Some Mechanisms of Supraventricular Tachycardia*

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Reciprocal rhythm was proposed about 30 years ago by Barker, Wilson, and Johnston as a probable mechanism for the explanation of supraventricular tachycardia. They proposed, on the basis of approximately 100 clinical records at the University of Michigan Hospital, that reentry through the A-V node, better known as reciprocal tachycardia, would account for approximately 40% of their cases. They also postulated that reentry through the S-A node might account for another 40%, and that 20% were probably due to ectopic foci. Ectopic focal activity is demonstrable in the laboratory, and it probably happens in man. Reentry or reciprocal activity through the A-V node can also be demonstrated, and it probably occurs in man.

Wilson and collaborators based their conclusions upon the termination of supraventricular tachycardia by brief periods of vagal stimulation induced by carotid sinus pressure. The postulate was that the effect of the vagus was to depress transmission or even block it within one part of the reentrant pathway. In those cases of reciprocal tachycardia in which the P waves were inverted, the postulate was that this was through the A-V node, and, therefore, the effect of the vagus terminating the episode was due to depressed conductivity or block within the A-V node. In another 40% in which the P waves were upright, the postulate was that the sinus node was the site of reentry and that the effect of vagal stimulation was to depress the conductivity in the sinus node. In others who had bizarre P waves and were unresponsive to vagal stimulation, it was postulated that these were in fact ectopic rhythms.

We have performed experiments through the years and arrived at the conclusion (much later than the clinical cardiologists) that dissociation can occur in the node. Reciprocation could conceivably be induced by premature stimulation of the atrium, which Dr. Scherlag has already shown you, or it could be induced by premature activity within the ventricle. Dr. Scherlag and I have both emphasized the use of a premature beat to initiate this kind of activity. That is not as artificial as it might seem; the only reason for introducing a premature beat, let us say to induce atrial reciprocation or an atrial echo, is to take advantage of the fact that during the relatively refractory period potentially dissociable pathways will be dissociated.

It is also conceivable, as Dr. Moore told you, to have concealed conduction. It is perfectly possible to have block below the site of the junction of two dissociated pathways, so that an impulse initiated in the atrium and returning to it fails to reach the ventricle because of the depressed conductivity below that junction. A premature atrial response which activates one pathway within the node returns to the atrium over an alternate route, reengages the first path within the node, and only then reaches the ventricle. Thus, we can have an impulse initiated within the atrium which takes, let us say, the alpha pathway down to the junction of the final common pathway which is still refractory and therefore fails to conduct to the ventricle. It nevertheless returns over the beta pathway of this y-shaped structure, activates the atrium, and reactivates the alpha pathway. By that time, the lower nodal pathway has recovered, and it is perfectly possible to have a 2:1 A-V block on the basis of circus movement within the node. Thus,

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paroxysmal atrial tachycardia with block does not exclude the possibility of circus activity.

I mentioned that Barker, Wilson, and Johnston proposed that some of these paroxysmal atrial tachycardias may be due to reentry within the sinus node. Dr. Han, in our laboratory, tackled this rather difficult problem seeking to demonstrate whether or not this was indeed possible. He explored the sinus node, rather laboriously, since this is a relatively difficult area to study over an extended period of time. It is easy to get responses from pacemaker cells within the sinus node, but it is difficult to hold them for a long enough period to get satisfactory evidence. At any rate, the technique here was to record an electrogram from atrial tissue within an excised scrap of muscle and to drive it for a time at a regular frequency, followed by a premature stimulus. Obviously, if this premature impulse is going to enter the sinus node and return, it had to fail to activate some elements of the sinus node; in other words, the one prime requisite for reentrant activity is that there has to be block somewhere. In Dr. Han's experiments, entry into the sinus node from the atrium and exit from the sinus node to the atrium were clearly not at the same sites. In other words, a loop was inscribed, and this accounts for the reentry. This is a possibility which was suggested by Dr. Hoffman about 15 years ago.

Atrial flutter has also been thought to be on the basis of a self-sustained reentrant circuit. The experimental technique for inducing flutter in dogs is first to crush an area of atrium in order to provide a circuit of suitable dimensions. The most convenient area lies between the superior vena cava and inferior vena cava. Thus, an obstacle is created which includes the crushed, nonconducting atrial tissue, plus the openings of the vena cava. Flutter can then be induced by stimulating the atrium at a rate more rapid than it can follow; in other words, to induce by electrical stimulation a brief period of atrial fibrillation. Upon terminating the stimulation, one of two things can happen—either the atria will stop momentarily until the sinus node resumes control of the activity or the atrial fibrillation will be replaced by flutter movement.

One of the characteristics of flutter is that sometimes it appears to drift into a state of fibrillation and back out. This has been taken as evidence that the same fundamental mechanism is involved both in flutter and in fibrillation, with the only difference being the rate of discharge of an ectopic focus. I think that one can understand that if the rate of discharge of an ectopic focus is sufficiently slow so that adequate time for recovery (and adequate does not need to be more than a few milliseconds) from the refractory state occurs between events, then the activation pattern of the atrium would be relatively uniform, abnormal but uniform. If, however, that pacemaker were to accelerate to a point where it impinged upon the refractory period of some elements within the atrium and not on others (and we call upon biological nonhomogeneity of the tissue to say that some elements may recover before others), then the activation pattern would become grossly irregular. Let us suppose that you have an impulse circulating around an obstacle, and that the dimensions of the obstacle and the refractory period of the tissue are such that the impulse is struggling to make it each time; that is, it just barely clears the refractory period. Now let us suppose we stimulate the vagus by carotid sinus massage. We will abbreviate the refractory period of atrial tissue which ought to make it easier for the circulating impulse to continue; it ought to accelerate. But when the vagus is stimulated there is not a uniform abbreviation of refractory period. The response is a spotty one because some fibers are closer to vagal endings than others; the effect of the vagus would be to abbreviate the refractory period and facilitate conduction in some areas of the circus loop, and to fail to affect it in others. Transmission will accelerate in those parts of the loop in which conduction is facilitated but will infringe upon fibers that are still totally refractory and cannot participate. This will fractionate the wave front and will generate fibrillation. This is the textbook picture of conversion of atrial flutter to fibillation by digitalis.