Inverted T Waves
An Electrocardiographic Marker of Stunned or Hibernating Myocardium in Man?

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In this issue of Circulation, Renkin et al have made an intriguing observation in patients with unstable angina. They describe a subgroup of unstable angina patients with persistent inversion of precordial-lead T waves who have resting regional left ventricular wall motion abnormalities. Six months after left anterior descending angioplasty, these patients demonstrated normalization of T waves and improvement in regional wall motion. In contrast, another group of patients with unstable angina, who had a similar extent of coronary artery disease but upright T waves, did not demonstrate wall motion abnormalities and had no change in T waves or wall motion after angioplasty. The results suggest that persistently inverted T waves in the setting of unstable angina and in the absence of documented myocardial infarction may help identify patients who have viable but abnormally functioning myocardium. Identifying such myocardium is important because it may recover function after restoration of blood flow.

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It is not clear from the study by Renkin et al whether the patients have myocardium that is truly stunned (postischemic left ventricular dysfunction) or is “hibernating” (chronic low-flow state). If the myocardium is stunned, this would imply that it had been subjected to multiple discrete episodes of ischemia in which recovery of function is slow or delayed after relief of ischemia. This certainly is conceivable in the setting of unstable angina as previously described by Nixon and coworkers. It would have been interesting to have known the time course of recovery of T waves and regional wall motion abnormalities after successful angioplasty. Did these parameters recover immediately, after a course of days, or only after weeks or months? Because the authors reported postangioplasty data at 6 months only, it is not possible to determine whether slow recovery of function occurred once ischemia was relieved with mechanical intervention.

An alternative explanation for their findings is that the myocardium of patients with negative T waves was hibernating. Rahimtoola defined hibernating myocardium “as a state of persistently impaired left ventricular function at rest that is due to reduced coronary blood flow.” The functional abnormality can be improved by either increasing coronary blood flow (as may have been the case in this study, after angioplasty) or by reducing oxygen demand. Evidence supporting the concept of hibernating myocardium in man has been reviewed. The concepts of hibernating and stunned myocardium are similar in that both conditions are characterized by viable myocardial cells (as opposed to infarcted) that demonstrate depressed function. However, with hibernating myocardium, blood flow remains reduced, and the persistent low-flow state with depressed regional function lasts months to years. However, once revascularization occurs and blood flow is restored, function improves; in some clinical studies, this improvement in function may be immediate. One theory for this phenomenon is that the heart downregulates its function to reduce oxygen demand in the setting of reduced supply. In contrast, in the situation of stunned myocardium, ischemia already has been relieved. Stunned myocardium is a temporary phenomenon that may last only hours to days and refers to a delay in return of function after relief of ischemia.

Whether patients with unstable angina, negative T waves, and wall motion abnormalities that are relieved after angioplasty demonstrate stunned myocardium, hibernating myocardium, or some combination of both of these entities is not clear from the present study. What is clear is that the authors have observed a potential electrocardiographic marker (inverted T waves) for identifying viable, but poorly contracting myocardium that has the potential to recover after restoration of blood flow.

How have such regions of myocardium been identified in the past? One technique that was used was to try to stimulate the stunned or hibernating myocardium...
dium to contract, which would favor tissue viability. Several experimental studies have shown that stunning of the myocardium can be overcome by administration of inotropic agents such as dopamine, dobutamine, and epinephrine and by the maneuver of postextrasystolic potentiation.\(^6\) \(^8\) One clinical study\(^9\) provided evidence that stunned myocardium could be recruited in man by a small bolus of isoproterenol after thrombolysis. Similarly hibernating myocardium can be stimulated to contact by inotropic maneuvers such as postextrasystolic potentiation,\(^10\) \(^11\) catecholamines,\(^12\) or by administering nitroglycerin\(^3\) (presumably decreasing preload and to some extent afterload, thus reducing oxygen demand). Studies have suggested that identification of hibernating myocardium by such maneuvers will predict ultimate recovery of left ventricular segments after revascularization procedures.\(^3\) \(^5\) Left ventricular segments that do not respond to such stimulation are likely to be infarcted (scarred).

Another technique that has been used to identify viable but nonfunctioning myocardium is positron emission tomography. Tracers such as rubidium are used to document the status of regional blood flow; with hibernating myocardium, one would expect reduction in regional flow by PET scanning, and with stunned myocardium, one would expect the presence of normal perfusion. Viability is determined by injection of tracers such as fluorodeoxyglucose that identify metabolically active and therefore living tissue.\(^13\) In hibernating and stunned myocardium, the affected myocardial segments should be capable of glucose uptake. Infarcted myocardium should not be capable of such metabolism. Tillisch et al used positron emission tomography and reported that metabolically active myocardium that had reduced perfusion demonstrated recovery of function after surgical revascularization.\(^14\)

The present study by Renkin et al\(^1\) provides a potential third marker of viable, poorly contracting tissue that has the potential for recovery—inverted T waves on the electrocardiogram. Of note, the first series of studies to describe stunned myocardium in experimental animals described prolonged abnormalities in the ST-T waves on the electrocardiogram.\(^15\) As noted by Renkin et al,\(^1\) one clinical study described transient Q waves associated with reversible wall motion abnormalities.\(^16\) While inverted T waves may indicate stunned or hibernating myocardium in the clinical syndrome of unstable angina, they may be lacking in other clinical entities associated with stunned or hibernating myocardium. For example, in a recent preliminary report,\(^17\) we observed prolonged wall motion abnormalities, assessed by two-dimensional echocardiography after exercise treadmill testing, at a time when electrocardiographic abnormalities had resolved. If Renkin et al's findings are confirmed by other investigators, the use of the flipped T waves as a marker of stunned or hibernating myocardium may prove to be an easy way of identifying those unstable angina patients whose left ventricular function will improve after angioplasty. Certainly using electrocardiography will be much cheaper and more convenient than using positron emission tomography or subjecting patients to boluses of inotropic agents.

References


(Circulation 1990;82:1060–1061)
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