ECG INTERPRETATION FOR THE EMERGENCY DEPARTMENT

Electrocardiographic ST segment elevation in adults with chest pain

William J Brady, Francis Morris

The accident and emergency (A&E) physician must be an expert in the interpretation of the electrocardiogram (ECG). Patients with chest pain and ST segment elevation on the 12 lead ECG who are experiencing acute myocardial infarction (AMI) may be candidates for urgent coronary revascularisation via either thrombolysis or primary angioplasty. Such treatment, to maximise benefit, must be delivered as early as possible after the onset of infarction. Other such patients with chest pain and electrocardiographic ST segment elevation may be suffering from a non-coronary chest discomfort syndrome. The correct identification of these patients—both clinically and electrocardiographically—must be made in order to offer the most appropriate treatments and to avoid potentially dangerous therapies. The A&E doctor is frequently the initial clinician who examines the chest pain patient, interprets the ECG, and makes these early therapeutic decisions. Accurate interpretation of the ECG and the correct diagnosis of AMI among the numerous causes of ST segment elevation is a mandatory skill. The ability of the doctor to correctly interpret the electrocardiographic findings in such patients directly and immediately impacts on management decisions as well as influences patient outcome.¹⁻⁴ The following discussion focuses on the differential diagnosis

of ST segment elevation in the patient with chest pain.

Case reports

CASE 1

A 38 year old African man presented to the A&E department with central chest pain. The pain had started at rest and was associated with sweating. No past history nor family history of ischaemic heart disease was noted. Examination revealed a harsh systolic murmur at the left sternal edge. The ECG (fig 1) was initially interpreted as demonstrating ST segment elevation in leads V1-V3, compatible with AMI. The patient was prescribed thrombolysis (streptokinase 1.5 megaunits) and referred to the coronary care unit. Subsequent evaluation revealed that he had a significant aortic stenosis. No evidence of AMI was found. Repeat ECGs did not reveal any evolving changes and cardiac enzymes were never raised. He had no further chest pain. The initial ECG was reinterpreted as demonstrating evidence of left ventricular hypertrophy. The ST segment elevation in leads V1-V3 was thought to be entirely compatible with this diagnosis and not suggestive of an AMI.

Department of
Emergency Medicine,
University of Virginia
School of Medicine,
Charlottesville,
Virginia, USA
W J Brady

Department of
Accident and
Emergency Medicine,
Northern General
Hospital, Sheffield
F Morris

Correspondence to:
Dr William J Brady,
Department of Emergency
Medicine, Box 523-21,
University of Virginia Health
Sciences Center,
Charlottesville, VA 22908,
USA (e-mail:
wb4z@hscmail.mcc.
virginia.edu).

Accepted 21 August 1999

Figure 1 Left ventricular hypertrophy: normal sinus rhythm with the cardinal features of left ventricular hypertrophy. The ST segment elevation apparent in leads V1-V2 is compatible with the repolarisation changes of left ventricular hypertrophy.

Figure 2 Benign early repolarisation (BER): normal sinus rhythm with ST segment elevation in leads V2–V4. The ST segment elevation is concave upwards and, in lead V3, a notch is evident at the J point. The ECG changes did not evolve over time. The final electrocardiographic diagnosis is BER.

CASE 2

A 48 year old man presented in the early hours of the morning with retrosternal burning discomfort. The pain had started six hours previously after a large meal and was unrelieved by antacids. The pain had prevented him from sleeping and he was particularly uncomfortable in bed. There was no significant past medical history apart from an acute attack of gout three months previously. Clinical examination was normal. The ECG (fig 2) was thought to demonstrate ST segment elevation compatible with an anterior AMI and he was referred to coronary care for thrombolysis. On further evaluation, it was thought that the ST segment elevation was a manifestation of benign early repolarisation (BER) rather than AMI. Serial ECGs did not reveal any evolution of the ST segment elevation and there was no cardiac enzyme rise. A stress test seven days after admission did not reveal any significant ischaemia.

CASE 3

A 64 year old woman presented to the A&E department with substernal chest pain, similar to previous attacks of angina. The pain was associated with nausea and sweating. She had a past history of AMI, diabetes, and hypertension. A 12 lead ECG showed sinus rhythm with ST segment elevation in leads III and aVF consistent with AMI (fig 3). The patient was referred to the coronary care unit for thrombolysis, which was applied without complication or apparent change in the patient's condition. Further study of her medical history revealed past inferior wall AMI with aneurysm. Serial ECGs did not demonstrate change and cardiac enzymes did not rise; an echocardiogram revealed inferior dyskinesia consistent with inferior wall aneurysm.

