Enhanced AV nodal conduction and Brenchenmacher tracts

Scheinman M.Enhanced AV nodal conduction and Brenchenmacher tracts. Pacing Clin Electrophysiol. 2013 Feb;36(2):135-6.

Early electrocardiographers focused on the relationship of a short PR, normal QRS, and supraventricular tachycardias. This entity was actually first described by Clerc et al. In 1938 (1) and reemphasized by Lown, Ganong, and Levine (2) in 1952. The LGL syndrome bears their eponym. Invasive electrophysiologic studies were subsequently used to scrutinize patients with short PR intervals. (3–5) These studies arbitrarily suggested the following criteria for enhanced atrioventricular (AV) nodal conduction based on invasive testing.

1. AV nodal conduction time less than 60 ms during sinus rhythm (normal 80–120 ms).
2. 1:1 AV conduction at atrial paced cycle lengths ≤ 300 ms.
3. An increment of atrial-His (A-H) ≤ 100 ms comparing the shortest atrial-paced A–H with that during sinus rhythm.

Further studies (6–8) emphasized that the AV nodal refractory period was short in these patients and that a small minority of patients would show an essentially flat A–H response with respect to atrial overdrive pacing with an unchanged A–H during programmed atrial extrastimuli. A seminal study was reported by Jackman et al. (9) in an invasive electrophysiologic study (EPS) of 180 patients. They found that enhanced AV nodal conduction could not be separated from normal AV nodal physiology. They clearly found a spectrum of A–H intervals in a uni-modal continuous distribution. These studies (6–9) clearly show that the LGL syndrome eponym should be abandoned since the presence of a short PR is not a necessary

feature for the tachycardia substrate. Specifically, patients with AV nodal reentry, atrial tachycardia, atrial fibrillation, etc., may suffer from these arrhythmias whether the PR is short or normal. A more interesting endeavor is the attempt to obtain anatomic correlates for patients who have the criteria for enhanced AV nodal conduction.

Most of the attempts to make this correlation were largely speculative. Specifically, this findingwas attributed to a hypoplastic AV node (10) or a small or fetal AV node. (11,12) Other attempts to correlate anatomy with pathophysiology involved attributing enhanced nodal conduction to the presence of James tracts. (13) These fibers course from the upper portion of the AV node or atrium and insert on the lower nodal region. These fibers might serve as a possible explanation for the phenomenon of enhanced AV nodal conduction in that conduction over these fibers might serve to bypass the AV nodal and explain the loss of intrinsic delay at the nodal level. The difficulty with this explanation is that these fibers are universally found in normal subjects, making it difficult to attribute enhanced conduction to a small subgroup of individuals. Moreover, Mahaim (14) described fibers that emanated from the AV node or His bundle traversed the central fibrous body to insert into the ventricle or fascicles. Antegrade conduction over these fibers should result in a pattern of preexcitation and could not explain the short PR and normal QRS configuration.

The only clear-cut anatomic electrophysiologic correlation for enhanced nodal conduction comes from the seminal observations of Brechenmacher et al.,(15,16) who clearly described the very rare finding of atrio-Hissian bypass tracts in patients who died suddenly. He described the finding in only two of 687 studied human hearts. In the first description (15) the atrial tract originated from the interatrial septum and descended vertically, bypassing the node to insert into the proximal portion of the His bundle. In the second case (16) the tract inserted into the distal His bundle at the site of branching into the right and left bundles. The first case had the expected short PR and normal QRS and suffered from paroxysmal tachycardia.

The second case had a PR interval of 160 ms and a left bundle branch pattern. Conceivably, the longer PR might have been due to infranodal disease since fibrosis was present in both the left as well as right bundle branches. No electrophysiologic studies were available for these cases. (15,16) In the present report by Professor Brechenmacher is yet one additional patient with sudden cardiac death. In this patient clear-cut abnormalities were found in the central fibrous body which did not cover the upper margin of the His bundle and was the site of insertion of the atrio-Hisian fiber. This case adds to the previous observations and incorporates important electrophsiologic findings that corroborate complete electrical bypass of the AV node. Since it is apparent that a dearth of correlations exists between anatomic findings and the presence of enhanced AV nodal conduction, cells. For example, the absence of Ik1 current (20) explains the lower resting potential which effectively inactivate Na+ channel and explains the central role of inward Ca++ currents. Enhanced inward currents could serve to explain more rapid AV nodal conduction. Other considerations include the type, density, and distribution of connexons which serve to modulate cell-to-cell conduction. (21) Finally, the autonomic nervous system pays a key role. The fat pad situated between the inferior vena and left atrium contains efferent fibers to the AV node and their stimulation serves to depress AV nodal conduction. (22) It is clear that absent any clearly defined anatomic cause (apart from the elegant and important observations of Professor Brechenmacher) for enhanced AV nodal conduction, more attention needs to be focused on changes of transmembrane currents, connex on physiology, as well as the autonomic nervous system to better explain facilitated nodal conduction. other explanations need to be explored. The AV junction is divided into the transitional cell area which is defined as the junction between atrial myocardium and the compact node, the compact node itself, and the lower nodal bundle. (17) Most of the conduction delay appears to occur within the ovoid cells of the compact node which is the site of decremental AV conduction. (18) Depolarization of the ovoid cells is dependent on slow inward current. (19)

Nodal membrane currents differ markedly from those found in “working” atrial or ventricular

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