Determination of Systemic Vascular Resistance
by a Noninvasive Technic

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SUMMARY
Measurement of systemic vascular resistance in man currently requires arterial and venous cannulation, so that cardiac output and mean arterial blood pressure may be simultaneously determined. This report describes a noninvasive technic for determining systemic vascular resistance (SVR), utilizing an ultrasonic method for cardiac output measurement, and a combination of sphygmonanometry and external carotid pulse tracing analysis for determination of mean arterial blood pressure. SVR was measured by this technic in 18 patients and compared with systemic vascular resistance determined by conventional methods at cardiac catheterization (SVR). There was excellent correlation, with SVR = 0.865 SVR + 216 and r = 0.85. The sensitivity of the method was verified in studies on 12 normal subjects in whom SVR while supine (1235 ± 61 dyne-sec-cm⁻², mean ± se) was less than SVR while standing (1416 ± 81, P < 0.01) and greater than SVR supine after amyl nitrite inhalation (652 ± 41, P < 0.0001).

It is concluded that this noninvasive, simple, and safe technic should prove to be a useful bedside method for routine clinical measurement of systemic vascular resistance.

Additional Indexing Words:
Echocardiography
LV internal minor axis
Sphygmonanometry

Although major abnormalities in systemic vascular resistance (SVR) are common in several diseases (e.g. hypertension, congestive heart failure, shock, thyroid disease, and A-V fistula), the clinical usefulness of this knowledge has been limited by the fact that standard methods for the measurement of SVR require arterial and venous cannulation.

Recently, a noninvasive method for estimation of cardiac output, employing ultrasonic measurement of the left ventricular internal minor axis throughout the cardiac cycle, has been described by several investigators.¹⁻⁴ It has also been shown⁵⁻⁶ that values of systolic and diastolic blood pressure, obtained by conventional sphygmonanometry, correlate fairly well with those found by direct intraarterial pressure recording. Although mean arterial blood pressure (blood pressure or BP) can be empirically estimated by adding one third of the pulse pressure to the diastolic blood pressure, this method is not always accurate, due to wide variations in the morphology of the intraarterial pressure curve.⁷ Given the striking resemblance found to exist between the intraarterial pressure curve and the externally recorded carotid pulse tracing,⁸⁹ it appeared probable that mean blood pressure could be indirectly approximated by integrating the carotid pulse curve, whose onset and peak would be taken to represent the diastolic and the systolic blood pressure, respectively, as they were determined by sphygmonanometry of the arm.

Thus, combined use of those two external technics for calculation of both cardiac output and mean arterial blood pressure might prove to represent a simple, noninvasive, bedside method for determination of the SVR in most disease states. It is the purpose of this study to examine the feasibility and test the accuracy and sensitivity of this approach.
Material and Methods

Initial studies were performed on 18 patients (group 1), 24 hours prior to routine diagnostic cardiac catheterization. Patients with aortic or mitral regurgitation, ventricular septal defect, atrial septal defect, significant tricuspid regurgitation, or regional abnormalities of left ventricular contraction were specifically excluded because echocardiographic determination of the forward stroke volume and cardiac output can be erroneous or impossible in these cases. The age, sex, and diagnosis for each patient of this group are listed in Table I. These studies were carried out in a fasting state and supine position, without sedation, and consisted of simultaneous recording of external carotid pulse tracing, echocardiogram, and blood pressure by sphygmanometry. The data obtained were subjected to the following analysis and calculation:

Calculation of Cardiac Output (CO<sub>ECHO</sub>). Echocardiographic studies of the left ventricle were performed with a Smith-Kline* Ekoline 20 echocardiograph, equipped with a 2.25-MHz transducer (0.5 inch in diameter, repetition rate 1000/sec). The end-diastolic (D<sub>ED</sub>) and end-systolic (D<sub>ES</sub>) internal minor-axis dimensions of the left ventricle were determined and were employed for the calculation of end-diastolic (EDV) and end-systolic (ESV) volumes, respectively, using the formulae developed by Fortuin et al.:

\[
\text{EDV} = 59 \times D_{ED} - 153 \quad (1)
\]

\[
\text{ESV} = 47 \times D_{ES} - 120 \quad (2)
\]

Stroke volume was: \( \text{SV} = \text{EDV} - \text{ESV} \). Cardiac output was then calculated as:

\[
\text{CO}_{ECHO} = \text{heart rate} \times \text{SV} \quad (3)
\]

Determination of Mean Blood Pressure (BP<sub>m</sub>). A mercurial sphygmomanometer† with its cuff snugly fitted around the left arm, was employed for the measurement of systolic and diastolic blood pressure, according to official recommendations. During slow deflation of the cuff (2–3 mm Hg/sec), the systolic blood pressure was read at the onset of the phase I, and the diastolic blood pressure at the onset of the phase IV of Korotkoff.

