Cardiac Motion in Patients with Pericardial Effusion

A Study Using Reflected Ultrasound

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The occasional finding of electrical alternation or alternans in patients with pericardial effusion has led to the theory that cardiac motion or rotation may be increased in the presence of excessive pericardial fluid.1-3 According to this theory, the pericardium together with the lungs and mediastinal structures normally exerts a restraining influence on the natural rotational movement of the heart. The introduction of fluid between the heart and the pericardium supposedly removes much of this restriction and allows the heart to rotate more freely.

This theory is primarily based on the assumption that electrical alternation is due to variations in cardiac position at the time of electrical depolarization.3 This assumption, however, is not universally accepted. Since some types of electrical alternation occur without pericardial effusion, this positional theory is apparently not feasible in all situations. Many authorities, seeking a more universal concept, feel that the electrical abnormality is probably not a result of positional changes, but is rather due to a disturbance of bioenergetic behavior of the myocardium.4 Some investigators have gone so far as to propose that the factor underlying all forms of cardiac alternans is a marked prolongation of the refractory phase of some part of the heart.5 This uncertainty regarding the mechanism responsible for electrical alternation casts a doubt on the entire theory of excessive cardiac motion with pericardial effusion and underscores the need for more direct information concerning the movement of the heart in patients with pericardial effusion.

Earlier reports described the feasibility of using reflected ultrasound in the diagnosis of pericardial effusion.6,7 One advantage of this diagnostic technique is that it permits the recording of heart-wall motion within the pericardial fluid.7 This procedure, therefore, provides a unique opportunity to study cardiac motion in patients with pericardial effusion. The result of such a study is the subject of this paper.

Method

The principles and technique of using reflected ultrasound in diagnosis have been described in previous publications.7,8 Since the recording of motion was vital to this study, all ultrasound recordings were made using a "slow-sweep" or "time-motion" display. With this method, an echo from a moving interface is recorded as a wavy line. In addition, the motion of any particular echo can be displayed on a strip-chart recorder by way of an analog output attachment. This diagnostic use of ultrasound thus permits the quantitative recording of movement of various cardiac structures. The resultant tracings are known as ultrasound cardiograms (UCG).

Ultrasoundcardiographic examinations were carried out on 23 patients with pericardial effusion, as confirmed by pericardiocentesis, surgery, autopsy, a roentgenographic contrast study,9 or a heart scan using radioactive albumin.10 These examinations were made with a commercially available ultrasonoscope utilizing a 0.75-inch diameter, 2.25-megacycle transducer and pulsed signals at 200 cycles per second. The patients were examined while in the recumbent

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position. The transducer was placed along the left sternal border in the fourth or fifth intercostal space; a sonic gel was used to ensure ailerless contact between the transducer and the skin. The probe was aimed almost directly posteriorly with the exact angle being adjusted so that echoes originating from both the anterior and posterior heart walls were recorded simultaneously. In some patients, the motion of the heart walls were recorded on a strip chart recorder simultaneously with an electrocardiogram and phonocardiogram.

Results

It is normally difficult to record the movement of the anterior wall of the heart because its echo is usually obscured by the many anterior chest-wall echoes. Figure 1, however, illustrates one of the few tracings in which both anterior and posterior heart-wall echoes could be demonstrated in a normal individual. It should be noted that the walls moved in opposite directions and motion of the anterior wall was considerably less than that of the posterior wall.

The 23 patients with pericardial effusion presented three distinct patterns of heart motion. Fifteen of the patients had ultrasoundcardiograms similar to that illustrated in figure 2. With the presence of pericardial fluid, the anterior heart wall was now clearly separated from the anterior chest-wall echoes and could be easily identified. In addition, the pericardial effusion divided the normal posterior wall echo into a dominant, non-moving posterior component, originating from the pericardium-lung interface, and a more anterior echo which originated from the intracardiac blood-endocardium interface. The latter echo was of lesser intensity and continued to move with cardiac action. The echo-free space between the two posterior echoes represented the pericardial fluid. The actual pattern of heart-wall motion was very similar to the normal. The two walls essentially moved reciprocally and the anterior wall motion was again the lesser of the two. All 15 patients had small or moderate amounts of pericardial effusion, and none showed any evidence of cardiac tamponade.

