Estimation of Stroke Volume Changes by Ultrasonic Doppler

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SUMMARY. The purpose of this study was to conduct a controlled evaluation of the continuous-wave Doppler technique for the estimation of stroke volume changes.

Six anesthetized dogs were studied. Aortic blood velocity was recorded from the suprasternal notch by a special continuous-wave Doppler unit. Cardiac output was varied by fluid infusion and exsanguination, and over 300 simultaneous records of aortic blood velocity and thermodilution cardiac output were taken. Average stroke volume and average systolic velocity integral (SVI), the area under the Doppler velocity curve, were calculated.

The relationship of SVI to stroke volume was evaluated for each animal using linear regression. Average results were: correlation coefficient 0.95 ± 0.04 SD; y-intercept 0.38 ± 0.14 cm (SD); standard error of fit 0.29 ± 0.03 cm (SD).

These data show that the systolic integral of aortic blood velocity was essentially directly proportional to stroke volume, even over a six-fold range. Thus, this technique will provide an accurate noninvasive estimate of changes in stroke volume.

THE CONTINUOUS-WAVE ultrasonic Doppler technique recently has been demonstrated to be potentially useful for noninvasive estimation of cardiac performance through the transcutaneous measurement of aortic blood velocity.1,2 This method utilizes an ultrasonic beam which originates from a transducer in the suprasternal notch and can be directed toward either the ascending aorta or the aortic arch. Both the magnitude and the direction of aortic blood velocity are indicated in real time.

While clinically useful information is provided directly by the velocity record, a question of additional interest is whether the Doppler technique gives a reliable indication of changes in stroke volume. Stroke volume is equal to the time integral of volume flow rate over each ejection interval. This is equivalent to aortic cross-sectional area times the integral of mean aortic velocity. The Doppler technique does not measure average velocity, but measures the highest instantaneous velocity observed in the ultrasonic field.3 It is reasonable to assume that the highest velocity is proportional to mean velocity over the physiologic range of flow rates, which is equivalent to assuming a constant shape of the spatial aortic velocity profile. If, in addition, the aortic cross-sectional area remains constant, or if area and velocity profile changes cause errors which cancel, then the time integral of the ultrasonically-measured velocity should vary in direct proportion to stroke volume. This measurement, the "systolic velocity integral" (SVI), is simply equivalent to the area under the time course velocity curve for each ejection.

If such proportionality can be shown to hold over a wide range of cardiac output, the Doppler-based SVI offers the promise of a simple, noninvasive method for assessing relative changes in stroke volume and cardiac output. The purpose of this study was to evaluate the extent to which the SVI provides a useful estimation of stroke volume change. Experiments were carried out using dogs so that cardiac output could be controlled over a wide range. The velocity measurements obtainable from the dog's ascending aorta are equivalent to those available in humans, so the results obtained should apply to patients.

Methods

Six dogs weighing 18–22 kg were anesthetized with sodium pentobarbital (25 mg/kg intravenously). Following endotracheal intubation, the animals were placed on assisted ventilation at a tidal volume of 15 cc/kg. Femoral arterial and venous catheters were placed, and a pulmonary artery thermodilution catheter (Swan-Ganz 7 Fr) was placed through a cutdown into the right jugular vein. The catheter was connected to an Edwards model 9510 cardiac output computer. A continuous electrocardiographic record (lead I) was obtained and recorded with the Doppler data.

Ultrasonic access to the ascending aorta was achieved by manual positioning of the Doppler probe in the suprasternal notch (fig. 1). In a preliminary animal (not one of the six experimental animals), this ultrasonic access was verified by the following procedure: 1) manually positioning the Doppler probe in the suprasternal notch and aiming to obtain the maximum observable systolic velocity, 2) right lateral thoracotomy, and 3) dissecting out the ascending aorta. From this preliminary experiment it was clear that maximum velocity was observed when the Doppler probe was aimed directly at the aortic root, and in this position the angle between the ultrasonic beam direction and the aortic axis was very nearly zero.

