



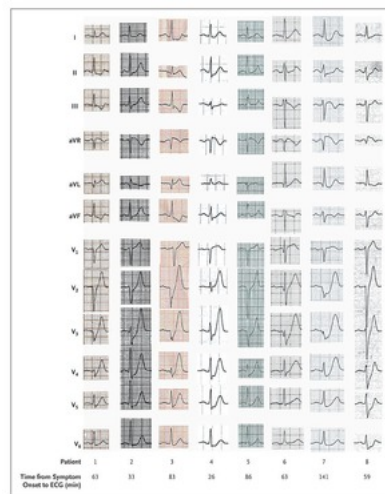
CORRESPONDENCE

A New ECG Sign of Proximal LAD Occlusion

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TO THE EDITOR:

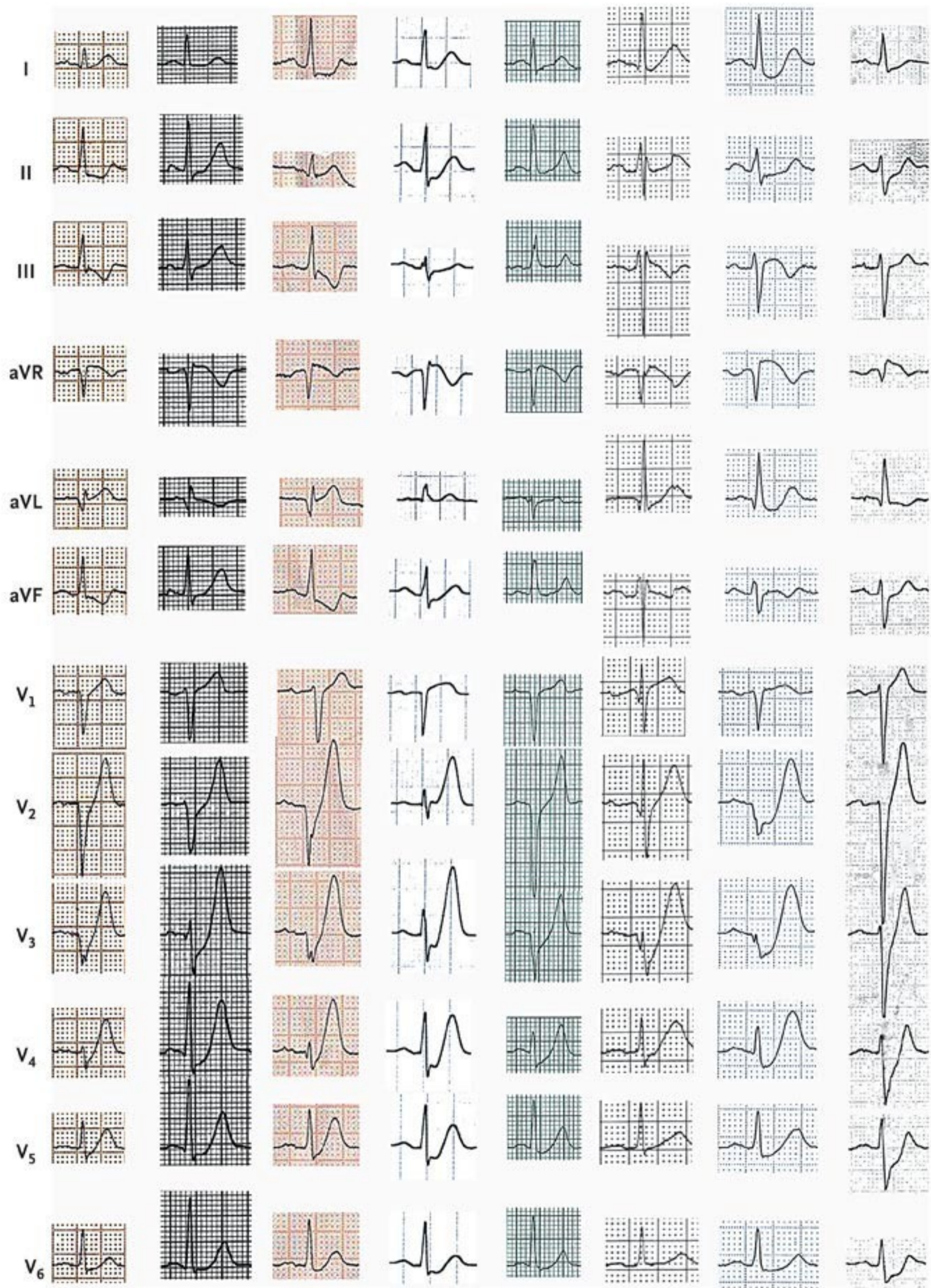
Figure 1.



Unique Precordial ST-Segment and T-Wave Morphology.

Recognition of characteristic changes in an electrocardiogram (ECG) that are associated with acute occlusion of a coronary artery guides decisions regarding immediate reperfusion therapy.¹⁻³ 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 V₁ to V₆ 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 [Figure 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 evidence of an occluded



Patient	1	2	3	4	5	6	7	8
Time from Symptom Onset to ECG (min)	63	33	83	26	86	63	141	59

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±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 µg 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_{ATP}) channels by ischemic ATP depletion, as has been shown in K_{ATP} knockout animal models of acute ischemia.⁴ It is of great importance for physicians and paramedics involved in the triage of patients with chest pain for reperfusion therapy to recognize this ECG pattern.

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