An example of the second type of heart-wall motion exhibited by the patients with pericardial effusion is illustrated in figure 3A.

Figure 1

Ultrasoundcardiogram of patient with no evidence of heart disease. The anterior heart-wall echo (AW) is faintly visible posterior to the echoes originating from the anterior chest-wall structures. The posterior wall echo (PW) represents a combination of echoes originating from the posterior myocardium, pericardium, and the posterior mediastinal structures or lung. The drawing to the right of the photograph shows the relative motion of the two heart walls.

Figure 2

Ultrasoundcardiogram of patient with mild pericardial effusion. The anterior heart-wall echo (AW) is a stronger signal and is separated from the chest-wall echoes by pericardial fluid. The effusion also divides the posterior echo into a fainter anterior component (PW), which originates from the posterior myocardium, and a stronger posterior echo (P) which stems from the pericardium and mediastinal structures. The vertical calibration dots are 1 cm apart.
serous pericarditis. Following surgical removal of his pericardium, cardiac motion also returned to normal.

Six patients demonstrated a third type of heart movement. The distinguishing feature of the ultrasoundcardiograms in these patients was that the anterior and posterior heart walls moved in the same direction at some time during the cardiac cycle. The extent of this abnormal pattern of movement varied among the six patients. Two patients exhibited only a slight amount of congruous heart-wall motion. One of these two patients was an 11-year-old boy with presumably viral pericarditis. The ultrasoundcardiogram recorded a sudden posterior displacement of both anterior and posterior heart walls at the peak of ventricular systole (fig. 4). The patient had no evidence of tamponade, and cardiac motion reverted to a normal pattern on removal of a mere 50 cc of clear serous pericardial fluid. The other patient with this pattern of heart motion also had no hemodynamic impairment; however, her pericardial effusion proved to be due to invasion of the pericardium by a malignant tumor.

The remaining four patients demonstrated a greater degree of abnormal heart-wall motion. The ultrasoundcardiograms revealed marked anterior wall motion with both heart

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**Figure 3**

Ultrasound tracings. (A) From patient with acute pericardial effusion and cardiac tamponade. The anterior wall echo (AW) is partially obscured by the anterior chest-wall echoes. The unlabeled echo posterior to the anterior wall probably originates from the interventricular septum. (B) From the same patient following pericardiocentesis.

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**Figure 4**

Ultrasoundcardiogram from patient with moderately large pericardial effusion. See text for description of heart-wall motion.
walls moving almost continuously in the same direction. In addition, all four patients had malignant pericardial effusion and possessed clinical evidence of cardiac tamponade. An example of the cardiac pattern exhibited by two of these patients is shown in figure 5A. The marked anterior wall displacement and the congruous motion of both heart walls can be readily appreciated. Figure 5B was recorded from the same patient and illustrates the marked diminution of heart movement following the removal of 450 cc of pericardial fluid.

The ultrasoundcardiogram of one of the last two patients is demonstrated in figure 6A. The excursions of this patient's anterior heart wall was even further increased to about 3 cm, and according to the crude simultaneous electrocardiogram, the heart-wall movement was not uniform for each electrocardiographic complex. One ventricular depolarization was followed by marked

![Figure 5](image1.png)

**Figure 5**

Ultrasoundcardiograms. (A) From patient with large malignant pericardial effusion and cardiac tamponade. The heart-wall echoes move almost continuously in the same direction. (B) Following pericardiocentesis. Note the marked decrease in heart-wall motion.

