

Radial artery pressures compared with subclavian artery pressure during coronary artery surgery

MICHAEL G. BAZARAL, PHD, MD; ALEXANDER NACHT, MD; JOHN PETRE, PHD; BRUCE LYTTLE, MD;
KAVITA BADHWAR, MA; FAWZY G. ESTAFANOUS, MD

■ Radial artery and subclavian artery pressures were measured in nine men undergoing coronary artery surgery requiring cardiopulmonary bypass. Radial artery pressures were measured simultaneously through short (10.2 cm) and long (106.9 cm) tubing using two transducers in a branched tubing system. Interaction between the branches was minimal in an in vitro test system. The radial artery pressures were compared with simultaneous subclavian artery pressures measured through a catheter introduced into the axillary artery. Pre-bypass, the systolic pressures were greater in the radial than in the subclavian artery ($P < 0.005$), by typically 10%. There was no significant difference between long and short branches of the radial artery tubing system; the exaggeration of systolic pressures under these conditions resulted from use of the radial artery per se. After weaning from bypass, the mean and diastolic pressures were lower in the radial artery than in the subclavian artery. Average systolic pressure was not significantly lower. The ratio of radial to subclavian systolic pressures showed increased variance. By the end of the operation, radial mean pressure was similar to the subclavian pressure, although the difference remained significant.

□ INDEX TERM: BLOOD PRESSURE DETERMINATION □ CLEVE CLIN J MED 1988; 55:448-457

THE PROXIMAL aorta supplies the coronary arteries and is the vessel into which the left ventricle pumps. In the absence of specific pathology, the aortic pressure is applied through short large-diameter arteries to the major organs. During major surgery and in the intensive care unit

the implicit assumption that the radial artery pressure is an adequate approximation to aortic pressure. However, the systolic pressure is generally exaggerated in peripheral arteries,^{1,2} and the tubing and transducer used to monitor the pressure can cause additional distortion of the pressure waveform.³ A second problem is that at the end of cardiopulmonary bypass, the systolic, diastolic, and mean pressures in the radial artery may be lower than the pressures in the aorta.⁴⁻⁷

■ See also editorial by Lamantia and Barash (pp 415-416)

the radial artery pressure is commonly monitored, with

We evaluated the errors in the radial artery pressure measurements during anesthesia for coronary artery surgery. The radial artery pressure was measured using two transducers and a branched tubing system. One transducer was located close to the cannulation site and connected via 10.2 cm of tubing; this approximated the true radial artery pressure. The second transducer was connected through a 182.9-cm length of fluid-filled

From the Departments of Cardio-Thoracic Anesthesia (M.G.B., J.P., F.G.E.), Cardiovascular Surgery (B.L.), and Biostatistics and Epidemiology (K.B.), The Cleveland Clinic Foundation, and Department of Anesthesia, New York University School of Medicine (A.N.). Submitted for publication June 1987; accepted Oct 1987.

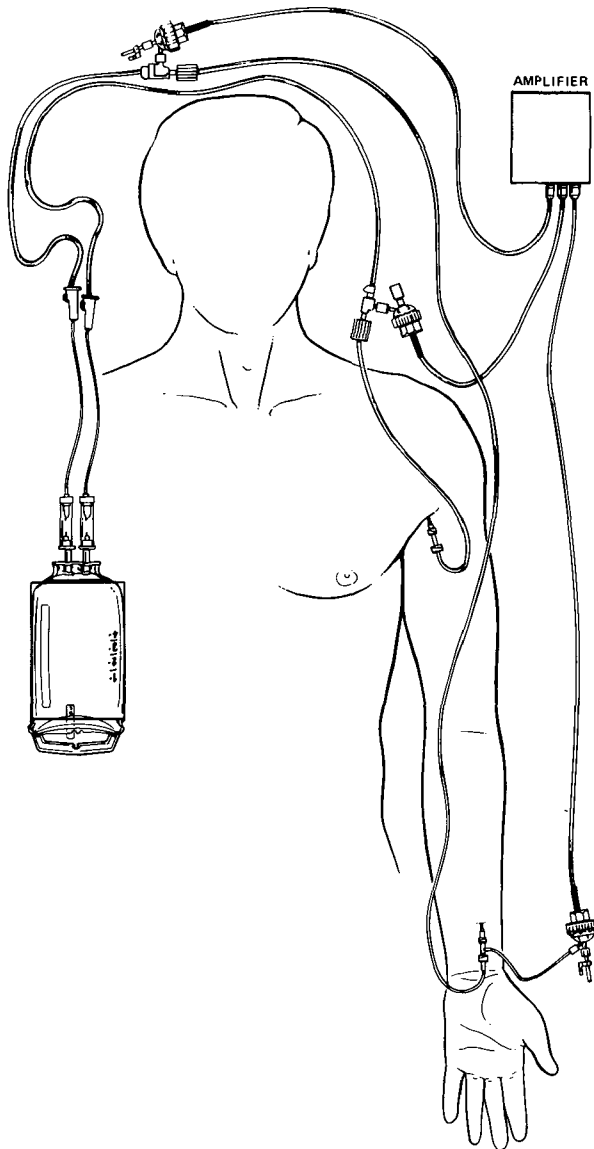


FIGURE 1. Tubing and transducer systems used for pressure measurements. Subclavian pressure was monitored through a 13.2-cm 20-gauge catheter inserted in the axillary artery and connected via a 32.4-cm length of 0.16-cm ID tubing to a transducer and continuous flush device. The radial artery pressure was monitored through a 5.1-cm 20-gauge catheter. The catheter was attached to a "t" connector and the side arm of the "t" connector (a 10.2-cm length of 0.25-cm ID tubing) was connected to a transducer (short-tubing radial). The through-port of the "t" was attached to a separate transducer and flush device via a 186.9-cm length of 0.16-cm ID tubing (long-tubing radial). Intraoperatively, the arms were at the patient's side, with the palms inward.