CASE 4

A 43 year old female without a past medical history presented with left chest pain. The pain

Figure 3 Left ventricular aneurysm: normal sinus rhythm with ST segment elevation in leads III and aVF. Comparison with serial ECGs as well as a previous ECG did not demonstrate any change. Review of past medical records revealed a history of inferior wall AMI with aneurysm.

Figure 4 Acute pericarditis: widespread ST segment elevation is seen in leads V2 to V6 as well as the inferior leads. The initial up-sloping portion of the ST segment is concave, suggesting a non-infarction cause of the electrocardiographic abnormality. Additionally, PR segment depression is seen in leads II, III, and aVF. Reciprocal changes are noted in lead aVR: PR segment elevation and ST segment depression.

had appeared approximately two to three days before arrival; the pain was worsened upon inspiration, assuming the supine position, and with upper extremity movement; no other associated symptoms were noted. The patient had recently experienced an upper respiratory infection. The physical examination was normal; no chest wall tenderness was found. An ECG (fig 4) revealed normal sinus rhythm with widespread ST segment elevation. The patient's pain lessened considerably on treatment with a non-steroidal anti-inflammatory agent and morphine sulphate. The patient was admitted to the cardiology service for 24 hour observation and discharged without incident.

Discussion

In the evaluation of patients with chest pain, the A&E doctor uses three principle tools: the history of the event, the 12 lead ECG, and serum markers of myocardial injury. The clinical history is the most vital assessment tool, yet the ECG is an important adjunct and may influence clinical management. ST segment elevation is perhaps the “most demanding” of the electrocardiographic features seen in the chest pain patient; it is “demanding” in that its presence must be explained and, if the aetiology involves AMI, urgent therapeutic decisions must be made.

Unfortunately, ST segment elevation is a not uncommon finding on the ECG of the patient with chest; its cause infrequently involves AMI. In fact, such patients may present electrocardiographically with ST segment elevation because of either AMI, confounding patterns, and/or masquerading syndromes. In most instances, ST segment elevation resulting from AMI is easily noted. Confounding patterns such as left bundle branch block, ventricular paced rhythms, and left ventricular hypertrophy may obscure the typical electrocardiographic findings of AMI as well as produce non-infarctional ST segment elevation which may lead the uninformed doctor astray. Other ST segment elevation patterns, including BER and acute pericarditis, occur in the individual with chest discomfort and may suggest the incorrect diagnosis of AMI. Refer to fig 5 for a depiction of ST segment elevation types for various clinical syndromes and tables 1 and 2 for a listing of these syndromes.

The occurrence of numerous other non-infarctional ST segment elevation syndromes only reinforces the point that ST segment elevation is an insensitive marker of AMI.⁵ One pre-hospital study of adult chest pain patients in North America demonstrated that the majority of patients manifesting ST segment elevation on the ECG did not have AMI as a

Table 1 Clinical syndromes in the chest pain patient which may cause ST segment elevation

Acute myocardial infarction
Variant angina
Acute pericarditis
Benign early repolarisation
Left ventricular hypertrophy
Bundle branch block
Cardiomyopathy
Acute myocarditis
Left ventricular aneurysm
Pre-excitation syndromes
Hyperkalaemia
Hypothermia
Central nervous system events
Acute abdominal disorder

Figure 5 Electrocardiographic syndromes presenting with ST segment elevation (AMI = acute myocardial infarction; BER = benign early repolarisation; BBB = bundle branch block; LVH = left ventricular hypertrophy).

Table 2 Causes of ST segment elevation among patients with chest pain in the emergency department: 902 patients presented with chest pain; 202 (22%) patients showed ST segment elevation on the ECG; the responsible aetiologies are listed below^a

Syndrome	Patients (%)
Left ventricular hypertrophy	25
Left bundle branch block	15
Acute myocardial infarction	15
Benign early repolarisation	12
Right bundle branch block	5
Intraventricular conduction delay	5
Aneurysm	3
Pericarditis	2
Undefined	18

Figure 6 ECG demonstrating left ventricular hypertrophy related repolarisation changes, particularly ST segment elevation in leads V1–V3 with ST segment depression in the lateral distribution. Prominent ST segment elevation is seen in the right to mid-precordial leads; the initial up-sloping portion of the ST segment is concave, which suggests a non-infarction cause of the ST segment change.

