Evaluation of Several Methods for Computing Stroke Volume from Central Aortic Pressure

By C. Frank Starmer, Philip A. McHale, Frederick R. Cobb, and Joseph C. Greenfield, Jr.

ABSTRACT

Six pulse-contour methods for estimating stroke volume from a single central aortic blood pressure were evaluated in 8 dogs and 17 patients. In the dogs, wide variations in stroke volume, measured with an electromagnetic flowmeter, were obtained by pacing the heart at various rates during a control period and during several pharmacologic interventions. Good correlations existed between measured stroke volume and most estimators when the data from each intervention were analyzed separately. However, regression analysis revealed considerable variation in the individual slopes and intercepts, and thus a poor correlation was obtained when all data for one dog were combined in a single analysis. Similar evaluations were carried out in two groups of patients in whom the pressure-gradient technique was used to measure stroke volume. In the group with minimal variations in hemodynamic status, the correlations between estimated and true stroke volume were reasonably good. In the patients having a wide range of hemodynamic conditions, considerable variation in both slopes and intercepts was observed, and the combined correlation coefficients were generally poor. Although pulse contour methods of estimating stroke volume may work reasonably well over a range of stroke volumes when the variation is induced by a single perturbing agent, none of these methods perform adequately when the variation is induced by multiple perturbing agents; thus their clinical usefulness is markedly limited.

KEY WORDS  human subjects  aortic pressure—pulse contour methods  pressure-gradient technique  cardiac output  dogs

The measurement of cardiac output is essential for an adequate evaluation of the hemodynamic status of the cardiovascular system. Continuous determinations of cardiac output are especially important for monitoring acutely ill patients and for documenting their response to therapy, since the hemodynamic status may vary markedly during a short period of time. Both the Fick and the indicator-dilution methods provide a relatively accurate estimate of cardiac output; however, these techniques are not generally practical for applications that require multiple, frequent determinations.

Various types of flowmeters can be used to measure aortic blood flow and compute stroke volume continuously in animals, but their routine use in humans is not feasible at the present time. Fry (1) has described a method for estimating pulsatile aortic blood flow which requires accurate measurement of the axial pressure gradient in the ascending aorta. The pressure gradient is used to solve a simplification of the Navier-Stokes equations to yield phasic blood flow. Precise measurement of the pressure gradient is extremely difficult, however, and with available manometry the pressure-gradient technique is not suitable for general use. An attempt to simplify this method (2) by substituting the time derivative of aortic pressure for the pressure gradient has been shown to have serious theoretical and practical limitations (3).

Warner et al. (4) described several methods for computing stroke volume based on the earlier work of Hamilton and Remington (5); these methods employ both the central aortic pressure and a peripheral arterial blood pressure. Later, by assuming a fixed pulse propagation velocity and modifying the model relating the mean distending pressure...
Methods

Eight adult mongrel dogs (20–26 kg) were anesthetized with sodium thiamylal (25 mg/kg, iv), and the sinoatrial node was removed through a right thoracotomy. This procedure was carried out so that the control heart rate at the time of study would be approximately 60 beats/min. After 10–14 days of recovery, the dogs were again anesthetized with sodium thiamylal (25 mg/kg, iv) and underwent a left thoracotomy. The root of the aorta was dissected free and reinforced with a patch of coarse Teflon mesh. A Statham TTQ electromagnetic flowmeter probe was placed around the aorta over the Teflon mesh and cushioned proximally and distally with Silastic sponge material. A bipolar epicardial pacing electrode was sutured to the right atrial appendage. The flowmeter probe and the pacing electrode wires were tunneled into a subcutaneous pouch at the base of the neck. Chronic placement of the flowmeter probe ensured that a noise-free signal with a stable base line would be obtained. The studies described in this paper were performed 10–14 days after the second operation. At that time, the dogs had a normal hematocrit and showed no evidence of ill health.