tubing, as used under typical clinical conditions. The waveform from the transducer close to the cannulation site is affected only minimally by the long tubing and second transducer.⁸ As a reference we used the pressure in the subclavian artery, measured through a 13-cm catheter introduced into the axillary artery and connected to a transducer through short tubing. Cannulation of the axillary artery for monitoring is known to be reasonably safe.⁹⁻¹³ We assumed that the catheter tip was in the subclavian artery, and that the pressure waveform approximated the pressure in the proximal aorta.

This system allowed us to evaluate the separate contributions of the long-tubing system and of the radial artery per se to the systolic overshoot in the radial artery waveform occurring during clinical monitoring. The systolic pressure overshoot contributed by the arterial system of the arm can be evaluated as the difference between the subclavian artery systolic pressure and the systolic pressure measured in the radial artery using the transducer connected via short tubing. The separate contribution to systolic overshoot arising from the use of long monitoring tubing can be evaluated as the difference between the radial systolic pressure measured through short tubing and the radial systolic pressure measured through long tubing.

A previous study of the radial pressure at the end of cardiopulmonary bypass showed that the pressures in the radial artery are, on the average, lower than the aortic pressures, and that the magnitude of the error varies widely from case to case.⁴ These pressure measurements could be made only while the chest was open. We used the subclavian artery pressure as a reference, and this permitted us to follow the relationship between central arterial pressure and the radial pressure from before the induction of anesthesia through to the end of surgery.

MATERIALS AND METHODS

Transducers, data acquisition, and data analysis

The arrangement of cannula tubing and transducers is shown in *Figure 1*. Transducers (Bentley Trantec) were used with nondisposable domes and were gas-sterilized before use. Transducers were matched for gain, and calibration was rechecked after each use. Recordings of patient data were made for 10 sec using three pressure channels (E for M) and an ECG channel, digitized at 200 samples/sec and recorded to disk with a DEC 11/23 computer. The data were reviewed and three consecutive beats from each recording, selected for absence of obvious noise artifact, were chosen for averaging. Character-

