

ECG Diagnosis: Hyperacute T Waves

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After QT prolongation, hyperacute T waves are the earliest-described electrocardiographic sign of acute ischemia, preceding ST-segment elevation.¹ Hyperacute T waves are broad-based and symmetrical, usually with increased amplitude and often associated with a depressed ST take off.¹ Hyperacute T waves are most evident in the anterior chest leads and are more apparent when a previous electrocardiogram is available for comparison.² Hyperacute T waves are noted early after the onset of coronary occlusion and transmural infarction and tend to be a short-lived structure that evolves rapidly into ST-segment elevation.³ The electrocardiographic differential diagnosis of the hyperacute T wave includes both transmural acute myocardial infarction and hyperkalemia as well as early repolarization, left ventricular hypertrophy, and acute myopericarditis.⁴

The principle entity to exclude is hyperkalemia—this T-wave morphology may be confused with the hyperacute T wave of early transmural myocardial infarction. In contrast to hyperacute T waves associated with myocardial ischemia or infarction, hyperkalemic T waves tend to be narrow and peaked with a prominent or sharp apex.⁴ For patients presenting with hyperacute T waves in the setting of suspected myocardial ischemia or infarction, treatment includes symptomatic control with nitroglycerin or morphine, oral antiplatelet agents (aspirin), consideration of anticoagulation with unfractionated heparin, and obtaining frequent serial 12-lead electrocardiograms (every 5 to 10 minutes). Prompt consultation with a cardiologist is indicated in these cases. ❖

References

1. Goldberger AL. Hyperacute T waves revisited. *Am Heart J* 1982 Oct;104(4 Pt 1):888-90. DOI: [http://dx.doi.org/10.1016/0002-8703\(82\)90038-2](http://dx.doi.org/10.1016/0002-8703(82)90038-2).
2. Nable JV, Brady W. The evolution of electrocardiographic changes in ST-segment elevation myocardial infarction. *Am J Emerg Med* 2009 Jul;27(6):734-46. DOI: <http://dx.doi.org/10.1016/j.ajem.2008.05.025>.
3. Morris F, Brady WJ. ABC of clinical electrocardiography: acute myocardial infarction—part I. *BMJ* 2002;324:831. DOI: <http://dx.doi.org/10.1136/bmj.324.7341.831>.
4. Brady W, Morris F. Electrocardiographic abnormalities encountered in acute myocardial infarction. *J Accid Emerg Med* 2000 Jan;17(1):40-45. DOI: <http://dx.doi.org/10.1136/emj.17.1.40>.

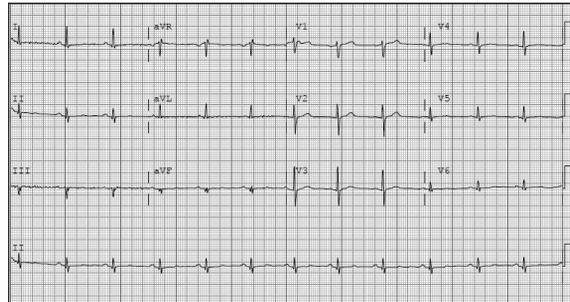


Figure 1. 12-lead electrocardiogram from a 47-year-old man presenting to the Emergency Department with left-sided chest discomfort, now resolved following sublingual nitroglycerin.

Demonstrates normal sinus rhythm with nonspecific ST-T wave changes including flattening of T waves in the inferior and lateral leads.

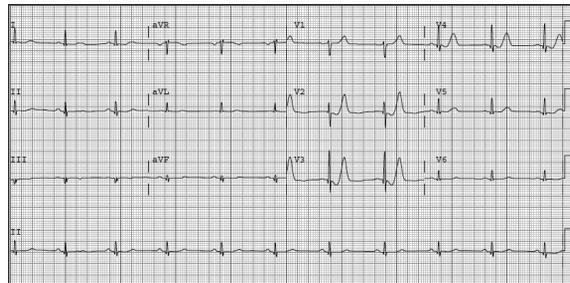


Figure 2. 12-lead electrocardiogram from same patient, obtained 90 minutes later, with patient now experiencing 5/10 left-sided chest discomfort.

Demonstrates large, symmetric T waves with depressed ST-segment take off consistent with hyperacute T waves in the anterior leads V₂-V₄.

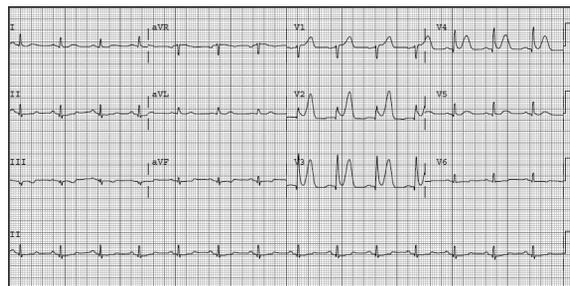


Figure 3. 12-lead electrocardiogram from same patient as Figures 1 and 2, obtained 10 minutes after the electrocardiogram in Figure 2.

Demonstrates > 1 mm ST-segment elevation in the anterior leads V₂-V₄, consistent with an acute anterior wall myocardial infarction.



Figure 4. 12-lead electrocardiogram from a 73-year-old man with end-stage renal disease and hyperkalemia (serum potassium 7.6 mEq/L).

Demonstrates peaked T waves in leads V₃ and V₄, consistent with hyperkalemia.

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