On the day of the study the dog was sedated with morphine sulfate (0.5 mg/kg, iv) and was positioned on its right side. Using enough lidocaine to produce local anesthesia, the flowmeter probe and the pacing electrode wires were exteriorized through a small skin incision. Also under adequate local anesthesia, the left carotid artery and the left external jugular vein were cannulated with 35-cm long, large-bore polyethylene catheters having lateral side holes and radiopaque tips. Under fluoroscopic control, the arterial catheter was advanced until the tip was under the aortic flow probe and the venous catheter was placed in the right atrium. Drug infusion was accomplished through a cephalic venipuncture. In some dogs, an additional cannula was placed in the femoral artery under adequate local anesthesia to permit rapid removal of blood. The aortic blood flow was monitored with a Statham M-4000 electromagnetic flowmeter, and the blood pressures were obtained through the polyethylene catheters attached to Statham P23Db transducers. The frequency amplitude characteristics of both the flowmeter and the pressure-recording systems were evaluated; the deviation in amplitude and the phase shift were negligible.
up to 25 Hz. The calibrations for the electromagnetic flowmeter probes were determined by allowing a measured volume of normal saline to flow through the probe for a known period of time. The calibration factor for the probes remained within a standard deviation of ±5% during the study. Lead II of a standard electrocardiogram was also obtained. All data were recorded on both an eight-channel direct-writing oscillograph (Hewlett-Packard 958-100) and a magnetic tape recorder (Hewlett-Packard 3917A FM). During the study the following interventions were used to obtain wide variations in blood pressure and cardiac output: infusion of phenylephrine (40–80 μg/min, iv), infusion of isoproterenol (4–8 μg/min, iv), and, in some dogs, acute removal of 750–1000 ml of blood. After achieving a steady state during the control period and during each experimental intervention, the heart rate was increased by right atrial pacing to obtain a wide range of stroke volumes using a Grass S-4 stimulator and isolation unit through a range of 60 to 210 beats/min in increments of 30 beats/min. The sequential order of the various pacing rates was randomized. Experimental data were recorded for 3 minutes at each pacing rate. At the conclusion of the study, the wounds were closed aseptically, and the dogs were returned to their cages.

HUMAN STUDIES

The pressure-gradient technique was used to measure simultaneously the aortic blood pressure and the blood flow in 17 patients. The pressure-gradient technique is based on the fundamental laws of fluid motion which are expressed mathematically by the Navier-Stokes equations (1). Under certain restrictions, which appear to be applicable in the ascending aorta, these equations may be reduced to the relationship

\[-\frac{dp}{dz} = \frac{Ld\eta}{dt} + Rq,\]

where \(p\) is the lateral pressure, \(z\) is the axial coordinate of the aorta, \(q\) is the instantaneous flow, \(t\) is time, \(L\) is the hydraulic inductance, and \(R\) is the hydraulic resistance of the blood vessel. The values of \(L\) and \(R\) are

\[L = 1.1 \frac{\rho}{\eta r^2},\]

\[R = 1.6 \frac{8\mu}{\eta r^2},\]

where \(g\) is the gravitational constant, \(r\) is the vessel radius, \(\rho\) is the density, \(\mu\) is the viscosity of the blood, and 1.1 and 1.6 are experimentally derived constants (1). In practice, the pressure difference, \(\Delta p/\Delta z\), is substituted in Eq. 1 for \(-dp/dz\) since the pressure gradient cannot be measured at a point (14). This equation can then be solved continuously for flow by an analog computer. The validity of this method has been demonstrated in a flow generator where the flows were known, in the aorta of dogs where the true flows were measured with an electromagnetic flowmeter, and in many studies in man in which the mean flow obtained with the pressure-gradient technique was compared with that measured with an indicator-dilution tech-
Dog Studies

