The second peak of the radial artery pressure wave represents aortic systolic pressure in hypertensive and elderly patients

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Background. Simultaneous measurement of ascending aorta and radial artery pressure shows that mean and diastolic pressures (DP) are in close agreement in normotensive adults, while systolic pressures (SP) are not. However, in the aortic pressure wave, a second systolic peak appears with increasing age and increases to represent the SP by age 32 yr, while in the radial artery, a second systolic deflection appears by age 40 yr. We suggest that the second radial systolic wave, sometimes seen during radial arterial pressure monitoring in older hypertensives, represents the aortic SP. We set out to evaluate whether the aortic and radial second systolic peaks agree, and since doubts exist about the agreement between aortic and radial DP in elderly hypertensive patients, we also assessed that relationship.

Methods. We compared simultaneously recorded radial and aortic pressures from 21 anesthetized adult patients using identical fluid-filled pressure measuring systems.

Conclusions. The second radial pressure peak agreed with that in the aorta within a mean of 0.6 (SD 1.5) mm Hg. The difference between DP in the aorta and radial artery was −1.4 (2) mm Hg. The radial–aortic SP and pulse pressure differences were 5.9 (7.6) and 7.3 (7.6) mm Hg, respectively. These results confirm that when the radial artery pressure wave shows a first and second, or only a second systolic deflection (on the right side of the pressure wave), the second represents the maximal ascending aortic SP, and that the radial and aortic DP are equivalent, even in older hypertensive patients.

Keywords: age factors; arterial pressure, hypertension; heart, radial systolic pressure; heart, systolic pressure differences

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Since the work and oxygen demand of the left ventricle are directly related to aortic systolic pressure (SP),1–4 and myocardial oxygen supply might be impaired in some patients,5 a measure of aortic SP would be of value during major surgery. Radial artery pressure is usually monitored, but this can be significantly greater than aortic pressure in normotensive adults.6 This may be because SP is taken as the first peak of pressure in the radial artery6 and by the second peak of pressure in the aorta in adults over 32 yr.8 However, a second systolic deflection is noted on the radial artery pressure wave by age 40 yr, which approaches the magnitude of the first by the eighth decade.7 Takazawa and colleagues8 found that nitroglycerin equally reduced aortic SP and the second systolic peak of radial pressure. This suggests that the second radial artery deflection represents aortic SP. The authors4 suggested direct comparison of aortic and radial artery pressure waves to confirm this, but did not carry it out. There have also been concerns recently about agreement between diastolic pressure (DP) measured in the radial artery compared with the aorta in elderly hypertensive patients.9

Using identical pressure systems to record pressure in the aorta and radial artery, we assessed agreement between aortic pressure and the second deflection of pressure in the radial artery, and the agreement between DP in the radial artery and aorta in older5 or hypertensive patients.
Methods

The study was approved by the institution’s Clinical Research Practices Committee, and written informed consent was obtained from all patients before they were considered for the study. We studied 45 patients undergoing coronary artery surgery in whom we noted a second systolic deflection in the radial artery pressure wave. The patients were aged 41–87 yr, with 70% over 60 yr. Isolated systolic hypertension (SP $>140$ and DP $<90$ mm Hg) was frequent (78%). All were receiving antihypertensive medication including angiotensin-converting enzyme inhibitors diuretics and β-blockers (atenolol or metoprolol). Before anaesthesia, we excluded patients with brachial SP differences of 5 mm Hg or more measured twice on both arms by sphygmomanometry, those receiving nitroglycerin infusion, and those in whom the radial artery was not cannulated before anaesthesia. During anaesthesia, we excluded patients in whom we could not see a second systolic peak on the monitoring oscilloscope (Fig. 1). Thus, we compared radial and ascending aorta pressure waves in 21 patients in whom a second systolic peak was seen in the radial artery following 60 (mean, SD 12) min of general anaesthesia, chest surgery and heparin administration in preparation for cardiopulmonary bypass (Table 1).