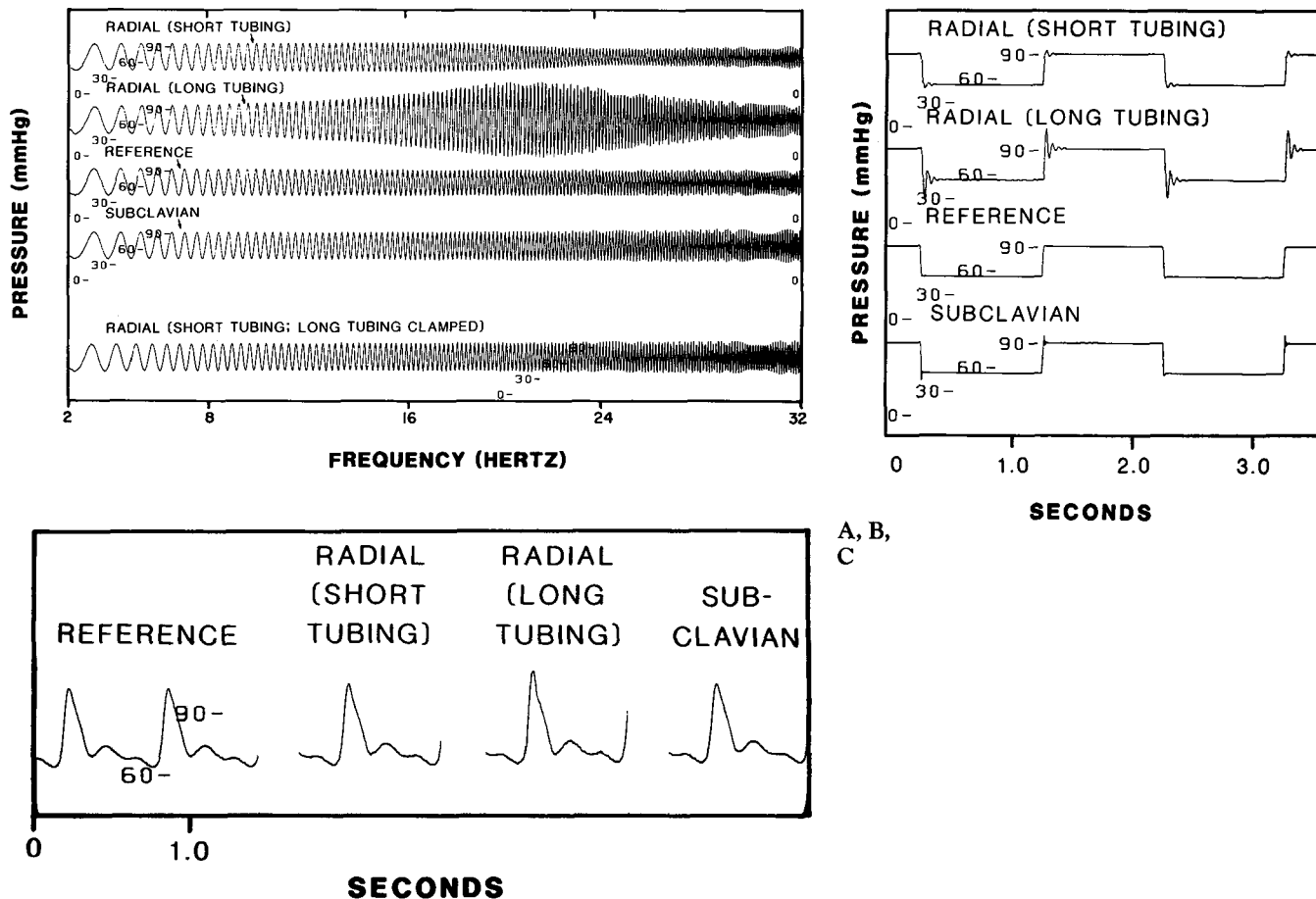


FIGURE 2A. Frequency response of the tubing and transducer system. A reference transducer, the radial artery catheter and “t” connector with associated short and long tubing and transducer assemblies, and a separate subclavian catheter with associated tubing and transducer were simultaneously connected to a pressure waveform generator. The subclavian transducer and tubing assembly displayed a nearly constant response between 2 and 32 Hz. The long-tubing branch of the radial system showed a nonlinear gain with a peak at 20 Hz. The short-tubing branch of the radial system showed a reduced gain above 20 Hz; the reduced gain at high frequencies did not occur when the long-tubing branch was clamped. **FIGURE 2B.** Square-wave response of the tubing and transducer system. The pressure generator was driven with 1.0 Hz square wave. The reference waveform contained no overshoot, and the subclavian waveform contained only a minor spike of overshoot. The radial branch with the long tubing showed overshoot (68% of the pulse pressure) with several cycles of ringing. The short-tubing radial branch showed minimal overshoot, which resulted from interaction with the long-tubing branch. **FIGURE 2C.** Response of the tubing and transducer system to a test waveform. The pressure generator was driven with the manufacturer’s “surgical aortic” waveform at 120 beats/min. The transduced pressure waveforms for the reference, subclavian, and the short branch of the radial system were identical. The waveform in the long branch of the radial system showed a 5-mmHg overshoot.

istics of the tubing and transducer systems (Figures 2A–C) were recorded using a digital recorder and monitor (Marquette 7100 and 7000RA). For these recordings a Bio-Tek model 601 A hydraulic pressure generator was driven by either an internally generated waveform (Figure 2B and C) or by an externally generated sine wave

(Figure 2) from a Tektronix model 501 function generator swept with a linear ramp. Calculations of natural frequency and damping coefficient were made from the sine wave frequency sweep tracing.¹⁴

All data are expressed as mean ± standard deviation. Statistical analysis was done using SAS software. Paired

TABLE 1
INTRAOPERATIVE RADIAL ARTERIAL AND SUBCLAVIAN ARTERIAL PRESSURES

Condition	Systolic (mmHg)			Diastolic (mmHg)			Mean (mmHg)		
	Subclavian	Long radial	Short radial	Subclavian	Long radial	Short radial	Subclavian	Long radial	Short radial
Preinduction	122±14	135±15‡	131±16†	69±8	69±8	68±8	90±9	91±9	89±9
Postinduction	110±12	121±13‡	119±12†	66±8	67±8	66±7	85±9	85±10	84±9
Postintubation	127±15	140±18‡	137±19†	75±5	75±6	74±6	97±9	97±10	96±10
Poststernotomy	114±13	126±11‡		71±7	70±7		88±10	87±10	
Postpericardotomy	106±11	117±13‡		70±10	70±9		83±10	84±10	
On bypass							61±10	60±9	
Cooled							67±6	64±7	
Warmed							66±11	59±12*	
Off bypass	105±15	94±23		58±8	53±7‡		76±10	68‡±11	
Closed	111±12	116±17		69±8	67±7†		86±10	83†±9	

* $P < 0.01$ relative to subclavian

† $P < 0.005$ relative to subclavian

‡ $P < 0.0005$ relative to subclavian

t tests were used for the statistical significance of differences between means. For evaluation of variances the test used was the generalized likelihood ratio to compare equal variances. In order to take the multiple testing into account, a difference was considered significant only if the P value was < 0.01 .

Patients, intraoperative management, and study protocol

Approval was obtained from the institutional committee on human research. Nine male patients scheduled for elective coronary artery bypass grafts who consented to the study continued to receive their oral medications until the time of surgery. Seven of the patients were receiving combined β -blocker and calcium channel agents, one a β -blocker alone, and one a calcium channel agent alone. Average age of the patients was 53 ± 11 years. Overall left ventricular function was graded according to the cardiologist's evaluation of the ventriculogram. Eight of the patients had normal or mildly impaired function, and one had moderate impairment. Premedication (scopolamine 0.4 mg and morphine sulfate 0.12 mg/kg, intramuscular) was administered 45 minutes before the patients arrived in the operating room.

Radial artery cannulation was done approximately 2 cm proximal to the styloid process, after local anesthesia, using a 3.4-cm 20-gauge Teflon cannula. The transducer for the short tubing was taped to the patient's forearm, and the transducer for the long tubing was mounted at the head of the operating room table (Figure 1). Eight of the nine radial artery cannulations were done on the left arm, and in no case was a site used that was distal to a

previous arteriotomy. Axillary cannulation was done after local anesthesia using in sequence a 3-cm 20-gauge cannula over a needle, a 0.25-mm spring wire, and a 13.4-cm 20-gauge Teflon cannula (Arrow Pediatric CVP Kit rs-254). The transducer was mounted on the operating room table near the patient's shoulder. Five of the nine axillary artery cannulations were on the left side. Both tubing systems were equipped with a continuous-flush device (Gould). Transducers were set at a common zero point using a spirit level after the arm was positioned at the patient's side. A peripheral intravenous cannula and a central venous or pulmonary artery catheter (via the internal jugular vein) were also placed after local anesthesia.