Mean aortic pressure range (mm Hg) | Stroke volume range (ml) | Peripheral vascular resistance range (dyne sec/cm²) |
--- | --- | --- |
Dog N | Stroke volume range (ml) | Peripheral vascular resistance range (dyne sec/cm²) |
1 | 151 | 94-240 | 9-80 | 900-4900 | 0.79-0.94 | 0.72-0.38 |
2 | 121 | 87-156 | 15-48 | 1400-3200 | 0.91-0.98 | 0.77-0.96 |
3 | 141 | 54-179 | 10-40 | 1200-5200 | 0.82-0.97 | 0.37-0.89 |
4 | 114 | 80-228 | 11-40 | 1500-6900 | 0.66-0.89 | 0.24-0.78 |
5 | 75 | 75-193 | 14-43 | 1400-4400 | 0.76-0.87 | 0.11-0.56 |
6 | 87 | 65-196 | 8-38 | 1700-5000 | 0.72-0.95 | 0.32-0.83 |
7 | 104 | 75-150 | 11-44 | 1900-3800 | 0.78-0.97 | 0.40-0.96 |
8 | 137 | 65-196 | 8-38 | 1400-6500 | 0.78-0.94 | 0.43-0.94 |

N = number of beats, r = range of correlation coefficients for the control period and interventions considered separately, and r correlation coefficient for control period and all interventions combined. Complete details of methods A–F are given in the text.

C: \[ SV_4 = K_4(P_{ae})(1 + \frac{T_2}{T_4}) \] (refs. 4, 8).

D: \[ SV_4 = K_4(P_{ae})(1 + \frac{S_e}{D_e}) \].

E: \[ SV_4 = K_4(P_{ae} - P_e) \] (ref. 11).

F: \[ SV_4 = K_0(PP)(T_2) \] (ref. 13).

G: \[ SV_4 = 460(T_2) - 47.3 \] (ref. 13).

Figure 1 graphically illustrates how the following parameters were obtained from the aortic pressure pulse.

\[ S_a = A_1 + A_2; \]
\[ D_a = A_3 + A_4; \]
\[ P_{md} = A_3 - A_1; \]
\[ P_{se} = A_2 + A_3 - [(P_2 - 20)(T_2)]. \]
\[ PP = P_e - P_d; \]
\[ T_2 = T_4 - T_2; \]
\[ T_d = T_2 - T_4. \]

\( P_a, P_d, \) and \( P_m \) are the systolic, diastolic, and mean aortic pressures (mm Hg), respectively. \( T_2 \) is the onset of ejection, and \( T_4 \) is the end of ejection. \( T_2^* \) is the onset of ejection of the following beat. \( T_3 \) and \( T_3 \) are points 80 msec before \( T_2 \) and \( T_4 \), respectively. \( A_1, A_2, A_3, \) and \( A_4 \) are the areas indicated in Figure 1 under the aortic pressure curve above 20 mm Hg. The constants \( K_1-K_4 \) are proportionality constants determined when each method is calibrated. Method C was obtained from the regression of true flow on ejection time for patient 5 (13) and then used for all patients. Recognition of the onset and the end of ejection from the aortic pressure wave was accomplished by the method described by Starmer et al. (15). In addition to these parameters, the mean central venous pressure and the true stroke volume were computed. True stroke volume was calculated by digital integration of the phasic aortic flow curve during ejection using the method described by Benson (16). Peripheral vascular resistance (dyne sec/cm²) was estimated for each pacing rate during each intervention by dividing the difference between mean aortic pressure and mean central venous pressure by the mean blood flow rate.

A summary computer card was punched for each beat analyzed containing \( SV_4 - SV_1 \), mean aortic pressure, peak systolic pressure, diastolic pressure, ejection time, and measured stroke volume. The resulting values were analyzed by computing correlation coefficients between measured stroke volume and estimated stroke volume obtained with methods A–G. Correlation coefficients were computed for each separate intervention in the dog studies and for all pooled interventions in both the dog and the human data. In addition, regression lines relating measured stroke volume and estimated stroke volume were computed for each intervention for each subject. The percent estimation error at the midrange of true stroke volume was also computed for the human data. Hotelling \( T^2 \) statistics (17) were used in testing the equivalence of various estimators.

