Disparities Between Aortic and Peripheral Pulse Pressures Induced by Upright Exercise and Vasomotor Changes in Man

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SUMMARY

Blood pressures were recorded simultaneously from the aortic arch and radial artery using two manometric systems with identical static and dynamic sensitivities. Measurements were made in four normal young men at rest and upright exercise requiring 29, 49, 78, and 100% of maximal oxygen uptake. Average radial arterial pressure rose from 133/66 mm Hg at rest to 236/58 mm Hg at maximal exercise. At the same time, average aortic pressures were 112/68 and 154/70 mm Hg, respectively. From rest to maximal exercise, pulse pressures at central and peripheral sites increased by factors of 1.95 and 2.60, respectively. Inducing reactive hyperemia in the arm abolished peripheral amplification. This amplification also diminished with time during prolonged heavy exercise. Mean pressures were nearly identical at the two sites at any oxygen uptake; mean pressures rose from 87 to 104 mm Hg from mild to maximal exercise. We conclude that estimates of stress on aortic and cerebral vessel walls and central baroreceptors would be grossly overestimated by use of peripheral pulse pressures.

Additional Indexing Words:

Central-peripheral pressure differences Pressure regulation Oxygen uptake Reactive hyperemia Radial arterial pressure Vasodilatation Vasoconstriction

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HE INCREASE in pulse pressure down-stream from the aortic arch is well known. This phenomenon has not been fully explained. Prevailing theories emphasize the importance of pressure waves reflected back from more peripheral vasculature. The theoretical aspects of this problem are not the concern of this paper.

It is known that the disparity between central and peripheral pulse pressures can be increased by tilting. Krocker and Wood felt this to be the result of peripheral vasoconstriction. However, central and peripheral pulse pressures have not been measured simultaneously in man at levels of exertion involving marked vasoconstriction in non-working peripheral regions (for example, the arms).

Recently Marx and co-workers observed almost constant aortic pulse pressure in upright man during moderate to severe exercise. In contrast, the marked and variable rise in
Aortic and peripheral arterial pulse pressures attending similar levels of exercise is well known. Since current theories stress the importance of vasoconstriction in the enhancement of peripheral

Figure 1

Aortic and radial arterial pressures during exercise requiring 28.9, 48.8, and 77.6% (averages) of maximal oxygen uptake. Lines are broken during periods when catheters were flushed. Periods during which cold or heat was applied to the arm with the radial arterial catheter or when the brachial artery of this arm was occluded (Occl) are marked by shaded bars (width representing time). Data are coded in the upper left hand corner (D.J.) showing the regions encompassed by radial (RA) and aortic (AO) systolic and diastolic pressures. Aortic pulse pressure is the shaded region with the heavy line through this region being both radial and aortic mean pressures.

The major points are as follows: (a) radial arterial systolic pressures never represent equivalent aortic values; (b) increments in aortic pressure with increased workload were variable; (c) radial systolic pressure tended to fall toward aortic values at high workloads; (d) mean pressures were identical at both sites; (e) mean pressure fell at the start of exercise in three out of four men (R.G. showed a rise first); and (f) pressures fluctuated periodically.
pulse wave amplification, elevations in peripheral pulse pressures with exercise may largely reflect such changes.

Our purpose was to measure and compare simultaneously measured central and peripheral arterial pressures with both static and dynamic accuracy to evaluate the extent of peripheral amplification in man over a range of cardiac outputs including the maximal level. We also attempted to determine whether this amplification could be affected by external stimuli known to change the local vaso-motor state.

Methods

Subjects and Procedure

The subjects were two sedentary and two physically active university students (table 1). One man (D. J.) had been a subject in previous catheterization studies in this laboratory. All details of the study were outlined to the subjects. After a thorough physical examination, maximal oxygen uptake was determined by using the procedure and criteria of Taylor and associates. After the subjects' responses to exercise had been standardized, all went through a mock run of the entire procedure.

A modified Seldinger percutaneous insertion technique was used to place catheters in the left brachial and right radial arteries. The radial arterial catheter was advanced to approximately mid-forearm. From the brachial artery, the catheter was advanced into the proximal aorta under fluoroscopic guidance. An end-hole catheter was positioned in the ascending aorta in subjects D. J. and M. M. In R. G. and S. M., an end-hole catheter with two side holes, located 4 and 8 mm behind the tip, was positioned in the descending aorta just below the origin of the left subclavian artery. Both aortic and radial arterial catheters were flushed periodically with heparinized saline during submaximal exercise.

