Sinoatrial reentry tachycardia: A review

History and electrophysiology

The concept of reentry within the sinus node is by no means new. In their 1943 report, Barker and co-workers postulated that “...a circus rhythm could be accommodated in auricular muscle and in one of the specialized nodes at known rates of conduction and with cycle lengths such as occur in paroxysmal tachycardia” (1). Lack of invasive electrophysiology at that time and subsequent failure to appreciate the heterogeneity of supraventricular arrhythmias, left their astute observation in the realm of conjecture. It was not until 1968 that Han, Malozzi and Moe finally demonstrated the existence of sinoatrial echoes (2).

In a superfused isolated rabbit right atrial preparation, they examined the response to premature extrastimuli with an 18 electrode grid at the sinus node and surrounding atrial tissue. They found that critically timed extrastimuli led to early re-excitation of the atrium, supposedly due to sinoatrial reentry. Sequential microelectrode measurements of sinus node and atrial transmembrane potentials were performed in an attempt to provide a temporal and anatomical map of the observed phenomenon. Based on findings, the authors concluded that sinoatrial reciprocation was caused by entrance block at one site, slow conduction within the node with disparate refractoriness, and re-excitation of the atrium at the original area of entrance block. They went on to suggest that repetition of this phenomenon may conceivably form the basis for clinically relevant tachycardia. In vivo confirmation of sinoatrial echoes was inferred from work by Childers (3) and Paulay (4). In the former study, programmed electrical stimulation was performed in
dogs. Three responses to premature atrial stimuli were noted: complete interpolation (i.e., the subsequent sinus complex was on time), incomplete interpolation (i.e., the subsequent beat was delayed), and sinus echo. In the latter, the first post-extrastimulus complex was electrocardiographically the same as sinus, but earlier than expected. The authors attributed this to sinoatrial reentry, and went on to describe each of the three responses to premature atrial beats in a 70-year-old man in an elegant electrocardiographic deduction of underlying mechanisms. An in vivo canine model was also employed in the report by Paulay et al. While confirming Childers' findings, Paulay offered two additional pieces of evidence implicating the sinus node/perinodal area: first, timing of the echo was independent of stimulation site (they performed pacing at the sinus node, Bachmann's bundle, right and left atrial appendages and low atrial septum), and second echoes were eliminated by crushing the sinus node. As has long been the case for AV nodal reentry tachycardia, it is apparent that considerable discussion also revolves around the issue of whether sinoatrial echoes do or do not involve perinodal atrial tissue as well as the sinus node itself. In 1979, Allessie and Bonke published work suggesting confinement of the re-entrant circuit to the node per se (5). The experimental set-up was similar to earlier work by Han (2), with the significant difference that resolution of the measuring electrodes was superior: 32 unipolar surface atrial electrograms were recorded during initiation of sinoatrial echoes as opposed to the 18 used by Han. In addition, up to 130 transmembrane sinus node potentials were recorded sequentially in cases of sustained reciprocation. These detailed observations led to the conclusion that sinus node reentry was confined to a circuitous pathway in an extremely small area (1–2 mm) with low conduction velocities (2.5 cm/s) within the node itself. It is of note that the same authors pointed out the inability to induce sustained arrhythmia in this model, and later concluded that sinoatrial reentry tachycardia (SART) may not be feasible without border zone atrial tissue. Given the limitations inherent to in vivo study in man, it may be fair to continue to use “sinus node reentry” and “sinoatrial reentry” synonymously for the time being. In the wake of in vivo studies in dogs, and reports of sinus node reentry in man by Paulay (6) and Childers (3), Narula first described sustained SART in two patients in 1974 (7). Out of 300 patients undergoing electrophysiologic study, he observed sinus node reentry beats in 20, and sustained tachycardia in two. The criteria for the diagnosis of SART proposed by Narula are still valid: 1) atrial activation and P-wave morphology are the same or highly similar to sinus rhythm, with activation from high to low right atrium, 2) the arrhythmia is inducible with atrial extrastimuli at specific coupling intervals, independent of AV nodal conduction intervals and site of stimulation, and 3) the arrhythmia can be terminated by atrial stimuli. SART is thus, by definition, a paroxysmal arrhythmia.

**Clinical presentation**

Symptoms of SART vary widely. Patients may present with paroxysmal palpitations, dyspnea, dizziness, (near-) syncope, chest discomfort and other symptoms (8). Given the electrocardiographic similarity to sinus tachycardia and the on average lower heart rates than, for example, circus movement using a concealed bypass (9), a degree of under-diagnosis is likely. Patient's symptoms can in fact be so reminiscent of anxiety disorders that a psychiatric diagnosis is initially entertained and referral to a cardiologist unnecessarily delayed. Again, electrocardiography is usually typical, with sinus rhythm morphology P waves and RP:PR ratio of greater than one. In one report, however, prolonged AV nodal conduction leading to superposition of the P wave on the preceding T wave or ST segment was shown to cause confusion with a diagnosis of AVNRT in 27% of a cohort of 65 patients undergoing EP study for symptomatic paroxysmal SVT (10). In general, there are only two serious electrocardiographic differential diagnoses: inappropriate sinus tachycardia, and atrial reentry tachycardia arising near the sinus node (11). The former differs primarily in its non-paroxysmal character, with an often elevated resting rate and gradual but excessive acceleration as a reaction to even mild exercise; the latter, also being a paroxysmal arrhythmia, can be extremely difficult to differentiate from sinoatrial reentry. Reaction to vagal maneuvers and adenosine may provide clues to the correct diagnosis, as both tend to terminate sinoatrial but not intratral reentry tachycardia. Differentiation between SART, inappropriate sinus tachycardia and atrial reentry tachycardia is detailed in Table 1. Reported incidence of SART varies widely, although it is by any standards a relatively uncommon arrhythmia. A study by Wellens and co-workers demonstrated SART in only 1.8% of 379 patients undergoing diagnostic EP study (12); however, the large proportion of patients included with VT and atrial flutter may cause underestimation of the overall prevalence in the general population. At the other end of the spectrum, Gomes et al.