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Editorial Comment

Atrial Tachycardia: A Rare Disease Sheds Light on Common Questions*

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Research in arrhythmias, like that in many other areas, has passed beyond the descriptive to the mechanistic. It would be extremely desirable to understand how an abnormality of either a single cell or cell-cell connections results in electrophysiologic abnormalities and how these abnormalities are expressed in a clinical arrhythmia. Atrial tachycardia seems simple enough. It is a regular, monomorphic arrhythmia that appears, clinically, to originate from a well localized area in the flat structure of the atrium. It should be a candidate for early complete understanding. In this issue of the Journal, McGuire et al. (1) have provided important information linking clinical, electrophysiologic and histopathologic findings in 18 patients with atrial tachycardia. This study, additionally, points out some of the remaining difficulties in arrhythmia research but provides wider ranging data that may help to answer important fundamental questions.

"Mechanisms" of arrhythmias. In the study by McGuire et al. (1) 9 of the 18 patients had paroxysmal episodes and 9 had incessant episodes, whereas 8 had inducible tachycardia in the electrophysiology laboratory and 10 did not. Further analysis of their data, however, reveals a statistical association between inducibility and clinical history. In almost 80% of those with incessant tachycardia, no arrhythmia could be induced in the electrophysiology laboratory, whereas in 67% of those with paroxysmal tachycardia, an arrhythmia could be induced. Does this represent a difference in underlying electrophysiologic substrate, or simply a lack of sensitivity and specificity in intracardiac electrophysiologic testing? It would be desirable to develop better clinical tests for the positive diagnosis of increased automaticity. At present, most of the conclusions about abnormal automaticity are reached by negative inference: a tachycardia that cannot be induced or terminated by premature extrastimuli may be automatic, but it may also be reentrant or triggered with entrance block. Perhaps one future direction for research on cellular mechanisms may be provided by examining the effects of drugs in combination with sophisticated catheter electrophysiologic techniques. For example, the drug ethmozine has many electrophysiologic properties, but a very specific property concerns the abolition of abnormal automaticity. This drug has been unusually effective in patients with presumed atrial automatic tachycardias and may indicate that the cellular mechanism for the arrhythmia is enhanced abnormal automaticity (2). The sophisticated application of cellular electrophysiologic techniques in the clinical arena is in its infancy with catheter recordings of "monophasic action potentials" (3) and "upstroke slopes" (4) in automatic areas. The development of more sensitive pharmacologic probes and catheters that record from smaller groups of cells will be invaluable in the assignment of a true mechanism to an arrhythmia, rather than an inference based on vagaries of extrastimulus testing.

Right atrial disease. In the study by McGuire et al. (1), in each patient the tachycardia appeared to be originating from a single focus. If this were the case, excision of that focus should have resulted in cure of the tachycardia. However, the operation failed in almost 30% of the patients. Failure to eliminate focal tachycardia by excision of a 10 cm^2 area implies that the area of disease may be more widespread. In terms of pathophysiology, this makes a discrete tumor an unlikely cause, but is more in favor of a diffuse process such as myocarditis or a developmental abnormality in atrial automaticity.

Four of the five patients who had arrhythmia recurrence had noninducible ("automatic"?) tachycardia. In our patients with atrial automatic tachycardia, we have had a similar problem with "late" recurrence in patients with right atrial tachycardias whose P waves were similar to sinus P waves. In the immediate postoperative period, most patients undergoing open heart surgery have varying degrees of true sinus tachycardia and, unless electrophysiologic mapping is carried out, it is practically impossible to differentiate sinus tachycardia from another focus of atrial tachycardia in patients who have had an operation for right atrial tachycardia. It was only after several weeks had elapsed and the "sinus tachycardia" should have disappeared that we began to suspect the presence of atrial tachycardia. Catheter electrophysiologic mapping after surgery in these few patients confirmed that the P waves were not originating from the sinus node and that there was, in fact, another area of atrial tachycardia.

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Since that time, we have performed detailed mapping of the right atrium immediately after surgery for atrial tachycardias and have found in approximately 50% that, rather than the sinus node, another focus adjacent to the area of excision was responsible for the atrial rhythm. This finding has necessitated wider excisions, extensive cryoablation and maneuvers such as infusion of isoproterenol and superfusion of the right atrium with warm water to attempt to bring out other ectopic areas before the patient leaves the operating room. Since we have increased our index of suspicion, we have ablated all the areas of tachycardia in each patient and have had no late recurrences. It is possible that with an increased index of suspicion of wider areas of disease, the surgical cure rate can be improved.

Is the sinus node necessary? Perhaps the most intriguing observation by McGuire et al. (1) is that, when the sinus node was excised in seven patients, significant bradycardia occurred in only one. This finding demonstrates that electrophysiologically adequate pacemaker cells are present in lower parts of the atrium. It brings to question previous reports, especially in relation to bradycardia after operations for congenital heart disease, that discuss direct sinus node damage as the origin of the bradycardia. It is more likely that in these operations, a wide area of atrium is destroyed; in a similar way, in patients with "structural" sinus bradycardia, it is not simply the sinus node but the entire right atrium, that is diseased.

McGuire et al. (1) are to be commended for a detailed reporting of several clinical observations. Perhaps with better tools and more studies such as these, we can discover a true etiologic-structure-function relation in many of the arrhythmias that we encounter in clinical practice.

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