During sphygmanometry, the carotid pulse tracing (CPT) was recorded on a Cambridge physiologic recorder, utilizing a Hellige* transducer. The sensing head of the transducer was manually held over the right carotid artery with moderate but steady pressure. The gain was adjusted so that the amplitude of the CPT was about one half the height of the oscilloscopic screen of the recorder. (That calculation of BP<sub>m</sub> by this method is not significantly affected by the gain employed in the recording of CPT was shown on five separate subjects in whom BP<sub>m</sub> was calculated using CPT’s recorded under five different gain settings: 2, 4, 6, 8, and 10.)

On the CPT (fig. 1), a straight line (L) was drawn connecting the onset of upstroke of two adjacent carotid pulse curves and represented the level of the diastolic blood pressure (BP<sub>d</sub>). The peak of the curve represented the systolic pressure (BP<sub>s</sub>). The distance from the peak of the curve to the line L represented the pulse pressure.

The surface (S) enclosed between the carotid pulse curve and the line (L) was determined by planimetry, and S was then divided by the length L yielding h, the mean height of the carotid pulse curve above the diastolic blood pressure. It followed that the mean blood pressure BP<sub>m</sub> would equal the sum of h and BP<sub>d</sub>:

\[
\text{BP}_m = h + \text{BP}_d \quad (4)
\]

Calculation of the Systemic Vascular Resistance (SVR<sub>n</sub>). Using the values of cardiac output and mean blood pressure obtained from equations (3) and (4), the SVR<sub>n</sub> (in dynes·sec·cm<sup>-5</sup>) was calculated as:

\[
\text{SVR}_n = \frac{\text{BP}_m \times \text{CO}_{ECHO} \times 80}{\text{SVR}_n} \quad (5)
\]

Subsequent conventional determination of systemic vascular resistance (SVR<sub>n</sub>) was done 24 hours later during cardiac catheterization. Under local anesthesia and light sedation (diazepam 5–10 mg im), standard right and retrograde left cardiac catheterization (via right antecubital vein and brachial artery, respectively) was performed in a fasting state and supine position, utilizing standard French no. 7 or 8 catheters. A polyethylene (PE 160, 25 cm long) catheter was percutaneously introduced into the left brachial artery.

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*Smith-Kline Instruments, Palo Alto, California.

†Baumanometer—W. A. Baum Co., Inc., New York, New York. Inflatable bag dimensions are 12 × 22.5 cm.

‡Fritz Hellige & Co., Freiburg, West Germany. This apparatus has a flat frequency response at frequencies from 50 Hz down to 0.3 Hz, and a time constant longer than 3 sec.

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Figure 1

Determination of mean arterial blood pressure. The area (S) of the external carotid pulse curve (hatched) equals the area of a rectangle (dotted) having the same base (L). The height of the rectangle (h) determines the level above diastolic arterial pressure (BP<sub>d</sub>) at which the mean arterial pressure lies. BP<sub>s</sub> = systolic arterial blood pressure.

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Table 1

**Hemodynamic Data of 18 Patients as Determined by Both Noninvasive Technic and Cardiac Catheterization**

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<th>Pt</th>
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<th>Heart rate (beats/min)</th>
<th>CO (liters/min)</th>
<th>Arterial blood pressure (mm Hg)</th>
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Mean ± se

| A   | 87.2 ± 4.9  | 74.4* ± 3.4  | 6.3* ± 0.33 | 126.1* ± 3.4 | 78.9* ± 2.1 | 99.2* ± 2.3 | 1309.5* ± 69.7 |
| B   | 72.1 ± 3.8  | 77.3 ± 3.2   | 5.5 ± 0.34  | 139.1 ± 6.9  | 72.6 ± 2.1  | 94.3 ± 2.8  | 1349.2 ± 70.5  |

Abbreviations: CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; A = noninvasive technic; B = cardiac catheterization; * = not statistically different from value at catheterization; pulm = pulmonary; se = standard error; RAPn = mean right atrial pressure.
for continuous arterial pressure monitoring. All pressures were recorded on an Electronics for Medicine DR-12 recorder, through Statham P23 Db strain-gauge transducers. Cardiac output (CO\textsubscript{FICK}) was measured by the standard, direct Fick method and oxygen content of blood samples was determined by the method of Van Slyke and Neill. Mean right atrial pressure (RAP\textsubscript{m}) was recorded before, and mean left brachial arterial pressure (BA\textsubscript{m}) during cardiac output measurement. Systemic vascular resistance (in dyne-sec-cm\textsuperscript{-5}) was:\textsuperscript{12}

$$SVR_e = \frac{BA_m - RAP_m \ (mm \ Hg)}{CO_{FICK} \ (liters/min)} \times 80 \quad (6)$$

Statistical comparison between SVR\textsubscript{a} and SVR\textsubscript{e} was made using the paired t test. P values higher than 0.01 were considered as nonsignificant.