![Figure 6](image2.png)

**Figure 6**

Ultrasoundcardiograms and electrocardiograms. (A) From patient with pericardial effusion and electrical alternation (see V4 electrocardiogram). The arrows indicate the QRS complexes of a technically poor electrocardiogram. See text for detailed description. (B) From same patient following pericardiocentesis.
posterior movement of both heart walls while a negligible displacement followed the next QRS complex. The patient's routine electrocardiogram demonstrated classical electrical alternation. Following the removal of 250 cc of pericardial fluid, the patient's tamponade was alleviated and the cardiac motion pattern markedly changed (fig. 6B). The total cardiac displacement was now less than 2 cm, one cycle of heart-wall motion corresponded to one electrocardiographic cycle, and electrical alternation was absent.

Figure 7 demonstrates an analogue presentation of the anterior heart-wall echo in the same patient. Electrical alternation is again visible in figure 7A. Shortly after the first heart sound the anterior wall moved posteriorly and did not begin to move anteriorly until shortly before the onset of the next ventricular systole. This second electrical depolarization thus occurred when the heart had not yet returned to its original anterior position, and the resultant electrocardiographic complex was altered. Following the next ventricular contraction, which produced only a minor posterior movement of the anterior heart wall, the heart promptly returned to its original anterior position. The complete cycle of alternating cardiac excursions was then initiated by an original appearing QRS complex. After pericardiocentesis, the anterior wall motion was not nearly as great, and the heart was able to return to its original starting position before the onset of each cardiac cycle (fig. 7B). All electrocardiographic complexes were now identical.

A similar analogue tracing of the anterior heart-wall echo recorded from another patient with electrical alternation is shown in figure 8. There was again an alternating pattern of cardiac motion with each electrocardiographic complex. The relationship was similar to that shown in figure 7A. In addition, the indirect carotid pulse tracing demonstrated some pulse variation with respiration, pulsus paradoxus, but not with alternate heart beats, that is, no pulsus alternans. Thus, in this patient, the alternation in cardiac motion did not seem to affect stroke output.

Figure 7

Analogue ultrasound recordings of anterior heart-wall echo (UCG), phonocardiogram (PCG), and electrocardiograms (ECG) on same patient whose ultrasound cardiograms (UCG) are shown in figure 6. (A) Before pericardiocentesis. See text for description. (B) Following pericardiocentesis.
heart walls did indeed move in opposite directions, with the posterior wall exhibiting the greater excursion. In the present study, most of the patients with small to moderate pericardial effusion and no cardiac tamponade exhibited this same pattern of heart motion. This finding does not necessarily exclude the possibility that some of these hearts may have been moving excessively; for it must be emphasized that the ultrasound technique used in this study allowed for the recording of anteroposterior motion only. Any rotational change that did not displace the heart either anteriorly or posteriorly would probably not alter the ultrasound tracing. Nevertheless, it is reasonable to conclude that most patients with slight to moderate amounts of pericardial effusion fail to demonstrate any significant alteration in anteroposterior cardiac motion.

Eight patients did demonstrate distinctly abnormal cardiac motion. The patients with fairly acute tamponade exhibited very limited heart-wall movement. This finding is consistent with impaired ventricular filling and decreased stroke volume resulting from the increased intrapericardial pressure. In the other six patients, however, the findings were decidedly different. The ultrasound recordings in these patients showed increased rather than decreased cardiac motion. The patterns varied in degree, but all six patients demonstrated increased anterior wall excursions, abnormal posterior wall patterns, and congruous motion of the heart walls during some phase of the cardiac cycle. These findings would have to be interpreted as indicating displacement of the entire heart.

