

Electrocardiographic Manifestations of Wellens' Syndrome

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Wellens' syndrome is a pattern of electrocardiographic T-wave changes associated with critical, proximal left anterior descending (LAD) artery stenosis. The syndrome is also referred to as LAD coronary T-wave syndrome. Syndrome criteria include T-wave changes plus a history of anginal chest pain without serum marker abnormalities; patients lack Q waves and significant ST-segment elevation; such patients show normal precordial R-wave progression. The natural history of Wellens' syndrome is anterior wall acute myocardial infarction. The T-wave abnormalities are persistent and may remain in place for hours to weeks; the clinician likely will encounter these changes in the sensation-free patient. With definitive management of the stenosis, the changes resolve with normalization of the electrocardiogram. It is vital that the physician recognize these changes and the association with critical LAD obstruction and significant risk for anterior wall myocardial infarction. (*Am J Emerg Med* 2002;20:638-643. Copyright 2002, Elsevier Science (USA). All rights reserved.)

When evaluating patients in the emergency department (ED), emergency physicians (EP) must be capable and proficient in the interpretation of the electrocardiogram (ECG). Among the many electrocardiographic findings indicative of acute coronary syndromes, the EP must be familiar with the characteristics of the preinfarction stage of coronary artery disease known as Wellens' syndrome. Wellens' syndrome consists of T-wave abnormalities in the anterior chest leads. These changes are associated with significant obstruction in the proximal left anterior descending (LAD) coronary artery. Patients with Wellens' syndrome are at high risk for the development of an extensive myocardial infarction of the anterior wall and death.¹ Although medical management may provide symptomatic improvement at first, the natural history of this syndrome is anterior wall myocardial infarction that, if not aborted, results in significant left ventricular dysfunction and/or

death. Once Wellens' syndrome has been recognized, urgent coronary angiography—not stress imaging of any sort—is required to evaluate the need for early angioplasty or coronary bypass surgery.² This article discusses the electrocardiographic findings suggestive of this potentially fatal condition.

CASE PRESENTATIONS

Case 1

A 49-year-old man presented to the ED with chest pain of 4 hours' duration that was associated with diaphoresis. Emergency medical technicians treated the patient with nitrates, morphine, and aspirin; he was sensation-free on arrival to the ED. The examination was unremarkable. The 12-lead ECG (Fig 1), performed when the patient was free of discomfort, showed deeply inverted T waves in leads V2 to V6; the ST segments were minimally elevated and convex in morphology in leads V2 to V4. The electrocardiographic pattern in a pain-free patient raised the possibility of LAD coronary T-wave syndrome. Serum markers were negative for myocardial infarction. The patient was admitted to a cardiology service for cardiac catheterization, which revealed a 95% proximal LAD artery occlusion. Angioplasty with stent placement was performed successfully.

Case 2

A 54-year-old woman presented to the ED with chest pain associated with diaphoresis and nausea. The examination was remarkable for diaphoresis. Before performing the ECG the patient's pain resolved. The ECG (Fig 2) showed biphasic T-wave inversions in leads V2 to V4; the T waves were biphasic in leads V2 and V3. She remained pain free in the ED; the initial serum marker was normal. She was admitted to the coronary care unit where the pain recurred approximately 6 hours later, associated with the development of obvious ST-segment elevation in leads V2 to V4 consistent with acute myocardial infarction (AMI). She received a thrombolytic agent with reperfusion confirmed by resolution of the pain and the ST-segment elevation. Cardiac catheterization revealed a near-complete proximal LAD lesion that was successfully opened and stented.

