Ultrasonic Cardiac Echography for Determining Stroke Volume and Valvular Regurgitation

By Richard L. Popp, M.D., and Donald C. Harrison, M.D.

SUMMARY

The ventricular dimensions of 51 patients with heart disease were determined by ultrasonic echography during cardiac catheterization. These data were used to calculate end-diastolic and end-systolic volumes and stroke volume, using a prolate ellipse as a geometric model of the left ventricle. In 30 patients without valvular regurgitation the stroke volumes determined by the echographic method were compared with those determined simultaneously by the standard Fick method with a correlation coefficient of $r = 0.966$. In 21 patients with valvular regurgitation, the severity of regurgitation was estimated by comparing the forward stroke volume determined by the Fick method with the total left ventricular stroke volume determined by the echographic method. These calculations of regurgitation correlated reasonably well with the degree of valvular regurgitation estimated from angiocardiographic study. It is suggested that these echographic determinations of stroke volume are an atraumatic, safe, and acceptable method in patients without valvular regurgitation. Moreover, these preliminary studies suggest that the severity of valvular regurgitation can be estimated by utilizing ultrasound echocardiography.

Additional Indexing Words:
- Prolate ellipse
- Ventricular dimensions
- Angiocardiographic ventricular volume
- End-diastolic volume
- Fick cardiac outputs
- End-systolic volume

Although the commonly used methods for determining cardiac output are reliable in most conditions, several limitations to their widespread use are apparent. The Fick and indicator-dilution technics require arterial puncture, cardiac catheterization, and blood sampling with analysis of specific elements within the blood, such as oxygen or indicator substances. The indicator-dilution methods may lose reliability when cardiac output is very low or when valvular regurgitation is present. Although the Fick method gives a reproducible measure of forward or effective cardiac output, it does not allow determination of the actual volume of blood pumped by the ventricle in the presence of valvular regurgitation. The only available methods of determining the volume of regurgitation in valvular heart disease and beat-to-beat variation in the stroke volume are the angiographic technics. Many objections to the routine use of volume angioigraphy are readily apparent. These include the dangers of multiple injections of hypertonic contrast medium into the heart and circulation, the effects of the contrast medium on ventricular function, the tedious nature of measurement technics, the need for costly angiographic equipment and the lack of the ability to perform multiple or repeated studies. With increasing appreciation of the limitations of the existing technics, several investigators have sought less traumatic and more readily repeatable methods for cardiac output calcula-
tion. These less traumatic methods have included assumption of arterial gas content from specialized methods of venous sampling, \(^6\) approximation of arterial CO\(_2\) content by end-tidal respiratory gas content and venous CO\(_2\) estimation by rebreathing techniques, \(^7\) and ballistocardiographic methods. \(^8\)

Recently the use of ultrasound for the measurement of stroke volume has been attempted. \(^9\) Ultrasonic echographic methods are safe, atraumatic, and require relatively inexpensive instrumentation, and therefore are attractive for general clinical use. While the initial results of these ultrasonic echographic studies of stroke volume were good, analysis of the basic rationale for achieving these good results and standardization of the procedure were lacking. Previous studies have attempted to compare echographic and angiographic measurements of ventricular dimensions (personal communication from H. Feigenbaum). The basic measuring techniques for echographic data employed in our studies were initially developed while the senior author (R.L.P.) worked with Dr. Feigenbaum and associates, and will appear in a paper to be published (personal communication from H. Feigenbaum). The standardized method for obtaining records which has been developed allows comparison of patients with various forms of heart disease, including those with right ventricular enlargement. \(^10\) Increases in right heart size had been a problem in the past. The present study was designed to evaluate these modified echographic techniques for determination of stroke volume in patients with various forms of heart disease. The initial problem in which these new methods were considered to be of potential use was in the assessment of actual left ventricular stroke volume in the presence or absence of valvular regurgitation.

**Methods**

Cardiac echograms were obtained from 54 patients who were undergoing cardiac catheterization and angiography for diagnostic evaluation. The first 45 patients were randomly selected, and the subsequent nine patients were selected for study because of the absence of valvular regurgitation by clinical examination. The final diagnoses of all patients are outlined in table 1. The cardiac echograms were obtained during the time of blood and expired gas collections used for cardiac output determination by the direct Fick method. Echograms were recorded both during resting and exercise Fick collections in 11 of the 54 patients. An electrocardiogram was recorded during the Fick and echographic studies for heart rate determination. The echographic examination was completed well within the 5 or 6-min period over which the Fick collection was made. Since echograms of technical quality adequate for measurement were not obtained in three subjects, data on these subjects were not included in this study. It appears that these three patients out of a total of 54 will probably represent the number of patients in whom it is not possible to obtain adequate records for calculation of ventricular size and volume.

All cardiac echograms were recorded using a Smith Kline Ekoline 20 instrument which produced 1,000 pulses/sec utilizing a 2.25 mega-Hertz transducer of 0.75 inch diameter. The transducer acts as a sound transmitter for 1 msec, and as a sound receiver for the remaining 999 msec until the subsequent pulse. The echo received from each interface between tissues of differing acoustical impedance is displayed on a cathode-ray tube. The elapsed time between transmission and reception of the signal allows measurements of the distance between the transducer and each interface since the ultrasound waves have a relatively constant velocity through most soft tissue. The echoes are displayed on the face of the tube at 0.5-sec intervals and a single lead electrocardiogram may be incorporated into the record. In addition, a scale of depth with markers spaced 2 mm apart allows accurate measurement of the scale within 1 mm.

All patients were studied in the supine position with the transducer placed in the fourth or fifth intercostal space at the left sternal border. The transducer was directed slightly inferolaterally until the strong posterior pericardial pleural and left ventricular wall signals were recorded. These echoes are recognized by their characteristic motion with reference to the electrocardiogram and cardiac cycle. The transducer was then rotated superomedially until the classic pattern of rapid mitral valve motion was observed. From this position the transducer was again rotated inferolaterally until the mitral valve was no longer in the beam. The sensitivity of the echography was adjusted to display all the echoes from the interventricular septum, as well as the left ventricular posterior wall, which are both recorded by the sound beam in this position (fig. 1). The face of the cathode-ray tube with its time-motion display was photographed for later analysis using Polaroid film. The transducer was
### Table 1

Summary of Results: Thirty Patients Without Valvular Regurgitation

| Patient | Study | Diagnosis | Dd (cm) | Ds (cm) | ΔL (cm) | SVE₁ (ml) | SVE₂ (ml) | SVF (ml) | SVE₁ − SVF | SVE₂ − SVF | SVE₁ − SVE₂ | Fick CO (L/min) | HR (beats/min) |
|---------|-------|-----------|---------|---------|----------|------------|------------|-----------|-------------|-------------|-------------|----------------|---------------|---------------|
| 1. C.G. | 1     | CAD       | 4.3     | 2.9     | 0.9      | 58         | 49         | 63        | −5         | −14         | 9           | 4.6           | 73            |
| 2. B.McC. | 2     | CMP       | 4.4     | 3.9     | 0.5      | 44         | 27         | 22        | 24         | +3          | −2          | 5             | 2.6           | 110           |
| 3. S.P. | 3     | AS        | 5.6     | 4.6     | 0.8      | 82         | 69         | 79        | +3         | −10         | 13          | 5.7           | 72            |
| 4. F.E. | 4     | AS        | 5.4     | 4.0     | 1.0      | 65         | 55         | 58        | +6         | −3          | 9           | 4.9           | 84            |
| 5. I.E. | 5     | MS        | 3.9     | 2.7     | 0.8      | 42         | 35         | 46        | −4         | −11         | 7           | 37            | 80            |
| 6. M.H. | 6     | HCVD      | 4.1     | 2.6     | 0.9      | 54         | 46         | 57        | −3         | −11         | 8           | 3.3           | 58            |
| 7. H.H. | 7     | AS        | 5.0     | 3.8     | 0.7      | 73         | 61         | 75        | −2         | −14         | 12          | 6.5           | 86            |
| 8. A.A. | 8     | AS        | 5.8     | 4.4     | 1.3      | 115        | 100        | 111       | +4         | −11         | 15          | 7.5           | 68            |
| 9. E.G. | 9     | Con. P    | 4.0     | 3.0     | 0.8      | 39         | 33         | 39        | 0          | −6          | 6           | 5.2           | 134           |
| 10. R.M. | 10    | AS        | 5.5     | 4.2     | 0.8      | 97         | 80         | 83        | +14        | −3          | 17          | 5.0           | 60            |
| 11. V.F. | 11    | AS        | 4.8     | 3.8     | 0.9      | 58         | 50         | 57        | +1         | −7          | 8           | 4.3           | 75            |
| 12. A.I. | 12    | CAD & VA  | 5.8     | 5.3     | 0.5      | 48         | 41         | 64        | −16        | −23         | 7           | 4.3           | 67            |
| 13. A.E. | 13    | MS        | 4.0     | 2.8     | 0.9      | 44         | 38         | 46        | −2         | −8          | 6           | 4.7           | 102           |
| 14. A.Y. | 14    | AS        | 4.7     | 3.2     | 0.9      | 74         | 63         | 65        | +9         | −2          | 11          | 4.1           | 63            |
| 15. M.R. | 15    | CMP       | 5.4     | 5.0     | 0.6      | 34         | 31         | 37        | −3         | −6          | 3           | 4.6           | 125           |
| 16. M.Z. | 16    | MS        | 3.9     | 2.2     | 1.3      | 51         | 46         | 59        | −8         | −13         | 5           | 4.1           | 70            |
| 17. J.D. | 17    | CAD       | 4.6     | 2.4     | 1.7      | 87         | 80         | 84        | +3         | −4          | 7           | 5.4           | 64            |
| 18. C.E. | 18    | CMP       | 4.8     | 3.6     | 0.9      | 60         | 51         | 56        | +6         | −5          | 9           | 4.4           | 78            |
| 19. M.J. | 19    | MS        | 3.0     | 2.0     | 0.8      | 41         | 35         | 48        | −7         | −13         | 6           | 4.0           | 84            |
| 20. G.S. | 20    | Coarct. & AS | 4.3   | 3.4     | 0.9      | 42         | 37         | 44        | −2         | −7          | 5           | 3.7           | 84            |
| 21. W.K. | 21    | CMP       | 5.5     | 4.9     | 0.8      | 51         | 46         | 43        | +8         | +3          | 5           | 3.4           | 79            |
| 22. B.P. | 22    | Funct. M  | 4.0     | 2.6     | 1.0      | 49         | 42         | 43        | −14        | −21         | 7           | 6.2           | 98            |
| 23. B.C. | 23    | Con. P    | 4.7     | 4.4     | 0.5      | 20         | 18         | 24        | −4         | −6          | 2           | 2.9           | 120           |
| 24. B.B.(R) | 24   | Cong. AVB | 5.4     | 4.0     | 1.2      | 98         | 84         | 99        | −1         | −15         | 14          | 3.3           | 33            |

Abbreviations: AR = aortic regurgitation; AS = aortic stenosis; CAD = coronary artery disease; CMP = cardiomyopathy; Coarct. = coarctation of the aorta; Cong. AVB = congenital atrioventricular block; Con. P = constrictive pericarditis; Dd = minor axis at end diastole; Ds = minor axis at end systole; Fick CO = cardiac output by the Fick method; Funct. M = functional murmur; HCVD = hypertensive cardiovascular disease; HR = heart rate; ΔL = change in major axis length; MR = mitral regurgitation; MS = mitral stenosis; SVE = echographic stroke volume; VA = ventricular aneurysm.
A cardiac echogram from the fourth intercostal space at the left sternal border is shown. The sound beam traverses the chest wall (CW), the lateral recess of the right ventricle (RV), the interventricular septum (IVS), left ventricular cavity (LV), and left ventricular posterior wall (LVPW). Dd = left ventricular dimension at end diastole; Ds = left ventricular dimension at end systole. Anterior is toward the top of the figure.

next placed on the chest wall at the point of maximal cardiac impulse and directed generally toward the right scapula. The transducer was adjusted to record the rapid motion of the anterior leaflet of the mitral valve. The sound beam was then directed medially and slightly anteriorly to record the echoes from the aortic root. The echo from the mitral aspect of the aortic root was preferentially placed on the film record for analysis (fig. 2).

The amplitude of this echo from the aortoventricular ring at the junction of the mitral and aortic valves as it moves toward the transducer at the physiologic cardiac apex is an approximation of the change in the apex to base length during the cardiac cycle. Although this procedure does not give a reliable measurement of the absolute apex to base length of the ventricle, it does allow measurement of the approximate change in length of the ventricular chamber.

Measurements

The left ventricular dimension (D), which approximates the diameter or minor axis, was measured from the endocardial echo of the posterior left ventricular wall to the endocardial echo of the left side of the interventricular septum (fig. 1). The phases of the cardiac cycle were determined from the electrocardiogram which was recorded simultaneously with the echogram or from the echographic pattern itself if no electrocardiogram was included. Analysis of the echographic patterns show a slight chamber enlargement as gauged by divergence of the posterior wall and septal echoes just after the P wave of the ECG, during atrial systole. At the peak of the R wave, the two echoes reverse their course and converge rapidly as the ventricular ejection period proceeds (fig. 1). Either the peak of the R wave or the point of reversal of the posterior wall and septal echoes prior to ejection was the point at which the end-diastolic dimension was measured (Dd, fig. 1). At end-systole the posterior wall and septal echoes have converged maximally. During isovolumic relaxation the interventricular septum echo usually shows a slight anterior motion and reversal of this motion which forms a notch in the echographic tracing. The posterior wall echo reaches its maximal anterior position at the onset of this notch in the septal motion and is the point at which the end-systolic dimension is measured (Ds, fig. 1). Extreme care must be taken to measure from the endocardial surface of the posterior wall rather than from the discrete echoes.
from the posterior chordae tendineae which are frequently recorded just anterior to the endocardial echo.\textsuperscript{11} The chordal echoes are recognized by their high intensity, sharpness, and lack of systolic increase in the distance to the myocardial echoes, which show an apparent increase in thickness from end diastole to end systole.

The change in the apex to base length, or major axis, was measured as the total excursion of the atrioventricular ring echo during the cardiac cycle ($\Delta L_1$, fig. 2). The pattern and amplitude of this echo is similar to the echo from the mitral ring described in detail by Zaky and associates.\textsuperscript{12} The average measurements from at least three cardiac cycles were used to determine the diastolic diameter, the systolic diameter, and the change in ventricular length. In those patients with atrial fibrillation and varying stroke volume, three or more cycles corresponding with cycle length of the average heart rate per minute were used. All measurements were made immediately after the records were obtained and before the results of the Fick cardiac output were known.

**Calculations**

Ventricular volumes at end diastole and end systole were calculated by using the end-diastolic dimensions ($D_d$) and the end-systolic dimensions ($D_s$) as ventricular diameters and the movement of the atrioventricular ring toward the apex ($\Delta L$) as ventricular length. Because of data from angiographic studies,\textsuperscript{13-15} the geometric shape of the ventricular shape was assumed to be a prolate ellipse (ellipsoid of revolution about the major axis). It had been demonstrated that the short diameters of the prolate ellipse are essentially equal for the left ventricular chamber.\textsuperscript{16}

Then the volume of a prolate ellipse is expressed as:

$$V = (\pi/6)LD^2 = (\pi/6)KD(D^2) = (\pi/6)KD^3$$

(1)

SVE\textsubscript{1} = $\pi/6(2)(Dd^3 - Ds^3)$

(1)

Stroke volume is the difference between the volumes of two such figures calculated using the end-diastolic dimension ($D_d$) and the end-systolic dimension ($D_s$) taken from the echogram (fig. 1):

$$SVE_1 = EDV - ESV = (\pi/6KDd^3) - (\pi/6KDd^3) = \pi/6K(Dd^3 - Ds^3),$$

(2)

where $EDV =$ end-diastolic volume, $ESV =$ end-systolic volume, $SVE =$ echogram stroke volume, and $K$ is a constant.

It is necessary to assume that the relationship of the chamber lengths and diameters remains the same during systole and diastole for stroke volume to be calculated. In a recent angiographic study\textsuperscript{17} of 20 subjects with heart disease, the ratio of diastolic length to diameter was the same as the ratio of the systolic length to diameter in those patients with significantly increased end-diastolic volume (greater than 300 ml). In subjects without significant increases in end-diastolic volume, the ratio of diastolic length to diameter averaged 1.9, and the ratio of systolic length to diameter averaged 2.3. When all subjects were considered together, the diastolic ratio of length to diameter averaged 1.8, and the systolic ratio 2.1.\textsuperscript{17} These observations confirm the findings of Gault and associates.\textsuperscript{18} Even if the diastolic length to diameter ratio changes during ejection, stroke volume calculation from the echographic measurements is still possible under these conditions. Since the change in the apex to base length ($\Delta L$) can be measured from the echogram (fig. 2), the end-systolic major axis length is found as follows:

$$\Delta L = Ld - Ls; \; Ls = Ld - \Delta L; \; Ls = Kd - L - \Delta L.$$  

(3)

The end-systolic volume calculated using this method is:

$$ESV = \pi/6Ls(Ds^2) = \pi/6Kd - \Delta L(Ds^2).$$

(4)

Since stroke volume is the difference between the end-diastolic and end-systolic volumes, it may be calculated by the formula:

$$SVE_2 = EDV - ESV = (\pi/6Kd^3) - (\pi/6Kd^3 - \Delta L) (Ds^2) = \pi/6[Kd^3 - (Kd - L - \Delta L)(Ds^2)].$$

(5)

Angiographic data from other studies\textsuperscript{16-19} prompted us to assume that the major axis is approximately twice as long as the minor axis, therefore the constant $K = 2$ in the formulae above. Using these values, the equation 2 can be rewritten as:

$$SVE_1 = \pi/6(2)(Dd^3 - Ds^3) = 1.047(Dd^3 - Ds^3).$$

(6)

Again using the constant $K = 2$ for the relationship of the major and minor axes at end diastole, the equation on line 5 can be reduced to:

$$SVE_2 = \pi/6 [(2Dd^3) - (2Dd - \Delta L)(Ds^2)].$$

(7)

Stroke volume was calculated from the Fick cardiac output as follows:

$$SVF = \text{Cardiac output} / \text{Heart rate}$$

where $SVF =$ stroke volume by the Fick method.
Figure 3

Stroke volume by the Fick method (SVF) plotted versus stroke volume calculated from the echographic measurements (SVE). The patients with valvular regurgitation show a larger stroke volume than was found by the Fick method. The amount of deviation from the Fick stroke volume was indicative of the severity of the regurgitation.

Results

Thirty-seven stroke volume determinations were performed in 30 patients who had no clinical or angiocardiographic evidence of valvular regurgitation. There was excellent correlation between the stroke volume calculated by the Fick method and that determined by the echographic methods SVE1 (equation 6) and SVE2 (equation 7) with a correlation coefficient r = 0.966 (figs. 3 and 4). The mean difference between the echographic method SVE1 and the Fick method was 0.5 ± 6.7 ml, with the echographic method giving slightly smaller values than Fick values, with a range of −16 to +22 ml (table 1). The mean difference between the echographic data SVE2 using equation 7 and the Fick method was 8.7 ± 5.8 ml, with the echographic method (SVE2) usually giving smaller values ranging from −23 ml to +3 ml (table 1). The echographic method, SVE1 (equation 6), gave values consistently greater than the echographic method, SVE2 (equation 7), with a range of +1 to +19 ml.

Since only the forward stroke volume is measured by the Fick method in the presence of valvular regurgitation, it is not possible to calculate the total volume of blood ejected during systole. Thus, it was not possible to evaluate the accuracy of echographic stroke volumes in patients with valvular regurgitation. On the other hand, the difference between the echographic stroke volumes calculated and those calculated by the Fick method would give an estimate of the regurgitant volume in these patients. It is necessary to assume the constants are the same and that equations 6 and 7 can be used in patients with valvular regurgitation.

To make these calculations as meaningful as possible, regression equations to express the relationship between the calculated echographic stroke volumes and the Fick stroke volumes were determined and used (figs. 3 and 4). These equations are:

\[
SVF = 0.864 \text{SVE}_1 + 8.7 \text{ ml} \quad (10)
\]

\[
SVF = 1.007 \text{SVE}_2 + 8.4 \text{ ml} \quad (11)
\]

The expected stroke volume was calculated from 24 studies in 21 patients in whom the presence and degree of valvular regurgitation were known. In each case, the stroke volume which would be expected from the echographic measurements was greater than the effective forward stroke volume measured by the Fick method (table 2). The difference between the echographic stroke volume and the Fick stroke volume correlated well with the degree of
Table 2

Summary of Results: Twenty-One Patients With Valvular Regurgitation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Study</th>
<th>Diagnosis</th>
<th>Dd (cm)</th>
<th>De (cm)</th>
<th>ΔL (cm)</th>
<th>SVE' 1 (ml)</th>
<th>SVE' 2 (ml)</th>
<th>SVF</th>
<th>SVE' 1 − SVF</th>
<th>SVE' 2 − SVF</th>
<th>SVE' 1 − SVF</th>
<th>SVE' 2 − SVF</th>
<th>Fick CO (L/min)</th>
<th>HR (beats/min)</th>
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<td>71</td>
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<td>105</td>
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<td>5.5</td>
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<td>75</td>
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<td>4.5</td>
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Abbreviations: SVE' = echographic stroke volume adjusted with the regression equation (see text for individual formulas for SVE_1 and SVE_2); mod. = moderate. Other abbreviations same as in table 1.
regurgitation judged by a rough quantitative method from angiographic studies (figs. 3 and 4).

**Discussion**

In these studies, left ventricular stroke volume was determined by using measurements of ventricular diameter and lengths obtained by echographic recordings utilizing ultrasound. The geometric shape which was assumed for the calculations of left ventricular volume was that of a prolate ellipse, as suggested by several angiocardiographic studies. The assumptions on which these calculations were made, however, emphasize three points which merit further discussion. First, the left ventricles of patients with and without cardiac disease do not strictly conform to an idealized geometrical shape, and show a variable relationship between the major and minor axes. There is evidence that the major-to-minor axis ratio is most reduced in markedly dilated hearts like those seen in patients with severe left ventricular failure or gross valvular regurgitation.

Secondly, while the relationship of the major and minor axes may change little throughout systole in patients, as previously discussed, with severe chamber dilation or ventricular failure, this relationship changes markedly during systole in patients with gross valvular regurgitation who have maintained good ventricular function. Thus, the geometrical figures used to calculate stroke volume in patients without regurgitation should be applied with some reservations to patients with large ventricles or severe regurgitation, or both.

