Does Radial Artery Pressure Accurately Reflect Aortic Pressure?*

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Study Objective: Our objective was to determine whether the systolic, diastolic, and mean arterial pressures measured in the radial artery accurately reflect corresponding pressures in the ascending aorta in narcotic-anesthetized patients with known obstructive coronary artery disease, before being subjected to cardiopulmonary bypass (CPB).

Design: This was a prospective study.

Setting: The cardiac operating room of a large, tertiary-care university medical center.

Participants: Fifty-one patients (45 men and six women; age range, 45 to 77 years) with documented atherosclerotic coronary artery disease were studied. All patients underwent elective coronary artery bypass grafting after the study.

Interventions: Patients were premedicated with lorazepam and morphine 60 min before administration of Fentanyl-pancuronium anesthesia. The radial artery was cannulated before induction of anesthesia and the aorta approximately 45 min later. Comparisons of radial and aortic pressures were then performed.

Measurements and Results: Radial and aortic pressures were recorded through standard, fluid-filled, high-pressure, 91-cm (36-in) long tubing and disposable transducers, meticulously cleared of air bubbles. Additional measurements included cardiac output, central venous pressure, core temperature, blood gas levels, and hematocrit reading. Radial-aortic pressure differences were as follows: systolic arterial pressure (SAP), 12±1 mm Hg; mean arterial pressure (MAP), −0.8±0.3 mm Hg; and diastolic arterial pressure (DAP), −1.0±0.3 mm Hg. All were significant (p<0.001), but the SAP difference was more than ten times that of either the MAP or the DAP values. The coefficients of determination (r²) indicated that the radial-aortic dependence was 0.44 for the SAP, 0.90 for the DAP, and 0.98 for the MAP relationship. Plotting the respective differences against the arithmetic mean of simultaneously measured pressures indicated that the radial SAP was 4 to 35 mm Hg higher than the aortic in 42 patients (82 percent) and was 10 to 35 mm Hg higher in 26 patients (51 percent); radial-aortic MAP differences clustered within 3 mm Hg in 47 patients (92 percent); radial DAP was ±3 mm Hg different from the aortic in 46 patients (90 percent). The largest MAP difference was −6 mm Hg in one patient. The largest DAP difference was ±5 mm Hg in three patients.

Conclusions: In this group of patients, who were studied before undergoing CPB, the radial SAP gave a poor estimate of that present in the ascending aorta, since in more than 50 percent of the cases, the radial SAP was 10 to 35 mm Hg higher than that in the aorta. The radial MAP and DAP are reliable, since in 90 percent and 92 percent of the patients, respectively, the pressure differences were within ±3 mm Hg of those in the aorta.

(Chest 1992; 102:1193-98)

In critically ill patients and in those undergoing major surgery, blood pressure is monitored invasively to ensure that the perfusion pressure needs of vital organs, eg, brain, heart, kidneys, etc, are satisfied. Ideally, we would like to know the pressure in the vessels that perfuse these organs or the pressure in the aorta, but since it is seldom feasible to measure the aortic pressure, the radial artery is commonly used. Since blood flows from the heart to the periphery under pressure, it is reasonable to expect the aortic pressure to be higher than the radial pressure. In fact, it has been shown that the mean arterial blood pressure (MAP) is consistently 1 to 3 mm Hg higher in the aorta than peripherally,1 except in cases of proximal stenosis or distal shunting.2 On the other hand, it has been shown in dogs3 and in awake humans4 that the systolic arterial pressure (SAP) could be up to 40 mm Hg higher in the radial artery than in the aorta. Recent work in humans and animals4-6 indicates that the systolic pressure wave can be markedly modified by summation of peripherally reflected waves, while work in an electronic model7 shows that in the absence of reflected waves, resonance of the brachio-radial artery tree can produce a considerable difference in radial-aortic values for SAP.

It seems then that there is sufficient published evidence indicating that the radial SAP is not a reliable variable by which to treat patients whose pathology or severity of operation indicates invasive monitoring of blood pressure; however, in our medical center, most discussions of the treatment of such patients, in clinical rounds or case conferences, center around the SAP and diastolic arterial pressure (DAP). In order to

DAP = diastolic arterial pressure; MAP = mean arterial pressure; PVR = peripheral vascular resistance; SAP = systolic arterial pressure; SVR = systemic vascular resistance