Case 3

A 54-year-old man presented via ambulance to the ED with chest pain. Examination revealed diaphoresis. He was

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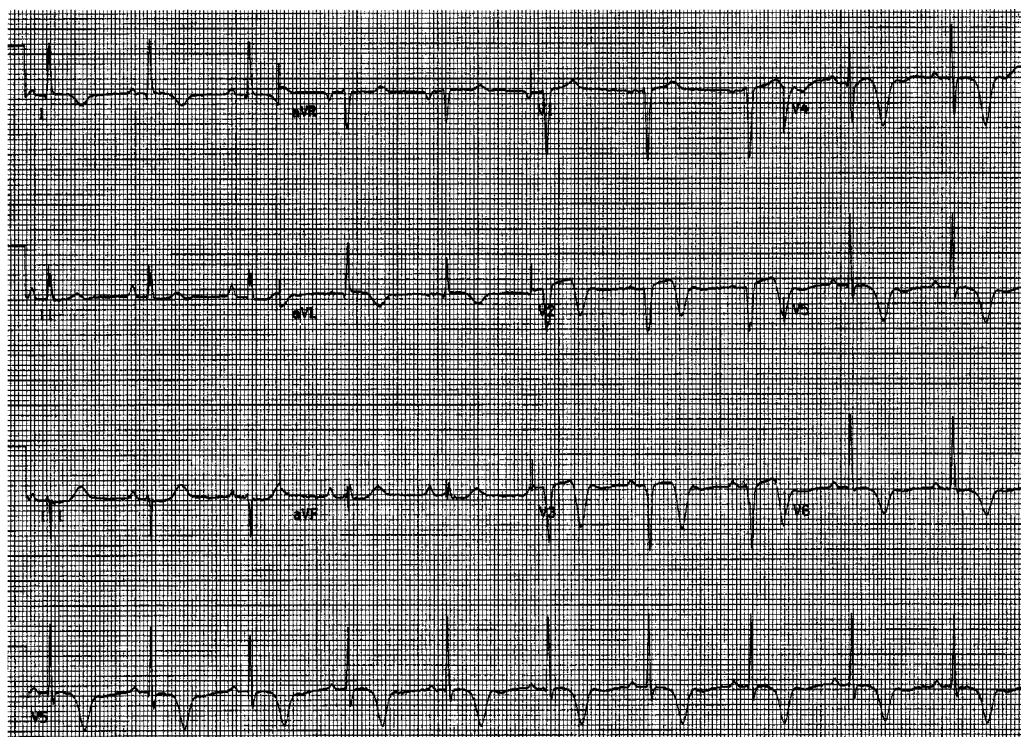
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FIGURE 1. Case 1, normal sinus rhythm with inverted T waves in leads V2 to V6. The T waves are deeply inverted. Note the abrupt angle of the descending limb of the T wave, accounting for its marked negative amplitude (ie, deeply inverted). Also note the minimal ST-segment elevation in leads V2 to V4 with a convex contour.



given nitrates and morphine with resolution of his pain. At this point, an ECG (Fig 3) showed normal sinus rhythm with biphasic T-wave inversions in the anterior distribution (leads V1-V4). The initial serum marker was negative for myocardial infarction. The patient's primary care physician examined the patient in the ED; a stress test was performed. Approximately 1 minute into the test, the patient developed severe pain in the midchest accompanied by diaphoresis and dizziness. The ECG

revealed ST-segment elevation in the anterior leads consistent with anterior wall AMI (Fig 4). He was taken to the catheterization laboratory where a proximal LAD lesion was found and successfully opened via angioplasty.

DISCUSSION

de Zwann et al¹ first described a subgroup of patients hospitalized for unstable angina that were at high risk for

