Artifactual Electrocardiographic Change Mimicking Clinical Abnormality on the ECG

CHRISTOPHER CHASE, MD AND WILLIAM J. BRADY, MD

Electrocardiographic artifact is a common finding in patients requiring evaluation and monitoring in the prehospital, emergency department, or intensive care unit settings. Artifact results from both internal (physiological) and external (nonphysiological) sources. In most instances, artifact is recognized as an incorrect electrocardiographic signal—its only impact producing interference in electrocardiogram interpretation; artifact may also produce electrocardiographic signals which mimick disease—these signals the physician must recognize as artifact. (Am J Emerg Med 2000;18:312-316. Copyright © 2000 by W.B. Saunders Company)

Artifact is a not uncommon finding in patients requiring electrocardiographic evaluation and monitoring in the prehospital, emergency department or intensive care unit settings. Artifact results from both internal (physiological) and external (nonphysiological) sources. Artifact may mimic the entire range of pathological electrocardiographic findings, ranging from arrhythmia to ischemic change.[1] Because of artifactual changes on the electrocardiogram (ECG), patients may receive unnecessary and potentially dangerous therapeutic interventions.[1,2] Often these masquerading deflections are apparent with careful, rational evaluation of both the patient and the ECG. Thus, the thoughtful clinician can accurately identify these artifactual pseudoabnormalities, eliminate the cause, and then acquire accurate electrocardiographic information while avoiding unnecessary and possibly dangerous therapies.

To identify and correct the artifact, an understanding of its origins is required. In general there are two sources of artifact: physiological and nonphysiological. Physiological sources may involve muscular activity and patient motion; these changes represent the most frequently encountered sources of artifact mimicking pathology. All muscle contractions are initiated by flow of electrically charged ions. This electrical flow produces a signal which can be recorded by ECG and is called an electromyographic (EMG) signal. Any and all muscle activity produces these signals which appear on the ECG monitor as narrow, rapid spikes simultaneous with muscle contraction which may mimic both atrial and ventricular arrhythmia.[3] These spikes are very different from the normal electrocardiographic pattern and can thus be electronically filtered out by current electrocardiographic monitors in many cases.

The effect of epidermal signals—motion-related change—is seen with large swings in the electrocardiographic baseline which occurs with position changes, coughing or ambulation; such effect actually originates from the electrocardiographic lead contact with the skin. In fact, it has been shown that simply by stretching the epidermis, a voltage difference of several millivolts is recorded by the ECG. This epidermal stretch-induced voltage change is the primary cause of motion artifact. This form of artifact cannot be filtered and must be prevented by having the patient be completely still during analysis.

Nonphysiological sources have numerous causes yet most commonly involve 60-Hertz interference. This interference is responsible for artifactual change when the ECG shows a wide, indistinct isoelectric baseline. The source of this interference is actually the alternating current in the power outlets, light fixtures, and other electrical apparatus in the hospital room and is usually filtered out by the electrocardiographic machine. Another source of artifact, cable and electrode malfunction, occurs with an insufficient amount of electrode gel, fractured monitoring wires, loose connections, and misplaced leads. These artifacts take many forms and must be prevented by vigilant quality control measures. Yet another cause, offset potential-induced artifact, occurs when stored potential energy in electrocardiographic cables prevents normal flow of the signal. This type of interference has been largely eliminated from current practice with the application of silver chloride electrodes which prevent the accumulation of any energy in the leads. Lastly, the actual flow of electricity through various appliances in the vicinity of the electrocardiographic machine, cables, electrodes, and other components of the system may disrupt the signal. This type of change has been reported in cutaneous nerve stimulatory, various medical pumps, patient and fluid warmers, and fluid chambers[4]; the accumulation of static electricity has also been described as another source of artifact.[5]

The first case involves an elderly man with chest pain. The patient had a history of Parkinson's disease. He was alert and in no distress. Continuous cardiac monitoring was in place, showing normal sinus rhythm. During the physical examina-

From the Department of Emergency Medicine, University of Virginia School of Medicine, Charlottesville, VA.

