Echocardiographic Measurement of Right Ventricular Wall Thickness
A New Application of Subxiphoid Echocardiography

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SUMMARY The feasibility of subxiphoid echocardiography to measure the thickness of the right ventricular wall (RVWT) was investigated. In 87 (90.6%) of the 96 patients studied, adequate visualization of the echoes from the right ventricular wall was obtained using the subxiphoid technique. RVWT averaged 0.34 ± 0.08 cm (mean ± 1 SD), ranging from 0.2 to 0.5 cm in 25 normal individuals. This was not significantly different from the values in the left ventricular overload group (0.36 ± 0.10 cm). However, the RVWT was increased significantly (P < 0.001) in the combined group (0.62 ± 0.18 cm), the right ventricular (RV) pressure overload group (0.60 ± 0.13 cm) and the RV volume overload group (0.53 ± 0.11 cm).

Thirty-two patients underwent diagnostic right heart catheterization which revealed a good correlation between the RVWT measured echocardiographically and the right ventricular peak systolic pressure (r = 0.84).

Subxiphoid echocardiography was considered to be useful in diagnosing right ventricular hypertrophy in adults.

THE RECENT DEVELOPMENT AND IMPROVEMENT of echocardiographic instruments has facilitated noninvasive assessment of cardiac function and evaluation of anatomical abnormalities of the heart. One of the most important achievements has been echocardiographic assessment of the dimensions of cardiac structures.1, 2 However, evaluation of ventricular wall thickness has been limited to the left ventricle. Assessment of the right ventricular wall remains outside the scope of echocardiographic study, except in infants and young patients who have a thin chest wall,3, 4 because of interference by the gaseous tissue of the lung and because of problems due to the physical properties of ultrasound. Only the right ventricular dimension described by Popp et al.4 was found to be a clue to the estimation of the size of the right ventricle, the value of which increases in patients with atrial septal defect or tricuspid insufficiency.

Chang et al.5 reported that subxiphoid echocardiography was useful in emphysematous or aged patients for recordings of the mitral valve and the left ventricular posterior wall. The present study was undertaken to examine the feasibility of echocardiographic measurement of right ventricular wall thickness by the subxiphoid method.

Patients and Methods

Ninety-six patients were examined; nine were excluded due to unsatisfactory echocardiographic visualization of the right ventricular wall. The clinical data of the remaining 87 patients are summarized in table 1. The patients were divided into four groups: normal, left ventricular overload, combined, and right ventricular overload. The normal group included 25 patients with no evidence of cardiopulmonary disorders. There were 15 males and 10 females ranging in age from 17 to 58 years (mean 32.9 years). The left ventricular (LV) overload group included 15 males and seven females aged 20–77 years (mean 56.4 years). The combined group...
The right ventricular (RV) overload group was subdivided into two groups: RV volume overload (12 patients) and RV pressure overload (10 patients). One patient with pericardial effusion was also examined to confirm echoes from the endocardium and epicardium of the right ventricular wall.

All echocardiograms were done with an Aloka SSD-90 using a 2.25 MHz, 10 mm diameter transducer. They were recorded on a strip chart recorder at 50 or 100 mm/sec with simultaneous recordings of the electrocardiogram and phonocardiogram.

For estimation of the right ventricular dimension (RVD), and the thickness of the left ventricular posterior wall (LVPWT) and interventricular septum (IVST), the transducer was placed at the left fourth intercostal space and angled inferiorly and laterally until a satisfactory echo-cardiogram could be recorded according to the standard method of Popp et al.8

Echocardiographic visualization of the right ventricular wall was accomplished by placing the transducer in the subxiphoid region and scanning from the base to the apex of the heart (fig. 1). The interventricular septum was easily visualized but the left ventricular wall and the mitral valve could be detected less frequently. The direction of the transducer was then carefully changed to obtain echoes from the right ventricular wall.

The RVD was measured between the right ventricular epicardium or, if not feasible, a depth of 0.5 cm from the chest wall and the right side of the interventricular septum. The LVPWT was measured between the endocardium and the epicardium of the left ventricular posterior wall. The IVST was measured between the right and left side of the interventricular septum. The RVWT was measured between the epicardium and the endocardium of the right ventricular wall. All measurements were made at the peak of the R wave of the electrocardiograms.

Results

Of the 96 patients examined, adequate visualization of the right ventricular wall was possible in 87 (90.6%) patients. Of the nine patients in whom visualization was unsatisfactory, three had mitral stenosis and six were normal. All of them were young and cardiac enlargement was mild or absent. Echocardiograms obtained by the standard method were available for measuring the RVD, the LVPWT, and the IVST in all patients but one who had atrial septal defect. Echocardiographic measurements of this patient were made with the subxiphoid technique alone.