Before induction of anaesthesia, we cannulated a radial artery and measured the pressure. We noted SP, DP and mean arterial pressure, and whether the wave showed a clear second systolic peak. Anaesthesia was established with fentanyl, isoflurane, midazolam and pancuronium for muscle relaxation, and heparin 300 units kg$^{-1}$ was given. We recorded radial and aortic pressure simultaneously for 30–40 s at 60 (12) min after induction of anaesthesia. Both pressures were measured through 5.1 cm, 20G Teflon catheters. The catheter in the ascending aorta was placed pointing against the flow at the aortic site chosen for cannulation. Both catheters were attached to individual high-pressure tubes, 91 cm long, 1.8 mm inner diameter (exposed over their whole length to the same air temperature) and to matched Transpac7 IV transducers (Abbott Critical Care Systems, Abbott Laboratories, North Chicago, IL, USA). The pressure measuring systems were calibrated statically to a mercury standard and kept meticulously bubble-free. The natural frequency and damping coefficients of the systems were measured by the flush method at the beginning and end of each recording in each patient, ensuring a natural frequency at least 20 Hz and damping coefficient of at least 0.2. Both transducers were maintained at the same vertical level, checked by their equal zero line display on the oscilloscope and recorder. The pressures were printed on a high-fidelity recorder (Siemens Medical Systems, Iselin, NJ, USA) which was linear between 0 and 500 Hz. The recorder was calibrated so that 100 mm Hg v$^{-1}$ output from the signal conditioner printed at 100 mm Hg per

### Table 1 Data from the 21 patients whose radial artery and aortic pressure waves were compared. Data are mean (sd)

<table>
<thead>
<tr>
<th>Control</th>
<th>Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr) (range)</td>
<td>64 (12) (41–87)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>82 (17)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170 (8)</td>
</tr>
<tr>
<td>Male/female</td>
<td>17/4</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>53 (10)</td>
</tr>
<tr>
<td>Heart rate (beats min$^{-1}$)</td>
<td>61 (10) 60 (12)</td>
</tr>
<tr>
<td>Core temperature (°C)</td>
<td>36 (1)</td>
</tr>
<tr>
<td>Arterial carbon dioxide tension (mm Hg)</td>
<td>39 (4)</td>
</tr>
<tr>
<td>Arterial oxygen tension (mm Hg)</td>
<td>236 (92)</td>
</tr>
<tr>
<td>Cardiac output (litre min$^{-1}$)</td>
<td>4.9 (1.6)</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>8 (2)</td>
</tr>
<tr>
<td>Diastolic pulmonary artery pressure (mm Hg)</td>
<td>11 (4)</td>
</tr>
<tr>
<td>Systolic pressure (mm Hg)</td>
<td>151 (19)</td>
</tr>
<tr>
<td>Diastolic pressure (mm Hg)</td>
<td>73 (13)</td>
</tr>
<tr>
<td>Mean pressure (mm Hg)</td>
<td>99 (12)</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>78 (21)</td>
</tr>
</tbody>
</table>
cm. This procedure included 50 mm Hg increments to 200 mm Hg per 2 cm for both radial and aortic recordings. Mean pressure was obtained by electronic integration every 9.6 s. Pulse pressure was calculated as the difference between SP and DP. To measure the difference between radial and aortic second systolic peaks, 10 s records from before the printed SP, DP and mean pressures (recorded at 25 mm s⁻¹ and 10 mm Hg mm⁻¹) were copy-enlarged onto 50 or 100 mm s⁻¹ and 5 or 2.5 mm Hg mm⁻¹ transparent celluloid as well as standard copy paper. These enlargements were superimposed on each other, so that the height of the radial and aortic second systolic peaks and the alignment of the gridlines on both recordings could be compared (Fig. 2).

