Reentry*

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There are two ways in which impulses can be generated: one is by the spontaneous discharge of a pacemaker cell in either the normal site or a subsidiary site, and the other is by reentry. The concept of reentry is a very old one postulated by clinical cardiologists many years before its demonstration.

The conditions that are necessary to permit this to happen have been known for at least 75 years. First of all, the conduction pathway must be blocked at some site; thus, an impulse arrives at a junction which is refractory or poorly excitable, and the margin of safety for continued propagation falls below the magic figure of 1. Second, there must be slow conduction over an alternate route to the tissue beyond the site of block. Since the cardiac tissue is largely syncytial in nature, there are always alternate routes around a localized area of block. Third, delayed excitation must occur beyond the site of the block. If that excitation is sufficiently delayed and if the tissue proximal to the site of the block is by that time recovered, it can then be excited from the opposite direction, and this would complete the circuit. Reentry can and probably does commonly happen. Reentry may be concealed; in other words, there may be localized reentrant activity which never escapes from that site because of refractoriness in the conduction pathway.

Therefore, it does not appear on the surface electrocardiogram, and it may not even appear in records from localized electrodes. So I repeat: of the four conditions necessary, the *sine qua non* is block. There has to be block for reentry to occur. Second, there is slow conduction over an alternate route to the tissue beyond the block; and third, there is delayed activation of that tissue beyond the block, and finally, reentry occurs.

* This is a transcription, edited by Dr. Charles L. Baird, Jr., of a lecture presented by Dr. Moe at the Symposium on Cardiac Arrhythmias, June 8, 1972, at Virginia Beach, Virginia.

PANEL DISCUSSION

Dr. Baird: Is it possible to differentiate from the scalar electrocardiogram whether the mechanism of ectopic beats is due to reentry or automaticity?

Dr. Moe: I do not think it is possible. The termination of supraventricular tachycardia or ventricular tachycardia by a single stimulus only suggests that reentry is the mechanism rather than automaticity. I am not nearly as comfortable as I was several years ago.

Dr. Hoffman: I feel at least as insecure as Dr. Moe.

Dr. Dreifus: Ventricular tachycardia due to digitalis toxicity is probably on an ectopic basis.

Dr. Moe: I agree, however, ectopic activity initiated by digitalis can also be the stimulus which generates circus movement. What starts something is not necessarily what continues it.

Dr. Baird: Can one differentiate the origin of ectopic beats from the scalar electrocardiogram?

Dr. Dreifus: I do not believe that this is possible.

Dr. Hoffman: Digitalis affects the cells of the specialized conduction system, and ectopic rhythms arise because of the depression of these specialized fibers which are more sensitive to the digitalis effects than ventricular muscle. Evidence is also accumulating that the internodal tracts of the atrium are more sensitive to the effects of digitalis than the atrial muscle fibers.

Dr. Moore: We have produced unifocal ventricular tachycardia experimentally in dogs, and mapping studies demonstrated that the left ventricle was the origin of the ectopic rhythm. Isolated studies of the Purkinje fibers from both the right and left ventricles also demonstrated that the left side was more sensitive.

Dr. Dreifus: Clinically, the ectopic beats occur at the site of the preexisting conduction block. However, it is very difficult to determine from where ventricle ectopic beats are arising, since the scalar electrocardiogram only provides the axis deviation.