SUMMARY  We have identified pericardial effusion by echocardiography in 174 patients. Seventeen had cardiac tamponade which was not always clinically obvious. Right ventricular narrowing or compression, occurring in the minor axis at end diastole and end expiration, to $7 \pm 2$ mm or less, was strongly associated with tamponade in patients with effusion. Right ventricular compression and signs of tamponade abated with pericardiocentesis. One patient was in tamponade without obvious right ventricular narrowing. Nonetheless, he demonstrated serial increases in right ventricular dimensions which paralleled hemodynamic improvement. Diminished left ventricular end-diastolic dimension, "swinging heart," electrical alternans, reciprocal respiratory variations in right and left ventricular end-diastolic dimensions and variation in amplitude of the mitral D-E slope were nonspecific for tamponade. Evaluation of right ventricular dimensions may be clinically useful to diagnose and monitor cardiac tamponade.

Echocardiography is the primary noninvasive tool to detect the presence and estimate the size of pericardial effusion. A recent report of three cases suggests that echocardiography may be helpful in identifying hemodynamically significant pericardial effusion. It became clear early in our experience with pericardial effusion that the patient with tamponade differed echocardiographically from the larger population. We believed that subsequent prospective analysis of these echocardiographic differences would allow us to identify patients in tamponade, a condition that was not always obvious because its symptoms and signs were frequently overshadowed by those of the patient's primary condition. We investigated the echocardiographic characteristics of the tamponade patient to test their specificity and to understand better the pathophysiology of the condition.

Methods

Three thousand consecutive echocardiographic studies were examined at the time of study for the presence or absence of pericardial effusion. Effusion was identified by the presence of an echo-free space between the epicardium and pericardium posteriorly, either alone or accompanied by a similar space anteriorly. Where possible, recordings were made with the patient both in the recumbent and left oblique positions.

Transducer location was usually in the fourth intercostal space near the left sternal border. The transducer was pointed superiorly and medially and swept inferiorly and laterally to provide images of all intracardiac chambers and valves. End-diastolic and end-systolic dimensions of the ventricles were measured in the minor axis, defined as the level of the superior margin of the mitral valve chordae tendineae. This level was slightly more basal than that suggested by Hagan and associates as being most accurate for evaluating septal motion. Right ventricular dimensions were also measured in the outflow tract at the level of the aortic valve leaflets. The right ventricular posterior wall was considered to be the anterior surface of the interventricular septum. Maximal right ventricular dimensions were obtained at both end inspiration and end expiration at end diastole with the patients in the left oblique position and with the transducer in a medical location. Left ventricular dimensions and the motion of the mitral valve (D-E excursion and E-F slopes) were routinely recorded.

The quantitation of right ventricular dimensional changes, limited by the resolution of the echocardiographic technique, at times was made more difficult by the prevailing clinical conditions. The studies were always performed in

From the Cardiovascular Division of the Department of Medicine and the Cardiovascular Research Institute, School of Medicine, University of California, San Francisco, California.

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Address for reprints: Nelson B. Schiller, M.D., Co-Director, Noninvasive Laboratories, Room 1186 Moffitt Hospital, San Francisco, California 94143.

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Right Ventricular Compression as a Sign of Cardiac Tamponade

An Analysis of Echocardiographic Ventricular Dimensions and Their Clinical Implications

NELSON B. SCHILLER, M.D., AND ELIAS H. BOTVINICK, M.D.
the presence of pericardial effusion, occasionally in the presence of intrapericardial air, and frequently in severely distressed patients. No patients were excluded for technical inadequacies. Optimal images of both ventricles were required to evaluate the axis of study and the ventricular dimensions were always measured from images whose morphology documented the transducer angulation. Although we attempted to localize the endocardial boundaries of the right ventricular cavity, the trabeculations of that structure occasionally brought an element of uncertainty by obscuring the distinction between endocardium and cavity. We attempted to minimize the influence of myocardial trabeculations by better definition of endocardial boundaries obtained with a transducer of higher frequency (3.5 MHz) or shorter focal length. Measurements were made within the resolution of the echocardiographic technique, assuming no better than a 2 mm resolution or a 4 mm range in the measured value.

Cardiac tamponade was defined as significant arterial hypotension (systolic pressure less than 100 mm Hg) resulting from pericardial effusion and relieved by pericardial drainage. All patients with documented pericardial effusion were studied for the presence of physical signs or symptoms of cardiac tamponade. These included dyspnea, tachycardia, neck vein distention, elevated venous pressure, narrow arterial pulse pressure and pulsus paradoxicus. Electrical alternans was sought. The clinical course of all patients with documented effusion was followed. All those with tamponade underwent pericardial drainage and special note was taken of the clinical and hemodynamic effects of the procedure. Right heart catheterization was performed during drainage in three patients in an attempt to monitor the hemodynamic status. The echocardiogram was repeated within one hour after drainage in nine patients. In five of these patients the echocardiogram was obtained continuously during pericardiocentesis.