Exercise was carried out on a treadmill at 3.5 mph on grades of 0, 7.5 and 15% and finally, at the lowest grade at 7 mph which was shown previously to elicit a maximal oxygen uptake. The duration of the submaximal workloads was 10 to 20 min. Oxygen uptake was measured between the fifth and tenth minutes of work at these three workloads and from 1.75 to 2.75 min of the run at maximal oxygen uptake. The protocol for pressure measurements during exercise in the different subjects is indicated in figure 1, including the periods during which hot or cold towels were wrapped around the arm or when

Table 1

<table>
<thead>
<tr>
<th>Subject</th>
<th>Oxygen uptake</th>
<th>% of max</th>
<th>Heart rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>L/min</td>
<td>ml/kg min</td>
<td>O2 uptake</td>
<td>(beats/min)</td>
</tr>
<tr>
<td>D.J.*</td>
<td>1.37</td>
<td>13.9</td>
<td>26.9</td>
</tr>
<tr>
<td>21 yr</td>
<td>2.57</td>
<td>26.1</td>
<td>50.6</td>
</tr>
<tr>
<td>98.4 kg</td>
<td>4.55</td>
<td>46.6</td>
<td>89.9</td>
</tr>
<tr>
<td>191 cm</td>
<td>4.97</td>
<td>50.7</td>
<td>98.3</td>
</tr>
<tr>
<td>M.M.</td>
<td>1.34</td>
<td>16.9</td>
<td>33.1</td>
</tr>
<tr>
<td>23 yr</td>
<td>2.24</td>
<td>28.3</td>
<td>55.5</td>
</tr>
<tr>
<td>803 kg</td>
<td>3.40</td>
<td>42.9</td>
<td>84.1</td>
</tr>
<tr>
<td>181 cm</td>
<td>4.04</td>
<td>51.0</td>
<td>100.0</td>
</tr>
<tr>
<td>R.G.</td>
<td>1.44</td>
<td>16.0</td>
<td>27.5</td>
</tr>
<tr>
<td>22 yr</td>
<td>2.19</td>
<td>24.3</td>
<td>41.8</td>
</tr>
<tr>
<td>89.5 kg</td>
<td>3.47</td>
<td>38.6</td>
<td>66.4</td>
</tr>
<tr>
<td>183 cm</td>
<td>5.20</td>
<td>57.8</td>
<td>99.5</td>
</tr>
<tr>
<td>S.M.</td>
<td>1.15</td>
<td>16.4</td>
<td>28.2</td>
</tr>
<tr>
<td>24 yr</td>
<td>1.92</td>
<td>27.4</td>
<td>47.2</td>
</tr>
<tr>
<td>69.9 kg</td>
<td>2.84</td>
<td>40.7</td>
<td>70.2</td>
</tr>
<tr>
<td>178 cm</td>
<td>3.91</td>
<td>56.0</td>
<td>96.6</td>
</tr>
</tbody>
</table>

*Age, weight, and height of subjects listed below initials.

*Hot and cold towels were used in an effort to attenuate radial pulse pressure by local vasodilation (hot) and accentuate amplification by vasoconstriction. Unfortunately, since the hand and wrist had to be excluded, most of the tissues stimulated were upstream from the tip of the radial arterial catheter. Hot towels had no effect and cold produced only brief transient increase.

Figure 2

Frequency responses for radial arterial (A) and aortic (A') catheters plus respective transducers. Curves B and C represent the responses of (A) and (A') plus the 24 Hz recording galvanometer. Curve D shows the response for both (A) and (A') plus the tape recording system with 20 Hz (RLC) filter.

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blood flow to the arm was occluded by inflating a pressure cuff to 300 mm Hg for 90 sec.

Between each workload the subject rested 10 to 15 min while seated on the treadmill as before. Zero pressure reference level was reset for changes in height of the subject's fourth interspace with changes in the treadmill grade. Gauges and recorders were then recalibrated, statically, at multiple pressures with a mercury manometer.

Oxygen uptake was determined from 2- to 3-min collections (1 min at maximal oxygen uptake) of expired air into a 350-L balanced spirometer via a low resistance valve and tubing. Expired gas samples were analyzed for oxygen and carbon dioxide by the Scholander microtechnique. Ambient temperature was 25 C.