Figure 7 (A) and (B) Right precordial leads with pronounced ST segment elevation and concave ST segment morphology. (C) The concept of appropriate discordance in the patient with left ventricular hypertrophy who has had an ECG states that the terminal portion of the QRS complex (arrowed B) and ST segment/T wave (arrowed A) are located on opposite sides of the isoelectric baseline. In the right to mid-precordial leads, QS complexes (predominantly negative QRS complexes), pronounced ST segment elevation is encountered as predicted by the concept of appropriate discordance. The initial, up-sloping portion of the ST segment/T wave is concave (small arrows), which suggests a non-infarction cause of the ST segment elevation.

final hospital diagnosis; rather, left ventricular hypertrophy and left bundle branch block accounted for the majority of the cases.⁶ Further, in a North American review of adult patients presenting to the emergency department with chest pain and ST segment elevation on the ECG, ST segment elevation resulted from AMI in only 15% of this population; left ventricular hypertrophy, seen in 30% of adult chest pain patients, was the most frequent cause of this ST segment elevation (W J Brady. Abstract presented at International Emergency Medicine Conference, Vancouver, Canada, 26 March 1998). In the coronary care unit population, Miller *et al* demonstrated that ST segment elevation was diagnostic for acute infarct in only half of patients with a past history of ischaemic heart disease with such ST segment changes.⁷ These syndromes causing ST segment elevation not related to AMI are not infrequently misdiagnosed as acute infarction, which then may subject the patient to unnecessary and potentially dangerous treatments and procedures. For example, a report by Sharkey *et al* noted that 11% of patients receiving thrombolytic agent were not experiencing AMI. The electrocardiographic syndromes producing this pseudoinfarct ST segment elevation included BER (30%), left ventricular hypertrophy (30%), and various intraventricular conduction abnormalities (30%).⁸

LEFT VENTRICULAR HYPERTROPHY

Electrocardiographic changes related to left ventricular hypertrophy are uncommonly encountered in the A&E chest pain patient. ST segment elevation is seen frequently in patients with pre-hospital chest pain and, in fact, is the leading cause of such non-infarctional ST segment change.⁶ Among patients with chest discomfort, left ventricular hypertrophy is responsible for approximately 25% of non-infarctional ST segment elevation (W J Brady. Abstract presented at International Emergency Medicine Conference, Vancouver, Canada, 26 March 1998). Its presence on the ECG, particularly the repolarisation changes that alter the morphology of the ST segment and/or the T wave, may confound the early evaluation of the chest pain patient. Larsen *et al* have shown that the electrocardiographic pattern consistent with left ventricular hypertrophy is encountered in approximately 10% of adult chest pain patients initially diagnosed with acute ischaemic heart disease.⁹ After hospital admission and more extensive evaluation, only one quarter of these patients were found to have AMI. The remainder of the patients—the vast majority—were ultimately diagnosed with non-ischaemic syndromes. The physicians caring for the patients in this study incorrectly interpreted the ECGs more than 70% of the time. They frequently did not identify the left ventricular hypertrophy pattern and therefore attributed the ST segment/T wave changes to ischaemia or infarction—when in fact the observed ST segment/T wave changes resulted from repolarisation abnormality associated with left ventricular hypertrophy.⁹

Figure 8 Various electrocardiographic complexes demonstrating BER. In each case, ST segment elevation is seen with prominent J point elevation and concavity of the ST segment.

