Editorial Comment

Atrioventricular Nodal or Atriojunctional Reentrant Tachycardia?*

MELVIN M. SCHEINMAN, MD, FACC
San Francisco, California

Ross et al. (1) in this issue of the Journal are to be congratulated on furnishing a bold and imaginative innovation in the surgical management of patients with drug-resistant 'atrioventricular (AV) nodal' reentrant tachycardia. Their seminal approach has wide implications both in terms of therapy and as potentially providing insights into the mechanism of this arrhythmia in humans. Although the diagnostic features of the arrhythmia have been defined, the basic mechanisms are still unclear.

Mechanisms

Role of atrial link in the tachycardia circuit versus intranodal reentry. Mendez and Moe (2) in 1966 presented cogent evidence for the existence of a dual AV nodal conduction system to explain atrial echoes in the isolated rabbit heart. These investigators also hypothesized the need for an atrial link in the tachycardia circuit. These observations were further supported by the studies of Wit et al. (3), in which an in vitro animal preparation was used. A series of both clinical and laboratory observations raised serious questions relative to the need for an atrial link in the tachycardia circuit. The experimental observations of Mignone and Wallace (4), together with those of Janse et al. (5), were interpreted as showing intranodal reentry. In addition, a series of invasive clinical electrophysiologic studies (6–9) in patients with 'AV nodal' reentry supported the concept that the atrium was not a critical link in the tachycardia circuit. Denes et al. (6) demonstrated the presence of dual AV nodal curves in the majority of patients with paroxysmal supraventricular tachycardia. Josephson and Kastor (7) elegantly demonstrated that the mass of atrial tissue could be dissociated from the tachycardia. These experimental and clinical observations painted a rather tidy schema of AV nodal reentrant tachycardia, namely, dual AV nodal conduction with reciprocation within the AV node. These observations gave birth to the term AV nodal reentrant tachycardia (a designation interestingly used by Ross et al. [1]).

Role of atrial perinodal fibers. The concept of sole reentry within the AV node was challenged by a number of subsequent observations. Gomes et al. (10) in a masterful, reasoned essay pointed out the consistent relation of the His deflection to the retrogradely conducted atrial impulse and suggested that this consistency was related to impulse propagation by way of an extranodal retrograde pathway. This consistency was corroborated by other investigators (9,11). In addition, Limura et al. (12), using an isolated rabbit heart preparation, emphasized the critical role of the atrial perinodal fibers in sustaining AV nodal reentrant tachycardia. In addition, the histologic basis for arrhythmias related to atrioHisian fibers was clearly confirmed by the studies of Brechenmacher (13).

Effect of surgery. In the report by Ross et al. (1), an imaginative approach is used to both cure the tachycardia and implicate the essential role of atrial fibers in clinical arrhythmias. Have the authors actually proved that a portion of the atrium is an essential portion of the tachycardia circuit? I think not. The surgical dissection used to expose the AV node and its approaches was rather extensive and may have produced nonspecific 'curative' trauma to the node itself. Such nonspecific damage may result from interruption of the neurovascular supply of the node incident to surgical trauma. The data provided could equally well be interpreted as showing nonspecific trauma to the node. While all patients showed disturbance of retrograde ventriculoatrial conduction, most also showed altered anterograde conduction. Could the latter reflect nonspecific trauma to the node with relative sparing of the fast pathway owing to its anatomic position relative to the dissection? The strongest supporting argument for atrial involvement in the tachycardia is detailed in Case 4, in which clearly AV nodal function remains intact but the tachycardia is no longer inducible. In the vast majority of patients (8 of 10) the critical AH interval required for preoperative initiation of the tachycardia could not be achieved!

Intralodal Versus Atriojunctional Tachycardia

The careful observations of Ross et al. (1) notwithstanding, the fundamental mechanism of this fascinating tachycardia in humans still remains a mystery. The ultimate solution to this important riddle may finally be clarified by three different approaches. 1) One approach may be the use of pharmacologic agents that have selective action on AV nodal cells. Neither verapamil nor beta-adrenergic blocking
agents would appear suitable, but preliminary studies (14) suggest that adenosine may, in fact, be such a drug. 2) Careful operative multielectrode mapping and atrial stimulation studies to discern the tachycardia pathway before and after selective atrial dissection should provide an excellent approach. 3) Although many patients with this arrhythmia have undergone careful electrophysiologic studies, there is a paucity of anatomic information. Because these patients age and die (hopefully of causes other than tachycardia), efforts should be made to obtain histologic evidence for or against the presence of extranodal pathways.

Role of surgery and other therapy. If the initial favorable results are maintained, then clearly the authors have provided an important new approach to the nonpharmacologic management of patients with so-called AV nodal reentry tachycardia. The authors are to be further commended for providing the details of their postoperative complications. Clinicians sorely need these data to best assess the benefit versus risk from among a variety of available nonpharmacologic approaches. These approaches include antitachycardia pacing (15) as well as catheter ablation of the AV junction (16).

Once again, a surgical approach has appeared to pave the way for a potential breakthrough in the management of patients with a frequently troublesome arrhythmia. Surely, the nimble-fingered catheter ablationists will not be far behind.

References