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RESEARCH ARTICLE

ECHOCARDIOGRAPHY IN PERICARDIAL EFFUSION

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ABSTRACT

Echocardiography has been established as the procedure of choice for the detection, confirmation and serial follow-up of patients with pericardial effusion. The criteria and their sensitivity for the diagnosis of pericardial effusion are reviewed.

INTRODUCTION

Although it was known separation of echo of the anterior heart wall from that of the chest wall in patients with pericardial effusion, it was not until Feigenbaum and colleagues published a series of papers in the mid sixties that the usefulness of echocardiography in pericardial effusion was established. Other papers confirmed their findings. Today, echocardiography is considered the procedure of choice for evaluating patients suspected of having pericardial effusion. This high place afforded to echocardiography is due to its being an accurate, easy to perform and non-invasive method. It must be remembered, however that the results obtained are entirely dependent on the knowledge, experience and technical skill of the examiner. The echocardiographer must be familiar with the ultrasonic cardiac anatomy and various intracardiac landmarks. Many previously published false-positive and false-negative echocardiograms in pericardial effusion can be directly attributed to faulty techniques.

ECHOCARDIOGRAPHIC CRITERIA FOR DIAGNOSIS OF PERICARDIAL EFFUSION (Spodick, D. H, 1991):

PERICARDIAL EFFUSION

A) Echo-free space-Posterior to LV (Small to moderate effusion).
Posterior and anterior (moderate to large effusion).
Behind left atrium (large to very large effusion).

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B) Decreased movement of posterior pericardium-lung interface.
C) RV pulsations brisk (with anterior fluid).
D) Swinging heart" (large effusion)
Periodicity 1:1 or 2:1
RV and LV walls move synchronously
Mitral/tricuspid pseudo prolapse.

Alternating mitral EF slope and aortic opening excursion

The following changes appear in the echocardiogram with the development of pericardial effusion. The presence of pericardial effusion is indicated by the demonstration of an echo-free space between the right ventricular anterior wall and the chest wall and/or between the left ventricular posterior wall and the pericardial lung interface. It has now been well established that the pericardial fluid first appears posteriorly in the dependent portion of the pericardial cavity. Therefore, the earliest manifestation of the presence of a tiny pericardial effusion is a slight separation, through systole and part of diastole, of the left ventricular posterior wall epicardial echo from a relatively stationary pericardial echo. With increasing accumulation of fluid, this posterior echo-free space becomes evident throughout the entire cardiac cycle. Absence of an anterior echo-free space; should suggest the presence of a small amount of pericardial effusion. With further increase in the pericardial effusion, the posterior space widens, with larger effusions the fluid also appears anteriorly as an anterior echo-free space. In the presence of anterior effusion, amplitude of motion of the anterior wall of the right ventricle (RV) becomes exaggerated. A large pericardial effusion is diagnosed when there is a further increase in the depth of the anterior and posterior echo-free spaces. Frequently, but not invariably, when a large anterior

and posterior effusion is present, the heart may be observed to be moving freely in the pericardial sac-"Swinging heart". "Swinging heart" was initially described by Feigenbaum and associates in 1966. The free motion of the heart attributed to the absence of the restraining function of the pericardium. The heart, suspended by the great vessels, swings freely within the pericardial sac, causing both the anterior and the posterior walls of the heart to move synchronously that is, anteriorly during systole and posteriorly during diastole and vice versa. Generally, but not invariably, greater swing occurs with larger effusions and especially those of malignant origin. As a consequence of the free swinging of the heart, abnormal motion of the various structures, such as the septum, mitral and tricuspid valves, and the aortic and the pulmonary valves, has been noted. As the heart swings anteriorly during systole, both the anterior and posterior walls of the heart, as well as the ventricular septum, also moves anteriorly, and therefore an abnormal ventricular septal motion is not frequently recorded in this situation. In patients with swinging heart syndrome various patterns of mitral valve prolapse have also been described. The most common pattern is the typical late-systolic mitral valve prolapse. Vignola *et al* noted a distinct effect of heart rate on the type of mitral valve prolapse. They prolapse in early or late systole if the heart rate exceeded 120/min and a pansystolic-type prolapse when the heart rate was less than 120/min. That is mitral valve prolapse is a pseudo prolapse phenomenon has been proved by the fact that, after pericardiocentesis or pericardiactomy, the mitral valve motion returns to normal. Occasionally anterior motion of the mitral valve in systole may be recorded. Similarly abnormal motion of the aortic valve and of the pulmonic valve has been noted in these patients. (Lemire *et al*, 1976 and Nanda NC *et al*, 1975).

