EDITORIAL COMMENT

Diagnosing the Mechanism of Supraventricular Tachycardia
Restoring the Luster of a Fading Art*

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In an era focused on voluminous data entry and efficient, “definitive” diagnostic tests, establishing a correct arrhythmia diagnosis on the basis of the fundamentals of the doctor-patient relationship, a good history, and physical examination has frequently become relegated to an afterthought of historical significance. The battle cry of “Let’s take the patient to the EP lab and figure it (the mechanism of supraventricular tachycardia) out” has frequently replaced the sleuth-like pleasure that accompanies making a correct diagnosis from a rigorous analysis of the surface electrocardiogram (ECG) and a thoughtful history. We all marveled when shown the translated report of Karel Frederik Wenkebach’s description of Type 1 second degree atrioventricular (AV) block preceding the advent of ECG on the basis of his careful analysis of the patient’s arterial and venous pulsations, and yet when I recently sampled one-half dozen of the “best and brightest” cardiology fellows to ask whether they had asked about neck pulsations during a supraventricular tachycardia (SVT), they all gave a negative response (1). Although optimistic that my cajoling on the importance of such an effort might have some influence, we all can be more confident that the important analysis described by Gonzalez-Torreccilla et al. (2) in this issue of the Journal will reignite the fire of enthusiasm that should exist for establishing the correct diagnosis on the basis of the analysis of the 12-lead ECG during the SVT and a basic assessment of bedside clinical variables.

In their report, the correct ECG diagnosis of the SVT mechanism was established with the presence of a pseudo r' deflection (V1) and/or pseudo s-wave (inferior leads) to favor atrioventricular nodal re-entrant tachycardia (AVNRT) and a distinct, identifiable P-wave after the QRS complex and QRS alternans to favor atrioventricular reciprocating tachycardia (AVRT) in 68% of the patients (2). The presence of important clinical variables such as age, sex, and presence of palpitations in the neck could be used to help confirm the ECG diagnosis. Female sex, older age, and the presence of neck palpitations all favored the diagnosis of AVNRT. As stated, the predicted probability increased to >90% when >2 positive clinical findings are present in conjunction with the appropriate ECG clues consistent with the diagnosis of AVNRT (2). Importantly, the presence of neck palpitations was a positive predictor for the diagnosis of AVNRT even in the absence of a definitive ECG diagnosis.

Clinical Merit of Establishing the Correct Diagnosis

Diagnosing the probable mechanism of SVT before an electrophysiological evaluation and ablation procedure can not only produce the pleasure of correctly solving the puzzle—it has important clinical merit. Precise probabilities for a correct diagnosis associated with combinations of simple ECG and clinical criteria improve the accuracy and effectiveness of information shared with the patient and their family. Patients can be better counseled on the risk of the appropriately anticipated ablation procedure, and ablation tools required for success and safety can be selected in advance. The modest risk of heart block can be detailed for patients undergoing AV nodal modification for AVNRT. Given the fact that most bypass tracts are left-sided and confirmed with a discrete negative p-wave in lead aVL during SVT, patients can be told about the possible need for transseptal puncture with its attendant bleeding and the subsequent, fortunately rare, thromboembolic risk of a left-sided ablation (3). Outcome with respect to successful ablation is somewhat lower with a posterior septal accessory pathway, and the described diagnostic clues coupled with an assessment of the p-wave morphology in ECG leads V2, V3, and aVF (negative in posterior septal pathway) and lead aVL during the SVT should allow for an approximation of the ablation target and better prediction of anticipated outcome even with a concealed accessory pathway.

