

Acute Pericarditis

Diagnostic Cues and Common Electrocardiographic Manifestations

Vignendra Ariyaratnam, MD,*† and David H. Spodick, MD, DSc‡

Abstract: Acute pericarditis (AP) is basically a clinical diagnosis. Although specific electrocardiographic (ECG) manifestations may indeed point to its diagnosis, sole reliance on such findings in isolation of the clinical setting, however, is often the common pitfall that could lead to a misguided diagnosis. We briefly review the anatomy of the pericardium and the pathophysiology of pericarditis to highlight common signs and symptoms as well as clinical findings that may assist in the diagnosis of AP. We also feature the characteristic evolution of its ECG manifestations and point out some of its typical and atypical features to help better differentiate AP from commonly confused conditions.

Key Words: acute pericarditis, electrocardiogram, clinical diagnosis

(*Cardiology in Review* 2007;15: 24–30)

The pericardium, devoid of impulse-generating capacity, does not produce detectable deflections on the electrocardiogram (ECG). Yet, in pericardial disease, ECG changes remain a common phenomenon, making the ECG a very useful diagnostic tool.^{1–3} Part of the mechanism for some such ECG manifestations, however, lies in its concomitant inflammation of the subepicardial myocardium.^{1,2} Hemodynamic changes and increased pericardial fluid, fibrin, blood, clot, or scar could also insulate the heart and dampen the voltage of the surface ECG.¹

RELEVANT PERICARDIAL ANATOMY AND FUNCTION

The pericardium is a flexible double-walled membrane that envelopes the heart, consisting of the superficial fibrosa and deep serosa. The serosa is in turn made up of 2 layers: the

parietal pericardium, which abuts the fibrosa, and the visceral pericardium (epicardium), which directly adheres to the heart. Between these dynamic serous layers exists a potential space that contains a film of 15 to 35 mL of pericardial fluid, an ultrafiltrate of blood plasma, and cardiac lymph that acts as a lubricant to reduce interlayer friction during cardiac contraction.^{1–4} Mechanically these layers, particularly the parietal pericardium, limit excessive cardiac dilatation by exerting a contact stress on the heart during diastole.^{1,5} This maintains an efficient cardiac cycle by limiting atrial and ventricular overdistension, especially at the right atrium and ventricle where, as a result of reduced myocardial thickness, reliance on pericardial constraint for normal pressures and dimensions probably occurs maximally. The pericardial layers also act as a barrier to a modest degree to shield the heart from friction and contiguous inflammation as well as spread of infiltrative disease from adjacent structures.^{1–3} Ligaments from the parietal pericardium limit cardiac displacement through attachments to the sternum, vertebral bodies, and the diaphragm.^{1–3}

PERICARDIAL INFLAMMATION

Pericarditis can manifest as acute, subacute, or chronic.^{1–3,6} Given the intimacy of the visceral pericardium and myocardium, inflammation of either structure is rarely confined to that structure without involvement of the other.^{1,2} Pericarditis, be it acute or chronic, may or may not produce an effusion. Since any degree of pericardial inflammation will produce “effusions” into the pericardial space no matter how minuscule, clinically, the term “dry” pericarditis is reserved to describe manifestations in which fibrinous pericardial inflammatory exudation (“bread-and-butter” pericarditis) is clinically undetectable or insignificant.^{1,6} However, increased pericardial vascularity and infiltration with a symphony of inflammatory mediators and leukocytes is a common theme in this clinical picture.^{1,3,4}

ACUTE PERICARDITIS

AP is diagnosed in approximately 0.1% of hospital admissions.^{7,8} It is more common in adults (commonly aged 20–50 years) and in men.⁷ Mortality varies considerably depending largely on the severity of the affliction and the underlying cause, death rarely occurring with viral etiologies but common in (untreated) suppurative pericarditis.^{1–3} Most of the time, however, no specific etiology can be uncovered, but a wide variety of diseases can be associated with AP (Table 1).^{1,9}

From the *Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), Veterans Affairs Boston Healthcare System, Boston, Massachusetts; the †Department of Medicine, Brigham and Women’s Hospital, Harvard University, Boston, Massachusetts; and the ‡Department of Medicine, Division of Cardiovascular Medicine, University of Massachusetts Medical School, Worcester, Massachusetts.