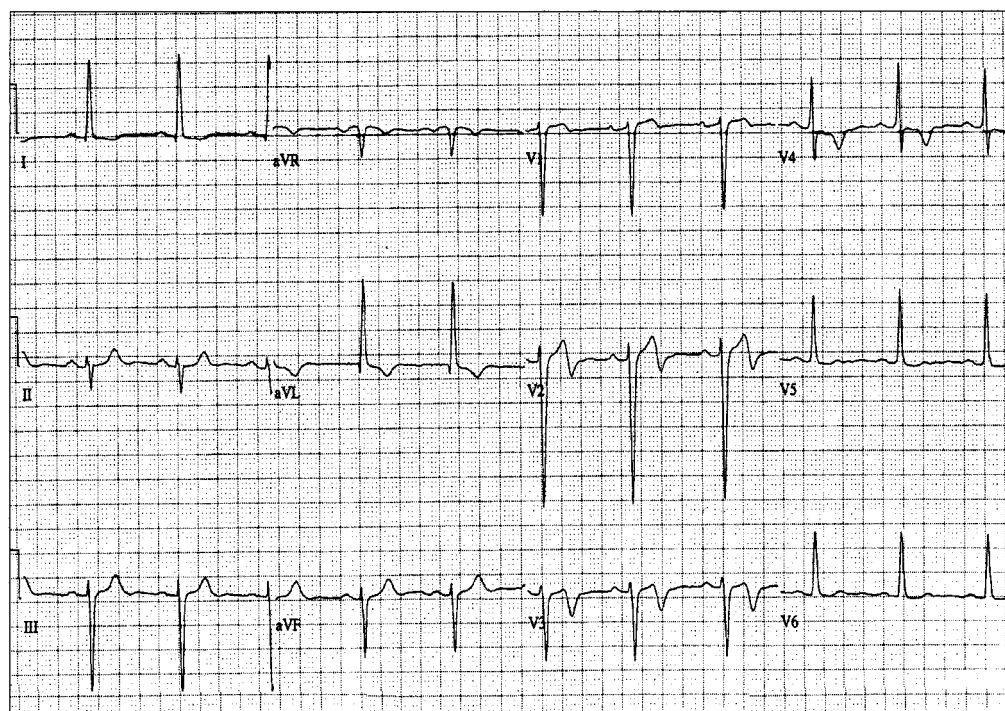


FIGURE 2. Case 2, normal sinus rhythm with biphasic T-wave inversions in leads V2 and V3 as well as T-wave inversion in lead V4. The ST segment is also elevated in leads V2 and V3 with a concave morphology.

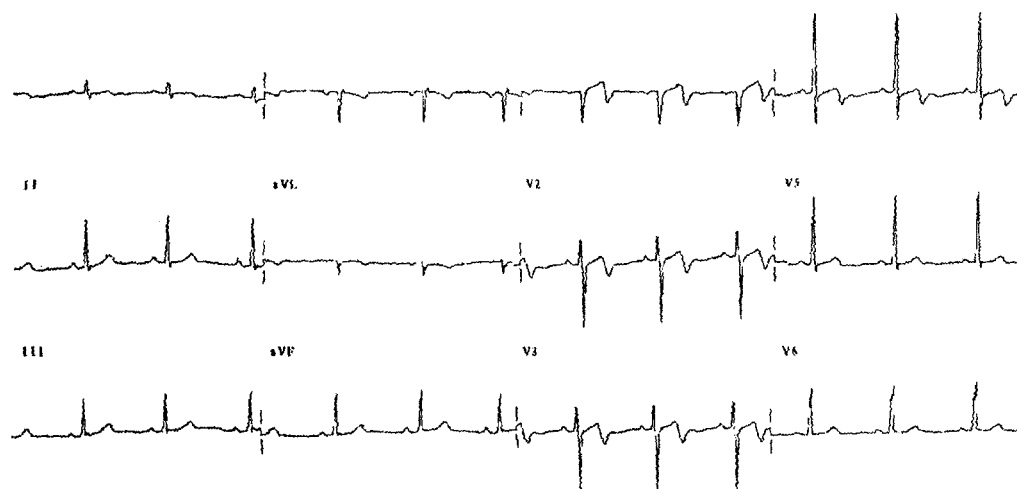


FIGURE 3. Case 3, normal sinus rhythm with biphasic T-wave inversions in leads V1 to V4 and concave ST-segment elevation.

the development of an anterior wall myocardial infarction. This subgroup could be recognized by characteristic ST-segment–T-wave changes in the precordial leads. These changes involve the T wave with occasional involvement of the ST segment. Two basic patterns of electrocardiographic change are encountered: (1) isoelectric or minimally elevated (ie, less than 1 mm) ST segment with a straight or convex morphology that leads into a negative (inverted) T wave at an angle of 60° to 90° (Figs 1 and 5);² (2) biphasic T waves in the right to midprecordial leads (Figs 2, 3, and 5).³