In patients with left ventricular hypertrophy, ST segment/T wave changes are encountered in approximately 70% of cases, resulting from altered repolarisation of the ventricular myocardium.¹⁰ These ST segment/T wave abnormalities are the new "norm" in patients with electrocardiographic left ventricular hypertrophy and may mask and/or mimic the early findings consistent with acute coronary ischaemia.¹¹ Left ventricular hypertrophy is associated with poor R wave progression and loss of the septal R wave in the right to mid-precordial leads, most commonly producing a QS pattern. In general, these QS complexes are located in leads V1 and V2, rarely extending beyond lead V3 (figs 1, 6, and 7A, B). As predicted by the concept of appropriate discordance (that is, the terminal portion of the QRS complex and ST segment/T wave are located on opposite sides of the isoelectric baseline; fig 7C), ST segment elevation is encountered in this distribution along with prominent T waves. The ST segment elevation seen in this distribution may be greater than 5 mm in height (figs 6 and 7A, B). The initial, up-sloping portion of the ST segment/T wave complex is frequently concave in left ventricular hypertrophy compared with the either flattened or convex pattern observed in the AMI patient (fig 7C).¹¹ This morphological feature is imperfect; early AMI may reveal such a concave feature. Another feature of left ventricular hypertrophy related ST segment/T wave change is its relative permanence; these changes are very unlikely to change during the initial evaluation. This relative difference in the longevity of the ST segment/T wave changes in the two syndromes lends itself to differentiation using serial electrocardiographic monitoring.

Figure 9 A comparison of ST segment elevation in (A) pericarditis, (B) BER, and (C) AMI. (A) Acute pericarditis is seen here with STE and PR segment depression. (B) BER with ST segment elevation starting at the J point. (C) AMI with convex ST segment elevation. Note the concave nature of the ST segment elevation in acute pericarditis and BER compared with the convex elevation in AMI.

BENIGN EARLY REPOLARISATION

The syndrome of BER, first described in 1936 by Shipley and Hallaran, is felt to be a normal variant, not indicative of underlying cardiac disease.¹² BER has been reported to occur in approximately 1% of the general population, in persons of all age groups and varying racial and ethnic backgrounds.¹³ In a large population based study, the mean age of patients with BER was 39 years with a range of 16–80 years. BER was seen predominantly in those patients less than age 50 years and rarely encountered in individuals over age 70 years (3.5%).¹³ Men manifest BER significantly more often than women.¹³ For unknown reasons, BER is more often encountered in black men age 20–40 years,¹⁴ though some dispute this finding.¹³

An ECG of BER (figs 2, 8, and 9B) includes diffuse or widespread ST segment elevation (fig 2), upward concavity of the initial portion of the ST segment (figs 2, 8, and 9B), notching or slurring of the terminal QRS complex (fig 2; lead V3), and symmetric, concordant T waves of large amplitude (figs 9, upper and middle examples, and 9B).¹⁵ The ST segment elevation begins at the "J" (or junction point of the QRS complex and ST segment). The degree of ST segment elevation encountered in BER is usually less than 2 mm but may approach 5 mm in certain individuals. Eighty to ninety per cent of individuals show ST segment elevation less than 2 mm in the precordial leads and less than 0.5 mm in the limb leads; only 2% of cases of BER manifest ST segment elevation greater than 5 mm.^{13 16} The ST segment looks like it has been lifted upwards evenly from the isoelectric baseline at the J point, preserving the normal concavity of the initial, up-sloping portion of the ST segment/T wave complex (figs 2, 8, and 9B).¹⁵ This ST segment elevation morphology is a very important feature that distinguishes BER related ST segment elevation from that associated with AMI. The J point itself is frequently notched or irregular in contour and is considered highly suggestive—but not diagnostic—of BER (fig 2; lead V3).^{13 15} The degree of J point elevation is usually less than 3.5 mm.¹³ Prominent T waves of large amplitude and slightly asymmetric morphology are also encountered; the T waves may appear "peaked," suggestive of the hyperacute T wave encountered in AMI. The prominent T waves are concordant with the QRS complex and are usually found in the precordial leads. The height of the T waves in BER ranges from approximately 6.5 mm in the precordial distribution to 5 mm in the limb leads.^{13–15}

The degree of ST segment elevation in BER is usually greatest in the mid to left precordial leads (leads V2–V5), and less often found in the limb leads. One large series reported that the limb leads show ST segment elevation in only 45% of cases of BER. "Isolated" BER in the limb leads (no ST segment elevation in the precordial leads) is a very rare finding.^{13 16} Such "isolated" ST segment elevation in the inferior (II, III, and aVF) or lateral (I and aVL) leads should prompt consideration of another explanation for STE.

Figure 10 Left ventricular aneurysm in lead V2 with concave ST segment elevation.