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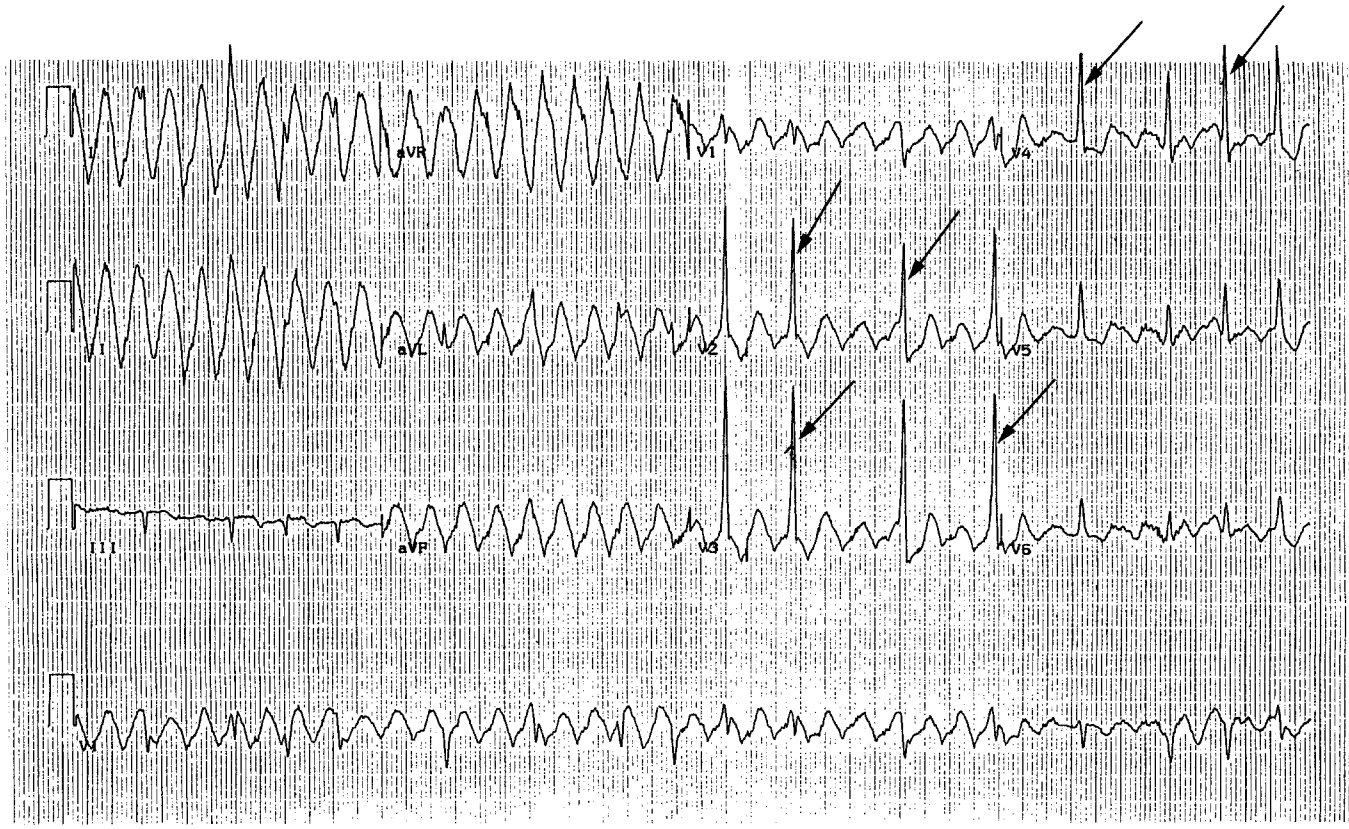


FIGURE 1. Wide-complex tachycardia consistent with ventricular tachycardia. Lead III reveals a narrow QRS complex rhythm at a slow rate while the remainder of the limb leads demonstrate a wide QRS complex tachycardia. Several narrow complex beats are seen in the precordial leads (arrows), representing the true cardiac rhythm while the wide QRS complexes are artifactual.