Correspondence: David H. Spodick, MD, DSc, Department of Medicine, Division of Cardiovascular Medicine, University Campus, University of Massachusetts Medical School, 55, Lake Avenue North, Worcester, MA 01655. E-mail: spodickd@ummc.org.

Copyright © 2006 by Lippincott Williams & Wilkins

ISSN: 1061-5377/07/1501-0024

DOI: 10.1097/01.crd.0000210645.89717.34

TABLE 1. Common Etiologies of Acute Pericarditis

I. Idiopathic
II. Infectious
a. Viral
Coxsackie virus
Influenza
Human immunodeficiency virus
Hepatitis virus B, A, C
Echovirus
Epstein-Barr virus
b. Bacterial
Staphylococcus
Pneumococcus
Haemophilus
Salmonella
Tuberculous
Meningococcus
Syphilis
c. Fungal (mycotic)
Histoplasmosis
Blastomycosis
Coccidioidomycosis
Aspergillosis
III. Connective Tissue Diseases/Vasculitic
a. Rheumatoid arthritis
b. Systemic lupus erythematosus
c. Scleroderma
d. Sjögren syndrome
e. Sarcoidosis
f. Dermatomyositis
g. Polyarteritis nodosa
h. Wegener granulomatosis
i. Ankylosing spondylitis
IV. Diseases of Contiguous Structures
a. Myocardial infarction
Acute infarction
Dressler syndrome
Postpericardiotomy syndrome
Ventricular aneurysm
b. Dissecting aneurysm
c. Pleural and pulmonary diseases
V. Metabolic Disorders
a. Renal insufficiency
“Dialysis” pericarditis
Uremia
b. Myxedema
c. Gout
VI. Neoplastic
a. Metastatic
Sarcomas
Breast
Lung
Lymphoma
Melanoma
Leukemia
b. Primary
Mesothelioma
Sarcomas
Fibroma
Lipoma

(Continued)

TABLE 1. (Continued)

VII. Traumatic
a. Direct
Pericardial perforation (gastric or esophageal perforation, chest trauma)
Cardiac injury (cardiac surgery or catheterization)
b. Indirect
Radiation
Blunt, nonpenetrating chest trauma
VIII. Drug-induced
a. Hydralazine
b. Procainamide
IX. Immune-mediated
a. Inflammatory bowel disease
b. Stevens-Johnson syndrome
c. Loeffler syndrome
d. Congenital anemias

From Spodick DH. Pericardial diseases. In: Braunwald E, Zipes DP, Libby P, eds. *Heart Disease: A Textbook of Cardiovascular Medicine*, 6th ed. Philadelphia, PA: WB Saunders Co; 2001.

COMMON SIGNS AND SYMPTOMS

Chest pain is by far the most common symptom of AP.^{1-3,10} Pain can be sharp, stabbing, pleuritic, or aching and can mimic symptoms associated with myocardial infarction (MI), especially when dull or pressure-like. Although intensities vary among individuals, classically, almost all patients report pain relief with sitting up or leaning forward. The onset of the pain may also suggest the etiology, for example, dramatic yet crescendoing pain is often noted with viral infections but often “silent” with rheumatologic etiologies.¹ Pain is usually retrosternal but may radiate to the shoulder, neck, and jaw, further mimicking angina and broadening the differential diagnosis. However, trapezius ridge pain, usually on the left, is often associated with pericardial irritation and is mediated through the phrenic nerve.^{1-3,10} Also, unlike angina, pain during AP is usually constant, unrelated to exertion and poorly, if at all, relieved with nitrates. Systemic reactions may ensue after the initial pericardial inflammatory response. Fevers, when high or spiking, often indicate an infectious etiology, ie, usually bacterial, but a prodrome including myalgias and malaise can in fact be present with any cause of AP, particularly in young adults.¹ A thorough history of the presentation, therefore, is essential for the physician.