LEFT VENTRICULAR ANEURYSM

Left ventricular aneurysm (LVA), also described as dyskinetic ventricular segment, is defined as a localised area of infarcted myocardium which bulges outward during both systole and diastole. LVAs most often are noted after large anterior wall events but may also be encountered after inferior and posterior wall AMIs. In most cases, the LVA is manifested electrocardiographically by varying degrees of ST segment elevation, which may be difficult to distinguish from ST segment changes due to AMI—particularly in the patient with chest pain with known past AMI.¹¹ The actual ST segment elevation itself is felt to result from either a current of injury originating from viable yet ischaemic myocytes in the aneurysm or from mechanical wall stress caused by traction on the normal, adjacent myocardium.

LVA is characterised electrocardiographically by persistent ST segment elevation seen several weeks after AMI. Because of the frequent anterior location of LVA, ST segment elevation is most often observed leads I, aVL, and V1–V6. Of course, the inferior wall LVA would be manifested on the surface ECG by ST segment elevation in the inferior leads as seen in case 3 (fig 3); such ST segment elevation, however, is usually less pronounced than the ST segment changes seen in the anterior leads. The actual ST segment abnormality due to the LVA may present with varying morphologies, ranging from obvious, convex ST segment elevation (fig 3) to minimal, concave elevations (fig 10). The distinction from ST segment elevation in the AMI patient may be difficult.¹¹ Reciprocal change, which is a not infrequent electrocardiographic feature of AMI, strongly suggests that the current ST segment changes results at least in part from acute infarction. Patients with LVA, usually the result of extensive anterior wall infarction, will frequently also have significant Q waves in the same distribution. The simultaneous appearance of the Q wave is not a feature that unquestionably supports the electrocardiographic diagnosis of LVA; the doctor must recall that Q waves may appear as early as two hours after the onset of transmural AMI. The dynamic nature of AMI related ST segment elevation compared with the often static ST

segment character in LVA makes both a comparison with past ECGs as well as the use of serial ECGs of considerable diagnostic value.

ACUTE PERICARDITIS

Acute pericarditis, a diffuse inflammation of the pericardial sac and superficial myocardium, has a number of underlying causes including infections, immunological disorders, uraemia, trauma, malignancy, cardiac ischaemia, AMI, as well as a multitude of other aetiologies. The clinical presentation of acute pericarditis can be confused with acute coronary ischaemia. The difficulty in distinguishing acute myopericarditis from AMI, coupled with an increasing emphasis on rapid revascularisation treatments in patients with acute infarction, has led to inadvertent administration of thrombolytic agents to patients with acute pericarditis.^{17 18}

It has been reported that up to 90% of patients with acute myopericarditis have abnormalities on the ECG.¹⁹ Classically, several stages of electrocardiographic change have been described involving ST segment elevation, PR segment depression, T wave inversion, and normalisation.¹¹ In general, stages 1–3 are seen over hours to days but stage 4 may not be reached for several weeks. Further, patients may not manifest all characteristic electrocardiographic features of pericarditis—that is, the chest pain patient may present with stage 1 findings with progression directly to stage 4. Fewer than half of the patients with acute myopericarditis, however, evolve through all stages.²⁰

ST segment elevation seen in patients with stage 1 pericarditis is usually less than 5 mm in height, observed in numerous leads, and characterised by a concavity to its initial up-sloping portion (fig 4). In some instances, the ST segment elevation may actually be obliquely flat (inferior leads of fig 4). The ST segment elevation caused by acute pericarditis is usually noted in all leads except V1 (fig 4); reciprocal ST segment depression is also seen in lead aVR (fig 4) and occasionally in lead V1. The ST segment elevation is most often seen in many leads simultaneously, though it may be limited to a specific anatomic segment. If the process is focal, the inferior wall is often involved with leads II, III, and aVF commonly affected.

PR segment depression associated with pericarditis perhaps is the most helpful feature in arriving at the correct electrocardiographic diagnosis; such a finding has been described as “almost diagnostic” for acute pericarditis (figs 4 and 11; upper and lower examples).¹¹ Reciprocal PR segment elevation is seen in lead aVR—and may be easier to view in certain cases (fig 10).