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discard this practice as a local anachronism, we interviewed 11 senior faculty members of our anesthesia and surgical departments who had participated in at least two visiting professorships at teaching institutions in the United States during the last two years. We approached the faculty members individually and explained our interest in finding out the prevalence of blood pressure presentation. We clarified that we were interested in invasively monitored patients when their management was being discussed either during intensive care rounds or in anesthesia case conferences. The faculty members were asked which pressures were routinely mentioned and which were mentioned on special occasions. In 19 of 20 visited institutions, the SAP and DAP were presented first. The MAP was mentioned only in relation to perfusion pressures and when the systemic vascular resistance (SVR) was deemed important. Interestingly, an orthopedic surgeon, a trauma surgeon, and an intensive care specialist volunteered that when only one number was given, it was understood to be the SAP. One reason for this preference might rest on tradition, but another could be the lack of recent, simultaneous radial-aortic clinical comparisons, other than after cardiopulmonary bypass, when the SAP at the radial artery is sometimes lower than the aortic, for reasons that are still unclear.

Additionally, the radial DAP is commonly used as an indicator of aortic DAP, when estimating the required pressure for coronary perfusion; however, to our knowledge, simultaneous measurement of DAP at these two sites has been done only in ten patients in cardiac failure.5 The object of this study was to assess whether augmentation of the aortic SAP at the radial artery persisted during narcotic anesthesia and whether the radial DAP was lower than the aortic.

**Materials and Methods**

After this study was approved by the institution’s Clinical Research Practices Committee, written consent was obtained from 51 consecutive patients who were to undergo coronary bypass grafting. All had obstructive coronary artery disease. They were studied before being placed on cardiopulmonary bypass. Radial artery and ascending aortic pressure waveforms were recorded simultaneously. No patient required isotropic support or vasodilators before induction of anesthesia or during the study. Patients with unequal brachial pressures as determined by left and right cuff pressure measurements, symptoms of peripheral artery disease (ie, claudication), or ejection fraction of less than 50 percent, as well as those with aortic arch calcification, were excluded. Preanesthetic medication consisted of lorazepam (0.05 mg/kg) and morphine (0.1 mg/kg) administered approximately 60 min before placement of monitoring lines. These lines consisted of radial and pulmonary artery cannulation after local infiltration of lidocaine. Before induction of anesthesia, radial blood pressures were recorded to appraise the change produced by anesthesia and to explore their possible association with the radial-aortic pressure discrepancies during anesthesia. Fentanyl (25 μg/kg to 47 μg/kg) was the primary anesthetic at the time of the study. Pancuronium was given for muscle relaxation.

Radial and aortic pressures were measured through 5.1-cm, 20-gauge Teflon catheters. For the aortic measurements, approximately 2.5 cm of the catheter was inserted into the ascending aorta, pointing distally. Both catheters were attached to individual tubes that were 91.4 cm long, 1.8 mm in inner diameter, and 0.9 mm in wall thickness and to transducers (Spectromed model T36AD-R). Both pressure measuring systems were meticulously free from air, calibrated statically to a mercury standard; and their natural frequencies and damping coefficients were determined, before and after each comparison, by the flush method described by Gardner.11 Recordings were obtained simultaneously within a period of 30 to 50 s, during which time the surgical procedure was restricted to avoid displacement of the heart, the great vessels, or the monitoring catheters and connecting tubing. Data were recorded in a thermoelectronic recorder, so labeled by the manufacturer (Siemens Medical Systems, Inc.) because it prints thermally and can register linearly signals from 0 to 500 Hz. The systolic and diastolic deflections were averaged every 9.6 s. This method allowed us to secure a minimum of three consecutive identical intervals during 30-s to 50-s recording periods. The MAP was obtained by electronic integration. Vascular resistance was calculated by the following standard formula:

\[
\text{resistance (dynes-cm}^{-2}\text{)} = \left(\frac{\text{MAP} - \text{CVP}}{\text{CO}}\right) \times 80
\]

We called it SVR when the aortic MAP was used in its calculation and called it peripheral vascular resistance (PVR) when the radial artery MAP was used.

**Statistics**

The differences within each category of data (radial-aortic SAP, MAP, DAP, PVR-SVR, frequency response, and damping coefficient) were compared by the paired two-tailed t-test. A level of p < 0.05 was considered significant. Pearson correlation coefficients were calculated for the radial-aortic SAP, MAP, DAP, and PVR-SVR relationships. Because the radial-aortic SAP difference was ten times greater than the MAP or the DAP differences, we also correlated its relationship to age, weight, core temperature, temperature of the palm of the hand, finger temperature, hematocrit reading, radial SAP before induction of anesthesia, and radial SAP during the period of study. For the same reason, a two-sample t-test on the differences between radial-aortic SAP discrepancies for patients with a history of hypertension and those without such history was performed.