On physical examination, the pericardial rub is pathognomonic.¹⁻³ Rubs, caused by friction between the inflamed pericardial surfaces, are usually triphasic (occurring sequentially during ventricular systole and early diastole and atrial systole) and are frequently evanescent.¹¹ Hemodynamic change is often noted if an associated pericardial effusion becomes excessive.¹ In such instances, presence of tachycardia, tachypnea, pulsus paradoxus, and sometimes Beck triad (jugular venous distension, hypotension, and muffled heart sounds) are hallmarks of cardiac tamponade.¹² Cardiac tamponade impairs diastolic ventricular filling by decreasing venous return and, if uncorrected, greatly diminishes cardiac output to eventually result in cardiovascular collapse. Physical findings associated with constrictive pericarditis include

a loud protodiastolic “knock” (reflecting a rapid deceleration of left ventricular filling) and a paradoxical rise of jugular pressure during inspiration (Kussmaul sign).¹³

DIAGNOSTIC STUDIES

A complete blood count and blood chemistry evaluation should accompany the basic workup. Although leukocytosis is indeed common, blood chemistries could in fact suggest renal disease and help unmask an underlying uremic cause. Inflammatory hematologic markers such as the erythrocyte sedimentation rate and C-reactive protein could be elevated and, depending on the extent of subepicardial myocardial involvement, serum cardiac isoenzymes such as creatine phosphokinase, MB creatine kinase isoenzyme, and aldolase may also rise.¹⁻³ As such, when the clinical presentation mimics an acute coronary syndrome, ischemia and MI must be ruled out first. With large pericardial effusions and especially with a history of thyroid disorders, thyroid function should be checked. High or spiking fevers, especially with rigors or when a bacterial etiology is suspected, necessitate blood cultures to be drawn promptly. In tuberculous pericarditis, the purified protein derivative skin test is often positive and should supplement acid-fast staining of any pericardial fluid.⁹ Human immunodeficiency virus antibody testing (enzyme-linked immunosorbent assay [ELISA] and Western blot) should be conducted after exclusion of common specific etiologies and especially in individuals with high risk of exposure.³ With connective tissue disorders, especially when pericarditis is prolonged, severe, or recurrent, evaluation of rheumatoid factor antinuclear antibody, antideoxyribonucleic acid, and antismooth muscle antibodies could be indicated.¹⁻³ Direct pericardial biopsy can also be used for recurrent or persistent pericardial effusions: visceral pericardial biopsy through a scope is always much more productive than parietal pericardial biopsy.

Hemodynamic evaluations of right atrial, right ventricular end diastolic, pulmonary artery diastolic, and mean pulmonary wedge pressures in AP are usually unchanged, but with constrictive pericarditis or cardiac tamponade, the hemodynamic signature is that of an elevation and equalization of such diastolic pressures.³ The chest x-ray (CXR) film is often normal in

patients with AP unless accompanied by a large pericardial effusion.¹⁻³ In such cases, an enlarged, often water bottle-shaped cardiac silhouette is common. Pleural effusions may also be noted with tamponading or nontamponading pericardial effusions,^{1-3,14} whereas pericardial calcification suggests constrictive pericarditis.^{1,2} The echocardiogram is relatively insensitive to pericardial inflammation in “dry” pericarditis. However, it sometimes depicts pericardial fibrin deposition occasionally with a ragged “sunburst” appearance but usually as strands and thus suggests AP in the presence of a pericarditic clinical picture.¹ Echocardiography is particularly important in detecting pericardial effusions, although retrocardiac and loculated effusions may sometimes be missed.¹⁻³

