ABNORMALITIES of the electrocardiogram are extremely useful in the recognition of heart disease, but they also occur in a variety of states in which the primary pathology is extracardiac. Among these, neurologic disease is of special interest. The abnormalities which sometimes occur in neurologic disease are among the most striking deviations from normal that have been observed. In addition, neurogenic changes in the electrocardiogram furnish valuable insights into the physiologic basis of this record.

Like other electrocardiographic abnormalities with an extracardiac basis, those of neurogenic origin should not be taken as evidence of primary heart disease. The most marked abnormalities are characteristic and unlikely to be mistaken for effects of heart disease provided their association with neurologic lesions is appreciated. Findings consist of large T waves of either normal or abnormal polarity, prominent U waves, and prolongation of the Q-T or Q-U interval. The association of these ECG features with neurologic disease is well established, and the large T waves in particular distinguish the findings from those in other states. It has also been established that less striking and less characteristic abnormalities of the ST-T deflection occur as a result of neurologic disease. These cannot be differentiated from abnormalities due to other causes, including heart disease, on electrocardiographic grounds alone. It is important that the possible neurogenic origin of nonspecific electrocardiographic abnormalities be considered in patients with neurologic disease to avoid the erroneous diagnosis of heart disease.

The physiologic mechanism by which lesions of the nervous system influence the electrocardiogram is most likely the direct effect of the autonomic system on electrophysiologic behavior of the heart. Other possibilities include hypotension or other hemodynamic factors and disordered electrolyte levels, but no consistent hemodynamic or electrolyte patterns have been noted. Furthermore, the most marked electrocardiographic abnormalities associated with neurologic disease do not resemble those which occur with hypotension or electrolyte disorders.

Findings in experimental animals strongly favor altered sympathetic tone as the mechanism of changes in the ST-T form associated with neurologic lesions. Stimulation of many brain stem and some cortical areas have been demonstrated to alter the electrocardiogram. In the dog, stimulation or destruction of cardiac sympathetic nerves at the level of the stellate ganglion results in systematic changes of the ST-T deflection. These experiments on dogs are especially informative since the cardiac distribution of right and left sympathetic systems differ and localized
effects can be produced. Sympathetic stimulation shortens and sympathetic ablation prolongs ventricular recovery time. In the case of the right stellate ganglion these effects occur in the anterior ventricular wall while those of the left stellate affect the posterior wall. Stimulation of the right stellate produces a state with shorter recovery times in the anterior than in the posterior ventricular wall. Ablation of the left stellate produces a similar relationship due to prolongation of recovery time posteriorly, and the electrocardiographic effects resemble those associated with stimulation of the right stellate. Likewise, left stellate stimulation or right stellate ablation results in similar temporal relationships of recovery times in anterior and posterior ventricular walls and similar electrocardiographic effects.

Electrocardiographic effects of experimental alterations of sympathetic tone in dogs resemble those observed in patients with lesions of the central nervous system. In particular, the effects of experimental left stellate stimulation or right stellate ablation result in large T waves and prolongation of the Q-T interval similar to findings characteristic of neurologic disease in man. Right stellate stimulation or left stellate ablation results in low or inverted T waves which although less characteristic of neurogenic origin are known to occur on this basis. Unilateral alteration of sympathetic tone also provides an explanation for a particular feature of the electrocardiogram associated with neurologic disease. ECG tracings with nonspecific abnormalities of neurogenic origin have notched T waves more frequently than electrocardiograms which are abnormal for other reasons. This finding is compatible with asymmetric alteration of sympathetic tone resulting in two populations of ventricular recovery times. Other clinical findings closely related to the experimental findings summarized here have been reported by Hugenholtz. Marked ECG abnormalities similar to those previously observed in association with central nervous system lesions were noted in a patient after right radical neck dissection with probable destruction of sympathetic fibers.

Although neurogenic electrocardiographic abnormalities are explicable on the basis of functional alterations of ventricular recovery, there is also evidence that structural cardiac changes are involved. Subendocardial petechial hemorrhages have been noted in cases of subarachnoid hemorrhage and in experimental animals made hypertensive by stellate stimulation. Morphologic changes in the myocardium have been produced by experimentally induced intracranial hemorrhage and by the intracranial injection of blood in mice. Repeated stimulation of the midbrain reticular formation in cats has resulted in small scattered myocardial lesions most marked in the subendocardium. Similar scattered lesions were reported in 8% of autopsies from a neurosurgical service. In another study actual myocardial infarcts were present at autopsy in 13 of 160 patients with cerebrovascular accidents and the clinical course suggested that the infarcts were secondary to cerebral disease.

At present, it is established that both characteristic and nonspecific abnormalities of electrocardiographic form occur as a result of neurologic lesions. The most likely mechanism of neurogenic ECG abnormalities is altered autonomic tone which may affect the ECG as a result of functional alterations of ventricular recovery time, by the production of anatomic lesions, or by a combination of these mechanisms. Whatever physiologic mechanisms operate, neurologic lesions should be added to the already long list of states in which the ST-T deflection may be abnormal. The possible neurogenic origin of such abnormalities should be considered in appropriate patients to avoid the erroneous diagnosis of primary heart disease.

J. A. Abildskov, M.D.

References
3. Korteweg GCJ, Boelen JTF, Cate JT: Circulation, Volume XLI, March 1970
Influence of stimulation of some subcortical areas on the electrocardiogram. J Neurophysiol 20: 100, 1957


Lament of a Lexicographer (1755) Pertinent to Editors Too?

It is the fate of those who toil at the lower employments of life, to be rather driven by the fear of evil, than attracted by the prospect of good; to be exposed to censure, without hope of praise; to be disgraced by miscarriage, or punished for neglect, where success would have been without applause, and diligence without reward.

Among these unhappy mortals is the writer of dictionaries; whom mankind have considered, not as the pupil, but the slave of science, the pioneer of literature, doomed only to remove rubbish and clear obstructions from the paths through which Learning and Genius press forward to conquest and glory, without bestowing a smile on the humble drudge that facilitates their progress.—Samuel Johnson: Preface to the Dictionary (1755).
Electrocardiographic Wave Form and the Nervous System
J. A. ABILDSKOV

Circulation. 1970;41:371-373
doi: 10.1161/01.CIR.41.3.371

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/41/3/371.citation