The significance of the difference between each of the three categories of blood pressure data (SAP, MAP, and DAP) was evaluated by plotting the differences within each category (radial SAP-aortic SAP, etc) against the plot of their average value ([radial SAP + aorta SAP]/2). Then the McNamara's test was employed. First, we deemed the radial and aortic SAP, MAP, and DAP to be in agreement if they differed by less than 4 mm Hg (units) in view of technical aspects of aortic pressure measurements.15 Secondly, we considered that a measurement error of 10 mm Hg was clinically significant in view of previous reports.16 For calculations of the group's radial-aortic pressure differences, each patient contributed one blood pressure determination (SAP, DAP, or MAP) from the aortic and radial sites. All data are expressed as the mean ± SEM.

**Results**

Of the 51 patients studied, 45 were men, and six were women (age range, 48 to 77 yr). The radial-aortic frequency response differences (p = 0.8) and the damping coefficient differences (p = 0.4) were not statistically significant. The following tabulation presents descriptive statistics for the 51 patients:
Table 1—Comparison of Radial Artery and Aortic Pressures

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Radial Artery</th>
<th>Aorta</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAP, mm Hg</td>
<td>111 ± 2</td>
<td>99 ± 2</td>
</tr>
<tr>
<td>DAP, mm Hg</td>
<td>60 ± 1</td>
<td>61 ± 1</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>76 ± 1</td>
<td>77 ± 1</td>
</tr>
<tr>
<td>Vascular resistance, dyne·s·cm⁻²</td>
<td>1.434 ± 58 (PVR)</td>
<td>1.450 ± 60 (SVR)</td>
</tr>
<tr>
<td>Frequency response, Hz</td>
<td>24.8 ± 0.6</td>
<td>25.0 ± 0.7</td>
</tr>
<tr>
<td>Damping coefficient</td>
<td>0.3 ± 0.1</td>
<td>0.3 ± 0.1</td>
</tr>
</tbody>
</table>

Table 1 presents comparative data for the 51 patients studied. Radial SAP and DAP were 141 ± 3 and 69 ± 1 mm Hg before induction of anesthesia and decreased to 111 ± 2 and 60 ± 1 mm Hg (p<0.001) after induction of anesthesia.

Radial SAP was 12 ± 1 mm Hg higher than aortic SAP (p = 0.0001). Radial MAP was 0.8 ± 0.3 mm Hg lower than the aortic (p = 0.0066). Radial DAP was 1.0 ± 0.3 mm Hg lower than the aortic (p = 0.0007). The PVR (radial MAP) was 16 ± 5 dynes·s·cm⁻² lower than SVR (aortic MAP) (p = 0.004). Radial SAP was 4 to 35 mm Hg higher than the aortic in 42 patients (82 percent) and was 10 to 35 mm Hg in 26 patients (51 percent). The radial-aortic MAP differences clustered within 3 mm Hg in 47 patients (92 percent); in three patients, it was ± 4 mm Hg, and in one patient, it was −6 mm Hg. Radial DAP was ± 3 mm Hg different from the aortic in 46 patients (90 percent). The highest DAP difference was ± 5 mm Hg in three patients.

Significant correlations (p<0.001) between radial and aortic values were as follows: radial and aortic SAP, r = 0.66; radial and aortic MAP, r = 0.99; and radial and aortic DAP, r = 0.90. The coefficients of determination, which indicate the percentage of dependence of one parameter upon another (not necessarily the cause) were as follows: SAP, r² = 0.44; MAP, r² = 0.98; and DAP, r² = 0.81; therefore, the radial SAP was 44 percent dependent on the aortic SAP, the radial MAP was 98 percent dependent on the aortic MAP, and the radial DAP was 81 percent dependent on the aortic DAP. Only a marginal relationship existed between the value of the radial SAP and the radial-aortic SAP difference: r = 0.4, and r² = 0.19. This indicates that the radial-aortic SAP difference is poorly dependent on the actual value of the SAP; and this was illustrated by a patient with a radial SAP of 108 mm Hg, aortic SAP of 73 mm Hg, and identical MAP and DAP. There was no significant correlation between the radial-aortic SAP difference and the preanesthetic SAP, PVR, SVR, age, weight, height, hematocrit reading, core temperature, and palmar or finger temperature (r = 0.08 to 0.19, and p>0.1). The radial-aortic pressure discrepancy in patients with a history of hypertension was 14 ± 2 mm Hg, and that discrepancy in patients with no history of hypertension was 10 ± 2 mm Hg; however, the difference between the two values was not significant (p = 0.11).