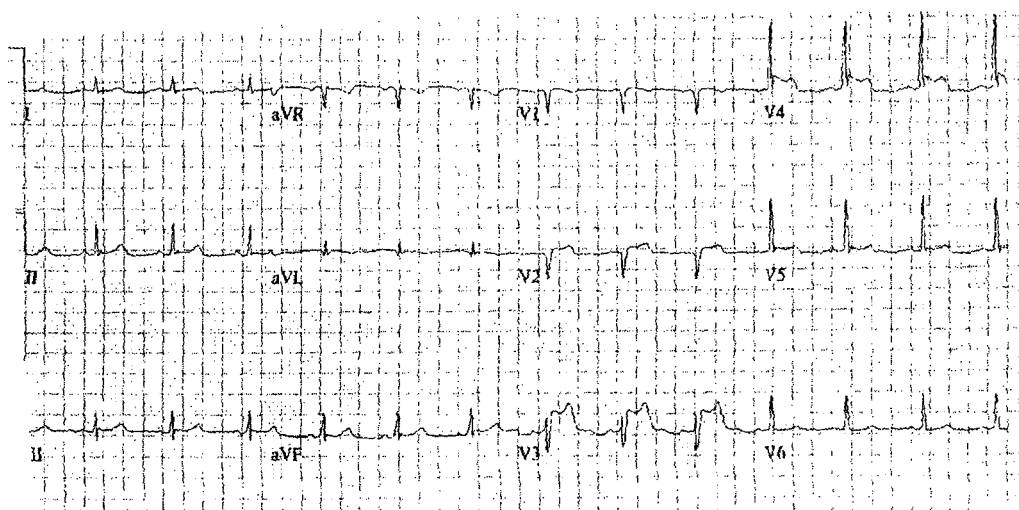
In Wellens' first study, 26 of 145 patients admitted for unstable angina (18%) had this electrocardiographic pattern.¹ In a second prospective study, 180 of 1,260 hospitalized patients (14%) showed the characteristic electrocardiographic changes.² Furthermore, all of these patients had significant disease of the proximal LAD. In the first study, 12 of 16 patients (75%) with electrocardiographic changes who did not receive coronary revascularization developed an extensive anterior wall infarction within a few weeks after admission.¹ In the second study, urgent coronary angiography was implemented, and all of the 180 patients with

electrocardiographic changes were found to have blockage of the LAD, varying from 50% to complete obstruction.²

Early detection of these electrocardiographic changes is also important because of the clinical presentation of a patient with Wellens' syndrome. The characteristic electrocardiographic pattern often develops when the patient is not experiencing angina. In fact, during an attack of chest pain, the ST-segment–T-wave abnormalities usually normalize or develop into ST-segment elevation.² Cardiac serum markers are often normal or minimally elevated. In Wellens' prospective study, only 21 of 180 patients (12%) with electrocardiographic changes had elevated cardiac enzymes. These elevations were always less than twice the upper limit of normal.² Therefore, the ECG may be the only indication of an impending extensive anterior wall myocardial infarction in an otherwise asymptomatic patient—as seen in Case 3 (Figs 3 and 4).

The electrocardiographic findings include significant involvement of the T wave with occasional alterations of the ST segment. The ST segment itself is often normal (ie, isoelectric); if abnormal, it is minimally elevated, usually less than 1 mm with a high take-off of the ST segment from the QRS complex. If the ST segment is elevated, it is either

FIGURE 4. Case 3, acute ST-segment elevation myocardial infarction of the anterior wall.



