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Assessment of the Delta Wave: Wolves in the 90's

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Introduction

When Wolff, Parkinson and White first described the syndrome that carries their name in 1930, it was thought to be a sign of good health.[1] Since then, WPW syndrome has been shown to be rarely associated with sudden cardiac death (SCD),[2] in addition to its more common manifestation with tachyarrhythmia symptoms. Generally, the main clinical question concerns quality and not quantity of life. Up to a third of persons who have this syndrome are asymptomatic.[3] The clinician must face questions with respect to those who are asymptomatic and suggest management options in the symptomatic patient.

The Asymptomatic Patient

The prevalence of ECG pattern suggestive of WPW has been estimated to be 0.1-0.3% of the population, and the incidence has recently been reported around 4 per 100,000 persons per year. Many patients have an accessory bypass tract documented, as part of a routine computerized ECG interpretation. The first question to be addressed is whether the delta wave is real or a non-specific artifact due to delayed conduction in the proximal septum often seen in the setting of left ventricular hypertrophy or incomplete left bundle branch block. Unlike many computer algorithms, we generally expect to see a true delta wave to be present in 2 out of 3 orthogonal (lead 1, V1, AVF) leads. As well, since a delta wave is a fusion beat one may be able to increase the amplitude of a delta wave (with no concomittant change in the PR interval) with carotid sinus massage or adenosine infusion.

The chief concern in the setting of an asymptomatic delta wave concerns the risk for sudden cardiac death (SCD). