Use of Ultrasound to Measure Left Ventricular Stroke Volume

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SUMMARY
Several empirical observations made during ultrasound examinations for pericardial effusion have led to the possibility that this diagnostic technique might be used to measure left ventricular stroke volume in man. The hypothesis that left ventricular stroke volume was proportional to the amplitude of an echo originating from a portion of the left ventricle near the mitral ring (MREa) times the distance between the echoes from the anterior and posterior ventricular walls (LVD) has been validated.

Ultrasound examinations were performed simultaneously with cardiac output determinations using the direct Fick method on 16 patients proven to have competent mitral and aortic valves. Correlation between the two methods of measuring left ventricular stroke volume was excellent \( r = 0.973; P < 0.001 \). When ultrasound measurements were used in the regression equation to predict stroke volume, the calculated values were within 11 ml or 15\% of those determined by the Fick method.

The fact that an ultrasound examination is simple, can be done as a bedside determination in a matter of minutes, and is totally harmless to the patient make it a promising diagnostic procedure. The measurement of left ventricular stroke volume represents another addition to the growing list of medical uses for this intriguing technique.

Additional Indexing Words:
Cardiac output, Ultrasound myocardial function, Echography

In the course of earlier studies using diagnostic ultrasound to detect the presence of pericardial effusion,\(^1,2\) several observations were made which suggested the possibility of using this technique to measure left ventricular stroke volume. The first of these observations was the finding of a consistent and strong ultrasound signal or echo near the echo from the anterior leaflet of the mitral valve\(^2,3\) (fig. 1D). This new echo was initially of interest because it was confused with the true mitral valve echo.\(^3\) Secondly, this echo offered some difficulty when we were attempting to diagnose pericardial effusion.\(^2\) On correlating the location and pattern of this echo with those of echoes originating from the cage of Starr-Edwards mitral valves,\(^4\) this echo appeared to be originating from a portion of the left ventricle near the mitral ring or annulus and, thus for convenience, was designated as the mitral ring echo (MRE).\(^3\) A similar echo has been recognized by other investigators, and they too believed that it originated from an area near the mitral ring or atrioventricular wall.\(^5\) Upon further investigation of the MRE, it was noted that the curve which it inscribed was surprisingly similar to known left ventricular volume curves with the amplitude varying directly with left ventricular stroke volume and inversely with left ventricular volume.\(^2\) Edler\(^5\) likewise noted that the amplitude of the atrioventricular wall echo varied with different disease states. It thus seemed pos-

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there is some functional relationship of MREa to left ventricular stroke volume and left ventricular volume, this ventricular dimension possibly could be substituted for left ventricular volume, and thereby MREa could be used to calculate left ventricular stroke volume.

This study attempts to test the validity of these observations and assumptions by using the amplitude of the mitral ring echo and the distance between the anterior and posterior heart wall echoes to calculate left ventricular stroke volume in the intact human subject.

Method

Studies were done on 16 patients undergoing diagnostic heart catheterization. The patients included both males and females, and their ages ranged from 15 to 61 years. No patient had any evidence of mitral or aortic insufficiency. In 15 of the patients valvular insufficiency was excluded by left heart catheterization and selective cineangiography. The one patient who had only right heart catheterization had no heart murmurs and was being studied to evaluate a recent pulmonary embolus.

The principles of diagnostic ultrasound have been reviewed in previous papers.7–10 The ultrasound examinations were performed with a commercially available ultrasonoscope utilizing a 2.25-mc, 0.75-inch transducer with a repetition rate of 1,000 impulses per second. For recording the echoes, the “slow sweep” or “time-motion” presentation was used whereby distance is plotted against time and moving echoes are plotted as wavy lines. The patients were examined in the recumbent position and Aquasonic gel was used as a coupling medium to ensure airless contact between the transducer and the skin. To record the anterior and posterior heart wall echoes, the transducer was placed along the left sternal border at the fourth intercostal space and pointed directly posteriorly (fig. 4). Just as is done when examining for pericardial effusion,2 the gain, damping, and reject modalities, as well as the exact positioning of the transducer, were adjusted to record the single most dominant echo originating from the vicinity of the posterior wall of the heart. In addition, the gain controlling the near field was adjusted to record the nonmoving anterior chest wall echoes plus the fuzzy thin echo just immediately posterior to the chest wall echoes. In many patients, despite its somewhat indistinct character, this anterior heart wall echo could be seen to move in a direction opposite to the posterior heart wall (fig. 3).
Posterior heart wall echo (P) in a normal subject (A) and in a patient with a dilated left ventricle (B). T represents the echo originating from the transducer. (A) The posterior heart wall echo (P) is 10 cm from the transducer. (B) The echo P is nearly 14 cm from echo T. (From Feigenbaum, H., Zaky, A., and Waldhausen, J. A.: Ann Intern Med 65: 443, 1966, by permission of the American College of Physicians.)