Thermodilution cardiac outputs were obtained by the infusion of 5 cc or 10 cc of 5% dextrose in water (depending on the calibration factor) through the proximal lumen of the Swan-Ganz catheter. Integration of the resulting temperature curves from the pulmonary artery thermistor was carried out automatically by the cardiac output computer. The measurement of cardiac output by this method is generally regarded to be accurate to within 10–20%.3,4

Cardiac output was varied by sequential intravenous fluid infusion and exsanguination. First, lactated Ringer's solution was infused in 300 cc increments via the venous catheter to increase cardiac preload. After each incremental infusion, 3–5 minutes were allowed for stabilization. This process was continued until the cardiac output (by thermodilution) could no longer be raised with fluid infusion. Generally 1200–1500 cc of fluid was required. Exsanguination was then carried out via the arterial catheter in 500 cc decrements of blood volume with equivalent stabilization periods. When thermodilution cardiac outputs of less than 1.00 L/min were reached, the experiment was terminated.

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Simultaneous records of ultrasonic aortic blood velocity and thermodilution cardiac output were taken at each state of blood volume (3-5 recordings at each level). The Doppler data, ECG, and voiced comments were recorded on magnetic tape for later playback and analysis, using a Gould model 2400 strip chart recorder.

The blood velocity data corresponding to each thermodilution measurement were analyzed from the time of injection until the output calculation was complete. For each Doppler recording, the average values of heart rate, peak velocity during ejection, and systolic velocity integral were determined from five or more beats. Although a simple electronic circuit was ultimately developed to calculate SVI values, all the data presented here were obtained by manual counting of the area under each velocity curve, in order to assure consistency. The cardiac output values obtained by thermodilution were divided by heart rate to yield values of mean stroke volume.

For each animal, these values of peak velocity and SVI were plotted vs average stroke volume. A linear regression line, correlation coefficient, and standard error of fit were calculated for each graph. Combined data for the series of six animals were used to calculate the mean and standard deviation values for correlation coefficient, intercept, and standard error of fit.

Results

Figure 2 shows a recording of one ejection as it appears at the output of the CW Doppler unit. Doppler shift frequency (proportional to aortic blood velocity) is represented on the ordinate vs time on the abscissa. The systolic velocity integral and peak velocity are indicated.

The regression line data of SVI vs stroke volume are shown in figure 3 for one of the animals. For all six experiments the systolic velocity integral correlated strongly with stroke volume: $r = 0.95 \pm 0.04$, Y intercept $= 0.38 \pm 0.14$, standard error of fit 0.29 $\pm 0.03$. The slopes of the regression lines were not tabulated, since, as we expected, these showed great variability from animal to animal.

On the other hand, peak velocity did not correlate well with stroke volume in this series of experiments. It did appear to vary in proportion to stroke volume at low values of stroke volume, but a definite plateau effect was noted at higher levels of stroke volume. This effect is well shown in figure 4, a plot of peak velocity vs stroke volume for one of the six animals. The reason for the disparity between peak velocity and SVI is indicated in figure 5, which shows velocity records obtained at low and high levels of stroke volume. At low stroke volumes, the ejection period is abbreviated and the velocity record is symmetrical, while at high values of stroke volume there is prolongation of ejection and a loss of symmetry. Other investigators have also...
observed changes of ejection shape and duration in response to altered hemodynamic conditions or heart rate. It is apparent that increased flow is accomplished by both a broadening of the ejection time course and an increase of peak velocity.

Two out of the six animals displayed a sudden and striking phenomenon which was noted only at very low levels of stroke volume. In these animals, a sudden rise of the indicated aortic blood velocity occurred. These velocity levels were far in excess of those expected from the measured stroke volume (by thermodilution). Figure 6 shows the regression data from one of these animals, with the data in question represented by open circles. It can be clearly seen that these data points do not fall on the normal regression line for the rest of the data. Furthermore, the occurrence of this phenomenon 1) was paroxysmal in nature, 2) was readily recognizable, 3) occurred only when the animal was in severe shock, and 4) was reversible by the infusion of volume to bring the stroke volume back up to higher levels. What actually caused this dissociation of the two measurements in these animals is not clear. In fact, it is not apparent whether either or both of the measurement techniques are in error, or whether the physical basis for their comparison (e.g., the size or orientation of the great vessels) changed suddenly in response to the progressive decrease of blood volume. Because of these characteristics and the fact that this behavior occurred in only two animals, these data points were eliminated from the statistical analysis of the cumulative data for all six experiments. If, however, all the points are included in the regression analysis, the correlation coefficient for the data in figure 6 becomes \( r = 0.91 \), that for the other experiment displaying this phenomenon becomes \( r = 0.95 \), and the average for all six experiments becomes \( r = 0.93 \). Although this phenomenon has not been reported in humans, further study may be indicated to determine the etiology and situation of occurrence of this effect.