A clearer estimate of the clinical significance of the radial-aortic pressure differences was obtained by plotting the radial-aortic pressure differences against the average of these two pressures (Fig 1). The mean differences (± 2 SD) for the SAP, MAP, and DAP were 12 mm Hg (range, −7 to 31), −0.8 mm Hg (range, −5 to 3), and −1.0 (range, −5 to 3), respectively.

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21656/ on 06/21/2017)
Although most differences fitted within the average difference ±2 SD, the SAP and MAP had two outliers, and the DAP had three outliers; thus, the distribution of the differences did not fit a normal distribution. Therefore, we tested the significance of the differences by the McNamara's test. By this test, when a difference of 4 mm Hg or more was considered a significant error, that of the SAP was significantly higher (p<0.0001) than that of the MAP and the DAP, while the MAP and DAP differences were not statistically different (p=0.083). Only one set of MAP measurements and four sets of the DAP measurements differed by more than 4 mm Hg, whereas 38 sets of SAP measurements differed by more than 4 mm Hg. When a difference, or error, of 10 mm Hg was considered clinically unsatisfactory, SAP vs MAP and SAP vs DAP were statistically significant (p<0.0001). Neither the MAP nor the DAP differences reached 10 mm Hg; however, the radial artery SAP was 10 mm Hg or more higher than the aortic in 26 comparisons (51 percent).

**Discussion**

This study, conducted in narcotic-anesthetized patients, shows that the radial SAP is higher than that of the ascending aorta, as has also been found in dogs and awake humans. It also confirms that in most patients the MAP is 1 to 3 mm Hg higher in the aorta than peripherally. Considering that a radial-aortic pressure difference of 4 mm Hg or more is required in order to regard these two pressures as dissimilar, because of the effect of kinetic energy on the aortic lateral pressure, the SAP at both sites was similar in only 18 percent (9 of the cases), while the MAP was similar in 92 percent (47). Clinically, this means that during narcotic anesthesia, the radial MAP reading was 4 to 6 mm Hg lower than the aortic pressure in 8 percent (4) of the patients, while the radial SAP underestimated that in the aorta by 4 to 35 mm Hg in 82 percent (42) of the patients and by 10 to 35 mm Hg in 51 percent (26) of the patients. These differences may be very important and could alter therapy in some patients; eg, one of the patients with the highest radial-aortic systolic pressure difference (35 mm Hg) had a radial SAP of 108 mm Hg and identical MAP values (62 mm Hg). Because he was extensively monitored, this finding did not alter his management, but in less fully monitored patients, these differences could produce some uncertainties.

The results of this study are in agreement with those of Schwid et al, who studied the behavior of the aortic and radial pressure waveforms on an electronic model. The electronic circuit modeled the resonant networks for the aorta and axillo-brachial-radial arterial system. They found that the radial artery pressure waveform could reproduce only the aortic MAP and DAP. This model, unlike ours, excluded wave reflection and the effect of fluid-filled catheters; however, their radial and aortic pressure tracings were very similar to ours. These authors also demonstrated that the natural frequency of a normal radial artery lies around 3 Hz and that of a stiff artery around 6 Hz, the stiff artery being more underdamped than the normal radial artery. Considering that a manometer system with a natural frequency of 7 Hz or less is unsatisfactory for measuring central pressures because it will distort the measured pressure waveform, the radial artery pressure waveform is a reproduction of that present in the aorta, recorded through an underdamped catheter-manometer system. The radial artery's own oscillations distort the aortic pressure waveform in the same way that an underdamped catheter-manometer system can distort the radial or any other pressure waveform.

Because any degree of atherosclerosis and high sympathetic tone can alter wave reflection and resonance in the arterial tree, thus distorting the similarity between the aortic and radial pressure waveforms, we attempted to find some correlations with parameters suggestive of vascular stiffness. We found that the radial-aortic pressure difference was greater in patients with a history of hypertension (although not statistically significant) than in those without such a history; that there was a positive correlation between this difference and the radial systolic pressure, although the coefficient of determination was only 0.19, and the patient with the widest difference had an SAP of 108 mm Hg. There were no other correlations, although sympathetic tone, age, hematocrit reading, core temperature, etc, are factors capable of distorting the radial SAP. Thus, at present, it is not possible to predict with accuracy which patients will show the greatest radial-aortic systolic difference. Therefore, the most reasonable conduct would be to change our clinical trust from the radial SAP to the radial MAP.

Although we studied patients who were to undergo coronary artery bypass operations, these observations were carried out before institution of cardiopulmonary bypass. Therefore, these results could be applied to patients in similar age groups undergoing major operations, eg, resection of an abdominal aortic aneurysm, carotid endarterectomy, or abdominal or thoracic surgery, or those in whom vasopressors are used during major surgery or in the intensive care unit; however, direct confirmation of these findings in other patients, particularly in those not anesthetized, is required.

