I want to emphasize that the vagus has effects upon both the sinus node and the A-V node. It slows sinus discharge, and it depresses, delays, or blocks atrioventricular transmission. It is also known that under certain conditions the discharge of vagal fibers to the heart may be phasic. From the physiological standpoint, it ought to be phasic, particularly at slow heart rates. The reason it should be phasic is that when the systolic pulse wave arrives at the baroreceptor regions of the aorta and of the carotid sinuses, it elicits a discharge of afferent fibers in those regions which goes to the central nervous system and leads to enhancement of vagal activity. By enhancement I mean an increase in the frequency of discharge. An increase in pressure in the pressure-sensitive regions of the arterial tree leads to an increase of vagal impulses to the heart. Since the systolic pulse rate is phasic, the changes in pressure in these baroreceptor areas are also phasic. One might reasonably expect that this is a closely coupled reflex arc; the discharge from the vagus nerve fibers would also be phasic and more or less locked to the systolic pressure cycle.

This has been recognized for a long time, particularly in one of the clinical situations that has aroused the curiosity of electrocardiographers through the years. When there is atrioventricular block so that the atria and the ventricles respond separately and independently, the condition known as ventriculophasic sinus arrhythmia has been observed. That is, if the basic rate of the ventricle following the development of A-V block is very slow, perhaps 30 beats per minute, and if the sinus rate is considerably faster, then it can be observed that the sinus discharge which follows a ventricular contraction is somewhat delayed. When the ventricle is beating once every two seconds, it will eject a large stroke volume with a wide pulse pressure, and there will be a sharp rise in systolic pressure. Thus, there will be a discrete stimulation to the baroreceptors, a resulting reflex discharge of vagal fibers to the heart, and a decrease in sinoatrial frequency. There are many possible ways of explaining ventriculophasic sinus arrhythmia, but the reflex vagal explanation makes the most sense and almost certainly is responsible. It has not been generally recognized that there might also be time-locked phasic changes in vagal activity to the atrioventricular conduction system; yet, this is also a possibility. Changes in conduction time or intermittent block, as in Wenckebach periodicity, could be reflexively induced through this baroreceptive mechanism, but the possibility of vagal activity has been rejected because there is often not a corresponding change in the sinoatrial cycle. In the process of another investigation, we discovered that the time course of vagal effects upon the sinus node and upon the A-V node are distinctly and discretely separate. These effects occur out of phase with one another, depending upon the heart rate. If the baroreceptor reflex produces alterations in sinus nodal activity which are out of phase with effects on A-V nodal activity, then one might expect that at one heart rate effects upon one system would...
predominate, and that at another heart rate effects upon the other might predominate. This is precisely what happens. Therefore, in the analysis of complex arrhythmias it is necessary to consider the time intervals between the possible phasic discharge in the vagus and the next event in the cardiac cycle. Experimentally, the overall time from a QRS complex to the expected effect upon sinus discharge is approximately 600 msec; the latency for effects on A-V transmission is approximately 400 msec.

Analysis of complex arrhythmias could very often be made much simpler if the electrocardiograms were recorded with a simultaneous record of arterial pressure, so that it would be possible to time the electrical events relative to the time and amplitude of the systolic pulse wave.