The third point for discussion in the evaluation of the echographic stroke volume technic is that of obtaining a standard ventricular dimension from patient to patient. Using the procedure described in "Methods," several references are employed to insure that the sound beam takes a very similar path through the ventricle of each patient. By directing the sound beam just below the mitral valve, the line of measurement is kept near the midportion of the ventricle. The posterior wall echoes are recorded easily by directing the transducer over a wide arc in several planes with the point of contact with the skin remaining fixed in the fourth intercostal space at the left sternal border. The echoes from the interventricular septum are recorded through a small range of transducer.

**Figure 5**

Frames from cineangiocardiograms taken in the frontal and lateral projections are shown. In the frontal projection, a metal disk marks the position of the transducer during echographic study. A metal rod has been placed alongside the patient at the level of the fourth intercostal space and directed along the plane of the transducer during an echographic study. The path of the sound beam through the ventricle is visualized. LV = left ventricle; RV = right ventricle.
motion, however. This is probably due to a limited area of the septum being oriented perpendicular to the path of the sound beam, since this portion of the ventricle has a surface which is convex toward the transducer. This convex surface gives a smaller area for reflection of the sound back to a given point than does the rather concave surface of the posterior left ventricular wall. Therefore, the requirement to record echoes from both the interventricular septum and the posterior left ventricular wall at a level just inferior to the mitral valve seems to allow comparison of the dimensions obtained among patients. The path of the sound beam has been confirmed previously by angiograms performed with a radiopaque marker in the position of the transducer and oriented in an identical axis (fig. 5).

Although there are limitations of the echographic method for determining stroke volume in patients with significant degrees of valvular regurgitation and ventricular dilation, as discussed previously, comparison of the SVF and SVE allowed us to predict the severity of regurgitation noted on cineangiography. In this series few patients with moderate and severe disease were studied; however, the geometrical considerations discussed above suggest that the more severe degrees of regurgitation associated with marked ventricular dilation will lead to an overestimate of the SVE, and an underestimate of the regurgitant volume when the SVE is compared to the SVF. Thus, this should magnify the separation between those patients with relatively normal ventricular shape and mild regurgitation and those with more severe regurgitation. The echographic technic is not proposed as an alternate to angiographic evaluation of valvular regurgitation, as diagnosis of the specific valvular abnormality is out of the realm of echography. However, the echographic method does appear to lend itself to the quantitation of regurgitation. Also, the ease and speed of stroke volume determination and the lack of need for inserting specialized instrumentation into the patient allow the application of this technic to the repeated study of monitoring of patients in many physical positions and physiologic conditions.

For purposes of repeated calculation or monitoring of stroke volume, we point out an expedient method for use in the clinical setting. Calculation of SVE requires only one echogram (fig. 1) and therefore is preferable to SVE. The regression equation using SVE may be reduced to a direct formula using Dd and Ds:

\[
SVF = 0.864 \times SVE_1 + 8.7 \, \text{ml} = (0.864) \times \left[ 1.047 \times (Dd^3 - Ds^3) + 8.7 - 0.905 \times (Dd^3 - Ds^3) \right] + 8.7 \, \text{ml}
\]

Within the range of practical values, a variation of 10% is obtained by using Dd = Ds to approximate the predicted SVF.

Our experience using these echocardiographic technics permit three preliminary conclusions. First, accurate calculation of stroke volume over a wide range of values is possible by using ventricular measurements obtained by cardiac echography. Secondly, the total stroke volume of the left ventricle can be estimated and compared with the effective forward stroke volume in patients with valvular regurgitation to evaluate the degree of regurgitation. Thirdly, this safe, simple and painless technic warrants further investigation, in order to refine the procedure described. Specifically, a standard echographic method for measuring the absolute apex-to-base dimension of the left ventricle would be of value. If this major axis could be measured, the true major and minor axis measurements could be used directly for each patient and the echographic stroke volume calculation would be more accurate for patients with large dilated ventricular chambers. The potential of the echographic technic will be known only after further validation of the method and discovery of its limitations.

**Acknowledgment**

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