**Manometry**

Aortic pressures were recorded via a specially fabricated 70-cm catheter* plus a Statham P23Gb strain gauge. A 29.5-cm, 18-gauge, thin wall Teflon catheter and Statham P23Db strain gauge were used at the radial artery. By using a variable frequency sinusoidal pressure generator with high frequency (500 Hz) monitoring gauge, the resonant frequencies of the aortic and radial gauges plus catheters were found to be 120 and 130 Hz, respectively (fig. 2); damping was less than 5% of critical. The dynamic response of the entire manometric system (catheter, strain gauge, Honeywell 131-2C carrier amplifier, and Honeywell M24-350 galvanometer) is also shown in figure 2. The five-harmonic (130 Hz/24 Hz) separation of the resonant frequencies of the catheter-strain gauge system and low frequency galvanometer provided a manometric system with a flat amplitude response to within ±5% to 20 Hz, damping 64% of critical, and linear phase shift in this pass band. Thus, five to six harmonics of the pressure pulse wave could be accurately transmitted even at heart rates up to 200 beats/min.

Pressures were also recorded on magnetic tape† Outputs from strain gauge carrier amplifiers were fed to DC amplifiers (8875A) via input filters. These 20-Hz second-order filters served a function analogous to the low frequency galvanometers in the parallel systems. Radial and aortic channels had unity gain within ±5% to 13 and 15 Hz, respectively. Both channels had 70%

*No. 5 French, Radiopaque Teflon. Cook Inc., Bloomington, Indiana.
†Model 3907. Hewlett-Packard.

**Figure 3**

Simultaneous recordings of aortic and radial arterial pressure waves during rest (A), 28.2 (B), 47.2 (C), and 70.2% (D) of maximal oxygen uptake in subject S.M. These tracings were taken at the peaks of slow pressure oscillations and show the relationship of these extreme peripheral values to the severity of exercise.
of critical damping and linear phase shift in these frequency ranges.

Immediately after two of the experiments, catheters were quickly removed from the arteries and inserted into the chamber of the pressure generator. Responses to square waves and sine waves of pressure to 150 Hz were recorded. Responses to pressure transients were determined similarly in a third experiment.

Mean pressures were obtained by playing tape recorded data through low pass (0 to 0.16 Hz) active filters consisting of two cascaded sections, each acting as a first order lag with a time constant of 1 sec.

Pressures from both sites were recorded at full scale sensitivity on the Visicorder (10 mm Hg/cm). Systolic, diastolic, and mean pressures were averaged over 15 sec at every 30 sec of measurement.

**Results**

**Aortic and Radial Arterial Pulse Pressure During Exercise**

Differences between aortic and radial pulse pressures during exercise tended to increase with the severity of work (fig. 1); however, these differences diminished as the duration of submaximal exercise increased, particularly at the highest workload (which averaged 77.6% of maximal oxygen uptake). At this workload, radial pulse pressure was maximal at 2 to 4 min (except in D. J.) and fell toward aortic values thereafter.

The disparity between aortic and radial pulse pressures was closely related to the severity of exercise if pulse pressures during the peaks of slow oscillations in the early minutes of exercise were compared (figs. 3 and 4). These simultaneous, in phase oscillations in the aorta and radial artery varied in frequency from 2 to 13 cycles/min. The difference in magnitude of these oscillations at the two sites was proportional to the difference in pulse pressures at these sites. These oscillations were most striking at maximal oxygen uptake; they varied in amplitude from 42 to 100 mm Hg in the radial artery (fig. 5). They tended to be cancelled out in figure 1 by the manner in which the data were plotted; that is, pressures were averaged over 15-sec intervals.

Additional high frequency oscillations in aortic pressure occurred during high levels of exercise (fig. 5). These, no doubt, reflect relative motion of the catheter with respect to the aorta due to high rates of ventilation and jarring running movements. Relative motion generates pressure artifacts due to inertia and variation of the angle between the catheter and velocity axes. Since catheter whip occurs at frequencies within the band pass of the recording system, the resultant “noise” cannot be eliminated without destroying the fidelity of the recording system.* Motion artifacts were minimal in M. M. and S. M.