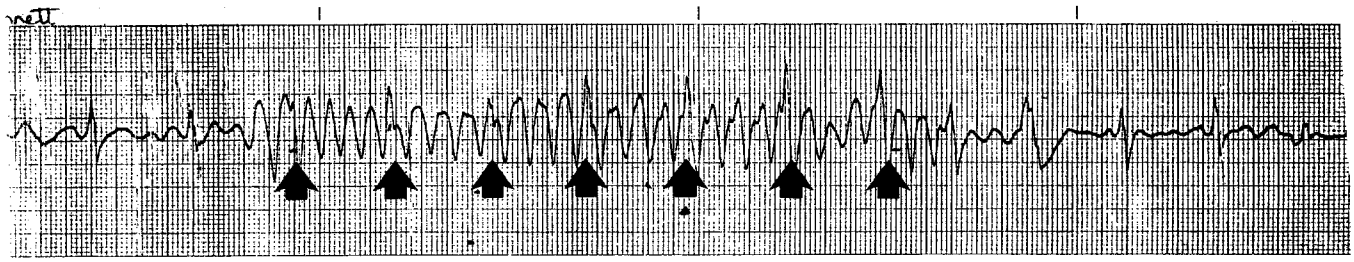


FIGURE 2. Rhythm strip showing wide-complex tachycardia. This artifactual arrhythmia was initially misidentified as nonsustained ventricular tachycardia. Notice the taller QRS complexes (arrows) which occur at regular intervals.

