

recording from the patient's back. Since V1 and V2 are attached to the patient's front, they will record changes reciprocal to changes seen from the back, which are ST depression and tall R waves. These uncharacteristic signs make the diagnosis of posterior MI difficult without heightened vigilance. Suffice it to say, pure posterior wall infarctions are rare. Most extend to involve the inferior wall or lateral wall and leads II, III, and aVF should be examined for characteristic signs of this extension in the former and leads I, aVL, V5 and V6 in the latter.

MI in the presence of LBBB

The presence of LBBB complicates the ECG diagnosis of acute MI. This is because LBBB alone can produce signs that may be confused with those of infarction: deep QS waves in the right chest leads and ST depression and T wave inversion in the left chest leads. Furthermore, the Q wave of left ventricular MI may be buried within the widened QRS complex. Therefore, the diagnosis of acute myocardial infarction should be made circumspectively in the presence of pre-existing LBBB. On the other hand, the appearance of new LBBB should be regarded as sign of acute MI until proven otherwise.

Electrolyte Abnormalities

Hyperkalemia and Hypokalemia

Serum potassium is the major intracellular ion that participates in the depolarization and repolarization of myocardial cells. Hence its serum concentration has a profound effect on the QRS and ST-T complex.

Narrow and tall peaked T wave (A) is an early sign of hyperkalemia. It is unusual for T waves to be taller than 5 mm in limb leads and taller than 10 mm in chest leads. Hyperkalemia should be suspect if these limits are exceeded in more than one lead. As serum potassium concentration