ECG was monitored with leads II and V6. Anesthesia was induced with 30 $\mu\text{g}/\text{kg}$ fentanyl; 0.04 mg/kg pancuronium bromide and 0.16 mg/kg metocurine iodide were used for muscle relaxation. Patients were ventilated with 100% oxygen, and after tracheal intubation were mechanically ventilated at 10 breaths/min to a PaCO_2 between 35 and 42 mmHg. Anesthesia was maintained with increments of fentanyl and pancuronium. Enflurane up to 1.5% inspired concentration was used to supplement the anesthetic as necessary, but was discontinued at the initiation of cardiopulmonary bypass and was not again administered until after the post-bypass data were recorded. The target mean arterial pressure (MAP) was 75 to 90 mmHg. To limit the blood pressure to this range, nitroglycerin was used up to a maximum of 50 $\mu\text{g}/\text{min}$, and if this was inadequate, sodium nitropruside was titrated as required.

Cardiopulmonary bypass was conducted through an

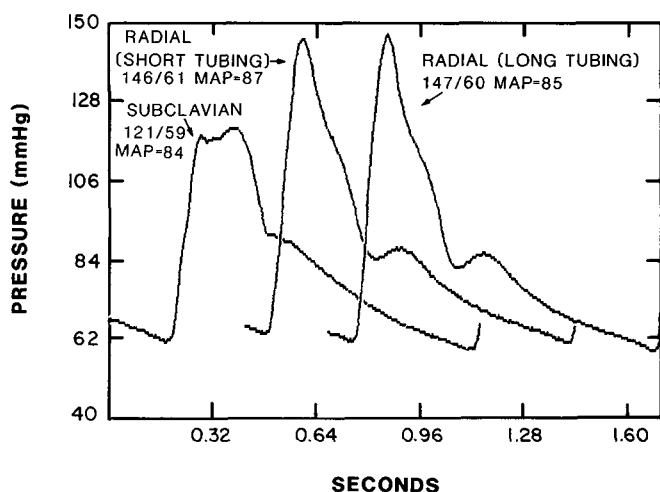


FIGURE 3. Pressure waveforms in a patient with systolic overshoot. The recordings were made before induction of anesthesia. Relative to the subclavian recording, the origin of the traces from the radial artery using short tubing and using long tubing are shown displaced by 0.256 sec and 0.512 sec respectively, to permit the individual curves to be clearly seen. In this patient the systolic pressure in the radial artery relative to the subclavian pressure is exaggerated, and the error is similar in both the long- and short-tubing branches of the radial tubing system.

ascending aortic cannula and a single venous cannula, using a roller pump system and membrane oxygenator primed with 2.5 L Ringer's lactate. Flow was 45 mL/min/kg. Average duration of bypass was 99 ± 31 min. Crystalloid cardioplegia and systemic hypothermia were used for myocardial protection; the average minimum bladder temperature was $28.2^\circ\text{C} \pm 1.2^\circ\text{C}$ and the patients were rewarmed to an average bladder temperature of $36.6^\circ\text{C} \pm 0.5^\circ\text{C}$ at the end of cardiopulmonary bypass. The hematocrit at the end of bypass was $26\% \pm 4\%$. Anesthesia during bypass was maintained with fentanyl, and sodium nitroprusside or Neo-synephrine (phenylephrine hydrochloride) was used to maintain the subclavian MAP between 40 and 80 mmHg. No vasoactive drugs were used during the last 20 minutes of bypass.

Pressure recordings were made before induction of anesthesia, after induction, after tracheal intubation, after sternotomy, after pericardotomy, after establishing cardiopulmonary bypass, after cooling to the target temperature, after rewarming was complete, immediately after terminating bypass (within three minutes), after the chest was closed, and at the end of surgery. The

TABLE 2
NORMALIZED SYSTOLIC PRESSURES

Condition	Location		
	Subclavian (mmHg)	Long-tubing radial* (mmHg)	Short-tubing radial* (mmHg)
Preinduction	121.9 \pm 14.0	133.9 \pm 14.6 \dagger	131.8 \pm 15.6 \dagger
Postinduction	110.0 \pm 12.2	120.9 \pm 11.7 \dagger	119.3 \pm 12.2 \dagger
Postintubation	127.4 \pm 14.5	138.8 \pm 17.4 \dagger	137.5 \pm 17.6 \dagger

* Systolic pressures were corrected for zero offset errors under the assumption that the short-radial, long-radial, and subclavian mean pressures were the same: normalized radial systolic = radial systolic + (subclavian mean - radial mean).

\dagger The difference between subclavian and short-tubing radial pressure is greater than the difference between short-tubing radial and long-tubing radial pressures ($p \leq 0.003$).

transducer systems did not reliably retain a zero point (± 2 mmHg) beyond one hour.¹⁵ Because the transducer for the short tubing, positioned at the patient's side, could not be routinely re-zeroed, measurements using this transducer were used only up to the postintubation sample.