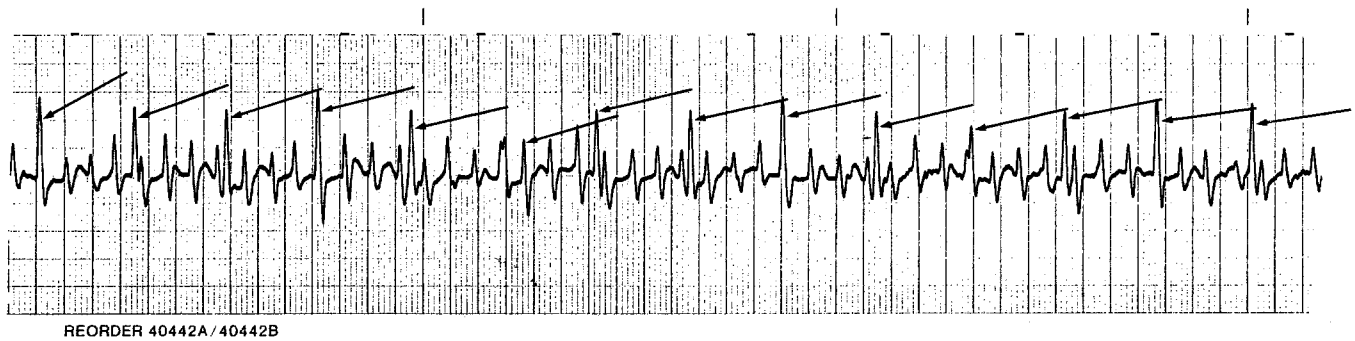
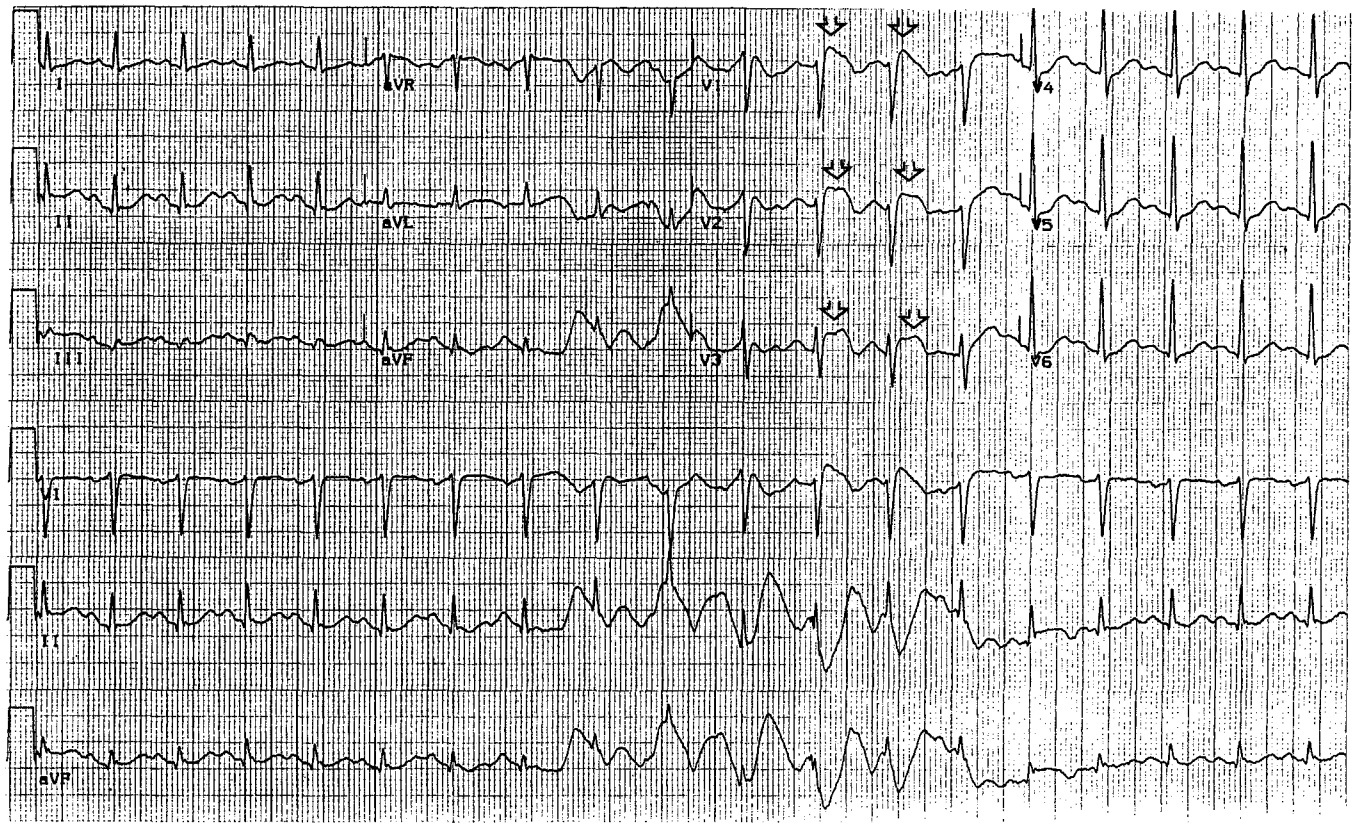
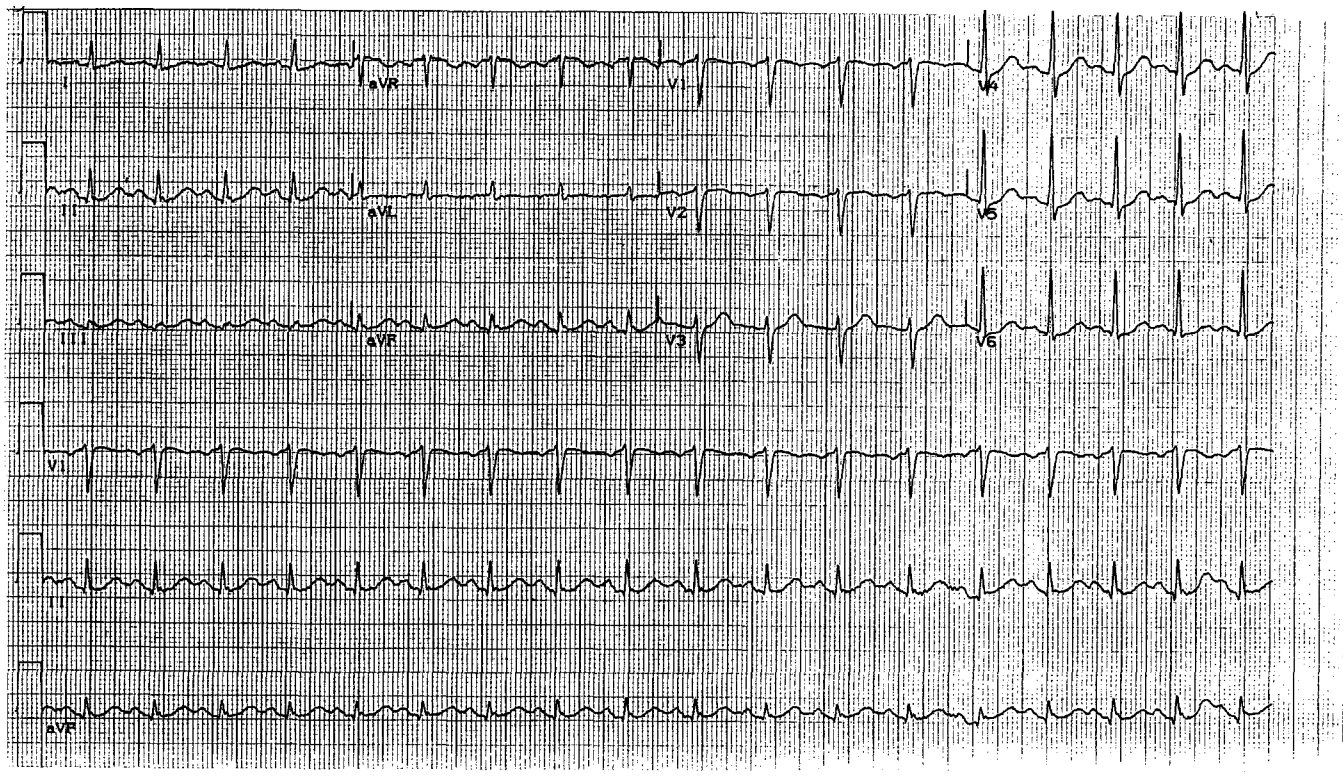


FIGURE 3. A rhythm strip showing normal QRS complexes (arrows) with overlying EMG artifact.