M-mode echocardiograms were obtained with either a Picker EV-10 or a Smith Kline Ekoline 20A instrument and were recorded either directly on Polaroid film or by a Honeywell or Irex stripchart recorder. The transducers were 2.25 or 3.5 MHz and were nominally focused at either 5 or 7.5 cm. Transducers of higher frequency and shorter focal length were used whenever possible to facilitate the recording of detailed right ventricular anatomy.

In order to document further ventricular dimensional changes in tamponade, we serially studied one patient by two dimensional echocardiography. The instrument employed was a prototype of a Varian Associates 32-element phased array, wide angle (80°) sector scanner. Two-dimensional images were obtained in the long axis by directing the echo sweep between the apex and base of the heart. End-diastolic images were recorded at end expiration on Polaroid film by gating the camera shutter to the electrocardiographic R wave peak.

Results

Of those patients studied, 174 had echocardiographically identifiable pericardial effusion. Of these, 17 patients were in tamponade. In addition to arterial hypotension most of the 17 also had respiratory difficulty, tachycardia, diminished arterial pulse pressure and elevated venous pressure. Tamponade was associated with pulsus paradoxicus greater than 10 mm Hg in 15 of these 17 patients. Twelve of the patients with tamponade had malignant disease, one had collagen disease, another had right ventricular failure, two cases occurred postoperatively and one was without demonstrable cause.

Tamponade patients demonstrated ventricular dimensional changes which distinguished them from others studied. Our results are shown in table 1. Sixteen of the 17 patients with clinical evidence of tamponade had abnormally small right ventricles, as measured in the minor axis at end diastole at end expiration. Right ventricular end-diastolic dimension at end-expiration in 25 normal subjects was 24 ± 5 mm in the left oblique position and varied little with respiration. The mean right ventricular end-expiratory end-diastolic dimension in 17 patients with tamponade was 4 ± 2 mm, and less than 7 ± 2 mm in 16 of these patients. This dimension was significantly different from both normal subjects (P < 0.001) and those with effusion but without tamponade (P < 0.001). Right ventricular end-inspiratory dimensions in tamponade were larger, often falling into the normal range. Patients with effusion without tamponade did not have a significantly diminished right ventricular minor axis end-diastolic dimension when compared to normals. Right ventricular end-diastolic dimensions could be artificially reduced in those patients with large effusions and accentuated in those with tamponade by sweeping near the apex, by moving the transducer laterally from the sternal edge, or by using a low intercostal space.

| Table 1. Echocardiographic Dimensional Abnormalities in Patients with Tamponade |
|------------------------------------------|-----------------|-----------------|-----------------|
|                                           | Normal subjects | Patients with effusion | Patients with tamponade |
| (RVED)d| end-expiration (mm) | N = 25 | N = 157 | NS | N = 17 | <0.001 vs normals & others with effusion |
|        | (mean ± sd)         | 24 ± 5 | 20 ± 4 | 4 ± 2 | |
| Number of patients with right ventricular narrowing | 0 | 0 | 16 |
| (end-expiratory (RVED)d| ≤7 ± 2 mm) | |
| Right ventricular end-diastolic dimension-outflow tract | 43 ± 6 | 41 ± 5 | NS | 29 ± 3 | <0.001 |
| (LVED)d - end-expiration (mm) | (mean ± sd) | 46 ± 4 | 47 ± 4 | NS | 40 ± 4 | <0.001 |

*Significance of difference was computed by chi-square analysis.

Abbreviations: (RVED)d = right ventricular end-diastolic dimension in the minor axis; (LVED)d = left ventricular end-diastolic dimension in the minor axis.
Right ventricular end-diastolic dimension was increased by sweeping toward the outflow tract, but was still generally less at this level in those with tamponade (29 ± 3 mm) than in normal subjects (43 ± 6 mm, \( P < 0.001 \)). All patients who had narrowing of the right ventricular outflow tract also had narrowing in the minor axis. Clinical symptoms were more advanced in those with narrowing of the right ventricular outflow tract than in those who had only right ventricular narrowing in the minor axis.