RESULTS

Tubing and transducer system

Evaluation of the transducer and tubing system using a sine wave generator with a linearly increasing frequency demonstrated that the frequency response of the subclavian tubing and transducer system was essentially flat between 2 and 32 Hz. The long branch of the radial system had a resonant frequency of 19.7 Hz and a damping coefficient of 0.213. The short branch showed decreased response above 18 Hz, which was a result of the interaction between the two branches (Figure 2A). The square wave response demonstrated that the subclavian and short-tubing radial arteries had minimal overshoot. The long-tubing radial arterial square wave response was marked by substantial overshoot and several cycles of ringing (Figure 2B). The test arterial waveform was reproduced accurately by the subclavian and short-tubing radial systems. The systolic overshoot in the long-tubing radial system was 5% of the reference systolic pressure (Figure 2C).

Short-tubing radial data

Short-tubing radial data, as well as long-tubing radial and subclavian data, were obtained for the preinduction,

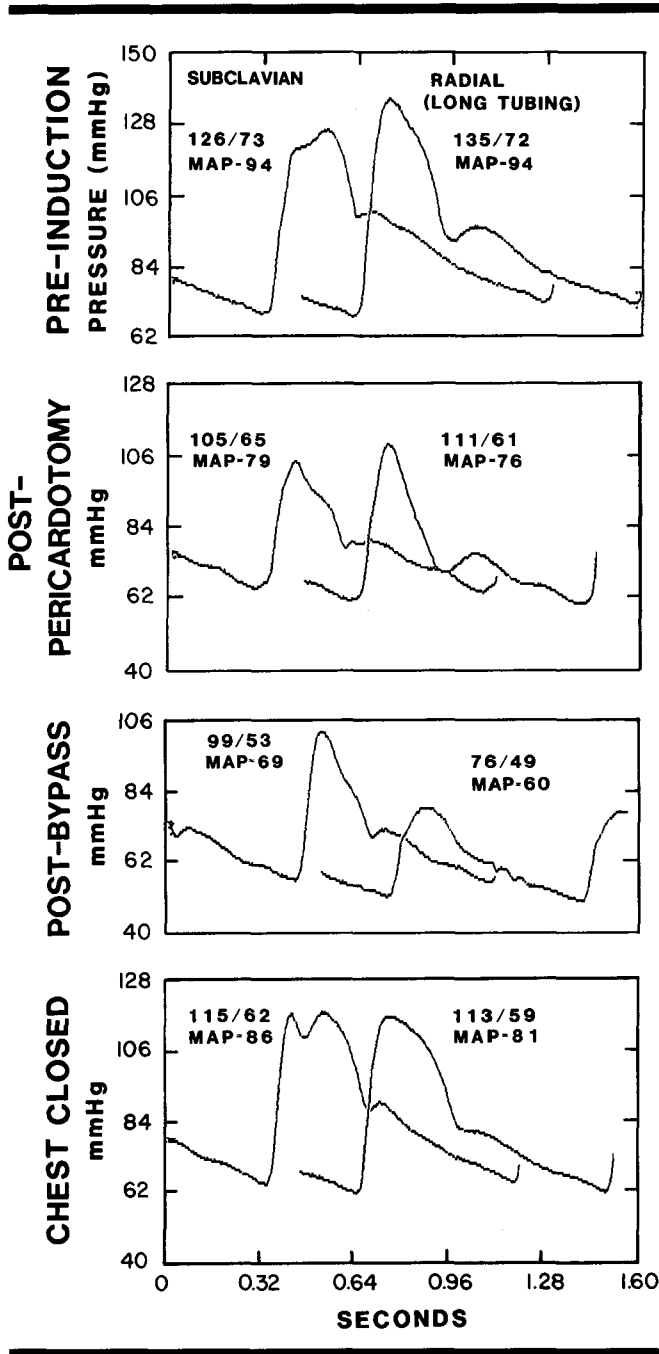


FIGURE 4. Intraoperative pressure waveforms from the subclavian artery and radial artery. Relative to the subclavian recording, the origin of the trace from the radial artery using long tubing is shown displaced by 0.256 sec to permit the individual curves to be clearly seen. This patient exhibited pre-bypass systolic overshoot in the radial artery. Post-bypass the radial artery pressure was substantially lower than the subclavian artery pressure, and this discrepancy had partly resolved by the end of the operation.

postinduction, and postintubation sample times. The diastolic and mean pressures were the same at all three transducers at these times (Table 1). The systolic pressure was of particular interest. In the raw data, for example at the preinduction time, average systolic pressure measured through the long radial tubing was significantly greater than the pressure in the subclavian artery, by an average of 13 mmHg. The average overshoot measured through the short radial tubing was 9 mmHg, also significant, whereas the difference between the short- and long-tubing pressures for the radial artery was substantially less (4 mmHg). The relative contributions to systolic overshoot in a selected patient are illustrated in Figure 3.

For detailed analysis the systolic pressures were corrected for small errors in referencing under the assumption that the mean pressures were identical (Table 2). At all three measurement times the 8–10-mmHg contribution of the radial artery per se to the systolic overshoot (long-tubing radial pressure–subclavian pressure) was significantly greater than the contribution of the long-tubing system (long-tubing radial pressure–short-tubing radial pressure). The difference between the systolic radial pressures measured through short and long tubing was 2–3 mmHg and was not significant.

Long-tubing radial data

Analysis of patient data (Table 1) showed that from before induction of anesthesia until institution of cardiopulmonary bypass, long-tubing radial artery pressure and the subclavian artery pressures were the same with respect to mean and diastolic pressure. Systolic pressures were significantly different; the average radial systolic pressure was typically 10% greater than the subclavian pressure, and, as noted above, this difference was primarily related to the radial artery measurement site, not to the tubing length.