Computer-assisted tomography and magnetic resonance imaging may show pericardial thickening (often ≥ 5 mm), calcification, or accompanying effusions even if loculated.¹⁵ Technetium-99 pyrophosphate scans may also be positive in patients with AP, whereas gallium-67 and indium-111 can display characteristics of purulent pericarditis.¹ However, such investigations are usually unnecessary in AP when the ECG suffices as a cheap and efficient diagnostic tool to supplement the clinical suspicion. (Leukemic infiltrations can mimic inflammatory cellular exudates with gallium and indium.)

ELECTROCARDIOGRAPHIC MANIFESTATIONS

Acute pericardial inflammation causes characteristic 12-lead ECG changes that have typically evolved sequentially through 4 stages (although much less seen today in treated patients).¹⁻³

Stage 1

Chest pain is predominant in this phase. Its onset usually coincides with widespread ST-segment elevations that are concave-upward, appearing in all leads except aVR and usually V1, in which ST-segment depression (or isoelectric ST in V1) is the usual finding. This involvement of virtually all ECG leads is a cardinal finding in AP (Fig. 1). T waves are concordant in leads with ST elevation and the ratio of ST junction to T-wave amplitude in leads V5 and V6 is ≥ 0.25 as measured using the PR segment as baseline.¹⁶ PR-segment depressions are quasispecific for pericarditis in this stage (noted in $>60\%$ of patients

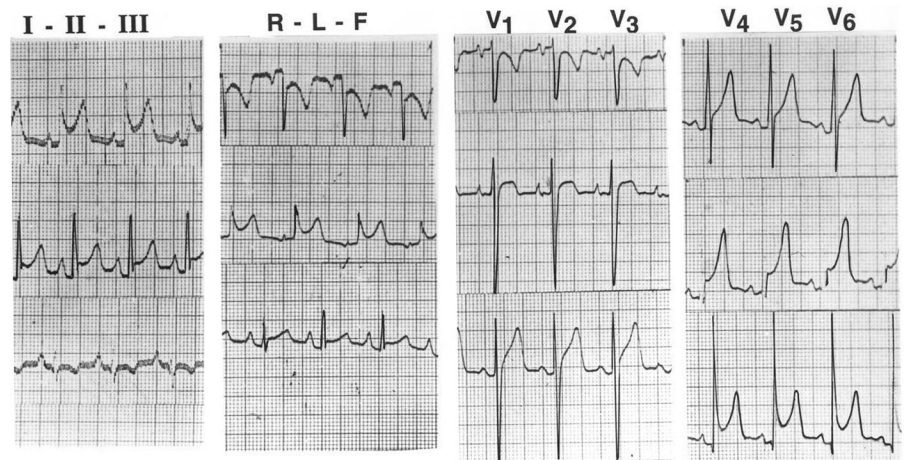


FIGURE 1. Stage 1 electrocardiogram in acute pericarditis. Diffuse J-point elevation (except in aVR and V1) with pronounced PR-segment deviations.