Although we compared three differences in blood pressure, they belong to three different categories of data, and their distributions did not follow a normal curve; therefore, statistical comparison by analysis of
variance was not appropriate. Results of the paired t-
test, when applied to assess the significance of the
radial-aortic differences in each category, indicated
that the three differences were highly significant;
however, the SAP difference was ten times that of the
MAP and DAP. This is not surprising, since in 41 of
the 51 patients, the aortic MAP and DAP were higher
than the radial values, although the differences were
very small. The correlation coefficients show a much
higher coefficient of determination for the MAP and
DAP values than for SAP values. Comparison of the
differences following the method of Bland and
Altman for two methods of measurement has the
advantage of introducing clinical judgment to accept
or reject these results. Under the conditions of the
study, where the radial SAP had already decreased
from 141 mm Hg before induction of anesthesia to
111 mm Hg, a radial SAP 10 mm Hg in error could
be considered unacceptable. This method allows us
to see that the radial and aortic pressures could be
considered identical in 18 percent (9) of the patients
for the SAP, 92 percent (47) for the MAP, and 90
percent (46) the DAP. Furthermore, while the radial
SAP was at least 10 mm Hg in error, neither the MAP
nor the DAP reached this magnitude.

We used fluid-filled systems to compare the pres-
sures at the radial and ascending aorta in order to
display the radial blood pressure as it is commonly
monitored in a clinical environment. By decreasing
the length of the pressure tubing from the standard
122 cm (48 in) to 91 cm (36 in) and by meticulously
removing air bubbles from the transducer-tubing sys-
tem, we obtained frequency responses that exceeded
those proposed by Sykes and associates (26 Hz for a
heart rate of 180 beats per minute) and Shinozaki et
al. (20 Hz for a heart rate of 120 beats per minute).
Extrapolating from these values, the maximum fre-
quency response for a heart rate of 65 beats per minute
would be 10 Hz, and 15 Hz for a heart rate of 90 beats
per minute. The heart rate in our patients was 65 ± 2
beats per minute. In two patients where the heart rate
was 90 beats per minute, the frequency responses in
their pressure measuring systems were 25 and 27 Hz,
respectively. It could be argued that in this age of
advanced technology, blood pressure studies should
be done using high-frequency response micromanome-
ters (>100 Hz). This assertion would be pertinent to
our study had we attempted to evaluate the role of the
brachial-radial resonance and of wave reflection on the
aorta-radial pressure difference. This would be a
challenging task, since the natural frequency of the
isolated radial artery lies between 3 and 6 Hz, while
the minimal frequency response required to measure
central pressures satisfactorily is 7 Hz. Thus, a
micromanometer would measure a waveform already
deformed by the brachial-radial resonance at the wrist.

This task is beyond our present expertise.

This study was not concerned with the cuff methods
of blood pressure monitoring; however, these tech-
niques, using either auscultation or oscillometry, are
frequently used for the assessment of the hemody-
namic state of patients. These methods, unlike the
radial artery cannulation, measure lateral pressure,
and provided that the cuff is of appropriate size and
properly positioned, systolic, diastolic, and mean
pressures should be unaffected by wave summation or
by the brachial-radial artery tree resonance. The
main causes of SAP deformation are wave reflection
and wave distortion by resonance of the
brachial-radial tree; and because cuff methods are not
affected by these factors, the SAP so measured should
be less distorted than that measured directly in the
radial artery. The oscillometric method, which is used
in some commercial automatic monitors (Dinamap
model 845), determines reliably SAP, DAP, and MAP
values, provided the cuff is not subjected to displace-
ment during the measurement. The auscultatory
method, using the Korotkoff sounds, determines only
SAP and DAP values and derives from them the
MAP.

This study shows that the higher radial than aortic
SAP pressure known since 1955 is to be present in
awake humans is also present in Fentanyl-anesthetized
patients. The reassuring findings of clinical signifi-
cance are that the radial MAP reading is equal to or 3
mm Hg lower than that measured in the aorta in 92
percent of the patients under narcotic anesthesia;
secondly, the radial DAP correctly measures the aortic
DAP in 90 percent of the patients and differs at most by
± 5 mm Hg in 10 percent of the cases. On the other
hand, the radial SAP is not only inaccurate, but also
grossly overestimates that at the ascending aorta in
more than 50 percent of the patients. Worst of all,
the highest overestimation may occur in patients with
already borderline radial SAP, as pointed out in our
example.

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