During the first or second minute of recovery from maximal exercise the aortic catheter was slowly withdrawn to the brachial

* Catheter-tip manometers would minimize this problem. We felt the additional surgical procedures necessitated by the use of these probes precluded their use in our normal subjects.
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Continuous record of aortic and radial arterial pulse and mean pressures during the runs at maximal oxygen uptake (replayed from magnetic tape). Note the magnitude of oscillations in pulse pressure in all four subjects as well as the marked variation in their frequency, particularly in D.J. and S.M. These two subjects showed striking changes in the period of oscillation as they approached exhaustion. Catheter motion noise was considerable in D.J. and R.G., making aortic pulse pressures appear greater than they were. These effects were absent in S.M. Arrows mark start and end of exercise. The third arrow (after end of exercise) in D.J. and M.M. marks the beginning of pull-back of the catheter into the brachial artery and shows the amplification of pressure.

Aortic and Radial Arterial Mean Pressures

During submaximal exercise, mean (that is, filtered) pressures from the two sites were identical within ±2 mm Hg, a level of discrepancy within the error of measurement considering factors affecting the kinetic energy component of mean flow. In the aorta, this component would be eliminated in the downstream side-hole catheters and could reach a maximum of 4 to 5 mm Hg in the unlikely event that the upstream open-end catheters happened to remain parallel to the mean flow axis. Since radial catheters were all open-end and pointed upstream, flow probably contributed a relatively constant but insignificant positive increment in pressure. Average mean pressures from both sites at rest and four levels of oxygen uptake in order of increasing values of the latter were 87, 95, 94, 104, and finally 107 mm Hg at maximal oxygen uptake. At maximal oxygen uptake, mean pressures rose progressively through the 2.75 min of work (2.17 min for D. J.) from 89 to 117 mm Hg at the end.

Aortic mean pressure was generally halfway between aortic diastolic and systolic.
Simultaneous recordings of radial arterial (lower) and aortic to brachial arterial pressure (upper) as the aortic catheter tip was slowly pulled back to the brachial artery during recovery from maximal exercise (replayed from magnetic tape). Systolic pressure rose from 135 to 185 mm Hg in moving from the former to the latter site while the shape of the wave changed progressively.

pressures (fig. 1). The relationship between mean, diastolic, and systolic pressures measured in the radial artery varied with the time and severity of exercise. For example, as subjects became hot and tired at the highest submaximal workload, falling radial arterial systolic pressure was not attended by falling mean pressure at that site (fig. 1). The difference between mean and diastolic radial arterial pressures varied from a minimum of 20 to 25% of pulse pressure to a maximum of 40% at the end of the third level of exercise.

Transitions from Standing Rest to Exercise

On standing, both mean pressure and pulse pressure rose abruptly and then quickly fell nearly to the previous levels (fig. 7). The same phenomenon was observed at the start of exercise. Within 10 to 30 sec after starting exercise, pressures returned to the pre-exercise levels recorded during seated rest (fig. 1). Occasionally, pressures rose several seconds before exercise began.

Postocclusive Hyperemia

Immediately following 90 sec of occlusion of blood flow to the arm, radial arterial pulse pressures were initially lower than, or equal to, aortic values (fig. 8). Peripheral pulse pressures gradually increased above aortic values, and within 15 to 20 sec after release of the cuff reached levels measured before occlusion (fig. 1).

Discussion

Previous observations of aortic pulse pressure during exercise have shown similar or greater increments with work than ours but are not strictly comparable. Body posture, the severity, or the type of exercise (bicycle), or both, and the subjects' ages preclude direct comparison. Also the degree to which the dynamic response of the manometric systems may have affected previous results cannot be evaluated. The degree to which blood pressure is affected by seated as opposed to upright posture is not known. Addition of
either dynamic or static arm work (especially the latter) to leg exercise (as often occurs during cycling, especially at higher workloads) would elevate aortic pressure. The high aortic pressures noted by Nagle and associates in two subjects may be related to the subjects' ages (40 and 57 years).

Basically this study focused on the question of whether increments in peripheral pulse pressure during exercise actually reflect changes in the aortic arch—one site of baroreceptor regulation. Theoretically, the magnitude of aortic pulse pressure depends upon whether the kinetic energy component, $\frac{1}{2} \rho V^2$, is included in the measurement. At a cardiac output of 20 to 25 L/min this component could increase systolic pressure as much as 40 to 50 mm Hg. This assumes the end-hole of the catheter to be pointed directly at the flow axis, a factor which could not be evaluated at rest, of course, even with the most careful fluoroscopy. In any event, the orientation could not be expected to remain static during the large respiratory movements and other movements of heavy upright exercise.

