

Nitzkin is wrong to claim that the currently pending bill was negotiated between Philip Morris and Matthew Myers of the Campaign for Tobacco-Free Kids. The current bill was the work of Senator Ted Kennedy (D-MA) and then-Senator Mike DeWine (R-OH), who have consistently been among Congress's strongest supporters of tobacco control.

The pending legislation differs markedly from the proposals previously put forward by Philip Morris. The bill has provisions that would require serious changes in the marketing and manufacture of cigarettes in the decades ahead, including the authority for the FDA to monitor and reduce levels of dangerous additives and nicotine. The

improved warning labels — just one provision of the bill — could save thousands of lives.

Nitzkin is certainly entitled to oppose the legislation, but he fails to offer any evidence for his claim that the bill will lead to increases in teen smoking and tobacco-related mortality. His position conflicts with the positions of virtually all the leading public health and medical organizations that are committed to reducing the burden of disease that cigarettes generate.

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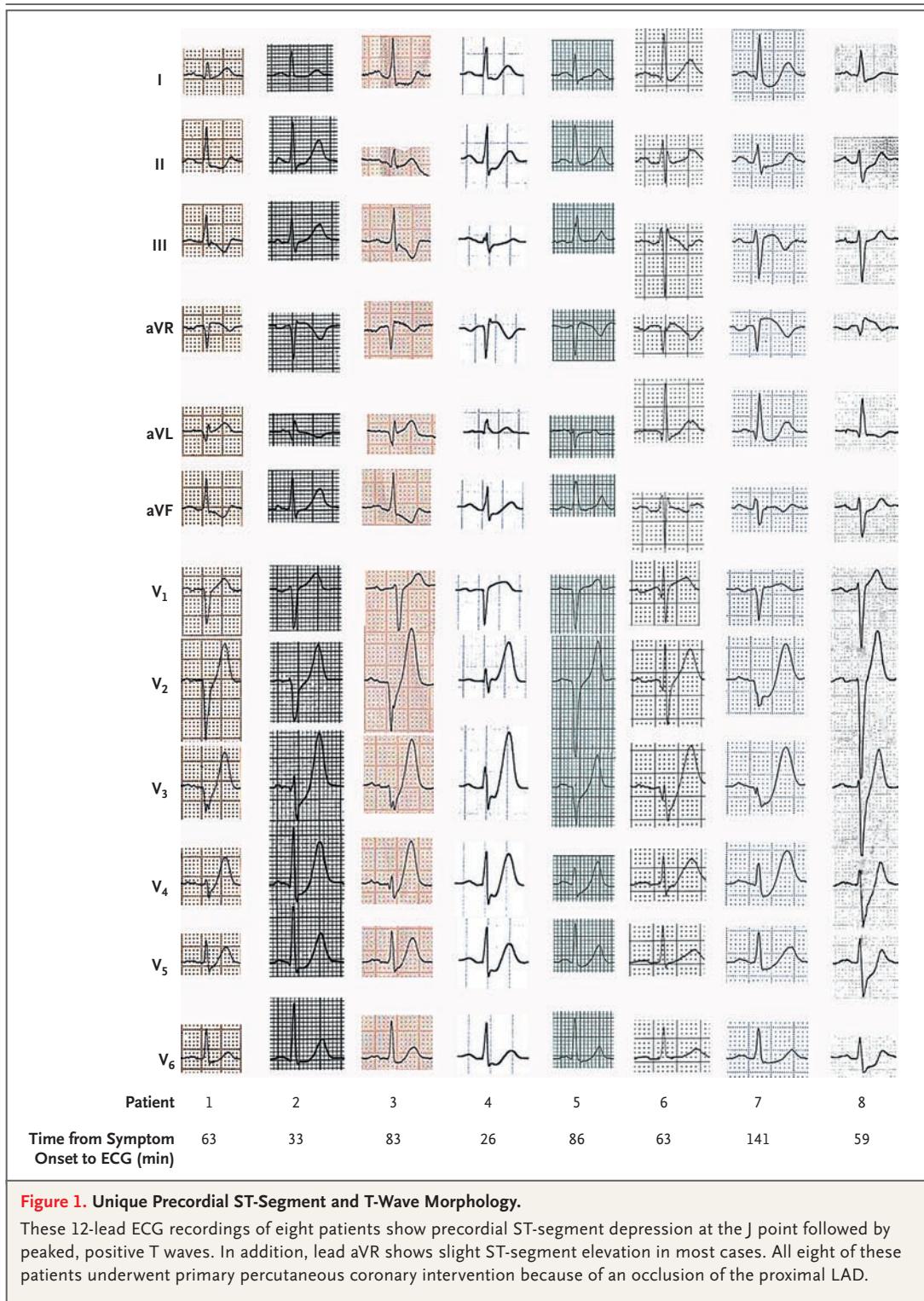
## A New ECG Sign of Proximal LAD Occlusion