In nine patients echocardiograms were obtained during or immediately after drainage of the effusion. Right ventricular minor axis end-diastolic dimension increased dramatically and into the normal range after removal of relatively small amounts of pericardial fluid (\( \geq 100 \) cc). Immediate symptomatic and hemodynamic improvement accompanied expansion of the right ventricular minor axis end-diastolic dimension. Figure 1A illustrates right ventricular minor axis narrowing in a patient with tamponade and subsequent expansion following pericardiocentesis (fig. 1B). The dimensional and morphologic changes occurring in the right ventricle in this patient were further elucidated with the aid of a two-dimensional sector scanner as illustrated in figure 2. As in the conventional echocardiographic study the right ventricular outflow tract remains patent, but the body of the right ventricle abruptly narrows below the level of the aorto-septal junction. Initially the anterior or free right ventricular wall is seen to be flat or slightly concave. The postpericardiocentesis study (right panel) reveals expansion of the body of the right ventricle and re-establishment of its outward convexity.

One of our 17 patients, illustrated in figure 3, had normal end-expiratory right ventricular end-diastolic dimension. However, this dimension was much diminished from values obtained before effusion developed. This patient, who had chronic obstructive lung disease, pulmonary hypertension and right ventricular failure, initially had echocardiographic evidence of right ventricular enlargement. No effusion was present. Over a period of four weeks respiratory difficulty increased and hypotension and pulsus paradoxus occurred, believed to be related to the pulmonary disease. A second echocardiographic examination (left panel) revealed a considerable decrease in right ventricular size and a large

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**Figure 1.** Top) Shown is an echocardiographic sweep from the left ventricular cavity to the aortic root (Ao) (left to right), in a patient with cardiac tamponade. Right ventricular (RV) narrowing is seen in the minor axis. Only minimal cavitary expansion occurs with inspiration. The right ventricular cavity opens at the outflow tract (RVOT) as the level of the aortic root is approached. Bottom) Echocardiograms from the same patient as figure 1A are shown following pericardiocentesis. The sweep, left ventricular cavity to aortic root, now runs right to left. Note the expansion of the right ventricular end-diastolic dimensions in the minor axis. The effusion is now difficult to identify. The calibration factors shown are identical. \( \text{ARVW} \) = anterior right ventricular wall; \( \text{CW} \) = chest wall; \( \text{EFF} \) = pericardial effusion; \( \text{IN} \) = end-inspiration at peak of respiratory trace; \( \text{IVS} \) = interventricular septum; \( \text{LA} \) = left atrium; \( \text{PLVW} \) = posterior left ventricular wall; \( \text{MV} \) = anterior leaflet of mitral valve.
pericardial effusion. Right heart catheterization showed elevated and equalized diastolic pressures typical of tamponade. Right ventricular systolic pressure was elevated to 58 mm Hg and after removal of only 100 cc of clear fluid, symptoms abated, right ventricular pressure rose to 95 mm Hg and arterial pressure rose. There was considerable increase in the right ventricular dimension immediately after pericardiocentesis (right panel).

Two other patients with tamponade had cardiac catheterization. Both showed right ventricular narrowing similar to that described above. Removal of small amounts of pericardial fluid resulted in amelioration of their symptoms and signs and resolution of right ventricular narrowing observed echocardiographically.

Also seen in the tamponade patients were: a small left ventricular size with left ventricular end-diastolic diameter

![Figure 2](https://example.com/figure2.png)

**Figure 2.** Shown are the two dimensional echocardiograms performed in the same patient as illustrated in figure 1, before (left) and after (right) pericardiocentesis. These images, obtained in the long axis view, encompass all portions of the heart seen in the standard M-mode echocardiographic sweep. Before drainage the right ventricular cavity is barely discernible below the aortoseptal junction, narrowing abruptly at this level. Following pericardiocentesis the right ventricle expands and is well visualized to the midseptal level. Note diminished effusion size after drainage. Both studies were performed at end expiration and at end diastole (R + 0.00). The bright spot (arrow) represents the timing of camera gating at end diastole. The calibration factor is the same for the two studies. However, the posterior gain setting is reduced in the initial study, obscuring the pleural effusion (PEFF) seen later. LV = left ventricular cavity.

![Figure 3](https://example.com/figure3.png)

**Figure 3.** Shown is the echocardiogram in the minor axis before (left) and after (right) removal of a small amount of pericardial fluid, illustrating expansion of the right ventricular end-diastolic dimension after drainage. Along with this relative change in right ventricular end-diastolic diameter, systolic pressure rose from 60 to 95 mm Hg and symptomatic improvement occurred. Although gains vary somewhat between studies shown, septal and anterior right ventricular wall thicknesses were measured and were confirmed on another record performed prior to pericardiocentesis which showed left ventricular anatomy in a less optimal manner. The calibration factor is the same for both studies.
in the minor axis at end expiration, at or just below the normal limits (table 1); "swinging heart," characterized by parallel motion of the anterior right ventricular wall and posterior left ventricular wall; and electrical alternans. These findings were not specific and were seen in several patients with large effusions without tamponade.