Statistics
We used the paired t-test to compare radial and aortic second systolic peaks, SP, DP, pulse and mean arterial pressures. P<0.05 was considered significant. We plotted the difference between radial and aortic pressure against the arithmetic mean of these values to display graphically both the size of the difference between the radial and aortic pressures measured and their maximal differences (Bland–Altman plot). All data are expressed as mean (SD).

Results
No patients had unequal brachial arterial pressures out of 50 candidates for this study. However, three patients were given i.v. nitroglycerin overnight and in two of these patients, the radial artery was cannulated after induction of anesthesia; these five patients were not included in the study. In the remaining 45 patients, the radial artery pressure had a second systolic peak before anesthesia, which was greater than the first in 13, equal in 10 and lower in 22. During the next 60 (20) min of anesthesia and surgery, when the aortic pressure could also be recorded, the second radial systolic peak decreased, losing its definition in 24 patients (Fig. 1). In the remaining 21 patients (71% were over 60 years), the second radial artery systolic peak kept its definition, being higher than the first in 4, equal in 4 and lower in 13. In these patients, the radial–aortic second systolic peak difference was 0.6 (1.5) mm Hg and non-significant, whereas the differences between radial and aortic SP and pulse pressures were large and significant (P<0.01 and P<0.001, respectively) (Table 2). The aortic DP and pulse pressures were 64 (9) mm Hg and 60 (17) mm Hg, respectively, and heart rate was 60 (12) beats min⁻¹ (Table 2).

Comparisons of SP, pulse pressure, DP and mean pressures in the radial artery and aorta by Bland–Altman plots showed similar disagreements (SP and pulse pressure) and agreements (DP and mean pressure) found in two previous studies. The differences between the radial and
Aortic second systolic peaks showed that all differences fitted within the average difference ± 2SD (Fig. 3).

Figure 1 shows the changes in the radial artery pressure waveform in one of the 24 patients excluded from the study during anaesthesia. This patient was selected for this illustration because he was one of three whose radial artery pressure wave was distorted to the point of resembling the aortic pressure wave when recorded at the beginning of anaesthesia, and reverted to that seen in normotensive older patients during anaesthesia. This patient was a 65-yr-old hypertensive being treated with atenolol, who had coronary artery disease and unstable angina but no other known health problems. Control SP and DP (not shown in the figure) were 176 and 80 mm Hg, respectively, and the heart rate was 52 beats min⁻¹. Fifteen min after induction of anaesthesia (panel A), the radial pressure showed a discrete first systolic peak and a well-defined late systolic peak, which was greater. Thirty-five min after chest incision (panel B), the pressure wave had two systolic peaks, the second slightly lower than the first, but still identifiable as a shoulder. Five min later and 2 min after administration of heparin 300 units kg⁻¹ (panel C), the second systolic shoulder became a convexity, while SP and DP decreased. Three min later (panel D), pressures were minimally lower than in trace C, the second systolic peak was completely absent and the incisura was noticeably lower than its counterpart on the simultaneously recorded aortic pressure (panel E). Full details are given in Table 3. The most obvious changes in Fig. 1 from panels A to B were the decrease of the second systolic deflection, markedly reducing SP with minimal decrease in DP, the smoothing of the postincisura deflection and the increase in heart rate. Another noticeable change was the gradual loss of detail in the radial pressure wave as anaesthesia proceeded, so that by the time it could be compared with the aortic pressure wave, there was little resemblance between the two, as can be seen from panels D and E. The dissimilarity between the radial and aortic pressures was common to all patients in whom the second radial systolic deflection was blurred or absent. In the aortic pressure, the second systolic deflection represented SP in all 21 patients who had a clear second radial systolic shoulder/peak and in 18 of the 24 in whom the second deflection was replaced by a convexity.

