Pericardial Effusion and Compressive Disorders of the Heart

Influence of New Technology on Unraveling its Pathophysiology and Hemodynamics

Ralph Shabetai, MD[a,†], Jae K. Oh, MD[b,*]

KEYWORDS

• Pericardium • Pericardial effusion • Cardiac tamponade • Constrictive pericarditis

KEY POINTS

- Constrictive pericarditis and cardiac tamponade cause severe diastolic dysfunction, but do not depress systolic function until an extreme, end-stage state has been reached.
- Although physiologic and hemodynamics features of pericardial disorders have been studied for more than two centuries, recent advances in cardiovascular imaging (in particular echo-Doppler) have revolutionized the diagnosis and understanding of pericardial diseases.
- The concept of ventricular interdependence is the hallmark of both cardiac tamponade and constrictive pericarditis. However, their underlying pathophysiology is significantly different.
- Techniques used for pericardiocentesis have signicantly evolved the past decades and echocardiographic-guidance has markedly improved its safety.

"This introduction is a revised article originally written by the late Dr Shabetai for a pericardial diseases textbook which was not published. He was the editor of previous Pericardial Diseases issue for Cardiology Clinics in the 1980s, it is most appropriate to begin our issue with his insights. The remaining articles describe advances in diagnosis and management, focusing on clinically important aspects of pericardial diseases."

Clinicians now take it for granted that when the question of a pericardial effusion arises, it is quickly settled by echocardiography. Before the advent of echocardiography, one relied on the notoriously inaccurate physical examination, an unexplained increase in heart size on a chest radiogram, or evidence of pericarditis. The chest radiogram alone, despite signs such as a double density along the cardiac edge, or a posterior bulge on the lateral view, is seldom sufficient for a firm diagnosis of pericardial effusion. Other techniques used in the preechocardiographic era included cardiac fluoroscopy, which disclosed absence of cardiac pulsations, and cardiac catheterization, during which the catheter failed to reach the edge of what

[a] Department of Medicine, University of California, San Diego, La Jolla, CA 92093, USA; [b] Department of Cardiovascular Diseases, Mayo Clinic, 200 First Avenue Southwest, Rochester, MN 55905, USA
[†] Deceased
* Corresponding author.
E-mail address: oh.jae@mayo.edu

Cardiol Clin 35 (2017) 467–479
http://dx.doi.org/10.1016/j.ccl.2017.07.001

seemed to be the right atrial border. Angiography disclosed a stationary water density surrounding an actively beating heart. Another method for diagnosing pericardial effusion that has been replaced by echocardiography was to inject carbon dioxide intravenously. The patient was placed in the left decubitus position and the radiolucent bubble was observed fluoroscopically and documented by a cross-table radiograph. The size of the effusion was estimated from the width of the water density over the gas bubble. Compare the effort and discomfort of the procedure with that of performing a limited echocardiogram to rule out pericardial effusion. In fact, reliable detection of pericardial effusion was the first clinical application of A-mode echocardiography by Dr Feigenbaum and colleagues[1] (**Fig. 1**).

The etiology of pericardial effusion has changed over the recent decades, in part because of the development of effective antibiotics. Tuberculosis and other pyogenic infections have become much less common as the cause of pericardial effusion, with or without tamponade; purulent pericarditis is now rare, although only a little less sinister than it used to be. Pericardial effusions complicating invasive cardiac procedures and therapy, or in acquired immune deficiency syndrome (AIDS) have become more common. Moreover, nowadays pericardial effusion occurs more often against a background of myocardial or systemic disease, a factor that may make evaluation and treatment more difficult. Young cardiologists will not know this, but cardiac tamponade used to be the scourge of dialysis units, until about a decade ago when the changed dialyzing membrane blocked transmission of whatever agent was responsible. On a medical service and in the clinic, cardiac tamponade is now more often subacute than either acute or chronic.

Serial echocardiography has demonstrated that silent pericardial effusion is common in pregnancy and after myocardial infarction or cardiac surgery. Although Dressler's syndrome is becoming less frequent, cardiac tamponade continues to be an important complication after heart surgery. Tamponade, often occurring after the patient has been discharged from hospital, may be a manifestation of pericardial injury, or owing to hemorrhage, often localized and sometimes organized, most commonly behind the right atrium. Echocardiography and computer-assisted tomography have been useful in characterizing localized effusions. Even after pericardial effusion resolves, the pericardium remains inflamed, resulting in constrictive hemodynamics. The extent of pericardial inflammation can be determined from delayed

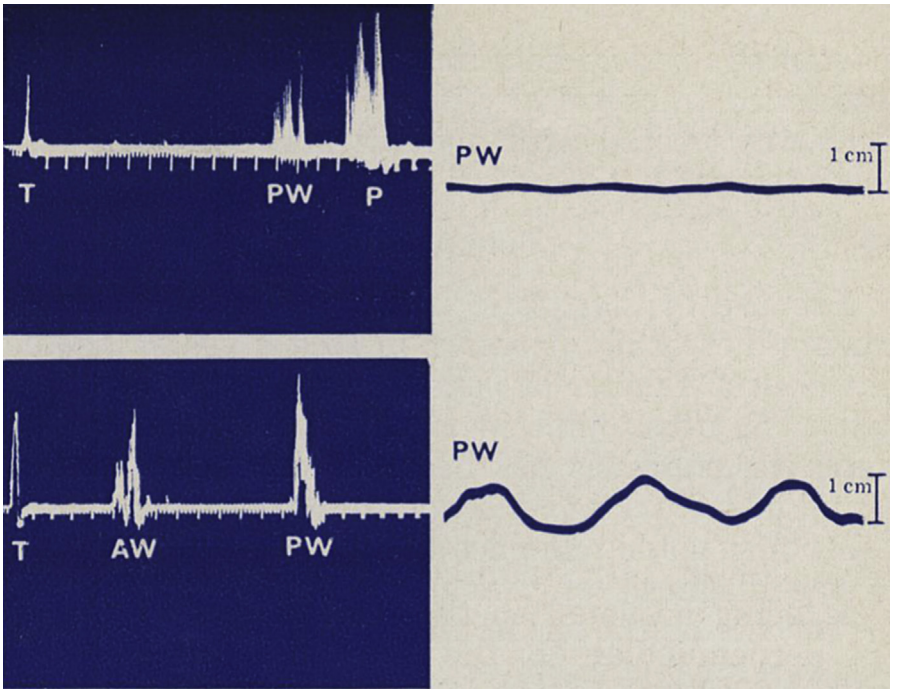

Fig. 1. (*Top*) A-mode echocardiography detecting pericardial effusion between posterior wall (PW) and the pericardium (P). The systolic and diastolic motion of PW is lost. (*Bottom*) After draining of pericardial effusion, the space is no longer present and the motion of PW is restored. (*From* Feigenbaum H, Waldhausen JA, Hyde LP. Ultrasound diagnosis of pericardial effusion. JAMA 1965;191:713; with permission.)