**Discussion**

Continuous monitoring of stroke volume and cardiac output is desirable in severely ill patients. Currently available invasive techniques for monitoring carry significant risk, are expensive and require trained personnel with special expertise. In this experiment we have utilized a relatively inexpen-

![Figure 4](image-url)  
**Figure 4.** Plot of peak velocity vs average stroke volume for one animal.

![Figure 5](image-url)  
**Figure 5.** Tracings of aortic blood velocity (A) at low, and (B) at high levels of stroke volume in one animal.

![Figure 6](image-url)  
**Figure 6.** Systolic velocity integral vs average stroke volume in one animal. Unexpectedly high values for SVI at low stroke volume levels are shown as open circles.
sive, reusable instrument which can be operated by most nursing and paramedical personnel.

Previous studies have demonstrated the feasibility and potential utility of measuring aortic blood velocities non-invasively in man using the continuous wave Doppler technique.1, 2, 7 The possibility of estimating changes in stroke volume from velocity measurements has also been indicated in an earlier study.2 Although general proportionality between Doppler and dye based measurements of stroke volume was shown in patients, several reasons for uncertainty were apparent. These include: 1) the small number of data points accumulated on any one patient, 2) the relatively small range of stroke volume studied on any patient, and 3) the use of pacing to vary cardiac output. The purpose of the present study was to evaluate the CW-Doppler technique throughout a wide range of stroke volume under controlled conditions in animals.

Two developments were necessary for the execution of these experiments. The first was the design and construction of a CW Doppler instrument which utilizes new signal processing techniques to discriminate the highest frequencies from the rest of the Doppler shift spectrum.5 Small size and ease of operation make this unit ideal for use in both the experimental and clinical environments. Second was the determination that excellent velocity information could be obtained from the ascending aorta. Previous investigators have emphasized measurements in the aortic arch since the ultrasonic beam from the suprasternal notch subtends an angle of approximately zero with respect to the red blood cell velocities in the arch. Thus, the cosine term in the Doppler shift equation should be equal to unity, and the measured velocities should be identical to the actual red cell velocities. We have observed, however, that ultrasonic access to the aortic arch is sometimes difficult or impossible in patients. On the other hand, while the angle subtended by the ultrasonic beam with respect to blood velocity in the ascending aorta may not be zero, it is constant and therefore the proportionality of Doppler velocity to actual blood velocity should hold.

The results of this study conclusively demonstrate that the integral of systolic aortic blood velocity, SVI, is very closely correlated with stroke volume over a wide range of cardiac outputs. The strong correlation coefficients obtained, and the relatively small intercept values, show that the SVI is essentially directly proportional to stroke volume.

It is important to note that no absolute estimate of stroke volume is provided by the Doppler data. As would be expected from variations in aortic size and orientation relative to the ultrasonic beam, the proportionality coefficients were highly variable between the animals. Thus, relative changes of stroke volume can be accurately assessed with the Doppler technique, but absolute quantitation of stroke volume requires that a point calibration be made with another technique such as dye dilution or thermodilution. For most clinical circumstances, however, this index of relative changes in stroke volume should provide an adequate monitor of alterations in cardiac output while avoiding repetitive invasion of the blood stream.

These results also indicate that the measurement of peak velocity alone is not reliably indicative of changes in stroke volume. Although a fair correlation between peak velocity and stroke volume is evident at low cardiac outputs, this is not true as stroke volume increases to higher values. This is because at high stroke volumes the peak velocity approaches a limit and the increased flow is apparent as a broadening of the time course of ejection rather than an increase in absolute aortic blood velocity.

Of further note is the sudden and significant change in the relationship of SVI to stroke volume which occurred in two animals. In these experiments, this readily recognizable phenomenon occurred only when animals were in a state of severe shock. It was also reversible by the infusion of lactated Ringer's solution in sufficient quantity to raise stroke volume above the threshold at which it occurred. Thus, the effect, even if present in humans, is not expected to hinder the clinical utility of the CW Doppler technique.

In conclusion, the noninvasive ultrasonic determination of aortic blood velocity has been shown to provide an accurate index of changes in stroke volume over a wide range of cardiac outputs. Our early experience with the use of this instrument in intensive care patients indicates that the results of this experiment may be simply and directly applied to humans. The clinical potential of this technique is vast, and may extend the benefits of accurate monitoring of cardiac output to virtually all hospital patients over the entire course of hospitalization.

References

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