An M-mode scan with the transducer placed in the subxiphoid region and scanned from the base to the apex of the heart is useful to the examiner in interpreting subxiphoid echocardiograms and in determining the correct direction of the transducer for measurement of the RVWT. As seen in figure 2, the left two-thirds of the tracing where the tricuspid valve leaflets or the chordae tendineae attached to them can be visualized, are suitable for measurement of the RVWT. The echo beam passes through the right ventricular wall obliquely and the papillary muscles (arrow) can be visualized in the right one-third.

Figure 3 is a subxiphoid echocardiogram obtained from a

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**Table 1. Clinical Data**

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Age (mean ± 1 sd (range))</th>
<th>Sex (male:female)</th>
<th>BSA (m²) (mean ± 1 sd (range))</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>25</td>
<td>32.9 ± 11.5 (17-58)</td>
<td>15:10</td>
<td>1.55 ± 0.15 (1.27-1.81)</td>
<td></td>
</tr>
<tr>
<td>LV overload</td>
<td>22</td>
<td>56.4 ± 16.5 (20-77)</td>
<td>15:7</td>
<td>1.57 ± 0.18 (1.26-1.81)</td>
<td>Hypertension: 18, AS: 1, Aortic: 1</td>
</tr>
<tr>
<td>Combined</td>
<td>18</td>
<td>45.9 ± 15.7 (24-70)</td>
<td>4:14</td>
<td>1.44 ± 0.14 (1.09-1.61)</td>
<td>MS+AI: 9, MS+ASI: 1, MSI: 2, VSD: 3, COLD: 3</td>
</tr>
<tr>
<td>RV overload</td>
<td>22</td>
<td>40.5 ± 12.6 (10-70)</td>
<td>7:15</td>
<td>1.49 ± 0.22 (1.25-2.09)</td>
<td>ASD: 10, TI: 2, MS: 9, VSD+PS: 1</td>
</tr>
</tbody>
</table>

Abbreviations: LV = left ventricular; RV = right ventricular; AS = aortic stenosis; AI = aortic insufficiency; MS = mitral stenosis; MSI = mixed mitral stenosis and insufficiency; VSD = ventricular septal defect; COLD = chronic obstructive lung disease; ASD = atrial septal defect; TI = tricuspid insufficiency; PS = pulmonary stenosis; SD = standard deviation.
patient in the normal group (the right ventricular peak systolic pressure was 25 mm Hg). The right ventricular wall is seen below the thick multiple echoes originating from the abdominal wall and moves slowly toward the transducer during ventricular diastole and away from the transducer during systole. The left ventricular wall moves toward the transducer during ventricular systole. The interventricular septum moves toward the left ventricular wall and reverses the direction of its motion during diastole as seen in an echocardiogram using the standard method. The RVWT of this patient was 0.4 cm.

Figure 4 shows a subxiphoid echocardiogram from a patient with ASD. The motion of the interventricular septum is in a direction opposite to that of the right ventricular wall and parallels the motion of the left ventricular wall (paradoxical septal motion). The RVWT of this patient was 0.4 cm.

A subxiphoid echocardiogram obtained from a patient with PS associated with VSD with a right ventricular peak systolic pressure of 74 mm Hg is shown in figure 5. The right ventricular wall had thickened to an estimated 0.9 cm.

Figure 6 shows a standard M-mode scan of a patient with pericardial effusion of long duration. An echo-free space is clearly shown behind the left ventricular posterior wall. The right ventricular wall could not be visualized clearly in this

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**Figure 2.** An M-mode scan with the subxiphoid technique. Scan was performed by directing the transducer superiorly and to the left laterally. Measurement of the thickness of the right ventricular wall should be made where the tricuspid valve (TV) or the chordae tendineae are visualized (left two-thirds of the tracing). If measured on the right one-third of the tracing, the presence of the echoes from the papillary muscle (arrow) and the oblique passage of the echo beam cause an erroneous measurement. IVS = interventricular septum; MV = mitral valve; LVW = left ventricular wall; RVW = right ventricular wall.