OTHER SYNDROMES

Numerous other clinical syndromes may present with electrocardiographic ST segment elevation, including the various ventricular paced rhythms, cardiomyopathies, acute myocarditis, hypothermia, hyperkalaemia, pre-excitation syndromes, and acute abdominal

Figure 11 Acute pericarditis with ST segment elevation in all three examples (upper lead III (left), middle lead V2 (middle), and lower lead V4 (right)) as well as PR segment depression in the upper (left) and lower (right) examples. Once again, note the concave morphology of the ST segment elevation, suggesting a non-infarction cause of the ST segment change. The shaded areas refer to the concave portion of the ST segment; the arrows indicate the PR segment depression.

and central nervous system disorders. These syndromes will be discussed in future reports; left bundle branch block has been discussed in an earlier report.

Conflict of interest: none.

Funding: none.

- 1 Muller DW, Topol EJ. Selection of patients with acute myocardial infarction for thrombolytic therapy. *Ann Intern Med* 1990;113:949–60.
- 2 Kleiman NS, White HD, Ohman EM, *et al*. Mortality within 24 hours of thrombolysis for myocardial infarction: the importance of early reperfusion. *Circulation* 1994;90:2658–65.
- 3 Lee TH, Weisberg MC, Brand DA, *et al*. Candidates for thrombolysis among emergency room patients with acute chest pain: potential true and false-positive rates. *Ann Intern Med* 1989;110:957–62.
- 4 GUSTO Investigators. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. *N Engl J Med* 1993;329:673–82.
- 5 Rude RE, Poole WK, Muller JE, *et al*. Electrocardiographic and clinical criteria for recognition of acute myocardial infarction based on analysis of 3,697 patients. *Am J Cardiol* 1983;52:936–42.
- 6 Otto LA, Aufderheide TP. Evaluation of ST segment elevation criteria for the prehospital electrocardiographic diagnosis of acute myocardial infarction. *Ann Emerg Med* 1994;23:17–24.
- 7 Miller DH, Kligfield P, Schreiber TL, *et al*. Relationship of prior myocardial infarction to false-positive electrocardiographic diagnosis of acute injury in patients with chest pain. *Arch Intern Med* 1987;147:257–61.
- 8 Sharkey SW, Berger CR, Brunette DD, *et al*. Impact of the electrocardiogram on the delivery of thrombolytic therapy for acute myocardial infarction. *Am J Cardiol* 1994;73:550–3.
- 9 Larsen GC, Griffith JL, Beshansky JR, *et al*. Electrocardiographic left ventricular hypertrophy in patients with suspected acute cardiac ischemia—its influence on diagnosis, treatment, and short-term prognosis. *J Gen Intern Med* 1994;9:666–73.
- 10 Huwez FU, Pringle SD, Macfarlane FW. Variable patterns of ST-T abnormalities in patients with left ventricular hypertrophy and normal coronary arteries. *Br Heart J* 1992;67:304–7.
- 11 Aufderheide TP, Brady WJ. Electrocardiography in the patient with myocardial ischemia or infarction. In: Gibler WB, Aufderheide TP, eds. *Emergency cardiac care*. 1st Ed. St Louis: Mosby, 1994: 169–216.
- 12 Shipley RA, Hallaran WR. The four-lead electrocardiogram in two hundred normal men and women. *Am Heart J* 1936;11:325–45.
- 13 Mehta MC, Jain AC. Early repolarization on scalar electrocardiogram. *Am J Med Sci* 1995;309:305–11.
- 14 Thomas J, Harris E, Lassiter G. Observations on the T wave and S-T segment changes in the precordial electrocardiogram of 320 young negro adults. *Am J Cardiol* 1960;5:468–74.
- 15 Wasserburger RM, Alt WJ, Lloyd C. The normal RS-T segment elevation variant. *Am J Cardiol* 1961;8:184–92.
- 16 Kabara H, Phillips J. Long-term evaluation of early repolarization syndrome (normal variant RS-T segment elevation). *Am J Cardiol* 1976;38:157–61.
- 17 Millaire A, De Groote P, Decoulx E, *et al*. Outcome after thrombolytic therapy of nine cases of pericarditis misdiagnosed as myocardial infarction. *Eur Heart J* 1995;16:333–6.
- 18 Khoury NE, Borzak S, Gokli A, *et al*. Inadvertent thrombolytic administration in patients without myocardial infarction: clinical features and outcome. *Ann Emerg Med* 1996;28:289–96.
- 19 Shabetai R. Acute myopericarditis. *Cardiol Clin* 1990;8:639–81.
- 20 Marinella MA. Electrocardiographic manifestations and differential diagnosis of acute myopericarditis. *Am Fam Physician* 1990;57:699–705.