The echo originating from the mitral ring (MRE) was obtained by placing the transducer over the apex of the heart and pointing the transducer posteriorly, medially, and a little superiorly (fig. 5). The exact direction was adjusted to record the strong echo which was just posterior to the mitral valve echo and which had a characteristic pattern of motion. The MRE moved toward the transducer with ventricular systole and away from the transducer with diastole. The diastolic motion was divided into three phases: rapid movement early in diastole, either less rapid or no motion in mid-diastole, and then rapid movement following atrial systole (fig. 5). A simultaneous electrocardiogram aided in identification of all echoes. By placing the transducer over the apex of the heart, the maximal amplitude of the MRE was recorded. This approach also helped to standardize the examination by allowing for differences in heart size from one patient to another.

The ultrasound recordings were obtained simultaneously with the determination of cardiac output as measured by the direct Fick method. A simultaneous electrocardiogram on a standard strip-chart recorder was used to measure the
heart rate. In the three patients with atrial fibrillation, an effort was made to utilize those ultrasound measurements which followed an R-R interval corresponding to the average heart rate during the determination of cardiac output.

**Results**

The ultrasound measurements which were used to determine left ventricular stroke volume (LVSV) were left ventricular diameter (LVD), which was the distance between the echoes from the anterior and posterior heart walls during diastole (fig. 4), and the mitral ring echo amplitude (MREa), which was the total amplitude of motion inscribed by the mitral ring echo from the beginning of systole to the beginning of diastole (fig. 5).

The formula used to calculate LVSV was derived primarily from empirical findings. As stated before, MREa seemed to vary with stroke volume for a given left ventricular end-diastolic volume (LVV). If LVV were increased and LVSV were not changed, then MREa decreased. Thus, MREa appeared to vary directly with LVSV and inversely with LVV, that is, \[ \text{MREa} = \frac{\text{LVSV}}{\text{LVV}} \] or \[ \text{LVSV} = \text{MREa} \times \text{LVV} \]. By using LVD as a function of LVV, the relatively simple formula \[ \text{LVSV} = \text{MREa} \times \text{LVD} \] was employed. When MRE \times \text{LVD} was plotted against the stroke volume, as determined by the Fick method, there was a straight line relationship between the two values (fig. 6). The correlation coefficient was \( r = 0.973 \) (\( P < 0.001 \)) with a regression equation \[ \hat{y} = 87.5 + 10.40 \times (x - 8.9) \]. Table 1 shows the results of using this regression equation to predict stroke volume from the ultrasound measurements (\( \text{SV}_a \)). The greatest discrepancy between the stroke volumes
Figure 5

Diagram and ultrasound recording demonstrating how the mitral ring echo (MRE) and its amplitude (MREa) is obtained.

Figure 6

Stroke volume as determined by the Fick method plotted against the product of the ultrasound measurements, LVD × MREa.

Discussion

Many attempts have been made to measure left ventricular stroke volume by utilizing various diagnostic procedures. Probably the most widely accepted method is measuring the cardiac output by either the Fick or the indicator-dilution technique and then dividing the cardiac output by the heart rate. This method has many obvious disadvantages. First, right heart catheterization is necessary. Secondly, this technique cannot be used to measure true stroke volume in the presence of mitral insufficiency or aortic insufficiency. Thirdly, this technique does not allow for beat to beat measurements of stroke volume.

Direct measurements of left ventricular volume by either the angiographic or indicator-dilution techniques are more recent procedures used to measure left ventricular stroke volume. These techniques again require cardiac catheterization. The angiographic procedure possesses several inherent objections. It introduces a foreign substance which may alter the hemodynamic situation; the technique offers some risk to the patient;
### Table 1

**Ultrasound Measurements Compared with Cardiac Catheterization Data**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
<th>Ultrasound measurements</th>
<th>Catheterization data</th>
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<td></td>
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<td>LVD(cm)</td>
<td>MREa(cm)</td>
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<tr>
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<td>1.2</td>
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</table>

**Mean**

|            |       |       |       | ±1.0   | ±0.3   | ±2.5   | ±2.1   | ±14 | ±26   | ±26   | ± 2   |

**Abbreviations:** LVD = left ventricular diameter; MREa = mitral ring echo amplitude; CO = cardiac output; HR = heart rate; SV = stroke volume; SV(a) = 87.5 + 10.40 (LVD × MREa) - 8.9; (AF) = atrial fibrillation.
it does not lend itself to many repeated examinations, and the actual calculations are tedious and time consuming. In addition, both the angiographic and indicator-dilution techniques are still being questioned as to their accuracy in truly measuring left ventricular volume.