The exact nature of this increased cardiac motion unfortunately cannot be completely determined from this study. As mentioned, the ultrasound technique was able to detect only anteroposterior movement. Nevertheless, excessive rotation remains an attractive explanation for the cardiac displacement. The spiral arrangement of the cardiac musculature, the asymmetry of left and right ventricular contraction, and the crossing of ejected blood into the pulmonary artery and aorta are all factors which might be expected to enhance...
the rotation of a heart that is suspended in fluid, has all restraining influences removed, and is free to rotate about its attachment to the great vessels. Furthermore, the primary shift in electrical axis with electrical alternation has been noted to be in the horizontal plane, \(^1\) again suggesting rotation about a superior-inferior axis. This study, however, does not exclude the possibility that the heart may merely rock or swing in an anteroposterior direction without any significant rotation.

No matter what the exact nature of the cardiac displacement is, the documentation of excessive cardiac motion in patients with pericardial effusion is significant for several reasons. First of all, the marked cardiac displacement in the two patients with electrical alternation provides firm support for the concept that when associated with pericardial effusion, the electrocardiographic abnormality is due to alternating changes in the position of the heart. The results of this study indicate that in these cases the cardiac displacement is so great that the heart is unable to return completely to its original position before the next ventricular contraction begins. As a result, the heart is in a slightly different location when the next electrical depolarization occurs. The following ventricular systole produces much less posterior displacement, and the heart finally returns to its original starting position prior to the subsequent cardiac systole.

Besides substantiating the positional theory of electrical alternation, this study also invites speculation as to what factors might be responsible for the observed alterations in cardiac motion. The reduced heart movement in the two patients with acute cardiac tamponade is not surprising. The increased intrapericardial pressure would certainly restrict ventricular filling and reduce stroke volume.\(^12\) Furthermore, in the patient with the perforated heart, the pericardium was essentially normal, and the pericardial effusion occurred acutely. In such a situation, the pericardium would be expected to stretch very little, and even a small amount of pericardial fluid would produce marked cardiac compression.

Multiple factors probably were responsible for the excessive cardiac motion exhibited by six of the patients. The actual amount of pericardial fluid is most likely of primary importance. As has been repeatedly demonstrated, the removal of even a small amount of fluid will correct electrical alternation, \(^2\) and as shown in this study, pericardiocentesis will dramatically decrease any cardiac displacement. In addition, assuming that a large pericardial effusion is a prerequisite for increased cardiac motion, then of necessity the pericardial sac must be large enough to accommodate this fluid without producing fatal tamponade. Thus, it may be that only with long-standing pericardial disease is there sufficient stretching of the pericardium to permit the accumulation of enough fluid for the removal of any restraining influence that the pericardium might have on heart motion.

The role that increased intrapericardial pressure plays in augmenting cardiac motion is not entirely clear. The four patients with the greatest degree of heart movement all had evidence of cardiac tamponade. Other investigators have repeatedly emphasized the coexistence of tamponade and electrical alternation.\(^13\) There remains the distinct possibility, however, that increased intrapericardial pressure may have nothing to do with enhancing cardiac displacement. As noted in this study, cardiac tamponade alone cannot be the sole explanation for excessive cardiac motion since two of our patients with tamponade exhibited diminished heart-wall movement. Furthermore, the coexistence of cardiac tamponade and excessive cardiac motion may merely be due to the fact that the amount of pericardial fluid required for cardiac displacement also of necessity produces an increase in intrapericardial pressure.

Another puzzling question is why electrical alternation occurs so frequently with malignancy.\(^2\) Five of the six patients who exhibited evidence of total cardiac displacement had malignant cells in the pericardial fluid. One possible answer might be that the quality of the pericardial fluid may be an important factor in influencing cardiac motion. Other au-

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tors have suggested that a pericardium invaded by tumor may be rigid and less likely to envelop the heart and restrict its motion. Furthermore, with malignancy there may be minimal inflammation and little or no adhesions between the visceral and parietal pericardium. Whatever the mechanism, it is difficult to deny a possible causal relationship between malignant pericardial effusion and excessive cardiac excursions.