The E-F slope of the mitral valve was difficult to measure accurately in tamponade, because the rapid heart rate present in this group of patients tended to superimpose the rapid phase of ventricular filling upon the atrial contraction phase of ventricular filling. Mitral D-E amplitude did appear to vary proportionately with left ventricular end-diastolic dimension during respiration and to increase after pericardiocentesis. Both variations of mitral D-E amplitude and reciprocal respiratory variations of ventricular size were nonspecific findings seen also in patients with effusion without tamponade, as shown in figure 4.

Discussion

Echocardiographic right ventricular narrowing was seen in 16 of 17 patients with hemodynamically significant pericardial effusion. There is a strong theoretical basis for the presence of right ventricular narrowing during tamponade. In 1897, Starling7 looking for a cause of diminished myocardial function, noted restriction of diastolic expansion in cardiac tamponade. DeCristofaro and Liu8 and Isaacs and associates9,10 believed that the increase in pericardial pressure during tamponade inhibited diastolic expansion of both ventricles. Fowler and coworkers postulated that the right ventricular chamber may be almost completely emptied by pericardial tamponade.11 They stated: "It seems likely that the thinner walled right ventricle may be compressed by tamponade to a relatively smaller volume than the thicker walled left ventricle." Shabetai and associates also referred to compression of the heart by pericardial effusion.12 Craig and associates found that reduction in left ventricular end-diastolic volume is one essential component of the mechanical changes associated with tamponade.13 The right ventricular dimensional decrease on M-mode echocardiography and the changes in configuration of the anterior right ventricular wall in our two-dimensional study suggest that right ventricular compression is an appropriate term to describe some of the echocardiographic findings in tamponade.

Accompanying right ventricular compression was sensitivity of the right ventricular end-diastolic dimension to respiration. Reciprocal respiratory variations in ventricular size and variations in the amplitude of the mitral D-E slope, originally noted in the tamponade setting by D'Cruz and coworkers, were most prominent in this condition, but were nonspecific and were seen in other patients studied (fig. 4).

Other findings not specific for tamponade seen in this group of patients included a low normal or diminished left ventricular end-diastolic dimension, "swinging heart" and electrical alternans. Occasionally, patients without effusion had reduced right ventricular dimensions in association with left ventricular hypertrophy or dilatation (fig. 5). In this setting, however, right ventricular size was rarely less than 1 cm at end diastole, was always smallest at peak systole and was not sensitive to respiration. On the other hand, left ventricular size in tamponade was always normal or less than normal. The presence of severe left ventricular hypertrophy and a moderate effusion could be confused with the echocardiographic features of hemodynamically significant tamponade. However, we have not encountered this combination of findings. Right ventricular narrowing might also appear with volume depletion, as in marked dehydration, blood loss, or hypovolemia and might aid in the diagnosis of these states. A false impression of critical right ventricular narrowing also could be obtained if the measurement were taken at or toward the cardiac apex.
Our findings suggest that directional change, as well as absolute echocardiographic right ventricular size, might be a more effective indicator of hemodynamic compromise than other clinical variables, including effusion size. One patient (fig. 3) who developed tamponade in the setting of pulmonary hypertension and right ventricular dilatation, illustrates the importance of the directional change in evaluating echocardiographic right ventricular size. In this patient it is not surprising that right ventricular narrowing could not be identified. We have also seen echocardiograms from a patient with an atrial septal defect who developed tamponade. Again, directional changes in right ventricular end-diastolic dimension, rather than its absolute value, appeared critical as arterial hypotension was associated with a considerable decrease in right ventricular size, which nonetheless remained above the normal range. Following drainage, right ventricular size was again considerably enlarged.

Right ventricular end-diastolic dimension varied with the hemodynamic significance of pericardial effusion. There appeared to be a critical value of right ventricular end-diastolic dimension indicating tamponade. It is likely that this value only approximates a critical dimension, which apparently varies from patient to patient and is influenced by conditions other than tamponade. Nevertheless, the value differed significantly from the measurement of right ventricular end-diastolic dimension in the same patient when relieved of tamponade and from the group of patients with effusion without tamponade. Although the exact quantitative measurement may sometimes be difficult to obtain, a small value below 1 cm, and certainly a diminution in right ventricular end-diastolic dimension between technically comparable studies, in a patient with effusion, indicates cause for concern about the presence or development of cardiac tamponade.

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N B Schiller and E H Botvinick

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