\[ = T_4 - T_2. \]

\( P_8, P_d, \) and \( P_m \) are the systolic, diastolic, and mean aortic pressures (mm Hg), respectively. \( T_2^* \) is the onset of ejection of the following beat. \( T_3 \) and \( T_3 \) are points 80 msec before \( T_2 \) and \( T_4 \), respectively. \( A_1, A_2, A_3, \) and \( A_4 \) are the areas indicated in Figure 1 under the aortic pressure curve above 20 mm Hg. The constants \( K_1-K_4 \) are proportionality constants determined when each method is calibrated. Method C was obtained from the regression of true flow on ejection time for patient 5 (13) and then used for all patients. Recognition of the onset and the end of ejection from the aortic pressure wave was accomplished by the method described by Starmer et al. (15). In addition to these parameters, the mean central venous pressure and the true stroke volume were computed. True stroke volume was calculated by digital integration of the phasic aortic flow curve during ejection using the method described by Benson (16). Peripheral vascular resistance (dyne sec/cm²) was estimated for each pacing rate during each intervention by dividing the difference between mean aortic pressure and mean central venous pressure by the mean blood flow rate.

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\[ = T_4 - T_2. \]
Results

The dog studies in this investigation were designed to determine whether any of the pulse-contour methods detailed above would provide estimates of stroke volume which were highly correlated with the value measured with an electromagnetic flowmeter. Table 1 is a summary of the hemodynamic and correlation coefficient data. Columns 3-5 show the total range in mean aortic pressure, stroke volume, and peripheral vascular resistance which were obtained during the control period and during all interventions. Obviously, a considerable range of hemodynamic data was available for analysis. The left column (ri) under each of the individual stroke volume estimation methods (A-F) shows the range of correlation coefficients relating the estimated and the measured stroke volumes obtained by analyzing the data from the control period and from each intervention separately. These correlation coefficients were quite good with the exception of method B, which was shown by a Hotelling T2 statistic to be significantly poorer (P < 0.05) than the other methods. No statistically significant difference was demonstrated among any of the remaining methods (P > 0.05). The right column (rc) under each of the methods shows the correlation coefficients obtained when data from the control period and from all interventions were combined in a single analysis. These combined correlation coefficients were, in general, quite poor and in over half the instances were lower than the poorest individual value. From the pooled correlation data, no significant difference (P > 0.05) was demonstrated among any of the six methods examined. The best combined correlations were seen in dogs 2 and 7, and it is of interest to note that these dogs experienced the least change in peripheral vascular resistance during the study.

Table 2 shows the complete hemodynamic, correlation, and regression data for dog 1 which was typical of all dogs included in this study. Inspection of the regression data for any estimation method shows a considerable range of intercepts, most of which are significantly different from zero (P < 0.05), and a wide range of slopes. In Figure 2A, the individual data points and the calculated regression lines are plotted for method F in this same dog. It was consistently observed that a completely different relationship existed between this estimator and the measured stroke volume during phentolamine infusion than that seen during either the control situation or the isoproterenol intervention. The control and the isoproterenol data seem to form a single, curvilinear relationship, but the phentolamine data lie on a separate regression line. The effect of combining all data into a single regression analysis is also seen in Table 2. Relatively homogeneous data sets, each with a high correlation coefficient but different regression parameters, were combined to create a heterogeneous data set which had a low correlation coefficient and a large standard error of estimate.

Hemodynamic data obtained in the two groups of patients are shown in columns 4-6 of Table 3. Group 1 included 4 patients in atrial fibrillation, who exhibited a wide range of stroke volumes due to variations in ventricular filling time, and 5 patients in complete heart block, in whom stroke volume variations were due to the varying contribution of atrial systole to ventricular filling and to the induced changes in heart rate from ventricular pacing. In this group of patients it was assumed that the peripheral vascular resistance, and hence the pressure-flow relationship, did not vary significantly during the 3-minute recording. Group 2 (Table 3) consisted of 8 patients in normal sinus rhythm in whom the stroke volume was altered by various interventions. It is obvious from the range...
Hemodynamic and Regression Data for Dog 1