A



B

FIGURE 4. (A) A 12-lead ECG showing ST segment elevation in leads V1-V3 (arrows), suggestive of an anteroseptal acute myocardial infarction. (B) A repeat ECG performed soon afterwards with resolution of the ST segment elevation. This ECG was performed when the patient was not experiencing tremor.

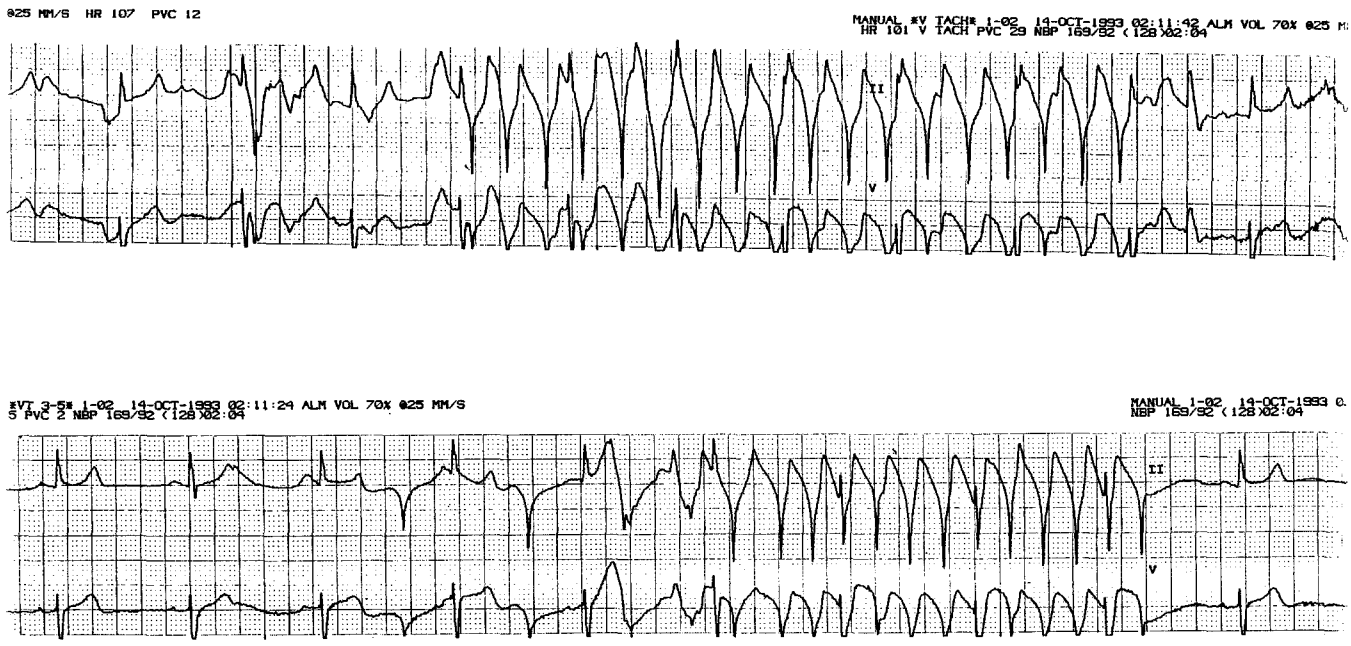


FIGURE 5. Rhythm strips showing normal sinus rhythm with intervening periods of an apparent wide-QRS complex tachycardia. This artifactual arrhythmia was initially misidentified as nonsustained ventricular tachycardia. During the apparent dysrhythmia, the patient did not experience a change in his condition nor show an alteration in his physical status; the vital signs remained normal as did his mental status. The cause of the artifactual arrhythmia was caused by placement of new electrical lines adjacent to the monitor apparatus with a break in the specific monitor's line insulation.

tion, a 12-lead ECG showed a wide-complex tachycardia consistent with ventricular tachycardia with rare narrow QRS configurations (Figure 1). As the ECG was being performed, the patient remained alert and conversant, with a palpable pulse of approximately 60 to 70 beats/min accompanied by telemetry monitoring showing sinus rhythm with a rate of 60 to 70 beats/min. It was clinically apparent that the electrocardiographic findings represented artifact produced by the Parkinsonian tremor and not ventricular tachycardia. This artifactual example shows physiological change caused by muscular contraction an patient motion.