In addition to marked cardiac displacement, other specific factors may be necessary to obtain electrical alternation. For example, heart rate has been considered to be important in the genesis of the electrocardiographic abnormality. It is fairly easy to anticipate that if the heart rate were slow enough, the heart would have sufficient time to return to its original position before the next cardiac cycle, and the electrocardiographic abnormality could not occur. Littmann and Spodick pointed out that in all of the reported cases of electrical alternation and pericardial effusion the heart rates were 100 beats per minute or more. They suggested that the heart might act as a pendulum and that alternans occurs when the heart rate is exactly twice the natural oscillatory frequency of the heart. Heart rate alone, however, is probably not sufficient to produce alternans since the heart rate with electrical alternation differed little from that without it in the patient whose recordings are illustrated in figure 7. Furthermore, as previously stated, all patients with electrical alternans have some degree of cardiac tamponade, and tachycardia is certainly to be expected. Nevertheless, in a patient with pericardial effusion and marked cardiac displacement, heart rate might be the critical factor as to whether or not electrical alternans occurs.

From this brief discussion, it may be logically concluded that no one factor is solely responsible for the abnormal cardiac motion observed among our patients with pericardial effusion. The amount of pericardial fluid is probably the single most important factor; however, further investigation is undoubtedly necessary to determine the etiological importance of intrapericardial pressure, type of pericardial effusion, and heart rate. In addition, more information concerning the exact nature of the cardiac displacement is a necessary prerequisite to any basic understanding of cardiac motion in patients with pericardial effusion.

**Summary**

Diagnostic ultrasound was used to record the movement of the anterior and posterior heart walls in 23 patients with proven pericardial effusion. Fifteen of these patients demonstrated heart-wall motion that could not be distinguished from the normal pattern. Two patients with acute cardiac tamponade exhibited markedly reduced cardiac motion. The ultrasoundcardiograms obtained from the remaining six patients indicated posterior displacement of the entire heart during systole. The extent of this cardiac displacement varied. Five of the six patients with excessive cardiac motion had malignant pericardial effusion, and four of the six had clinical evidence of cardiac tamponade. The two patients with the most marked excursions exhibited electrical alternation.

The results of this study substantiate the theory that excessive cardiac motion may occur in some patients with pericardial effusion. Objective evidence is also provided in support of the positional etiology of electrical alternation associated with pericardial effusion. An attempt is made to identify some of the factors responsible for the observed abnormal cardiac motion, and some speculation is offered as to what role rotation might play in the cardiac displacement.

This study also serves as an example of the type of cardiovascular investigation that can be performed with diagnostic ultrasound.

**References**


Experiments Illustrating the Operation of the Safety-Valve

Taking a human heart from a body dead within about 24 hours, and in which I could discover no material morbid change of form or substance, I proceeded to remove portions of the sigmoid or semilunar valves of the aorta and pulmonary artery, and to fix tubes into these vessels, with a view to inject the ventricles. I next cut away a large part of the auricles, so as widely to expose their communications with the ventricles: and now, upon injecting the left ventricle through the aorta, the mitral valve became at once firmly closed, and, as seen through the auricle, presented very much indeed the appearance of a continued suture, with a slight prominence or tense bulging sac on either side. The pressure of the syringe and distention of the ventricle being maintained, nothing escaped. Great force was required to disarrange this state, and produce a narrow thready jet. After this, proceeding to the injection of the pulmonary artery in the same manner, and slowly filling the right ventricle, it was quite impossible to produce a similar effect. No position in or out of water, no degree of gentleness or force, no state in anywise natural to the organ that I was able to induce, would prevent a considerable ribband-like stream of regurgitation between the ill-apposed edges of the valve.—T. W. KING: An Essay on the Safety-Valve Function in the Right Ventricle of the Human Heart: and on the Gradations of this Function in the Circulation of Warm-Blooded Animals. Guy Hosp Rep 2: 127, 1837.
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