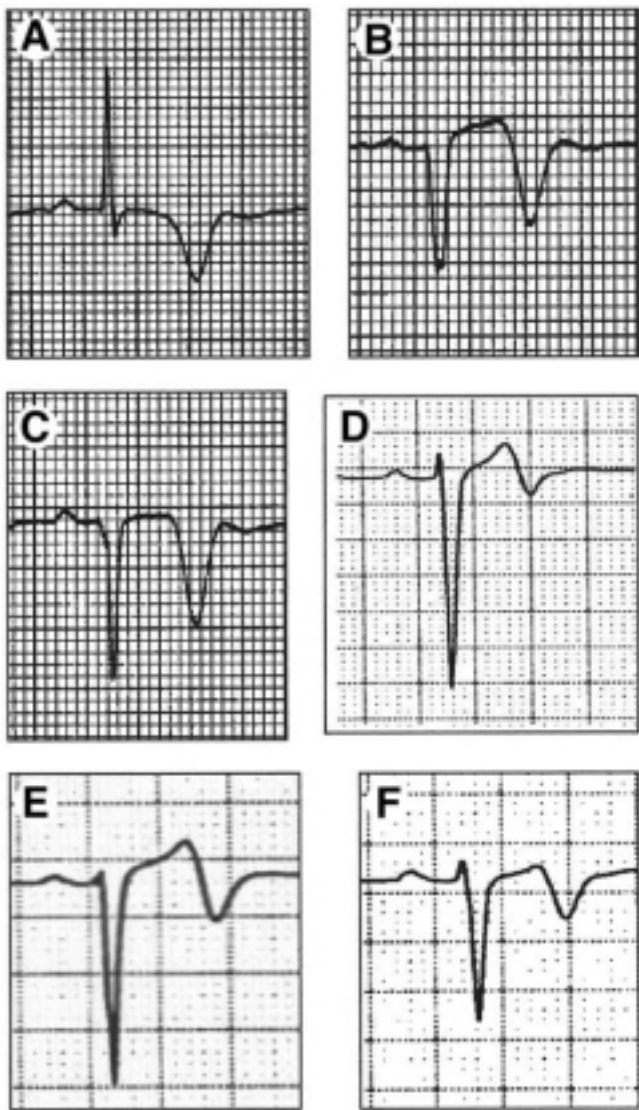


FIGURE 5. The T-wave inversions of Wellens' syndrome, the more common pattern of (A-C) deeply inverted T wave and the (D-F) less common biphasic T wave. (B-F) ST-segment elevation is present.

convex in contour (Figs 1 and 5) or obliquely straight in appearance (Figs 1 and 3); concave morphologies are also seen (Figs 2, 3, and 5).¹ T-wave findings, the key features of this electrocardiographic syndrome, may take the form of 1 of 2 patterns of T-wave changes. In the more common pattern, which comprises approximately 75% of cases, the T wave is deeply inverted (Figs 1 and 5A-5C). As the ST segment terminates, the T wave assumes a very negative angle relative to the isoelectric baseline; this angle may approach 90°. The inverted T wave is symmetric in contour. The less common variant, comprising 25% of Wellens' syndrome cases, presents with biphasic T waves (Figs 2, 3, and 5D-5E).^{1,2}

The ST-segment and T-wave changes are present in leads V2 and V3; in certain cases, the changes may also involve leads V1 and V4. In Wellens' prospective study, approxi-

mately two thirds of patients also had these changes in lead V1 and three quarters in lead V4. Patients with abnormalities in lead V4 occasionally will show similar abnormalities in leads V5 or V6.² The electrocardiographic pattern often develops when the patient is not experiencing angina, and the ST-segment-T-wave abnormalities usually normalize or develop into ST-segment elevation during an attack of chest pain. In Wellens' study, 60% of patients diagnosed with Wellens' syndrome had the characteristic electrocardiographic changes on admission. After admission, 56 (31%) developed the changes within 24 hours, 10 (5%) within 2 days, 5 (2.8%) within 3 days, and 1 (0.6%) within 5 days.²

Electrocardiographic T-wave inversions may be related to a number of clinical syndromes, ranging from life-threatening events such as acute coronary ischemia, pulmonary embolism, and central nervous system injury, to entirely benign presentations such as persistent juvenile T-wave pattern and the digitalis effect. The list of differential possibilities of the inverted T wave is quite extensive, including acute coronary ischemia (non-Wellenoid ischemia, Wellens' presentation, and non-ST-segment elevation AMI), past myocardial infarction, persistent juvenile T-wave pattern, left ventricular hypertrophy, bundle branch block, digitalis effect, acute myocarditis, pre-excitation syndromes, acute pulmonary embolism, cerebrovascular accident, and later stages of pericarditis.⁴ If the EP considers the following criteria in the interpretation of the ECG with T-wave inversion, the differentiation of Wellens' T waves from other causes of inverted T waves will be easily accomplished (Table 1).^{3,5-7}