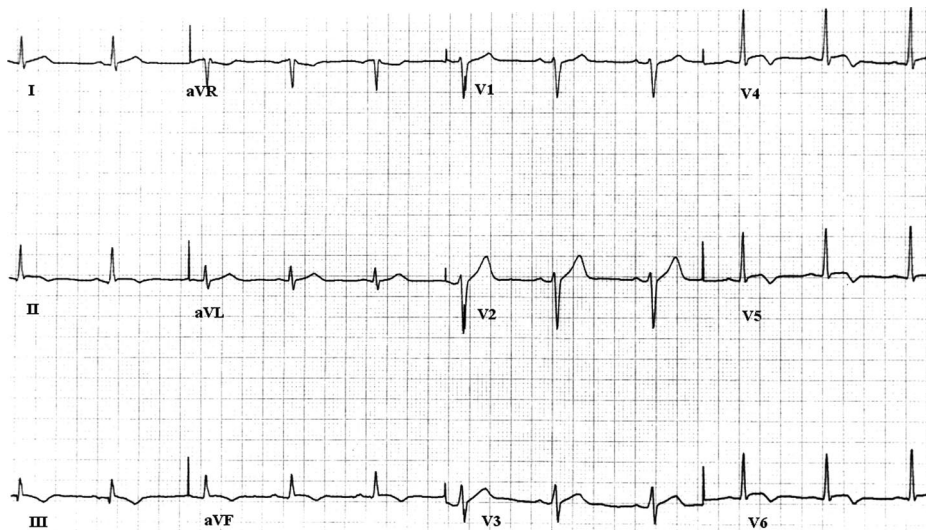


FIGURE 2. Electrocardiograms in acute pericarditis depicting stage 2 (late) evolving to stage 3. Continuation of evolutionary phase with T-wave flattening, common in stage 2, but T-wave inversion of stage 3 has begun.

with AP) and denote subepicardial atrial injury. Depressed PR segments are often horizontal or downsloping toward the QRS complex and can be noted in most leads, particularly II, aVF, and V4 to V6, but not in aVR and V1, where PR-segment elevation (upsloping) occurs.^{1,17} The ECG may also be of low voltage even without a clinically detectable effusion (“dry” pericarditis). This stage may last for up to 2 weeks after initial symptoms.

Stage 2

This is an evolutionary stage in which ST-segment changes return to baseline. In the early phase of this stage, T waves show little change, but J points (ST segments) undergo a normalizing pattern and return to baseline. In the late phase, T waves flatten and may even start inverting (Fig. 2). This stage occurs from hours to days after initial symptoms.

Stage 3

Generalized T-wave inversion is noted in almost all leads (opposite to the original direction of the ST segments). This stage arises from diffuse but possibly superficial myocardial injury or global myocardial inflammation. The onset

of this stage is often between the second and third weeks and ECG changes may continue for several weeks.

Stage 4

Gradual resolution of T-wave inversion occurs as the ECG returns to its prepericarditis state. Rarely, focal T-wave flattening or inversion persists, especially if underlying myocardial injury or scarring has occurred (cicatrical constriction is developing). This stage may last from days up to 3 months.

ELECTROCARDIOGRAPHIC DIFFERENCES BETWEEN ACUTE ISCHEMIA AND ACUTE PERICARDITIS

ECG changes occur in 90% of patients with AP.^{1,2} Occasionally, it may indeed be difficult to distinguish these ECG changes from those of acute ischemia and early repolarization. In such instances, serial ECGs depicting the previously mentioned pathognomonic evolutionary stages in AP may help make the distinction (Fig. 3).^{1,18} However, such classic sequential change in ECG pattern is now seen in only approximately 50% of cases. In addition to the history and

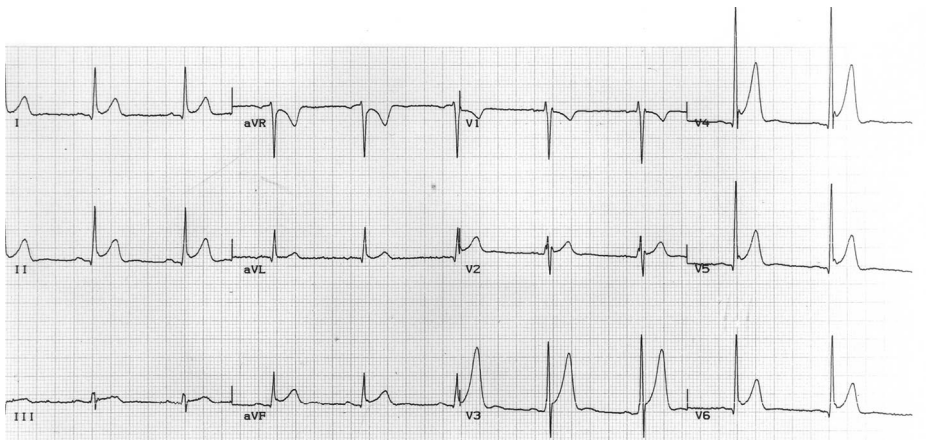


FIGURE 3. Electrocardiogram showing early repolarization.

