Atrioventricular Reentry
Lessons Learned From Radiofrequency Modification of the Node

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In a remarkable series of experiments, the late Gordon Moe and his colleagues hypothesized the existence of dual atrioventricular nodal pathways to explain atrioventricular nodal echo complexes elicited in a rabbit model. They postulated existence of an antegrade slow conducting pathway with a shorter refractory period compared with a fast conducting pathway. Thus, premature impulses that blocked in the fast pathway could conduct over the slow pathway and subsequently reenter the fast pathway to produce return impulses to the atrium. In this model, selective ablation of either pathway should prevent either echo complexes or tachycardia.

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The late Dr. Kenneth Rosen and his colleagues extended these concepts to humans, proving the existence of dual atrioventricular nodal function in patients with atrioventricular nodal reentrant tachycardia. They characterized fast and slow pathway conduction and also distinguished typical (slow-fast) from atypical (fast-slow) forms of atrioventricular nodal reentrant tachycardia. These pathways had different properties and different responses to drugs. Moreover, Josephson et al showed that all recordable atrial electrograms could be dissociated from the tachycardia, suggesting that the tachycardia circuit was entirely intranodal. The concept of atrioventricular node reentry tachycardia caused by an intranodal circuit with both an upper and lower common pathway was generally accepted.

This concept was challenged by Ross et al, who showed that atrioventricular node reentry tachycardia could be cured by dissection of atrial perinodal fibers without disruption of atrioventricular nodal conduction. Other surgical groups have confirmed these findings and raised the possibility that atrial perinodal fibers (originally proposed by Moe et al) are critical links in the tachycardia circuit. The proposed problems related to the implications of the surgical observations have been discussed elsewhere. In addition, the surgical approach failed to produce selective ablation of either pathway, and in some instances, atrioventricular node reentry tachycardia cure was achieved with maintenance of dual atrioventricular nodal pathways.

More recently, radiofrequency catheter ablation procedures have been introduced that have obviated the need for surgery and have provided important insights into the mechanism of this arrhythmia. Radiofrequency energy results in desiccation of very small amounts of tissue and, thus, should provide more precise localization of specific pathways. Lee et al for example, showed that radiofrequency lesions placed in the atrium just caudad to the area where the His bundle is recorded reproducibly results in selective ablation or modification of fast pathway function. This technique, however, is associated with a significant incidence of inadvertent complete atrioventricular block. Roman et al subsequently showed that ablative lesions placed between the coronary sinus os and the septal leaflet of the tricuspid valve selectively destroyed slow pathway function and essentially obviated the risk of complete atrioventricular block.

These seminal observations were confirmed and extended by Jazayeri et al and Kay et al, who found that selective slow pathway ablation not achievable by a strictly posterior approach could be successfully achieved by placement of radiofrequency lesions in the midseptal region (i.e., the region superior to the os of the coronary sinus and inferior to the His bundle). Both studies confirmed that successful slow pathway ablation was not associated with inadvertent atrioventricular block. Several additional recent observations have important implications with regard to the mechanism of atrioventricular node reentry tachycardia. Roman et al described discrete potentials thought to be associated with slow pathway activation. Ablation in the region of these potentials ablated the slow pathway. Confirmation that these potentials are associated with the tachycardia when the atria are dissociated would prove that a discrete tract (possibly atrionodal) insulated from surrounding atrial tissue is a critical component of the reentrant circuit. Jazayeri et al and Kay et al, however, showed that slow pathway ablation was possible without registration of these potentials.

Of note are the recent observations of Jackman et al showing that atrioventricular node reentry tachycardia could be reset by atrial extrastimuli delivered in the region of the slow pathway. These observations prove that the slow pathway input to the atrioventricular node is independent of a final common pathway. Confirmation of surgical evidence of the absence of an upper final common pathway was recently provided by McGuire et
al.16 Similarly, Chien et al17 showed that selective pacing from the earliest site of atrial excitation during tachycardia could result in dissociation of this area without affecting either the coronary sinus electrograms or the tachycardia cycle length.

What have we learned from radiofrequency modification of the atrioventricular node? First, the original concepts of Moe et al designating slow and fast pathway conduction have been conclusively validated in humans. In addition, to generate echo complexes or sustained tachycardia, a common lower pathway must link these two pathways. The site of this linkage would appear to be within the atrioventricular node, because the His bundle is not a necessary component of the reentrant circuit.18,19 The designation of a completely intranodal final upper pathway would appear to be overly simplistic. In fact, the tissue that constitutes the upper link between the atrioventricular nodal pathways is still a matter of debate. The ability to selectively destroy specific atrial areas that prevent tachycardia would prove that these sites are critical components of the tachycardia circuit. Still unclear is whether the ablative lesions are actually applied to the atrionodal region or whether alterations in the pattern of atrial input to the node determine whether tachycardia is still inducible.

The observations of Roman et al12 and more recently of Jazayeri et al13 and Kay et al14 prove that selective ablation of the slow pathway is the ablative procedure of choice for patients with atrioventricular node reentry tachycardia. It has also become clear that this technique is probably the treatment of choice for all patients with symptomatic tachycardia caused by atrioventricular node reentry tachycardia. Although the ability to selectively destroy the component pathways has extended our understanding of this fascinating arrhythmia, important gaps in our understanding still persist.

References


KEY WORDS • atrioventricular reentry • Editorial Comments
Atrioventricular reentry. Lessons learned from radiofrequency modification of the node.

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Circulation. 1992;85:1619-1620
doi: 10.1161/01.CIR.85.4.1619

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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http://circ.ahajournals.org/content/85/4/1619.citation

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