To test the sensitivity of the proposed noninvasive method for determination of SVR, determinations of SVR\textsubscript{a} were made in 12 healthy volunteers who formed the population of group II.

Making use of the knowledge that a change in body position from supine to standing results in an increase in SVR,\textsuperscript{13} whereas amyl nitrite inhalation results in a decrease in SVR,\textsuperscript{14,15} we determined SVR\textsubscript{a} by the proposed noninvasive method: (1) at the end of a 3-min period of standing still; (2) at the end of a 3-min period of lying supine at rest; and (3) 30–45 sec after inhalation of amyl nitrite while in supine position at rest.

SVR\textsubscript{a} values when standing, as well as after amyl nitrite, were statistically compared for each subject to those obtained in supine position at rest ("baseline" SVR\textsubscript{a}), using the paired t test. P values higher than 0.01 were considered as nonsignificant.

**Results**

**Group I**

Systemic vascular resistance (dyne-sec-cm\textsuperscript{-5}), as determined by the proposed noninvasive technic (SVR\textsubscript{a}), was 1310 ± 70 (mean ± se), whereas that determined by catheterization (SVR\textsubscript{e}) was 1350 ± 71. The observed difference, representing 3% of the SVR\textsubscript{e}, was insignificant (P < 0.5).

Cardiac output as determined by echocardiography was not significantly different from that measured at catheterization 24 hours later. Similarly, no significant difference was noted between systolic, diastolic, and mean blood pressures as determined noninvasively, and those measured intraarterially during catheterization (table 1).

Figure 2 illustrates the CO, BP, and SVR as they were determined by both the noninvasive technic and cardiac catheterization. Although the differences were statistically insignificant, it can be seen that noninvasively determined CO and BP\textsubscript{m} were greater than CO and BP\textsubscript{m} as determined by catheterization, by 15% and 5%, respectively. Yet, SVR\textsubscript{a} was underestimated by only 3% of SVR\textsubscript{e}.

SVR\textsubscript{a} and SVR\textsubscript{e} showed a close linear correlation which was expressed as a regression equation:

$$SVR_e = 0.865 \ SVR_a + 216 \quad (7)$$

($r = 0.85$, se of the slope = 0.131).

**Group II**

The sensitivity of the described noninvasive technic in detecting changes in SVR occurring upon change in the body position from supine to standing or produced by inhalation of amyl nitrite was tested, and pertinent data are listed in table 2 and figure 3.

As can be seen (fig. 3), SVR was 1235 ± 61 dyne-sec-cm\textsuperscript{-5} (mean ± se) in supine position and rose to 1416 ± 81 upon assumption of the standing position. This difference was significant (P < 0.01) and represented 15% of the value of SVR in supine position. The increase in SVR upon standing had a weak effect on CO, which fell by only 9.4% of its value in supine position. This was due to the fact that, upon standing, the significant fall in stroke volume was partially counterbalanced by a small increase in heart rate. Change in body position from supine to standing resulted in an insignificant increase in BP\textsubscript{m}.
Table 2

Effects of Positional Change and Amyl Nitrite Inhalation on Hemodynamics of 12 Volunteers, as Determined by Noninvasive Technic

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<th>Supine and Amyl Nitrite</th>
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<tbody>
<tr>
<td>Stroke volume (ml)</td>
<td>66 ± 2.3</td>
<td>87 ± 2.5</td>
<td>99 ± 7.0</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>82 ± 8</td>
<td>73 ± 4</td>
<td>109* ± 5</td>
</tr>
<tr>
<td>Cardiac output (liters/min)</td>
<td>5.7 ± 0.3</td>
<td>6.3 ± 0.3</td>
<td>10.5* ± 0.5</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>98 ± 2.1</td>
<td>95 ± 2.8</td>
<td>83* ± 3.8</td>
</tr>
<tr>
<td>Systemic vascular resistance (dyne-sec-cm^-5)</td>
<td>1416* ± 81</td>
<td>1235 ± 61</td>
<td>652* ± 41</td>
</tr>
</tbody>
</table>

*P < 0.01 in comparison to value in supine position. Values are mean ± se.

Abbreviations: MAP = mean arterial pressure.