While the patients' circulation was maintained by cardiopulmonary bypass, the flow was typically nonpulsatile. Although a pulse may be present if the heart is allowed to eject blood, only a mean pressure is reliably present, and our observations were therefore intentionally limited to the measurement of mean pressure during bypass. Upon initiation of cardiopulmonary bypass, there was no difference between the mean pressures measured in the radial and subclavian arteries. After the induction of systemic hypothermia was complete, there continued to be no difference between the sites. Measurements at the completion of rewarming showed the radial artery mean pressure was significantly less than the subclavian mean pressure. At this time the radial artery

pressure was, on the average, 91% of the corresponding subclavian pressure, and the standard deviation of the ratio was 11%. This standard deviation is significantly greater than the pre-bypass standard deviation of 5% ($P < 0.001$) and shows that not only is the pressure less in the radial artery after re-warming but also that after re-warming the radial artery pressure becomes a less consistent measure of the central arterial pressure.

Immediately after weaning from cardiopulmonary bypass, the pressure in the radial artery was generally less than the pressure in the subclavian artery (Figures 4 and 5). The radial artery mean and diastolic pressures at this time were significantly less than the corresponding subclavian artery pressures, and the reliability of these pressures was less than pre-bypass, as measured by a significant increase in the standard deviation of the ratio of radial to subclavian pressures. The systolic pressure in the radial artery after bypass is on the average 89%

of the subclavian pressure but the subclavian and radial pressures are not significantly different ($P = 0.03$). The standard deviation of the ratio of radial and subclavian systolic pressures is 14%, significantly different from the pre-bypass standard deviation of 3% to 5% ($P < 0.001$); this denotes substantial patient-to-patient variability of the radial artery systolic pressure relative to subclavian systolic pressure immediately post-bypass. By the end of the operation the pressures in the radial artery were similar to the pressures in the subclavian artery, although the differences in mean and diastolic pressures remained statistically significant.

The various errors in radial artery pressures relative to subclavian pressure at each measurement time were tested for correlation with patient data and hemody-

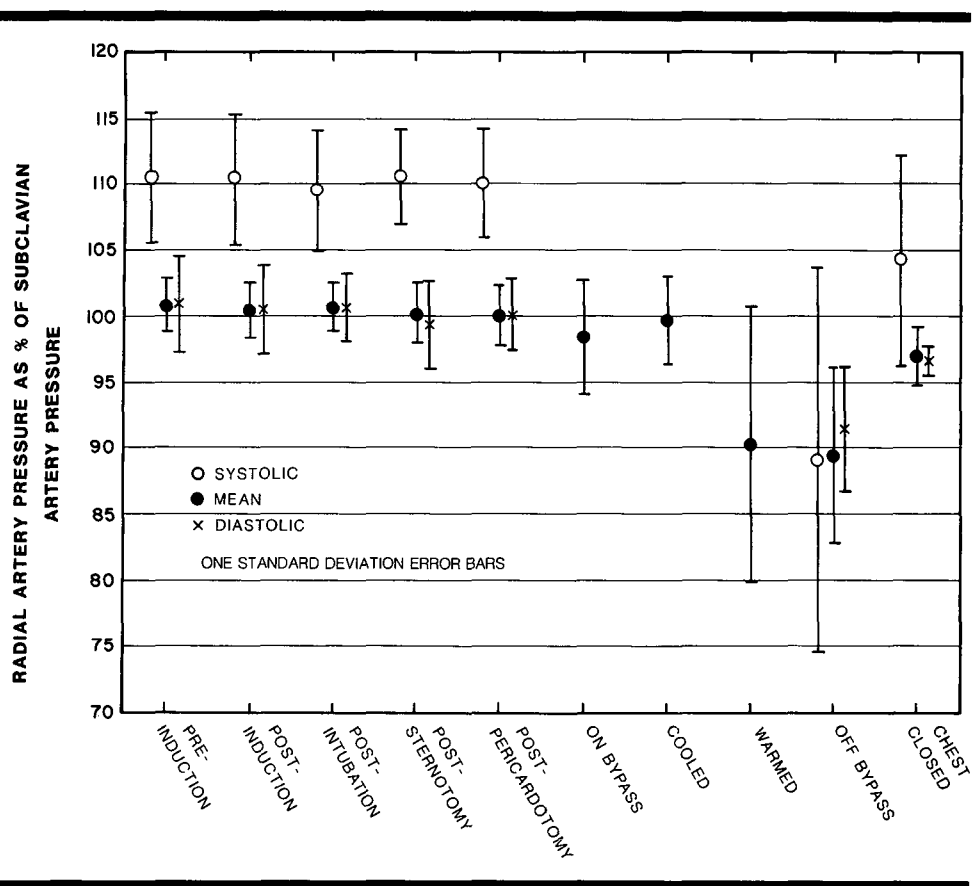


FIGURE 5. Graphical summary of intraoperative pressure recordings from nine patients. The pressure in the radial artery (using long tubing) is expressed as percent of the simultaneous pressure in the subclavian artery. Pre-bypass, the systolic pressures are generally exaggerated in the radial artery, and after re-warming and in the immediate post-bypass period, the radial artery pressures are typically lower than the corresponding subclavian artery pressures. At the end of the operation the relationship between the radial and subclavian pressures approaches the pre-bypass conditions.

namic findings. There was no significant correlation with duration of bypass, patient age, heart rate, or CVP (nine patients) and in the eight patients having pulmonary artery catheters, there was no correlation with cardiac output or pulmonary artery pressures. Intraoperative hemodynamic measurements (Table 3) demonstrated that the cardiac output was highest and systemic vascular resistance least, on the average, immediately post-bypass.