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>Mean aortic pressure range (mm Hg)</th>
<th>Stroke volume range (ml)</th>
<th>Peripheral vascular resistance range (dyne sec/cm²)</th>
<th>r</th>
<th>t</th>
<th>A</th>
<th>S</th>
<th>SEE</th>
<th>r</th>
<th>t</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>48</td>
<td>110-144</td>
<td>24-61</td>
<td>1500-2000</td>
<td>0.94</td>
<td>9.1</td>
<td>4.3</td>
<td>4.2</td>
<td>0.83</td>
<td>-28.3</td>
<td>5.6</td>
<td></td>
</tr>
<tr>
<td>Iso</td>
<td>49</td>
<td>94-142</td>
<td>34-80</td>
<td>900-1600</td>
<td>0.79</td>
<td>10.6*</td>
<td>3.7</td>
<td>6.9</td>
<td>0.72</td>
<td>-15.6*</td>
<td>4.6</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>54</td>
<td>185-240</td>
<td>9-48</td>
<td>3100-4900</td>
<td>0.90</td>
<td>7.5</td>
<td>2.0</td>
<td>3.3</td>
<td>0.79</td>
<td>-4.3*</td>
<td>2.4</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>151</td>
<td>94-240</td>
<td>9-80</td>
<td>900-4900</td>
<td>0.67</td>
<td>15.8</td>
<td>2.6</td>
<td>12.9</td>
<td>0.67</td>
<td>-6.4*</td>
<td>3.5</td>
<td></td>
</tr>
</tbody>
</table>

N = number of beats, r = correlation coefficient, I = regression intercept, S = regression slope, SEE = standard error of estimate. Conditions are C = control, Iso = isoproterenol, and P = phenylephrine. Complete details of methods A-F are given in the text.

*Regression intercepts that were not significantly different from zero (P > 0.05).

of peripheral vascular resistance values that the hemodynamic status in these patients, except for patient 10, was altered considerably.

The analyses relating the measured stroke volume to the various estimates of stroke volume are given in columns 7-17 of Table 3. The complete regression data for all methods examined were similar and only method F is shown in detail. Correlation coefficients are listed for the other methods. The correlation coefficients obtained in the patients with atrial fibrillation and complete heart block generally indicated good linear relationships between estimated and true stroke volume. The percent error at the midrange of estimated stroke volume varied from 11 to 20% in these 9 patients. In the 8 patients with normal sinus rhythm, the combined data from all interventions is shown. The correlation coefficients obtained were uniformly poor. The percent error at the midrange of estimated stroke volume varied from 12 to 29%. Note that in group 2, the patients are listed in increasing order of the magnitude of the changes in peripheral vascular resistance which occurred during the study and that the correlation coefficients generally decrease as this resistance change increases. In each of the 17 patients, the intercepts of the regression line relating estimated stroke volume to measured stroke volume were significantly different from zero (P < 0.05) for all methods, except in a few isolated instances. As noted in the combined data from the dog studies, no statistical difference among the various estimation methods (P > 0.05) could be determined.

Method G examined the relationship between the duration of ejection alone and the stroke volume. In the patients with complete heart block, method G provided correlation coefficients which were as good as those from any of the pulse-contour methods. However, in the patients with atrial fibrillation, the correlations were not so good. This finding is probably due to the nonlinear relationship between duration of ejection and stroke volume which has been described in patients with atrial fibrillation (18).

**Discussion**

The purpose of the present study was to determine whether any of the currently available methods for estimating stroke volume from a single central aortic pressure are sufficiently accurate to be used as a means of obtaining multiple, frequent determinations of cardiac output under clinical conditions. It was felt that none of these methods could be justified completely on biophysical grounds, and thus the empirical evaluation described in this paper was designed. Although relatively good correlation coefficients were obtained over a range of stroke volumes within each intervention in which the pressure-flow relationship remained relatively unchanged, these coefficients were quite poor when all data from one dog were combined in a single analysis. In addition, the data suggest that the simple linear models currently available are not adequate estimators of stroke volume over a sufficiently wide range of hemodynamic conditions governing the pressure-flow relationship. It is concluded that these pulse-contour estimation methods, in their present form, are of little value in monitoring acutely ill patients and following their response to therapy.

