

# Neural Effects on Cardiac Rate and Rhythm\*

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The importance of autonomic discharge emanating from high neural regions rostral to the medulla oblongata on heart rhythm was first recognized in the classical studies by Schiff (5) and Danilewsky (1) during the late nineteenth century. Only in the last two decades, however, has a precise description of these neural effects on heart rhythm become possible. Crucial to these more recent advances have been improvements in technology which have permitted application of discrete electrical stimuli to various regions of the brain as well as continuous monitoring of heart rate and arterial pressure. These studies, reported here, represent a portion of a broader research project carried out in the neurophysiological laboratory of the Medical College of Virginia for a number of years and aimed at clarification of the role of autonomic discharge from higher neural centers on organ function (2, 4).

Briefly, the experiments were performed in anesthetized dogs, cats, and monkeys. Steel electrodes were guided under stereotaxic control into regions of the cortex and subcortex of the brain, and stimulation was delivered at a constant electrical current. Heart rate and arterial pressure were monitored continuously throughout the procedures.

In one series of experiments, stimulation of an area of the cerebral cortex within the cingular gyrus consistently produced severe disturbances of heart rhythm consisting of sinus bradycardia, sinus and ventricular arrest, and, frequently, atrial flutter and fibrillation (fig. 1). Section of the vagi

bilaterally abolished these arrhythmias. Thus, it was clearly demonstrated that certain regions of the brain were capable of exerting considerable control on parasympathetic function.

In a second group of experiments, electrodes were similarly placed more deeply into the subcortex in a region within the mesencephalic reticular formation. Stimulation of this region evoked striking increases in sympathetic efferent discharge which produced all of the ventricular arrhythmias observed in the clinical ECG. Furthermore, the arrhythmias were always evoked in a sequence; for example, sinus tachycardia was followed by ventricular fusion contractions, ventricular premature contractions, ventricular tachycardia, and rarely, ventricular fibrillation (3). It is particularly noteworthy that a graded increase in stimulus intensity was also capable of producing this spectrum of ventricular rhythm disturbances (fig. 2). If we sectioned the vagus nerves bilaterally, no change occurred in the responses observed. Administration of propranolol in small doses totally abolished all of the effects. Although alterations in arterial pressure occurred in many experiments, the changes were not closely correlated with the rhythm disturbances. Thus, stimulation of regions in the subcortex in the first series of experiments produced marked changes deeper within the brain than those observed in cardiac rhythm as a result of enhanced sympathetic discharge.

These experimental studies demonstrate that higher regions of the brain superior to the medulla may produce significant changes in cardiac rhythm as a result of alterations in autonomic discharge. Furthermore, in various regions of the brain, either

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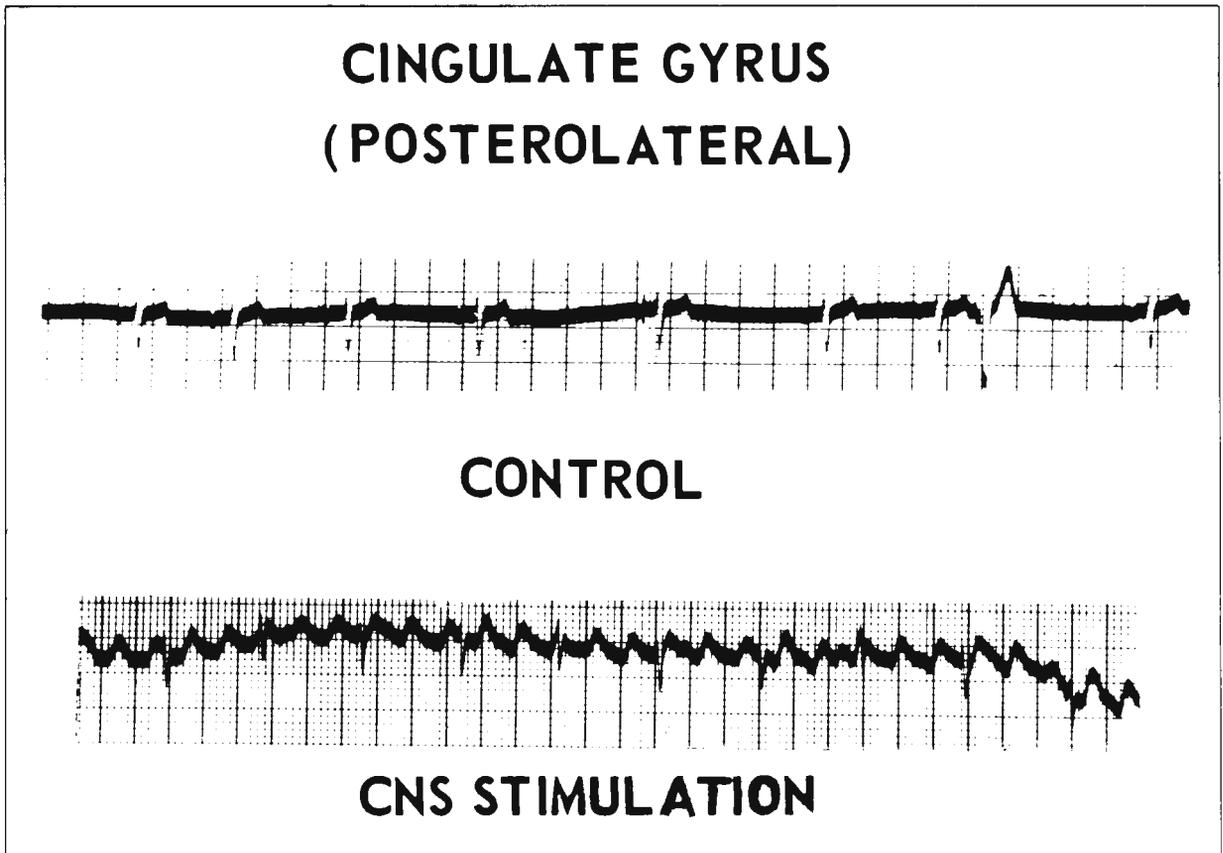


Fig. 1—The effect of stimulation of the cingulate gyrus on heart rhythm.

dominantly parasympathetic or sympathetic responses are produced. It is possible through these alterations in neural discharge to produce essen-

tially all of the arrhythmias observed by electrocardiography in the clinical setting.

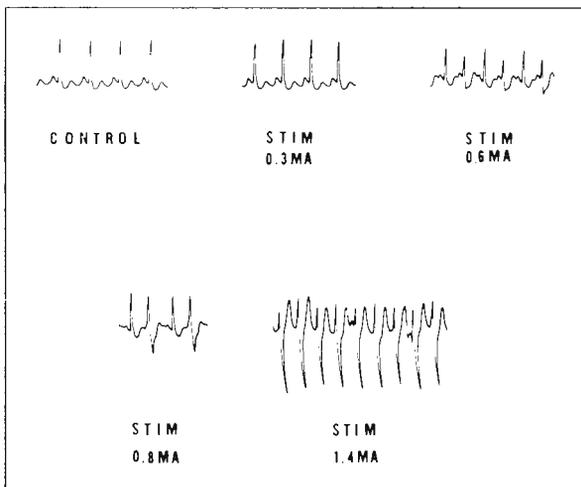


Fig. 2—The effect of graded increments in electrical stimulation of the mesencephalon on heart rhythm.

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