DISCUSSION

A review of the papers published in *Anesthesia and Analgesia* in 1986 showed 17 papers in which the radial

TABLE 3
INTRAOPERATIVE HEMODYNAMIC DATA

Condition	Time (min)	Mean arterial pressure (mmHg)*	Heart rate (beat/min)	Central venous pressure (mmHg)	Pulmonary wedge pressure (mmHg)†	Cardiac index (L/min)†	Systemic vascular resistance‡
Preinduction	0	90±9	59±11	9±3	14±4	2.4±.6	1379±242
Postinduction	6±4	85±9	66±15	11±4	14±4	2.5±.5	1133±242
Postintubation	11±5	97±9	75±16	12±4	15±4	2.7±.6	1294±266
Poststernotomy	39±13	88±10	69±16	10±3	15±3	2.4±.5	1203±280
Postpericardotomy	91±26	83±10	80±20	10±2	12±3	2.5±.5	1161±185
On bypass	112±20	61±10					
Cooled	147±29	67±6					
Warmed	205±37	66±11					
Off bypass	217±37	76±10	85±11	12±4	15±6	3.4±.6§	790±223§
Closed	255±39	86±10	80±7	13±3	15±4	2.5±.6	1194±340

* All pressures are in mmHg. Arterial pressure shown is the subclavian pressure.

† N = 8

‡ Dyne-sec-cm⁻⁵. Calculated as: 80 x (mean arterial pressure - central venous pressure) cardiac output.

§ Cardiac index at this time is greater, and systemic vascular resistance is less, than at all other times (P < .01)

artery pressure was an analyzed variable. In one study the dorsalis pedis artery was also used, and there were no studies in which arterial cannulas for monitoring during anesthesia were placed at other sites. In four of these papers the systolic pressure was an analyzed variable, and in four the post-bypass radial artery pressures were used to evaluate hemodynamics. The implicit assumption that radial artery pressure is an adequate approximation to central arterial pressures is frequently made, but there is ample evidence that this assumption is questionable. Distal pulse amplification is a well-recognized phenomenon, and while errors in systolic pressure measurements are frequently attributed to the tubing and transducer system, in our patients the use of the radial artery per se was a primary factor in systolic pressure exaggeration. The other error associated with the use of the radial artery for monitoring is the low radial artery pressure, relative to central arterial pressure, that occurs after rewarming on cardiac bypass. This has been previously observed,^{4,5} and appears to be related to transiently high blood flow in the forearm, resulting in a pressure drop distally.⁴

We interpret these results to mean that the radial pressures were not affected in cases where the proximal 20-gauge catheter was ipsilateral. This seems reasonable given the small diameter of the catheter relative to the size of the proximal artery. We do not believe that the low distal pressures post-bypass were an artifact of stretch or compression of the subclavian artery associated with sternal retraction. The relationship between proximal

and distal pressures did not change after the chest was opened or throughout the pre-bypass period. The post-bypass findings, where a significant change was found, were obtained while the position of the sternal retractor was generally the same as during the pre-bypass measurements.

The systolic pressure exaggeration in the peripheral arterial waveform presumably depends on the amplitude and phase relationships of high-frequency components in the central arterial pressure wave, as well as the tone in the distal arterioles. The systolic pressure overshoot will be related to the initial dp/dt of the systolic upstroke, and will vary among patients and from time to time in the same patient.¹⁶ The patients we studied were managed intraoperatively with the intention of avoiding episodes of high myocardial work, and all were managed up to the time of operation with a calcium-channel or β -blocking drug. Thus the observed pre-bypass systolic overshoot in this study is likely to be less than the overshoot that may occur under other circumstances. Similarly, our finding that the long-tubing system as used clinically contributes little to the systolic overshoot pertains specifically to the circumstances of this study. In other circumstances where the cardiac contractions may be brisk, the tubing system may contribute substantially to the systolic overshoot.⁸ The diastolic pressure, measured at a time when the pressure is changing slowly, is typically not affected by the frequency response of the arteries and the tubing system. The mean pressure is also unaffected by the high-frequency response, assuming no net energy loss.

Systolic overshoot arising in the tubing system can be minimized by the use of short tubing, but this is not practical in many circumstances. A mechanical device may be used to reduce the high-frequency response and to increase the damping in the tubing system.¹⁷ While the importance of obtaining an accurate radial artery pressure trace should not be minimized, our study demonstrates significant systolic errors resulting from the use of the radial artery per se, even in the absence of errors resulting from the use of long tubing.

Systolic pressure exaggeration in the radial artery may be particularly relevant when considering the systolic pressure as an index of myocardial work. The original studies that showed the rate-pressure product¹⁸ or systolic pressure¹⁹ to be correlated with myocardial oxygen consumption during anesthesia used central arterial pressure, whereas a similar more recent study showing no correlation used radial artery pressure.²⁰ The absence of correlation in the later study may simply be related to the error inherent in the use of the radial artery.

The second type of error occurs at the end of cardiopulmonary bypass, when the radial artery pressure is generally lower than the central arterial pressures. The extent of the error in any specific patient is variable and unpredictable. The inherent error in the radial pressure under these circumstances should preclude the use of radial artery pressures for studies of hemodynamics in the immediate post-bypass period, and we should critically review our previous findings about post-bypass hemodynamics in studies where these findings were based on the use of radial artery pressures.