*Peak flow velocity was estimated from aortic flow-meter traces to be three times mean velocity.
Although responses of D. J. and S. M. were consistent with some addition (in D. J.) or elimination of this effect, results from R. G. and M. M. were the reverse of those predicted.

Peripheral arterial pulse pressure showed amplification with increasing intensity of work. However, this relationship tended to be obscured in all subjects because of marked slow oscillatory variation in pulse pressures. On the average, aortic and radial arterial pulse pressures increased from rest (sitting) to maximal exercise by factors of 1.95 and 2.60, respectively. This calculation is based on the average rise in radial arterial pressure during the first 5 min of exercise. Thus, increasing systolic pressure observed during work of increasing severity was predominantly a peripheral phenomenon and occurred to a lesser degree in the aorta.

This disparity in pressures between the two sites has major clinical significance. Observations of abnormally elevated peripheral pulse pressure during exercise must raise the following questions. Is the elevation due to peripheral vascular amplification, presumably induced by local vasoconstriction, or to increased central blood pressure? If it is predominantly the former, as in our normal subjects during exercise, the pressure-volume work of the heart, the estimation of time-tension index from the product of heart rate times peak systolic pressure, or the mechanical stress on the aorta and cerebral vessels and central baroreceptors will be, of course, greatly overestimated by the use of peripheral arterial pulse pressures.

Since wave reflection is directly related to the vaso-active state in the region of pressure measurement, any condition causing peripheral vasodilatation or vasoconstriction must be considered as a factor potentially modifying peripheral arterial pulse pressure. For example, peripheral pressure measurements from vasodilated exercising regions are considerably less amplified at a given work intensity than those from nonworking, presumably vasoconstricted regions. Also, addition of external heat loads will lower pulse pressure strikingly in a resting limb during exercise, whereas aortic pressure is minimally affected. The response to reactive...
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hyperemia in this study illustrated this effect. Also, peripheral vasodilatation in the arm attending increasing internal heat load\textsuperscript{4} was most likely the cause of the progressive fall in peripheral pulse pressure during exercise (fig. 1).

Despite falling radial arterial pulse pressure during work, mean pressures at the central and peripheral sites remained essentially identical. Thus, though the shape of the radial arterial pressure wave was altered, the area inscribed by it (or the very low frequency components, that is, less than 0.16 Hz)\textsuperscript{*} was unaltered.

The cause of cyclic variations in pulse pressure, especially in the radial artery, during exercise is not known. Measurements of breathing and stepping frequency during work on R. G. and S. M. showed that these were not at the same frequency as pressure variations. They may be related to rhythmic peripheral vasomotor changes involved in thermal regulation.\textsuperscript{14} Cyclic changes in finger\textsuperscript{15} and arm blood flow\textsuperscript{16} over a range of frequencies, usually from one cycle every 10 to 20 sec, have been observed during rest and exercise. These variations appear to increase with severity of exercise; during exhausting exercise they possibly represent "hunting" of the peripheral circulation for optimal distribution between the skin for heat dissipation and the muscle for continued oxygen transport.

Studies on dogs by Warner\textsuperscript{17} led him to stress the role of falling peripheral resistance as a stimulus increasing cardiac output during the transition from rest to exercise. Holmgren\textsuperscript{18} has reviewed this problem extensively and has observed a transient fall in blood pressure during the first few seconds of exercise, but only if exercise was immediately preceded by rest, not if it was preceded by a lower level of work. Our data show changes in pressure at the onset of exercise similar to those reported by Warner. It is important to note, however, that these drops in pressure often followed an anticipatory rise.

We conclude that the increased disparity between aortic and radial arterial pulse pressures during exercise is sufficient to preclude meaningful evaluation of the pulsatile strain on central vasculature and baroreceptors from peripheral arterial measurements. Errors in these estimates from nonworking peripheral sites will increase with the severity of exercise and possibly other factors eliciting local vasoconstriction. A catheter with adequate frequency response will transmit dynamic characteristics of aortic pressure between that site and a transducer far more reliably than the brachial-radial arterial system since the latter has variable compliance and dynamic sensitivity.

**Acknowledgment**

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