**Figure 3.** A subxiphoid echocardiogram obtained from a healthy subject. The right ventricular wall moves toward the transducer gradually during ventricular diastole until the P wave of the electrocardiogram. There is a more abrupt motion toward the transducer during the P-R interval. Shortly after the R wave, the right ventricular wall begins to move away from the transducer or toward the left ventricular wall which moves in a direction opposite to the right ventricular wall. After the maximum movement away from the transducer before the second heart sound of the phonocardiogram, the right ventricular wall moves in an opposite direction toward the transducer. The motion of the interventricular septum shows a similar pattern to that of the right ventricular wall although it is more complex. The right ventricular peak systolic pressure was 25 mm Hg and the RVWT was measured as 0.4 cm. ECG = electrocardiogram; PCG = phonocardiogram; Epi = epicardium; End = endocardium; RVWT  = thickness of the right ventricular wall.
tracing but became clearer when the transducer was angled more accurately, as is experienced often in pericardial effusion (fig. 7A). Figure 7B is a subxiphoid echocardiogram from the same patient, which clearly revealed the right ventricular wall. In both panels, the echo from the epicardium of the right ventricular wall was denser than that from the endocardium and the effusion fluid separated the epicardium from the pericardium throughout the whole cardiac phase. The epicardium was visualized as a smooth echo in figure 7A although it appeared as rough, multiple echoes in some places in figure 7B. This is possibly due to the attachment of inflammatory products to the epicardial surface of the inferior portion of the right ventricular wall. The RVWT was measured as 0.6 cm in figure 7A and figure 7B.

The results of the echocardiographic measurements are shown in table 2. In the normal group, the mean RVWT was 0.34 ± 0.08 cm (mean ± 1SD) and was not significantly different from that in the LV overload group (0.36 ± 0.10 cm). The combined group, the RV pressure overload group and the RV volume overload group had increased RVWT values. They were 0.62 ± 0.18 cm, 0.60 ± 0.13 cm, and 0.53 ± 0.11 cm, respectively. For the RVD index (calculated from the RVD divided by the body surface area), the mean value in the normal group was 0.96 ± 0.27 cm/m². Only the RV volume overload group showed an increased mean value (2.49 ± 0.67 cm/m²). The remaining groups had normal or slightly increased values. The LVPWT was 0.78 ± 0.10 cm in the normal group and was increased significantly only in the LV overload group (1.16 ± 0.30 cm). The IVST was 0.82 ± 0.12 cm in the normal group and was also increased significantly in the LV overload group (1.18 ± 0.32 cm). The ratios of RVWT/LVPWT were calculated. The ratio was 0.44 ± 0.12 for the normal group and 0.33 ± 0.10 for the LV overload group. The ratios for the other three groups were increased but there were no significant differences among them.

Of the patients studied, 32 underwent diagnostic right heart catheterization within one month before or after the echocardiographic examinations. The RVWT was significantly correlated with the right ventricular peak systolic pressure ($r = 0.84$) as seen in figure 8.

**Discussion**

Echocardiographic measurements of the size of cardiac structures such as the left atrial dimension,² the left ventricular internal dimension,³,⁴,⁵ and the thickness of the left ventricular posterior wall⁶ have been reported by many investigators, and their accuracy has been confirmed by angiography² or at surgery.¹ However, echocardiographic estimation of the thickness of the right ventricular wall has not
been documented in adults. With the standard technique, that is, with the transducer positioned in the left fourth intercostal space, successful echocardiographic recording of the right ventricular wall has been disappointing in most adult patients, especially those with pulmonary emphysema. There are several reasons for this. The most important one is that with age the lung overinflates into the space between the chest wall and the heart, thus interfering with the penetration of the echo beam. Another reason is that multiple echoes from the chest wall are usually denser than the echo from the epicardium; thus, distinguishing between them is often difficult even with careful adjustment of near gain. Even if these difficulties are overcome, the echoes from the right ventricular wall often interfere with the chest wall echoes and measurement of the thickness of the right ventricular wall results in a lower value.

Chang et al. found that in emphysematous or aged patients detection of the heart valves and visualization of the left ventricular wall became easier when the transducer was in the subxiphoid region and there was little or no interference by the lung. They also noted comparable measurements of left ventricular dimensions, left ventricular wall thickness, and the interventricular septum with the standard and the subxiphoid techniques. They made no reference to measuring the right ventricular wall with this new method.

