Plot of forearm temperature *versus* time for all patients. Time zero is lowest forearm temperature.

TABLE 1. Systemic Vascular Resistance in dyn • s • cm^{-5} during Cardiopulmonary Bypass

Patient	Maximum SVR	Minimum SVR
1	1,280	720
2	3,200	1,050
3	3,200	1,818
4	3,824	840
5	1,400	573
6	2,187	960
7	1,600	1,150
8	2,560	1,040
9	2,533	967
10	2,773	1,850
11	2,258	596
12	2,769	582
13	2,873	1,389
14	2,267	867
15	2,923	960
16	2,720	640
17	2,377	567
18	2,357	667
Mean	2,526 (14)	958 (16)
SD	642 (22)	392 (8)

Values in parentheses are minutes between systemic vascular resistance (SVR) measurement and lowest arm temperature for the "Maximum SVR" column and minutes between SVR measurement and the end of bypass for the "Minimum SVR" column.
$P < 0.001$ from t test.

TABLE 2. Relative Forearm Vascular Resistance as Per Cent of Maximum

Patient	Immediately Before Bypass	Lowest Forearm Temperature	End of Bypass	Maximum ΔP
1	100	96	14	23
2	77	70	18	21
3	43	42	18	19
4	97	40	6	8
5	62	30	19	19
6	33	67	100	100
7	57	100	48	54
8	55	31	22	46
9	74	35	49	50
10	38	24	8	6
11	43	80	90	90
12	54	39	43	43
13	52	41	23	23
14	54	59	24	22
15	47	51	20	19
16	95	24	14	16
17	54	44	17	41
18	100	70	16	19
Mean	63 (0.1)	52 (−4)	31 (3)	34 (0.4)
SD	22 (4)	23 (8)	27 (6)	26 (3)

Values in parentheses are minutes between forearm vascular resistance measurement and event. Positive values mean the measurement was done before the event, negatives mean after.
$P < 0.001$ from ANOVA.
Intergroup comparisons by SNK showed "immediately before bypass" and "lowest forearm temperature" are each different from both "end of bypass" and "maximum ΔP" ($P < 0.05$).

tically significantly different from both end-of-bypass and maximum ΔP FVR by SNK procedure ($P < 0.05$).

Type of operation had no effect on ΔP. The operations included 11 coronary artery bypass grafts, four mitral valve replacements, and three aortic valve replacements. The percentages of each operation with ΔP > 12 mmHg were 73, 75, and 67%, respectively.

We attempted to plot ΔP against duration of CPB, total warming time, and various measures of arm temperature, including lowest temperature during CPB, total increase during warming, warming rate, and temperature when CPB pump support was discontinued. There was no clear relationship except that where the lowest arm temperature during CPB was below 29° C (9/18 patients), ΔP after CPB was always positive. Above 29° C ΔP was variable.

Indirect brachial blood pressures were closer to aortic than were radial pressures for the eight patients in whom all three pressures simultaneously were recorded (20 data points). Following CPB, the absolute value of systolic aortic less systolic radial pressure was 23 ± 11 (SD) mmHg, while the absolute value of systolic aortic less systolic indirect brachial was 15 ± 12 mmHg. A comparison of systolic (aortic–radial) and systolic (aortic–indirect brachial) by paired t test yielded $P < 0.002$.

Discussion

Systolic RP is normally higher than systolic AP.[1] Our finding that this was true before, but not in the period immediately after CPB, requires explanation.

We suspected that this represented either vascular changes in the arm or an artifact of the radial artery or aortic pressure monitoring system. In order to exclude the latter, we set up the same series of connecting tubing, transducer, and cannula (or needle) used in patients and obtained the frequency response and damping factor listed above. We found that the radial and aortic pressure monitoring systems yielded values similar enough to exclude this explanation. By measuring RP and AP before and after CPB, without changing the apparatus, we believe that any differences between the systolic pressures represent vascular system changes and not artifacts of measurement system. The finding of change in a similar direction in the relation between mean AP and mean RP also supports this contention, since mean pressure, a low-frequency phenomenon, is less affected by measuring system artifact.[6]

There are several possible vascular mechanisms for the observed pressure differences after CPB. O'Rourke has summarized the factors that change arterial pulse wave shape during transmission: damping or attenuation in travel, dispersion of the wave due to different frequency components traveling at different velocities, reduction or amplification of components of the pulse wave by reflected waves, natural vibrations or resonance in various parts of the arterial tree, and the progressive increase in stiffness of peripheral arteries.[7] It is impossible to determine which, if any, of these factors were operative in the patients studied. All of them, however, affect pulse pressure, not mean pressure. None would account for the observed change in mean pressure.