Physiologic Basis for Clinical Clues to SVT Diagnosis

Identification of clinical variables that have diagnostic predictive value should also make one seek a physiologic and anatomic basis for observed relationships. A pounding sensation in the neck during AVNRT is the result of cannon waves, when the right atrium contracts against a simultaneously contracting right ventricle and was perceived as such; 51% of the study patients with the typical form of AVNRT reported the sensation (4). The physical examination correlate of the continuous pulsing cannon A waves has been described as a “frog” sign for obvious reasons (5). Although the atrial contraction during AVRT will occur against closed AV valves, the longer ventriculo-atrial interval results in separate ventricular and then atrial contraction...
and a relatively lower right atrial and venous pressure (4). Of note, the presence of palpitations in the neck was still experienced by 17% of the patients in the current report, which is somewhat higher than previously noted (2,4). Perhaps the questions asked of the patients in the current study were too directed, and a more open-ended question related to palpitations might have resulted in fewer patients with AVRT identifying dominant pulsations in the neck. It would have been interesting to obtain confirmation of symptoms experienced during induced arrhythmias in the electrophysiology laboratory.

The striking >2:1 predominance of women for AVNRT is well-recognized (2,6,7). It has also been described for outflow tract ventricular tachycardia or frequent ventricular premature depolarizations and inappropriate sinus tachycardia (8,9). The anatomic and electrophysiological basis for the observed increased frequency in AVNRT in women remains puzzling. Women with AVNRT have shorter slow pathway refractory periods, AV block cycle lengths, and tachycardia cycle lengths than men with AVNRT (6). There are no reported sex differences in fast pathway effective refractory period and retrograde conduction. Therefore, women have a wider “tachycardia window,” representing the difference between the fast and slow pathway refractory periods, which might explain the higher incidence of AVNRT (6). An increase in the length of the posterior extension of the AV node and slow pathway has been associated with an increase in the width of the coronary sinus os and might predispose to AVNRT possibly by increasing the length of the posterior extension of AV nodal slow pathway. However, this anatomic finding that has been reported to be noted more commonly in patients with AVNRT was not more common in women (10).

**AV Nodal Re-Entry as an Acquired Disease**

It is interesting to speculate regarding the important relationship of the age-related increase in observed AV nodal re-entry. An average of more than 10 years separates the time of clinical presentation of AVRT versus AVNRT (2,7). AVNRT is virtually unheard of in children under 5 years of age. Furthermore, young children <13 years of age have an incidence of dual AV nodal physiology of 15% compared with 44% in the adolescent population (11). The increase in dual AV nodal physiology and AV nodal re-entry with age has been incompletely explained. Microfibrosis associated with aging produces nonuniform anisotropic conduction or changes in membrane ionic properties that could contribute to the development of re-entry. This has been described for the anterior portion of the AV node and contributes to an increase in His–atrial time noted with age (12). However, the disease typically initially manifests in early mid life, at an average age of 39 years in the current report; so, age-related changes cannot be the entire explanation. It is also possible that elderly patients are more likely to present clinically with atrial fibrillation rather than a sustained regular tachycardia with the triggering event AVNRT (13). Thus, the true incidence of AVNRT in elderly persons might be underestimated. Of note, there was a modest increase in heart disease in patients with AVNRT also observed in the current report (2). Perhaps AV nodal re-entry represents an acquired perivalvular post-inflammatory fibrotic process in some patients. Such perivalvular fibrosis seems to be the common link for the ventricular tachycardia substrate in nonischemic cardiomyopathy (14). There is no reason that the process cannot affect both sides of the valve structure and involve the slow pathway region. Of note, the link between AVNRT and outflow tract ventricular arrhythmias seems more than coincidental (15). An epicardial/myocardial inflammatory process can both alter the autonomies to favor enhanced sympathetic efferent activity and/or cause sufficient microfibrosis to result in both arrhythmias without necessarily causing depression of myocardial function. This would help explain both the increase in heart in patients with AVNRT versus AVRT and the strong relationship of AVNRT and right ventricular outflow tract tachycardias (16). Animal studies of experimentally created AV nodal re-entry support a possible anatomic basis for its development that is beyond normal development and simple alterations in autonomic balance and support the possibility of an acquired abnormality (17).

In summary, ECG analysis and clinical observations serve patients and the medical community best when they have clinical merit and force us to speculate and then investigate the physiologic basis for the observations made. The findings described by Gonzalez-Torrecilla et al. (2) accomplish both.

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**REFERENCES**


Key Words: clinics • diagnosis • electrocardiogram • supraventricular tachycardia.