The present study indicates that the thickness of the right ventricular wall can be measured with this method in 87 of 96 patients examined. The nine patients in whom good echocardiograms could not be obtained with the subxiphoid technique were all young and had thin thoraxes with no cardiac enlargement or had a tight abdominal wall as experienced by Chang et al. Identification of the epicardium and endocardium of the right ventricular wall was easier with the subxiphoid technique than with the standard technique. If good visualization of echoes from the right ventricular wall using the standard technique can be made, systolic separation of the epicardium from the pericardium or the chest wall should be noticed frequently without any evidence of pericardial effusion. This finding also was observed frequently with the subxiphoid technique, and both systolic and diastolic separations were found in a patient with pericardial effusion. This further supports the theory that an echo below the mul-

![Figure 7. Echocardiograms obtained from the same patient as in figure 6. Panel A shows the right ventricular wall with the standard method and panel B shows it with the subxiphoid method. The right ventricular wall can be detected easily with both methods because effusion fluid separates the epicardium from the pericardium during the whole cardiac phase. The thickness of the right ventricular wall was measured as 0.6 cm equally with both methods. Epi = Epicardium; End = Endocardium.](http://circ.ahajournals.org/)

### Table 2. Echocardiographic Data

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>RVDI (cm/m²)</th>
<th>RVWT (cm)</th>
<th>LVPWT (cm)</th>
<th>IVST (cm)</th>
<th>RVWT/LVPWT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>25</td>
<td>0.96 ± 0.27†</td>
<td>0.34 ± 0.08</td>
<td>0.78 ± 0.10</td>
<td>0.82 ± 0.12</td>
<td>0.44 ± 0.12</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.60-1.71)†</td>
<td>(0.2-0.5)</td>
<td>(0.6-1.0)</td>
<td>(0.6-1.1)</td>
<td>(0.22-0.67)</td>
</tr>
<tr>
<td>LV overload</td>
<td>22</td>
<td>1.04 ± 0.25</td>
<td>0.36 ± 0.10</td>
<td>1.16 ± 0.30</td>
<td>1.18 ± 0.32</td>
<td>0.33 ± 0.10</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.66-1.51)</td>
<td>(0.2-0.5)</td>
<td>(0.8-2.0)</td>
<td>(0.8-1.9)</td>
<td>(0.13-0.50)</td>
</tr>
<tr>
<td>Combined</td>
<td>18</td>
<td>1.10 ± 0.31</td>
<td>0.62 ± 0.18*</td>
<td>0.91 ± 0.21</td>
<td>0.91 ± 0.21</td>
<td>0.70 ± 0.23*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.65-1.84)</td>
<td>(0.4-1.2)</td>
<td>(0.7-1.3)</td>
<td>(0.7-1.3)</td>
<td>(0.40-1.33)</td>
</tr>
<tr>
<td>RV volume overload</td>
<td>12</td>
<td>2.49 ± 0.67*</td>
<td>0.53 ± 0.11*</td>
<td>0.84 ± 0.22</td>
<td>0.85 ± 0.19</td>
<td>0.71 ± 0.30*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.54-3.96)</td>
<td>(0.4-0.7)</td>
<td>(0.5-1.6)</td>
<td>(0.6-1.4)</td>
<td>(0.25-1.40)</td>
</tr>
<tr>
<td>RV pressure</td>
<td>10</td>
<td>1.02 ± 0.28</td>
<td>0.60 ± 0.13*</td>
<td>0.84 ± 0.22</td>
<td>0.85 ± 0.19</td>
<td>0.74 ± 0.18*</td>
</tr>
<tr>
<td>overload</td>
<td></td>
<td>(0.67-1.46)</td>
<td>(0.4-0.9)</td>
<td>(0.5-1.6)</td>
<td>(0.6-1.4)</td>
<td>(0.45-1.13)</td>
</tr>
</tbody>
</table>

*Significantly different from the normal group (P < 0.001).
†Mean ± 1 se.
‡Range.

Abbreviations: RVDI = right ventricular dimension index; RVWT = thickness of the right ventricular wall; LVPWT = thickness of the left ventricular posterior wall; IVST = thickness of the interventricular septum.
tiple echoes from the abdominal wall originates from the epicardium. Moreover, echoes originating from the abdominal wall are not so dense as those from the right ventricular wall and the depth at which echoes from the right ventricular wall could be visualized using the subxiphoid technique ranged from 2 to 8 cm from the transducer, but is less than 2 cm in depth with the standard technique. In the present study the mean value was 5 cm. Therefore, gain control could be done more easily to obtain an echo from the epicardium of the right ventricular wall. Identification of the endocardium is also difficult with the standard technique because of a narrow window through which the right ventricular wall can be detected and because of a weaker echo than that originating from the epicardium causing difficulties in the adjustment of gain or output. By the subxiphoid technique, the window is wide enough that an M-mode scan may be performed if necessary to obtain satisfactory echo from the endocardium. Moreover, gain adjustment is easier at a greater depth and the epicardium can be recorded without interference from the echoes from the chest wall or the abdominal surface.

