Electrocardiographic Changes Associated With Neurologic Events

To the Editor:

The timely and provocative editorial "Electrocardiographic Changes Associated With Neurologic Events" by Drs. Strauss and Samuels (CHEST 1994; 106:1316-17) illustrates the continuing enigma of altered myocardial repolarization. The clinical challenge it poses remains a vexatious problem. The perplexity of abnormal T waves produced by factors that alter the myocardial repolarization vector from its usual epicardial to endocardial direction in the intact heart is often a source of concern to the clinician. In this abnormally influenced state, repolarization proceeds in the same direction as ventricular depolarization. Myocardial repolarization changes are classified as primary changes, which are associated with direct influences on the basic biochemical physiology of the muscle cell, and secondarily—those which are the result of alteration in the sequence of myocardial depolarization. Repolarization is sensitive to numerous internal and external influences. The medical literature is replete with case reports of various pathologic states and conditions, which affect the T wave of the electrocardiogram. Factors believed to influence repolarization are the myocardial temperature gradient, the anatomic structure of the heart, and an intact, functioning pericardium.1 The T wave is recorded during mechanical systole, as opposed to the QRS, which occurs during isometric contraction. Repolarization is energy consuming, whereas depolarization is not. This energy expenditure is required to allow the reconstitution of the intra- and extracellular ion concentrations, which take place against an electrical gradient.

Although deeply negative T waves are the most dramatic and frequently described repolarization abnormality in patients suffering a major cerebrovascular event, the occurrence of tall (upright) T waves which can occur in some hemorrhagic stroke patients also are difficult to explain.2 These large, broad, upright T waves are unlike those associated with hyperkalemia (narrow, peaked, with normal or shortened QT interval). Patients exhibiting this pattern have not been found to be hyperkalemic; however, the relationship to intracellular and membrane electrolyte abnormalities has not been investigated. Prominent U waves are often present in the electrocardiograms of these patients but are often not appreciated as they are obscured by the terminal portion of the T wave. Electrocardiographic abnormalities are most often seen in patients with severe subarachnoid hemorrhage, but, can also be associated with hemorrhagic brain infarction, drug toxicity, septicemia, neoplasia, trauma, epilepsy, metabolic-electrolyte abnormalities, and other medical catastrophes.

The principal questions provoked by this enticing editorial follow:

What is the cause of the altered myocardial physiology in certain central nervous system disorders? What disease is produced? What is the prognostic significance of these events? And of ultimate importance—should treatment be instituted? Why should it be undertaken, and when, during the course of events is treatment considered appropriate and optimal? Presently, experimental and clinical evidence favors the implication of catecholamine excess in the development of the myocardial changes produced by central nervous system insults and other altered physiologic states.3 The repolarization abnormalities, conduction defects, and arrhythmias can all be explained on the basis of direct or indirect catecholamine influence. The catecholamine "storm" can be implicated in the reported subendocardial hemorrhages, the cardiac myocytolysis, and other cellular disruptions within the myocardium.4 The importance of the direct chemical toxic effect vs the indirect effects of vasospasm and diminished perfusion have not been fully elucidated. At the opposite spectrum of concern are the findings of a recent clinical study suggesting that global T wave inversion is a nonspecific primary repolarization abnormality generally believed to be of no intrinsic prognostic significance, even though they may be catecholamine related.5

The lesions associated with catecholamine excess have been shown to be minimized or prevented by beta-blockade, experimentally and clinically.6 The treatment of centrally or catecholamine-induced cardiac arrhythmia is basic and relatively simple to achieve in most cases. The relevance and necessity of treating the microscopic changes is still open to question and debate. Specific questions must be asked and ultimately answered. Do these changes affect the prognosis or longevity of a given subset of patients? Are those changes temporary and for the most part reversible, or are they progressive and productive of permanent sequelae? The final answer has yet to be written. For now, individual clinical judgment remains the mainstay of decision making—that decision which subsequently leads to preservation of an acceptable quality of life when each patient is individually considered. Only then can the question of therapy be appropriately addressed.

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REFERENCES
1 BuDusky BM, Soloff LA. The myocardial temperature gradient and ventricular repolarization. Angiology 1968; 19:257-67