TABLE 2. Clinical Characteristics of Acute Pericarditis Versus Acute Ischemia

	Acute Pericarditis	Acute Ischemia
Pain character	Sudden Sharp May radiate Could involve trapezius ridge Persistent; may wax and wane	Usually gradual, crescendo Dull, heavy, crushing, "pressure" sensation Usually radiates Never involves trapezius ridge Usually intermittent, ≤ 30 min each episode but may be longer in unstable, persistent angina
Pain exacerbation	Body movements and with recumbency Inspiration	Not related to body movement or position No effect
Pain relief	No effect with nitrates	Relieved with nitrates usually
Pericardial friction	Pathognomonic, usually triphasic	Present only with pericarditis but commonly days after acute event
Murmurs	Absent unless preexisting	May be present
S1	Intact	Often dull, attenuated (after first day)
Abnormal S3	Absent unless preexisting	May be present
Abnormal S4	Absent unless preexisting	Nearly always present
Pulmonary congestion	Absent, unless (rarely) with cardiac tamponade	May be present

From Spodick DH. Pericardial diseases. In: Braunwald E, Zipes DP, Libby P, eds. *Heart Disease: A Textbook of Cardiovascular Medicine*, 6th ed. Philadelphia, PA: WB Saunders Co; 2001.

TABLE 3. Electrocardiographic Manifestations in Acute Pericarditis versus Myocardial Infarction and Early Repolarization

ECG Manifestation	Acute Pericarditis	Myocardial Infarction	Early Repolarization
PR-segment deviation	Frequent Depressed (horizontal or upsloping)	Absent	Occasional Restricted distribution
Abnormal Q wave	None, unless with infarction	Common	None, unless with infarction
Loss of R-wave voltage	Absent	Present	Absent
RS complex slurring	Uncommon	May vary	Nearly always
J-ST axis	Diffuse elevation Reciprocal depressions absent Concave upward Evolution present	Localized deviation over area of involved artery Reciprocal depressions present Convex downward No "classic" evolution but changes present and may vary	Precordial leads Reciprocal depressions absent Concave upward Absent evolution
ST/T ratio (lead V6)	Usually ≥ 0.25	Not applicable	Usually < 0.25
T wave	Normal amplitude Can have blunt summit Inverted after J point returns to baseline	May vary May vary May vary	Usually tall Usually peaked summit Inverted while ST segment still elevated
Arrhythmia	Uncommon (in absence of heart disease)	Frequent	Occasional

From Spodick DH. Pericardial diseases. In: Braunwald E, Zipes DP, Libby P, eds. *Heart Disease: A Textbook of Cardiovascular Medicine*, 6th ed. Philadelphia, PA: WB Saunders Co; 2001.

physical examination, close attention to certain key points during these stages could help narrow the differential diagnosis (Tables 2 and 3).^{1-3,18}

P Wave

The P wave in AP is almost always unchanged because, typically, few ECG changes occur during depolarization; atrial depolarization relatively unaffected in AP. However, interatrial block is common in constrictive pericarditis, especially with chronicity, and depicts widened P waves (≥ 110 msec) and are usually notched.

PR Segment

In MI and early repolarization, PR-segment depressions as seen in AP are almost always absent unless infarct pericarditis is

present. Furthermore, this quasispecific finding in AP is diffuse, affecting virtually all leads (sparing V1 and aVR).¹⁷ PR-segment deviations also display a vector (axis) that is 180° opposite to the P axis (which is usually -120 to -150°).¹

Q Waves

Q waves are absent in AP, or rarely occur as a result of, concurrent underlying myocardial injury unrelated to pericarditis.