The Thickness of the Right Ventricular Wall

In the present study the average normal value of the right ventricular wall thickness was 0.34 cm. This was not so different from the values in neonates using the standard technique. The right ventricular wall was shown to increase in thickness in patients with right ventricular pressure overload without significant increases in the right ventricular dimension index. However, patients with right ventricular volume overload had increased mean values of both right ventricular wall thickness and right ventricular dimension index. This is considered to be due partly to secondary pulmonary hypertension, as shown in the good correlation between right ventricular wall thickness and right ventricular peak systolic pressure. Diamond et al. observed an increased right ventricular dimension index in patients with right ventricular volume overload, such as atrial septal defect or tricuspid insufficiency. But investigations concerning the thickness of the right ventricular wall have not been reported. In order to make a diagnosis of right ventricular hypertrophy, it is necessary to know the right ventricular wall thickness as well as the size of the right ventricular cavity. The present study indicates that subxiphoid echocardiography provides adequate visualization of echoes from the right ventricular wall facilitating the measurement of its thickness.

Other Echocardiographic Measurements

The thickness of the left ventricular posterior wall and that of the interventricular septum were increased only in patients with left ventricular overload. But the thickness of the left ventricular posterior wall exceeded 1.2 cm in two patients with right ventricular overload; one with atrial septal defect and the other with mitral stenosis. Neither showed any evidence suggesting associated abnormalities causing left ventricular hypertrophy. The patient with atrial septal defect was 70 years old and the echocardiographic examination could be made only with the subxiphoid technique. The other was also an aged patient and had frequent episodes of chronic bronchitis. Therefore, chronic hypoxemia due to respiratory disorders may be a cause of the increased thickness of the left ventricular wall secondary to left ventricular dysfunction although the possibility of mistaking papillary muscle echoes for the endocardium cannot be eliminated completely.

Critiques of the Subxiphoid Method

Two problems should be considered in applying this method to evaluate right ventricular hypertrophy. In the M-mode scan the right ventricular wall appears to increase in thickness from the base to the apex because of visualization of the papillary muscles and the oblique passage of the echo beam. An erroneous measurement can result if the thickness of the right ventricular wall is measured near the apex. Thus a criterion to determine the proper site for this measurement is necessary. In the present study, the direction of the transducer was set so as to visualize echoes from the tricuspid valve or the chordae tendineae attached to it in the same manner as the thickness of the left ventricular posterior wall is measured, because at such a location the right ventricular wall is uniform in its thickness and the endocardium is easily distinguished from the chordae tendineae.

The other problem is that when the transducer is placed in the subxiphoid region, the echo beam transects the portion of the wall of the right ventricular inflow tract. In the course of right ventricular hypertrophy, the wall of the outflow tract or the parts around the crista supraventricularis hypertrophies first, especially in patients with right ventricular pressure overload; these hypertrophic changes subsequently extend to
The Effect of Propranolol on Canine Myocardial CPK Distribution Space and Rate of Disappearance

JOHN A. CAIRNS, M.D., F.R.C.P.(C), AND GERALD A. KLASSEN, M.D., F R.C.P., (C)

SUMMARY  Canine myocardial CPK was partially purified and injected into 11 conscious mongrel dogs. From serial serum CPK measurements in each dog, mean Kd was calculated as $0.0047 \pm 0.0009 \text{ (\pm SD)} \text{ min}^{-1}$. Correlation coefficients indicated that CPK disappearance rate was well described by a single exponential expression. Kd measured on consecutive days in four dogs varied minimally. CPK distribution space ranged from 74 to 134% of plasma volume. Propranolol loading with 0.3 mg/kg or 2 mg/kg, followed by hourly maintenance doses, resulted in increased Kd in eight of ten dogs, mean Kd rising from 0.0048 min$^{-1}$ to 0.0059 min$^{-1}$ ($P < 0.02$). Propranolol appeared to increase plasma volume but had no significant effect on the relationship of CPK distribution space to plasma volume. If the serial CPK technique were used to measure infarct size, an average Kd, propranolol might produce artifactual reduction of infarct size measurement by increasing Kd and possibly by increasing plasma volume. The obligation to assess the effect upon CPK Kd and distribution space of an agent designed to limit infarct size is apparent.

SOBEL AND SHELL have described a technique for estimating the depletion of myocardial creatine phosphokinase (CPK) occurring in the course of acute myocardial infarction. The technique is based on the kinetics of the CPK released by the damaged tissue. The estimate of myocardial CPK depletion is dependent on the use of an appropriate parameter characterizing CPK disappearance from the vascular compartment and verification that a valid approximation of the parameter can be obtained in order to calculate aggregate release of CPK from serial serum CPK values. The CPK disappearance from the vascular compart-

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