### Table 3

<table>
<thead>
<tr>
<th>Time of event</th>
<th>SP/DP (mm Hg)</th>
<th>Peak pressure (mm Hg)</th>
<th>DP/SP ratio</th>
<th>Heart rate (beats min⁻¹)</th>
<th>Cardiac output (litre min⁻¹)</th>
<th>Diastolic pulmonary artery pressure (mm Hg)</th>
<th>Second systolic peak</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (RA)</td>
<td>164/78</td>
<td>86</td>
<td>0.48</td>
<td>49</td>
<td>4.7</td>
<td>13</td>
<td>&gt;first</td>
</tr>
<tr>
<td>B (RA)</td>
<td>125/70</td>
<td>55</td>
<td>0.56</td>
<td>74</td>
<td>5.0</td>
<td>11</td>
<td>&lt;first</td>
</tr>
<tr>
<td>C (RA)</td>
<td>96/55</td>
<td>43</td>
<td>0.55</td>
<td>71</td>
<td>4.6</td>
<td>11</td>
<td>undefined</td>
</tr>
<tr>
<td>D (RA)</td>
<td>95/52</td>
<td>43</td>
<td>0.55</td>
<td>76</td>
<td>4.7</td>
<td>12</td>
<td>absent</td>
</tr>
<tr>
<td>E (AA)</td>
<td>81/52</td>
<td>29</td>
<td>0.64</td>
<td></td>
<td></td>
<td></td>
<td>&lt;first</td>
</tr>
</tbody>
</table>

Three min later (panel D), pressures were minimally lower than in trace C, the second systolic peak was completely absent and the incisura was noticeably lower than its counterpart on the simultaneously recorded aortic pressure (panel E). Full details are given in Table 3. The most obvious changes in Fig. 1 from panels A to B were the decrease of the second systolic deflection, markedly reducing SP with minimal decrease in DP, the smoothing of the postincisura deflection and the increase in heart rate. Another noticeable change was the gradual loss of detail in the radial pressure wave as anaesthesia proceeded, so that by the time it could be compared with the aortic pressure wave, there was little resemblance between the two, as can be seen from panels D and E. The dissimilarity between the radial and aortic pressures was common to all patients in whom the second radial systolic deflection was blurred or absent. In the aortic pressure, the second systolic deflection represented SP in all 21 patients who had a clear second radial systolic shoulder/peak and in 18 of the 24 in whom the second deflection was replaced by a convexity.

### Discussion

In this study, the comparison of radial and aortic pressure waves in 21 patients shows that the second systolic peaks agree within 0.6 (1.5) mm Hg. Thus, these results support the view of Takazawa and colleagues⁴ that the second rather than the first component (shoulder/peak) of the radial pressure represents aortic SP in adult patients. These authors had compared the effect of nitroglycerin on the reduction of the aortic maximal SP and the second radial artery systolic peak in 24 adults, finding that this treatment reduced the aortic pressure by 22 (13) mm Hg and the second radial artery systolic shoulder by 24 (13) mm Hg, and that the difference between these reductions was statistically insignificant. However, they had compared aortic pressure recorded by microtip catheter, with aortic pressure derived (through a process of averaging digitized pressure waves and using elements of the fourth derivative to identify the first and second systolic peaks) from radial pressures recorded by applanation tonometry, and had not measured DP in the radial artery. Therefore, they did not compare the aortic and true radial pressure waveforms, and suggested that analysis of the simultaneously recorded analogue aortic pressure...
and radial pressures would permit recognition of their first and second systolic deflections, and confirmation of the relationship between aortic pressure and the second radial wave. Then, it would be seen that the lowering effect of vasodilator treatment on the aortic pressure agreed with that on the second radial wave. We have also derived aortic SP from digitized radial pressure and transfer function technology in 62 anaesthetized patients. In 80% of these patients the second radial systolic shoulder was absent, and aortic SP was represented by the first systolic deflection. The difference between the measured aortic SP and that derived from the radial artery was 0.0 (4.4) mm Hg, and well within Association for the Advancement of Medical Instrumentation guidelines for comparison of arterial pressures with different methods of 5 (8) mm Hg. However, the differences in individual patients ranged between −12 and 8 mm Hg. Thus, it seems that accurate identification or derivation of the aortic SP from radial artery measurements is possible only if the treatment (e.g. anaesthesia) does not decrease the second aortic systolic peak below that of the first, or the ability of the brachial and radial arteries to transmit the aortic pressure. In the present study, we compared simultaneous recordings of the aortic and radial pressures, and found that the maximal disagreement between the aortic SP and the second RA systolic shoulder was 2 in 19 patients, 3 in one patient and −4 in one of 21 patients (Fig. 3), confirming that the second radial systolic peak represents the aortic SP in older hypertensive patients.