RS Complex

The RS complex in AP is normal unless changes are preexisting. In MI, changes can occur over a wide range.

R Wave

Poor R-wave progression may be a finding in MI but is not a characteristic in AP.

ST Segment

ST-segment elevation in AP is concave-upward, whereas in (acute) MI, convex-upward segment elevations nearly always are noted. ST-segment changes are also diffuse in AP but are usually localized and territorially correspond to the coronary arterial supply involved in MI. Moreover, reciprocal changes are a feature of MI but not AP. The ST segment is almost always depressed in aVR. The mean J-ST vector (axis) in AP is usually directed left and inferiorly, between +30° and +60° in normal hearts.¹

T Wave

T wave is always concordant with ST-segment deviations on respective ECG leads in AP. Discordance during stage 1 is atypical of AP but characteristic of MI. An ST-segment to T-wave ratio <0.25, especially in lead V6, denotes early repolarization. T-wave summits can be blunt (normal amplitude) in AP but are usually peaked (tall) in early depolarization.

Voltage

Low voltage can be present in AP and is more pronounced when a pericardial effusion is present. This finding is also common in constrictive pericarditis.

Electrical Alternans

This can manifest in AP with a pericardial effusion that permits cardiac swinging virtually always with cardiac tamponade. The main mechanism of this spatial axis change of ECG deflections, particularly the QRS complex, is the oscillation of the heart cushioned in the buoyant pericardial sac.

Arrhythmias

These are uncommon in AP and, if present, commonly reflect underlying myocardial disease. However, in MI, arrhythmias are common.

TYPICAL AND ATYPICAL ELECTROCARDIOGRAPHIC VARIANTS IN ACUTE PERICARDITIS

Typical

One or more of the stages could be “missed” depending on when the ECG is done, particularly if there is rapid evolution between stages.¹ This is especially common of late with prompt recognition and use of newer, aggressive anti-inflammatory treatment in AP. ST segments in stage 1 may also be isoelectric and even depressed in lead III. Small voltage limb leads, however, may have no ST deviations. Stages 2 (late phase) and 3 could persist indefinitely or for long periods; T-wave changes indicating a more aggressive process.^{1,18}

Atypical

Extremely rapid evolution between stages may render the ECG ineffective in capturing any ST-T changes. “Absent”

ECG changes may also occur when the superficial myocarditis is very subtle or insignificant. J-ST changes may be localized to a few ECG leads and can mislead physicians into considering MI as a leading diagnosis. Discordant T waves, especially in stage 1, rarely occur in AP, but if present, mimic such changes as seen in MI. T-wave inversions could also be selective in stage 3 and occur only in some of the precordial leads.¹

A FEW FINAL WORDS

AP is a clinical diagnosis.¹⁻³ However, various circumstances may indeed make its recognition challenging and could dictate the speed as well as ease of proper diagnosis and treatment.^{1-3,18} As such, a thorough appraisal of the presenting clinical picture in its entirety is always best. The pitfalls of inappropriate interpretation of clinical cues in AP, be it subtle or atypical, can almost always be avoided if characteristic ECG findings are considered in concert with the patient’s history as well as signs and symptoms.¹⁸ A systematic approach of evaluating serial ECG manifestations and awareness of the existence of typical and atypical variants may help to differentiate AP from commonly confused conditions and rule out MI or early depolarization (Tables 2 and 3; Fig. 3).^{1-3,18} The etiology of AP, if not clearly evident, could sometimes be unmasked with laboratory blood evaluations and exclusion of specific causes (Table 1).¹ If suspicion of AP remains high or diagnosis is uncertain despite negative preliminary clinical, serial ECG, and laboratory blood evaluation findings, further investigation may be warranted for appropriate management. In such cases and especially in complicated or life-threatening AP, medical subspecialty consultation or multidisciplinary involvement could also be essential.