The second case shows pseudoventricular tachycardia produced by muscular tremor in a patient with chronic atrial flutter and bacterial pneumonia. During the interview, the patient experienced rigors accompanied by a wide-complex tachycardia on the monitor (Figure 2). During the apparent tachycardia, the patient remained alert and hemodynamically stable with a pulse of 60 beats/min. With resolution of the shaking chill, the tachycardia terminated and the baseline rhythm (atrial flutter) reappeared.

The third case is of a 29-year-old man with a past medical history significant for epilepsy controlled with dilantin presenting to the emergency department with a 3-day history of nausea, vomiting, and malaise. During the assessment the patient was found to be tachycardic with a pulse of 140 beats/min and was placed on oxygen, bedside heart monitoring and two large bore intravenous lines were placed for fluid resuscitation. After completing the primary and secondary surveys, the physician was once again called to the patient's room for a reported unstable cardiac rhythm. On arrival, the physician noted that the patient was having clonic movement of his extremities. Vitals showed pulse of the 110 beats/min and blood pressure 130 mmHg by palpation. A rhythm strip obtained at that time appeared to

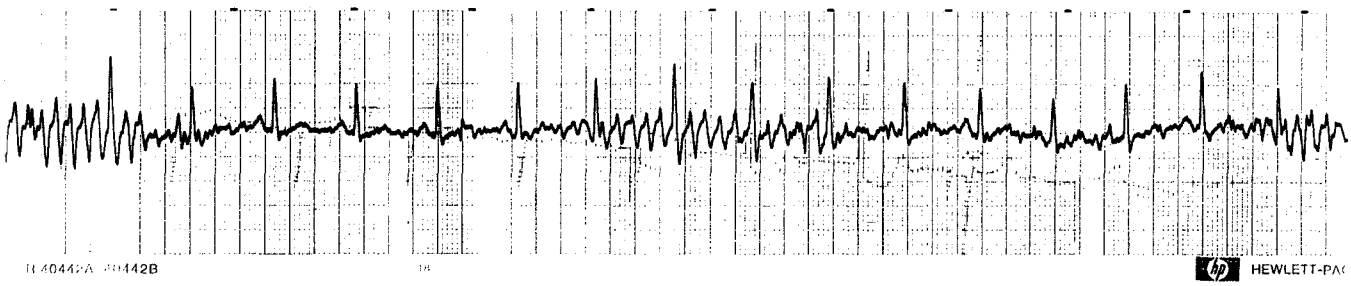


FIGURE 6. Rhythm strip showing normal sinus rhythm in a patient with environmental hypothermia and intermittent shivering. During the shivering, the electrocardiographic monitor revealed frequent, small spikes occurring between the larger narrow QRS complexes. This artifactual arrhythmia was initially misidentified as atrial flutter.

show a narrow-QRS complex tachycardia with intervening single large QRS complexes followed by several small QRS complexes (Figure 3).

A fourth case involves a 63-year-old man with a past medical history of alcoholic liver disease who presented with irritability, difficulty concentrating, and intermittently slurred speech. His wife stated that over the past several days he had been trying to quit drinking. During the physical examination, the patient developed a course tremor of both upper extremities. An ECG obtained shortly thereafter showed sinus tachycardia with a rate of 110 beats/min with ST segment elevation in leads V1-V3 (Figure 4A). Although treatment of a possible acute myocardial infarction was initiated lorazepam was prescribed for the alcohol withdrawal-related tremor. After the tremor resolved, the ST segment elevation resolved (Figure 4B). The patient was admitted to the hospital with successful treatment of the ethanol withdrawal syndrome; no evidence of myocardial ischemia or infarction was found. A nuclear medicine exercise stress test was unremarkable.

In all instances, a clinical correlation was sought between a stable patient and an apparently malignant electrocardiographic abnormality. None was found an case. Further review of the clinical situations revealed muscular tremor producing artifact and not arrhythmia or acute infarction, sparing the patients potentially dangerous medical therapies.

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