These data also confirm that the agreement between the aortic and radial DP in normotensive patients persists in older hypertensives. Thus, the large overestimation of directly measured brachial DP by cuff sphygmomanometry (18 (12) mm Hg in patients with isolated systolic hypertension) is not because of lower DP in the radial artery, but the inability of non-invasive pressure measuring systems to measure true DP in these patients.

Representation of the aortic SP by the second radial systolic peak implies that radial and aortic SP are equivalent when the second radial systolic peak is equal to or greater than the first. Thus, the radial and aortic SP were equal in 23 of the 45 patients (51%) before, and in 8 of 21 (38%) during anaesthesia. However, this study was not designed to assess the effect of age or treatment on the aortic and radial SP, so agreements of these estimates are provisional.

Estimates of the aortic SP and DP made from the radial artery pressure wave allow rapid assessment of the effect of reducing arterial pressure on aortic pressures in older hypertensive patients, in whom aging and hypertension increase the pulse pressure by increasing SP and decreasing DP. Arterial pressure reduction by a treatment may reduce pressures to a point where the reduction of DP (a major coronary perfusion pressure factor) is relatively greater than the reduction of SP (left ventricle work load). In the radial artery, the aortic pulse pressure is the difference between the second systolic deflection and DP, therefore observing it before and early on during anaesthesia would reveal whether the DP is greater than the pulse pressure for any SP, as it normally is in young and middle-aged normotensive adults, and what effect anaesthesia has on such a relationship. We incidentally noted in this study that aortic pulse pressure was greater than DP in more than 50% of patients before anaesthesia, and that anaesthesia improved this relationship by decreasing the pulse pressure in most patients (Fig 1, panels A, B and D), but had the opposite effect in three patients. The present findings may help in evaluating the effects of anaesthesia (or any vasoactive treatment) on the true aortic pulse pressure by comparing the DP/SP ratio produced by such treatment with known values in normotensive adults. This ratio estimates DP as a fraction of SP and is 0.66 for awake normotensive adults aged 35 (11) yr and 0.62 for normotensive, anaesthetized patients aged 54 (12) yr. In Figure 1 and Table 3, this ratio was 0.48 at the beginning of anaesthesia, increased to above 0.56 during anaesthesia, and reached 0.64 when assessed in the aorta. However, in three of the 45 patients, it decreased from 0.48 to 0.3–0.4. Although there are several possible causes of low DP in older hypertensives (low heart rate, central venous pressure and/or stroke volume), in these three patients a heart rate below 50 beats min⁻¹ seemed to be the reason.

Analysis of the radial artery waveform alone has usually been disappointing; however, simultaneous observation of radial and aortic pressure waves has shown that the late radial systolic deflection, which appears at age 40 yr and continues increasing to reach the height of the first peak by the eighth decade, is closely associated with aortic SP. Aging and hypertension increase aortic SP by increasing the second aortic systolic deflection and, coincidentally, the height of the second peripheral systolic peak also increases. Also, in older patients with arterial degeneration, the aortic pressure wave reaches peripheral arteries with minimal distortion. Thus, there are persuasive reasons for the presence of the second systolic shoulder/peak on the radial pressure wave in older hypertensive awake patients. In fact, the late tidal wave (second systolic peak) was recorded for the first time in hypertensive patients in 1872 using a mechanical sphygmmograph–polygraph applied on the radial artery at the wrist.