The high correlation between measured stroke volume and the estimates obtained by the single aortic pressure techniques when each intervention was analyzed separately indicate that, with the exception of method B, linear models can be used to approximate the relationship between these two quantities. Eqs. 4-9 imply that the pressure-flow relations.
relationships predicted by these models pass through the origin. However, the finding of significant nonzero regression intercepts suggests that we are dealing with a nonlinear system which, for some ranges of data, can be adequately represented by a linear system. This demonstration of nonzero regression intercepts is not new. Herd et al. (11) noted that positive intercepts were obtained when their data were analyzed as a simple linear model. Kouchoukos et al. (8) showed that the regression line relating method C to measured stroke volume had a nonzero intercept and, similarly, Alderman et al. (10) showed that both methods B and C had nonzero intercepts.

If it is accepted that a linear model is adequate to describe the relationship between measured and estimated stroke volume over a limited range, then the finding of nonzero regression intercepts has an important implication with regard to calibration of these methods. When it is assumed that the intercept is in fact zero, as Eqs. 4-9 imply, then the origin serves as one calibration point and only one determination of stroke volume by an independent method is required to calibrate the method in use. However, since nonzero intercepts were consistently observed, two separate determinations of stroke volume must be made by an independent method at points widely spaced with respect to the error of the particular pressure-pulse method to fix the calibration line accurately. Figure 2B demonstrates the magnitude of error which could be made in prospective estimation of stroke volume by assuming that the regression intercept is zero when it is not. The solid line represents the combined data for method F in dog 1, which had a nonzero intercept. If a calibration were performed at point C and a zero intercept was assumed, the stroke volume estimation would be accurate only at the point of calibration. As the estimation index varies about the calibration point, the true stroke volume would be overestimated by as much as 20% or underestimated by as much as 60%.

The control and the isoproterenol data in Figure 2A, which were obtained at comparable levels of mean aortic pressure for method F, demonstrate a consistent finding in the dog studies. The stroke volumes measured during the control situation included values which were smaller than the lowest values measured during the isoproterenol intervention, and the total range of values was less during the control period. This phenomenon usually resulted in the control regression slope being larger than the slope obtained from the isoproterenol data. In addition, the control regression intercept was usually lower than the corresponding value for isoproterenol. These findings again suggest that the relationship between measured and estimated values is nonlinear, particularly for low values of stroke volume.

The relationship between the data obtained during the various interventions (Fig. 2A) provides an explanation for the poor correlation coefficients found in the combined data. The data obtained during phenylephrine infusion were clearly separate from those obtained during the control period and during isoproterenol infusion. This separation was also true of the data recorded in four of these dogs during acute hemorrhage. Only a limited range of stroke volumes were available for analysis during hemorrhage, but these data tended to fall at the low end of the range of phenylephrine data and were separate from the control and the isoproterenol data. The combination of these heterogeneous data into a single analysis would clearly result in a low correlation coefficient.

In reducing a stroke volume estimation model which depends on measuring two pressures simultaneously to one which involves only a single pressure, it is generally assumed that the velocity with which pressure waves are propagated in the arterial system is fixed. In a series of dog studies,
Malindzak and Stacy (19) found that arterial pulse propagation velocity could be altered significantly from the control values by the administration of vasopressors and vasodilators. This finding indicates that any single-pressure method which depends on the assumption of a constant pulse propagation velocity would have serious limitations in situations where the pressure transmission characteristics varied to any appreciable extent. Therefore, statements supporting single-pressure methods can be made only if changes in the pressure transmission media can be represented by a suitable function of a single pressure.