Simple forearm arterial vasodilation or vasoconstriction could cause the observed difference in mean arterial pressure and could contribute to (possibly by changing one of the factors above) the observed difference in systolic pressure.

We investigated the simplest and most obvious possibility: radial artery vasodilation or vasoconstriction. Either could cause the observed difference in mean as well as systolic pressure. Our data are consistent with the hypothesis that lowered RP at the wrist results from diversion of flow to a vasodilated forearm vascular bed. We favor this explanation over one of radial artery vasoconstriction for several reasons.

First, both SVR and forearm resistance were found to be low, not high, when ΔP increased, implying vasodilation. Second, if radial vasoconstriction were present, we would not expect the increase in forearm temperature, which was seen in every patient.

Our finding that arm vascular resistance varied with ΔP was highly significant statistically ($P < 0.001$), but the r value was only 0.49. There are two possible explanations. First, the regression coefficient may be low because arm flow is not the sole factor influencing ΔP. The calculated r value of 0.49 translates to a coefficient of determination of 0.24. This means that only about 24% of the variability in ΔP is accounted for by the variability in forearm vascular resistance. Thus, reduced forearm vascular resistance is an incomplete explanation for the change in the relationship between aortic and radial systolic pressures. We do not know what factors account for the balance of the variability. Choosing simple linear regression reflects a first-step approach to the problem but also is quite rational, based on the fact that pressure drop is inversely proportional to resistance for steady laminar flow in a Newtonian fluid. This is an application of the familiar relation: "mean arterial pressure less CVP equals cardiac output times resistance," which is useful, although it, too, is applied during nonsteady flow in a non-Newtonian fluid. We suspect the areas delineated by O'Rourke above are where to look for a more complete answer; their investigation is beyond the scope of this clinical study.

Less importantly, the imprecision of the method of strain-gauge plethysmography may have induced some "scatter" in the data. The range of normal values of forearm vascular resistance found by Mason and Braun-

wald, using this technique,[3] is quite wide: 24–52 mmHg · ml^{-1} · 100 ml tissue · min. Previous work from this laboratory during steady state CPB produced similar results,[8] with mean control values of 33–42 mmHg · ml^{-1} · 100 ml tissue · min and standard deviations of 20–28 mmHg · ml^{-1} · 100 ml tissue · min. This wide range of values may be inherent in the technique. This is not surprising, since measured arm circumference changes are small, on the order of 10–40 thousandths of an inch over several seconds of measurement. Comparison of control measurements between patients in this study would not be meaningful, since ambient conditions (bypass flow, pressure, temperature) were not controlled.

The idea that warming might trigger forearm vasodilation is supported by the temporal association of low forearm temperatures with high forearm vascular resistances and *vice versa*.

Measurement of blood pressure by cuff deserves some comment. The indirect cuff pressure is a better indicator of aortic pressure than is radial pressure, at least when the radial pressure is much lower than aortic. But because of the wide scatter of the data, any single cuff pressure measurement is still a poor predictor of aortic pressure. While an adequate cuff pressure may suffice for reassurance when the RP is only marginal, direct AP measurement is indicated when RP is seriously low.

Dopamine was infused at the discretion of the attending anesthesiologist after CPB in 15 of 18 patients. It never was started prior to at least one determination of ΔP after CPB. Its effect on ΔP was unpredictable. Response varied from a return to pre-CPB ΔP within 10 min to an actual increase in ΔP after dopamine was started. Dopamine could not have caused the change in ΔP, since it was started only after ΔP was recorded.

We recommend direct measurement of the AP following CPB whenever RP is low enough that treatment is contemplated, since AP may in fact be adequate. If inotropic drugs are titrated for their effect on blood pressure, aortic pressure is likely to be of more importance than radial during circumstances in which the two are different.

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