SVR fell from a control value of 1235 ± 61 dyne-sec-cm^-5 (mean ± se) before to 652 ± 41 during inhalation of amyl nitrite. This decrease was highly significant (P < 0.001) and represented 47% of the pre-amyl nitrite value of SVR (fig. 3). The observed significant increase in CO (67%) was not adequate to sustain the mean arterial blood pressure which decreased by 12% of its pre-amyl nitrite value. It should be noted that both stroke volume and heart rate contributed to the increase in CO, although not to the same degree.

Discussion

This investigation has demonstrated that systemic vascular resistance can be determined by use of a totally noninvasive technic with a reasonable degree of accuracy. It has also shown that this technic was sensitive enough to detect changes in systemic vascular resistance resulting from change in body position or from amyl nitrite administration.

The proposed noninvasive technic for determination of systemic vascular resistance requires calculation of both cardiac output by echocardiography and mean blood pressure by a combination of sphygmomanometry and analysis of the externally recorded carotid pulse wave. It is therefore conceivable that its usefulness and limitations would be determined by the merits and inadequacies of the involved particular methods.

Echocardiographic determination of the forward cardiac output can not be performed in cases of mitral or aortic regurgitation or ventricular septal defect, where forward stroke output constitutes only a portion of the total LV stroke output. Also, it is unreliable in the presence of regional abnormalities of left ventricular contraction, and impossible in the presence of paradox motion of the interventricular septum due to significant volume overload of the right ventricle as in atrial septal defect or severe tricuspid insufficiency. Furthermore, in the presence of marked pulmonary emphysema or significant deformities of the thoracic wall, echocardiographic study of the left ventricle may be difficult or even impossible. In the absence of these conditions, however, echocardiographic resolution of both the left ventricular posterior wall and the interventricular septum, adequate to permit calculation of the cardiac output, is feasible in about 80% of cases.

The reproducibility of echocardiographic measurements of the left ventricular minor-axis dimensions between different observers, as well as on the same patient at different times, has been recently documented. Therefore, considering its convenience, simplicity, and reasonable accuracy, the echocardiographic technic for cardiac output estimation was deemed suitable for the purpose of this study.

Figure 3

Effect of postural change and amyl nitrite inhalation on systemic vascular resistance (SVR) determined by noninvasive technic in 12 subjects. Columns represent mean values and horizontal lines, standard error. See text for discussion.
Mean arterial blood pressure can be determined by integration of the intraarterial pressure curve.\(^7\) Since the external carotid pulse curve has been shown\(^8,\)\(^9\) to resemble closely the intraarterial pressure waveform, it was conceivable that integrating the latter might represent an approach to the calculation of the actual mean arterial blood pressure with one proviso: that the values of both systolic and diastolic blood pressure would be available to be assigned to the peak and the onset, respectively, of the external carotid pulse curve. Actually, conventional sphygmomanometry can provide reasonably accurate estimates of the systolic and diastolic blood pressure.\(^5,\)\(^6,\)\(^10,\)\(^17,\)\(^18\)

Either the onset of Korotkoff phase IV\(^10,\)\(^17-20\) (abrupt muffling of the sounds), or that of phase V\(^6,\)\(^21\) (disappearance of the sounds), should be used for the determination of the diastolic pressure. If Korotkoff phase IV is used, the estimate is usually 7-10 mm Hg higher than the true diastolic pressure.\(^5,\)\(^10,\)\(^17,\)\(^18\) If phase V is used, in most instances the estimate is much closer to the true diastolic pressure.\(^5,\)\(^10\) but under certain conditions this estimate can be substantially false.\(^10,\)\(^18\) We used the onset of phase IV as an index of the diastolic pressure, because making a standard in magnitude and predictable in nature error seemed to be much more safe than to adopt an index (phase V) whose accuracy may unpredictably change from excellent to poor.

It is known that usually the mean blood pressure equals the sum of the diastolic plus one third of the pulse pressure.\(^7\) However, significant changes in the configuration of the intraarterial pressure curve may render this empirical method useless in certain individuals.\(^7\) For example, patient 1 (J.H.) of group I (Table 1), had intraarterial blood pressure 210/88 (mean 110) mm Hg. Adding one third of the pulse pressure to the diastolic pressure would result in an overestimation of the true mean blood pressure by 19 mm Hg. Calculation of mean blood pressure by integration of the external carotid pulse curve is affected by its morphology and therefore is less likely to be wrong in these particular cases.

The proposed technic for determining systemic vascular resistance should not be considered as the most accurate one. It can not be applied in patients in whom echocardiographic determination of cardiac output fails for any reason. Furthermore, it is subject to substantial error in patients with significantly elevated mean right atrial pressure because the latter can not be measured externally and is ignored in formula (5). However, it represents a totally noninvasive, simple bedside technic that can repeatedly be applied to the same patient without any discomfort or risk.

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**References**


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