The chest pain patient who presents with a convincing clinical description of acute coronary syndrome and manifests electrocardiographic change involving the T wave in the anterior distribution will likely be managed in appropriate fashion in terms of initial therapy, diagnostic studies, and disposition—nitrates, aspirin, and β -adrenergic blockade, among other agents coupled with repeat ECGs and serum markers ending in an inpatient admission. Morphologically, Wellens' T waves are either deeply inverted or biphasic—both configurations are highly characteristic of the syndrome and unlike other T-wave inversions related to acute confusional state (Fig 6). The major issue here is the recognition of the syndrome and its relation to high-grade, proximal LAD obstruction—with the natural history of extensive anterior wall AMI. The avoidance of provocative testing, including stress imaging, is key in that such testing may precipitate an AMI with significant acute sequelae. Continuing on in the differential, such T-wave changes can

TABLE 1. Clinical and Electrocardiographic Criteria for Wellens' Syndrome

Symmetric and deeply inverted T waves, in leads V2 and V3, occasionally in leads V1, V4, V5, and V6;
or
Biphasic T wave in leads V2 and V3;
plus
Isoelectric or minimally elevated (<1 mm) ST segment
No precordial Q waves
History of angina
Pattern present in pain-free state
Normal or slightly elevated cardiac serum markers.

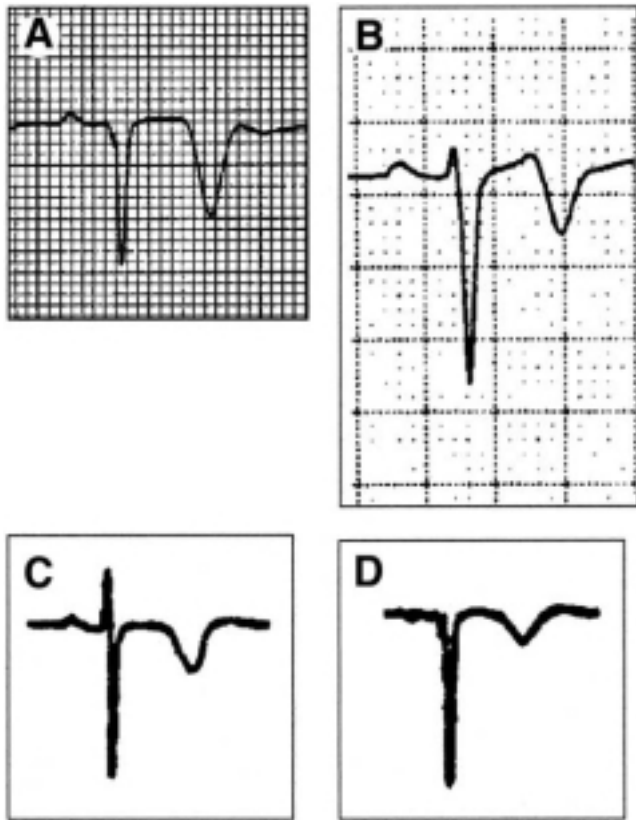


FIGURE 6. A comparison of abnormal T-wave inversions in acute ischemic heart disease. (A) Deeply inverted T wave of Wellens' syndrome. (B) Biphasic T wave of Wellens' syndrome. (C) Non-Wellens' ischemic T-wave inversion. (D) Non-Wellens' ischemic T-wave inversion. The common feature that is useful in distinguishing between these 2 types of inverted T waves and the related clinical syndrome is the depth of the inverted T wave—considerably more so in Wellens' syndrome.

occur owing to past myocardial infarction; once again, the morphology of such T-wave changes (inverted symmetrically with minimal negative amplitude) is quite distinct from the Wellens' T waves.