**TO THE EDITOR:** Recognition of characteristic changes in an electrocardiogram (ECG) that are associated with acute occlusion of a coronary artery guides decisions regarding immediate reperfusion therapy.<sup>1-3</sup> Working from our primary database of percutaneous coronary interventions, which includes records of the ambulance, or admission, ECG (performed on first medical contact with the patient), the preprocedural ECG, and the coronary angiogram, we describe a new ECG pattern without ST-segment elevation that signifies occlusion of the proximal left anterior descending coronary artery (LAD). Instead of the signature ST-segment elevation, the ST segment showed a 1- to 3-mm upsloping ST-segment depression at the J point in leads  $V_1$  to  $V_6$  that continued into tall, positive symmetrical T waves. The QRS complexes were usually not widened or were only slightly widened, and in some there was a loss of precordial R-wave progression. In most patients there was a 1- to 2-mm ST-elevation in lead aVR (see Fig. 1 for representative examples of this ECG pattern). We recognized this characteristic ECG pattern in 30 of 1532 patients with anterior myocardial infarction (2.0%).

Although tall symmetrical T waves have been recognized as a transient early feature that changes into overt ST elevation in the precordial leads, in these patients this pattern was static, persisting from the time of first ECG until the preprocedural ECG was performed and angiographic evi-

dence of an occluded LAD was obtained (i.e., 30 to 50 minutes). The ECGs with this pattern were on average recorded 1.5 hours after symptom onset. Collateral filling of the LAD ranged from Rentrop class 0 to class 3, and a wraparound LAD was present in 50% of patients. There was no evidence of involvement of the left main stem of the coronary artery, nor was there evidence of significant disease in the coronary arteries supplying the posterior or posterolateral myocardial territories. Potassium levels on admission were normal ( $3.9 \pm 0.5$  mmol per liter). Despite successful procedures in all cases, there was considerable loss of myocardium, with a median creatine kinase MB peak of  $342 \mu\text{g}$  per liter.

The electrophysiological explanation of the observed ECG pattern remains elusive. We could not establish patient characteristics, nor could we identify coronary angiographic characteristics that were unequivocally associated with the ECG pattern described as compared with a pattern of anterior ST elevation. Theoretically, an anatomical variant of the Purkinje fibers, with endocardial conduction delay, could be present. Alternatively, the absence of ST elevation may be related to the lack of activation of sarcolemmal ATP-sensitive potassium ( $K_{\text{ATP}}$ ) channels by ischemic ATP depletion, as has been shown in  $K_{\text{ATP}}$  knock-out animal models of acute ischemia.<sup>4</sup> It is of great importance for physicians and paramedics involved in the triage of patients with chest pain



**Figure 1. Unique Precordial ST-Segment and T-Wave Morphology.**

These 12-lead ECG recordings of eight patients show precordial ST-segment depression at the J point followed by peaked, positive T waves. In addition, lead aVR shows slight ST-segment elevation in most cases. All eight of these patients underwent primary percutaneous coronary intervention because of an occlusion of the proximal LAD.

for reperfusion therapy to recognize this ECG pattern.

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1. Engelen DJ, Gorgels AP, Cheriex EC, et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. *J Am Coll Cardiol* 1999;34:389-95.
2. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. *N Engl J Med* 2003;348:933-40.
3. Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. *N Engl J Med* 2003;349:2128-35.
4. Li RA, Leppo M, Miki T, Seino S, Marban E. Molecular basis of electrocardiographic ST-segment elevation. *Circ Res* 2000;87:837-9.

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