ECHOCARDIOGRAPHIC CRITERIA FOR DIAGNOSIS OF CARDIAC TAMPONADE (Spodick D. H., 1991):

CARDIAC TAMPONADE: Diagnosis is made by features of effusion as mentioned above along with:

A) RV Compression
   RV diameters decreased, especially outflow tract. Early diastolic collapse of RV.
B) RA free wall indentation (Collapse) during late diastole or isovolumic contraction.
C) L free wall indentation (Cases with fluid behind LA).
D) LV free wall Paradoxic motion.
E) SVC and IVC congestion (Unless volume depletion).
F) Inspiratory effects (With pulsus paradoxus)
   RV expands
   IV septum shifts to left.
   LV compressed
   Mitral DE amplitude decreased.
      EF slope decreased or rounded
      Open time decreased
   Aortic valve opening decreased; premature closure.
   Echocardiographic stroke volume decreased.
G) Notch in RV epicardium during isovolumic contraction.
H) Coarse oscillations of LV posterior wall.

Cardiac tamponade is a clinical syndrome characterized by hypotension, venous distention, tachycardia, pulsus paradoxus and a prompt favourable hemodynamic response to pericardiocentesis. Although echocardiography in such patients allowed easy detection of a large anterior and posterior pericardial effusion, until recently no echocardiographic criteria for the diagnosis of cardiac tamponade were available. Feigenbaum *et al*; 1965, 1966; noted that the motion of the posterior wall was diminished in patients with cardiac tamponade. This finding subsequently was noted, however, in only two of six patients with such a diagnosis. The next recognized echocardiographic criterion in cardiac tamponade was a swinging heart with an electrical and pulsus alternans (Gabor.GE *et al*; 1971 and Usher BW et al; 1972). D'Cruz *et al*; 1975 and Vignola *et al*; 1976 reported preliminary observations suggesting that changes in mitral valve motion and chamber dimensions may be indicative of tamponade. Decreased excursion of the mitral valve with diminished E-F slope was noted by both groups. D'Cruz *et al* also noted pronounced phasic variation in the dimensions of the right and left ventricles. During inspiration the dimension of the right ventricle (RV) increased whereas that of the left ventricle (LV) decreased. This variation in chamber dimension with respiration accounts for the pulsus paradoxus that is commonly seen in these patients. Vignola *et al*, in addition, described a notch on the epicardial surface of the right ventricle, which occurred during the isometric contraction phase of the cardiac cycle, and also noted coarse oscillation of the left ventricular posterior wall.

Recognition of chamber collapses has great importance, because they also appear in incipient tamponade, as well as advanced tamponade. Left atrial (LA) and biatrial collapse has 90% to 100% sensitivity and a somewhat lower specificity for cardiac tamponade. It occurs immediately following atrial systole and atrial wall remains invaginated to a variable degree during early ventricular systole but disappears by the end of ventricular systole. It often has a reverse "L" configuration, best seen in either the apical or subcoastal four-chamber or the parasternal short axis views. D'Cruz has quantified this phenomenon; the greatest normal reduction in echo area of either atrium is less than 16%. In tamponade, atrial collapse raises this to 20% or more, and the left atrium tends to collapse over a greater area than the right atrium. Duration of right atrial collapse beyond $1/3^{rd}$ of cardiac cycle has almost a perfect specificity and is highly sensitive as a sign of tamponade. Right ventricular collapse occurs as an inward motion of the free wall in early diastole, appearing as a distinct concavity or "dent" well seen in almost any imaging view displaying the free wall. It usually normalizes by the end of ventricular diastole. Right ventricular collapse has 80% to 90% specificity and 90% to 100% sensitivity for cardiac tamponade. Finally, while virtually all causes of pericardial inflammation or injury can cause tamponade, particular suspicion is required in patients with the most common causes.

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