Recently, second systolic peaks from the left ventricle, ascending aorta and peripheral arteries have been recorded with great fidelity using fluid-filled clinical catheter systems with frequency response of 11–12 Hz and damping coefficient of 0.15. Additionally, diastolic and the second systolic shoulder features of the radial pressure pulse are included within 4–8 Hz. In contrast, signals with a high frequency content such as rate of rise, first peak and incisura of pressure waves at heart rates of 120 beats min⁻¹ require pressure measuring systems with a frequency response above 20 Hz. Therefore, we maximized the frequency response of the systems used in this study to record undistorted aortic pressure waves, which retain greater
details than clinically used radial pressure, as can be seen in Figures 1 and 2. There are clinical states when the second radial systolic deflection is poorly defined or absent in awake patients. However, the most common cause of blurred or hidden second radial systolic deflection is pressure wave amplification caused by the observer, for example by changing a pressure wave displayed on four separate channels to full-screen display where all pressures show on the same scale. This manoeuvre increases the amplitude of the displayed signal from 100 mm Hg cm⁻¹ to 25 mm Hg cm⁻¹, which straightens the rises and falls of the pressure wave, reduces the separation between the first and second systolic peaks when they are at similar levels, and changes the second shoulder into a convexity when the second shoulder is lower than the first. This manoeuvre would have turned the second radial artery shoulder in Figures 1 and 2 into poorly defined convexities. This effect arises whether microtip or high-pressure fluid-filled catheter systems are used. We verified this distorting effect by amplifying digitized pressure waves from a previous study 2–4 times.

A practical approach to identifying second radial systolic peaks is to consider the SP wave, from its foot to the incisura, as three fractions. The first systolic peak is located on the first fraction (0.08–0.12 s from the foot of the wave), and the second on the third fraction on the right (approximately 0.25 s from the foot of the pressure wave).

Limitations
In this study, the pressure waves were compared after a long period of anaesthesia and administration of a large dose of heparin, which erased the second radial systolic peak in more than half the initially sampled group. This was unavoidable because this was the only chance to record radial and aortic pressure waves concurrently with minimal changes in clinical management. Use of paper recorder instead of A/D conversion did not permit recording radial artery pressures at 25 mm s⁻¹ for long periods of time in all patients because of the large amount of paper needed. However, these limitations did not hinder the evaluation of aortic SP and DP by the second radial systolic peak/shoulder and the radial diastolic pressure, respectively.

Clinical implications
At present, the incidence of isolated systolic hypertension is as high as 54% at 50–59 yr, and 87% of those aged 60 years and over; diastolic hypertension prevails in a small number of hypertensives below 50 yr of age. Systolic hypertension is more difficult to control in older than in younger patients. Thus, most treated older hypertensives may present for anaesthesia and major surgery with SP above 140 mm Hg and DP below 80 mm Hg (85% of 45 in the present group). Reduction of SP by anaesthesia to a desirable clinical level (about 120 mm Hg) is usually easy, but can cause a low DP (e.g. below 40 mm Hg), which, if ignored, could lead to myocardial ischaemia in elderly hypertensives. However, the present findings indicate that invasive radial artery pressure monitoring provides SP and DP measurements in close agreement with those in the aorta of older hypertensives. Thus, anaesthetists can accurately estimate aortic SP and DP using the radial artery waveform, unlike epidemiologists and other clinicians who have to guess, by sphygmanometry, whether an arterial pressure of 140/70 mm Hg is 140/70 or 140/30 mm Hg in the aorta. Whether reduction of arterial pressure from ≥160/≥80 to =120/≥40 mm Hg by anaesthesia is the cause of perioperative myocardial ischemia or of adverse outcomes from coronary bypass surgery in patients with isolated systolic hypertension remains to be investigated.

In summary, we found that the second radial artery systolic peak, when visible, represents the maximal aortic SP, and that the radial and aortic DP agree in hypertensives and elderly patients.

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