Considering noncoronary causes of T-wave inversions, the left ventricular hypertrophy and bundle branch block patterns should be readily recognized by their significant coexistent findings. For left ventricular hypertrophy, the presence of prominent electric forces in the QRS complexes of leads V1 to V6 is required to make the electrocardiographic diagnosis of left ventricular hypertrophy by voltage criteria. The EP must also realize that such repolarization change in left ventricular hypertrophy is usually limited to leads I, aVL, V5, and V6. Regarding the bundle branch block pattern, the widened QRS complex and characteristic morphologies of both right and left bundle branch block are obvious clues to its presence. Left bundle branch block pattern does not include T-wave inversion in leads V1 to V4; right bundle branch block pattern does include such T-wave changes.

From the perspective of electrocardiographically similar T-wave inversions, those changes associated with acute central nervous system insults are the most comparable.⁸ These T-wave changes are so similar, in fact, that they are described as Wellenoid in appearance (Fig 7). These T-wave changes are often very deeply inverted and/or biphasic in morphology. Of course, the clinical presentation of the patient (cephalgia, focal neurologic examination, and/or altered mental status) is likely to suggest the appropriate diagnosis and solve the electrocardiographic differential quandary.

Digitalis effect can be diagnosed electrocardiographically, of course, when the patient is using a digitalis compound. These T-wave changes most often are associated with diffuse ST-segment depression and have the characteristic gradual descending limb of the T wave with a rapid return to the baseline of the ascending portion. Acute pulmonary embolism, acute myocarditis, and pericarditis all have characteristic historic and examination findings that will suggest the appropriate clinical diagnosis.

CONCLUSION

Wellens' syndrome is a preinfarction stage of coronary artery disease. Patients with this syndrome present with

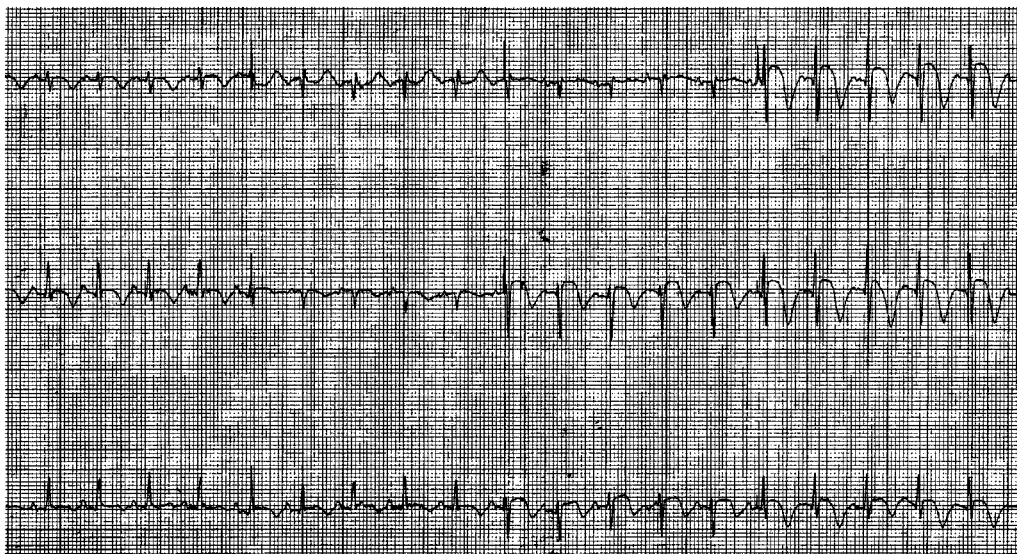


FIGURE 7. ECG showing extensive T-wave inversions with biphasic morphology in leads V2 to V6. This ECG was obtained in a 54-year-old patient who presented with altered mental status; cranial computed tomography scan showed extensive subarachnoid hemorrhage.

characteristic electrocardiographic findings in the precordial leads. These changes are associated with obstruction in the proximal LAD coronary artery. Two patterns of electrocardiographic changes are encountered, the deeply inverted T wave and the biphasic T wave. Once Wellens' syndrome has been recognized, cardiology consultation for possible coronary angiography is likely necessary to further evaluate the patient. Unless performed under the supervision of a cardiologist, stress imaging is strongly discouraged.

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