The same pulse-contour methods were evaluated in two groups of patients. Group 1 included nine patients in whom the peripheral vascular resistance, and hence pulse transmission characteristics, would not be expected to change significantly during the brief period of study; group 2 included eight patients in whom various interventions were used to alter the peripheral vascular resistance over a wide range. As noted above, reasonable correlation coefficients were obtained in group 1. Nonzero regression intercepts were also consistently observed. These results are analogous to the data obtained when the dog study interventions were analyzed individually. In group 2, however, the correlations observed were quite poor and these results were similar to those obtained when the dog data were analyzed by combining all interventions within each dog. The analysis of each separate intervention again revealed high correlations. Slopes and intercepts computed for each intervention were different. Therefore, the low correlations observed in the combined human data resulted from mixing multiple homogeneous data sets into one heterogeneous data set and are consistent with the results of the dog studies.

In the present study, evaluation of the various methods for estimating stroke volume from a single central aortic pressure was performed under controlled laboratory conditions. A separate trial of method F was conducted under clinical conditions on the surgical intensive care unit. Forty patients who underwent open-heart surgery were studied for up to 3 days. A Sorenson catheter was used to continuously monitor the central aortic pressure, and cardiac output was estimated by a dye-dilution technique. The first cardiac output determination was used to calibrate the pulse-contour method. The results indicated that subsequent estimates of cardiac output over a 24-hour period using method F were poorly correlated with the dye-dilution determinations. The wide variation in hemodynamic status in these patients, along with the difficulties inherent in the single-point calibration, contributed significantly to these poor results.

Two important conclusions can be made based on the results of the present paper. Several pulse-contour methods can provide reasonable estimates of stroke volume when the data are collected under...
### Table 3

**Patient Studies**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Condition</th>
<th>N</th>
<th>Mean aortic pressure range (mm Hg)</th>
<th>Stroke volume range (ml)</th>
<th>Peripheral vascular resistance range (dynes sec/cm²)</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>G</th>
<th>r</th>
<th>I</th>
<th>S</th>
<th>E.E.</th>
<th>Midrange % error</th>
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<tr>
<td>1</td>
<td>A.F.</td>
<td>45</td>
<td>69-89</td>
<td>11-65</td>
<td>1500</td>
<td>0.87</td>
<td>0.83</td>
<td>0.85</td>
<td>0.84</td>
<td>0.89</td>
<td>0.61</td>
<td>0.86</td>
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<td>3.2</td>
<td>6.3</td>
<td>18</td>
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<tr>
<td>2</td>
<td>A.F.</td>
<td>24</td>
<td>82-96</td>
<td>10-60</td>
<td>1200</td>
<td>0.89</td>
<td>0.82</td>
<td>0.88</td>
<td>0.88</td>
<td>0.95</td>
<td>0.92</td>
<td>0.96</td>
<td>6.2</td>
<td>4.8</td>
<td>5.7</td>
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<tr>
<td>3</td>
<td>A.F.</td>
<td>46</td>
<td>86-121</td>
<td>10-54</td>
<td>2200</td>
<td>0.90</td>
<td>0.88</td>
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<td>0.88</td>
<td>0.51</td>
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<td>8.9</td>
<td>3.0</td>
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*Intercept values were not significantly different from zero (P > 0.05).
controlled situations in which only minimal variations in the determinants of the relationship between pressure and flow occur. However, when stroke volume is estimated following a number of interventions which alter the relationship between pressure and flow, these estimates become unreliable. Thus, these estimation methods are of little value in monitoring the acutely ill patient in whom the hemodynamic status is variable. In addition, the finding of significant nonzero regression intercepts indicates that the linear models proposed thus far are not adequate to describe the relationship between the aortic blood pressure contour and the stroke volume.

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References

Evaluation of Several Methods for Computing Stroke Volume from Central Aortic Pressure
C. Frank STARMER, PHILIP A. MCHALE, FREDERICK R. COBB and JOSEPH C. GREENFIELD, Jr.

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