The results of the present study indicate that diagnostic ultrasound with its many inherent advantages may provide another way of measuring left ventricular stroke volume. As stated in many previous publications, ultrasound in the power ranges used for diagnostic purposes is entirely innocuous to the patient. The technique used in this study is simple and the equipment is portable. Thus, the examination can be performed as a bedside procedure in a short time. The ease of the examination is borne out by the fact that for each patient in this study the ultrasound recordings were obtained within the 4 minutes it took to measure the cardiac output by the Fick method. The close correlation between the calculated left ventricular stroke volume and the expected left ventricular stroke volume as measured by the Fick method indicates that the accuracy of this ultrasound technique is probably as good as, if not better than, any of the other currently available methods.

The advantages of a simple, accurate method for measuring left ventricular stroke volume are numerous. First of all, when such a technique is combined with a Fick cardiac output, theoretically a quantitative measure of mitral or aortic insufficiency can be obtained. In patients without mitral or aortic valvular incompetence, this technique provides a bedside method for measuring cardiac output. In addition, since the formula \( LVSV = MREa \times LVD \) seems to be valid, the fact that the curve inscribed by the MRE is similar to left ventricular volume curves is probably more than coincidental. Thus, the recording of this echo may lend itself to hemodynamic studies involving changes in left ventricular volume. The validation of this formula also indicates that the ultrasound measurement of left ventricular diameter (LVD) probably does represent some function of left ventricular volume and might prove to be of value as a qualitative or semiquantitative measure of left ventricular volume.

It is somewhat difficult to explain entirely why these two relatively simple ultrasound measurements should be directly related to left ventricular stroke volume. The logical tendency is to try to fit the measurements into some mathematical formula used to measure left ventricular volume. Obviously there are too few data to justify such an attempt. In fact, one cannot entirely justify placing cm\(^2\) or ml after the ultrasound calculations. Left ventricular diameter is actually measured in cm, as is MREa, thus the answer should really be in cm.\(^2\) The only justification one might have to use ml is that LVD supposedly represents a function of left ventricular volume in ml, and MREa is a function of how much left ventricular volume changes with ventricular systole. In any case, for the time being the findings must be considered entirely empirical. It is possible that future attempts to explain these findings may yield valuable information concerning the shape of the left ventricle and the manner in which this chamber ejects blood.

References

Elementary Approaches to Thrombosis

Various medical scientists look at thrombosis from different points of view. Only one, the clinician, studies the whole patient in whom a symptomatic clot resides. . . .

The pathologist notes that when a vessel is injured, a clumping of platelets quickly develops at the site of injury. Under the electron microscope, he can see that the clumped platelets tend to lose their "dense granules," perhaps releasing their clot-promoting contents.

The biochemist is interested in the chemical attraction between platelets and injured intima and the subjacent collagen; he tends to implicate the chemical, adenosine diphosphate. The biochemist has made significant advances in the chemistry of prothrombin and its active enzyme, thrombin, as well as in the biochemistry of the fibrinogen-fibrin reaction.

The physiologist is interested in clots that form in living vessels. He notes that undamaged vascular intima is electrically negatively charged, repelling platelets that are similarly charged. When a vessel is damaged, its electronegativity disappears or may be replaced by an electropositivity, which tends to attract platelets to the injured site.

The biophysicist is concerned with two rheologic principles as they may relate to vascular thrombi. First, fluid moving through a tube generates a negative "streaming" potential at the surface of the tube. This exaggerates the normal negative charge of the intima and is dependent upon the flow rate of the blood. Further, the flow rate in very small tubes, such as capillaries, varies with the fourth power of the caliber of the tube. If the diameter of a tube is reduced by one half, the flow rate diminishes to one sixteenth.—Charles A. Owen, Jr.: Hypercoagulability and Thrombosis. In Proc Mayo Clin 40: 830, 1965.
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