REFERENCES

1. Spodick DH. Pericardial diseases. In: Braunwald E, Zipes DP, Libby P, eds. *Heart Disease: A Textbook of Cardiovascular Medicine*, 6th ed. Philadelphia: WB Saunders Co; 2001:1823-1870.
2. Shabetai R. Diseases of the pericardium. In: Bennet JC, Plum F, eds. *Cecil Textbook of Medicine*, 20th ed. Philadelphia, PA: WB Saunders; 1996:336-342.
3. Roldan CA. Cardiovascular disorders. Pericardial diseases [pericarditis]. In: Beers MH, Berkow R, eds. *Merck Manual of Diagnosis and Therapy*, 17th ed, Centennial ed. West Point, PA: Merck & Co. Inc; 1999. Available at: <http://www.merck.com/mrkshared/mmanual/section16/chapter209/209b.jsp>. Accessed August 15, 2005.
4. Spodick DH. Pericardial macro- and microanatomy: a synopsis. In: Spodick DH, ed. *The Pericardium: A Comprehensive Textbook*. New York, NY: Marcel Dekker; 1997:7-14.
5. Hammond HK, White FC, Bhargava V, et al. Heart size and maximal cardiac output are limited by the pericardium. *Am J Physiol*. 1992;263:H1675-H1681.
6. Spodick DH. The normal and diseased pericardium: current concepts of pericardial physiology, diagnosis and treatment. *J Am Coll Cardiol*. 1983;1:240-251.
7. Grimm RA, Hesse B. Acute pericarditis. In: *Pericardial Disease*. 2003. Available at: <http://www.clevelandclinicmeded.com/diseaseandmanagement/cardiology/pericardial/pericardial.htm>. Accessed June 28, 2005.
8. Stein JH. Pericardial disease and pericardial heart disease. In: *Diseases of the Heart and Blood Vessels. IV. Specific Disease Entities. Internal Medicine*, 5th ed. Mosby, Inc; 1998. Available at: <http://online.statref.com/document.aspx?fxid=9&docid=459>. Accessed June 28, 2005.
9. Zayas R, Anguita M, Torres F, et al. Incidence of specific etiology and

- role of methods for specific etiologic diagnosis of primary acute pericarditis. *Am J Cardiol.* 1995;75:378–382.
10. Lange RA, Hillis LD. Clinical practice. Acute pericarditis. *N Engl J Med.* 2004;351:2195–2202.
 11. Spodick DH. Pericardial rub. Prospective, multiple observer investigation of pericardial friction in 100 patients. *Am J Cardiol.* 1975;35:357–362.
 12. Braunwald E. Pericardial disease. In: Fauci AS, Braunwald E, Isselbacher KJ, et al., eds. *Harrison's Principles of Internal Medicine*, 13th ed. New York, NY: McGraw-Hill; 1998:1334–1341.
 13. Bilchick KC, Wise RA. Paradoxical physical findings described by Kussmaul: pulsus paradoxus and Kussmaul's sign. *Lancet.* 2002;359:1940–1942.
 14. Weiss JM, Spodick DH. Association of left pleural effusion with pericardial disease. *N Engl J Med.* 1983;308:696–697.
 15. Breen JF. Imaging of the pericardium. *J Thorac Imaging.* 2001;16:47–54.
 16. Ginzton LE, Laks MM. The differential diagnosis of acute pericarditis from the normal variant: new electrocardiographic criteria. *Circulation.* 1982;65:1004–1009.
 17. Baljepally R, Spodick DH. PR-segment deviation as the initial electrocardiographic response in acute pericarditis. *Am J Cardiol.* 1998;81:1505–1506.
 18. Marinella MA. Electrocardiographic manifestations and differential diagnosis of acute pericarditis. *Am Fam Physician.* 1998;57:699–704.