The clinical effect of the error in radial artery pressures after rewarming on bypass and in the immediate post-bypass period will depend on the magnitude of the error

and on the patient's condition. Certainly a relatively healthy patient can sustain without harm actual central arterial pressures that are higher than the measured radial pressures. However, in patients capable of limited myocardial work, precise hemodynamic measurements may be important to a good result, and in such patients a substantial error arising from use of the radial artery can be a clinical problem. When an adequate arterial pressure is not easily achieved during weaning from cardiopulmonary bypass, confirmation of the radial pressure by measurement of a central arterial pressure is often indicated and may permit more appropriate management. In this study and in previous studies the radial and aortic mean pressures were similar by the end of the surgical procedure. Thus the radial artery appears to be an adequate measure of the central pressure for postoperative monitoring of MAP.

Alternative sites of cannulation for blood pressure measurement should be considered. Despite the proximity of important structures and the potential for major complications, axillary and femoral sites can be safely used.¹³ The best site for routine cannulation may prove to be the brachial artery since complications are infrequent and typically minor,²¹ and since the use of a long catheter (15 cm) placed in the brachial artery may avoid much of the error inherent in radial artery pressure measurements.⁷

MICHAEL G. BAZARAL, PHD, MD
Department of Cardio-Thoracic Anesthesia
The Cleveland Clinic Foundation
One Clinic Center
9500 Euclid Avenue
Cleveland, Ohio 44195

REFERENCES

- Wood EH. Physical response requirements of pressure transducers for the reproduction of physiological phenomena. *Trans American Institute of Electrical Engineers. I: Communications and Electronics* 1956; 75:32-40.
- McDonald DA. *Blood Flow in Arteries*. Baltimore, Williams & Wilkins, 1960, pp 199-274.
- Bruner JMR, Krenis LJ, Kunsman JM, Sherman AP. Comparison of direct and indirect methods of measuring arterial blood pressure. *Medical Instrumentation* 1981; 15:11-21.
- Stern DH, Gerson JL, Allen FB, Parker FB. Can we trust the direct radial artery pressure immediately following cardiopulmonary bypass? *Anesthesiology* 1985; 62:557-561.
- Gallagher JD, Moore RA, McNicholas KW, Jose AB. Comparison of radial and femoral arterial blood pressures in children after cardiopulmonary bypass. *J Clin Monit* 1985; 1:168-171.
- Becker B, LaFontaine E, Lin CY. Accuracy of radial artery pressure monitoring. *Society of Cardiovascular Anesthesiologists Abstracts*, 1983, p 198.
- Gravlee GP, Brauer S, Johnston WE, et al. Simultaneous comparison of aortic, femoral, and brachial arterial pressures during cardiac surgery. *Society of Cardiovascular Anesthesiologists Abstracts*, 1985, p 69.
- Boutros A, Albert S. Effect of the dynamic response of transducer-tubing system on accuracy of direct blood pressure measurement in patients. *Crit Care Med* 1983; 11:124-127.
- Adler DC, Bryan-Brown CW. Use of the axillary artery for intravascular monitoring. *Crit Care Med* 1973; 1:148-150.
- DeAngelis J. Axillary arterial monitoring. *Crit Care Med* 1976; 4:205-206.
- Schonstedt R, Oehmig R. Cannulation of the axillary artery for continuous blood pressure measurement and blood gas analysis. *SO Anesth-Intensivther Notfallmed* 1982; 17:237-239.
- Bryan-Brown CW, Kwun KB, Lumb PD, Pia RLG, Azer S. The axillary artery catheter. *Heart and Lung* 1983; 12:492-497.
- Gurman GM, Kriegerman S. Cannulation of big arteries in critically ill patients. *Crit Care Med* 1985; 13:217-220.
- Taylor BC, Ellis DM, Drew JM. Quantification and simulation of fluid-filled catheter/transducer systems. *Medical Instrumentation* 1986;

- 20:123-129.
15. Gordon V, Welch J, Carley D, Teplick R, Newbower R. Zero stability of disposable and reusable pressure transducers. *Medical Instrumentation* 1987; **21**:87-91.
 16. Bruner JMR, Krenis LJ, Kunsman JM, Sherman AP. Comparison of direct and indirect methods of measuring arterial blood pressure, part III. *Medical Instrumentation* 1981; **14**:182-184.
 17. Gardner RM. Direct blood pressure measurement-dynamic response requirements. *Anesthesiology* 1981; **54**:227-236.
 18. Wilkinson PL, Moyers JR, Ports TA, et al. Halothane and morphine-nitrous oxide anesthesia in patients undergoing coronary artery bypass operation. *J Thorac Cardiovasc Surg* 1981; **82**:372-382.
 19. Sonntag H, Merin RG, Donath U, Radke J, Schenk HD. Myocardial metabolism and oxygenation in man awake and during halothane anesthesia. *Anesthesiology* 1979; **51**:204-210.
 20. Moffitt EA, Sethna DH, Gray RJ, DeRobertis M, Matloff JM, Bussell JA. Rate-pressure product correlates poorly with myocardial oxygen consumption during anesthesia in coronary patients. *Can Anaesth Soc J* 1984; **31**:5-12.
 21. Mann S, Jones RI, Millar-Craig MW, Wood C, Gould BA, Raftery EB. The safety of ambulatory intra-arterial pressure monitoring: a clinical audit of 1000 studies. *Int J Cardiol* 1984; **5**:585-597.

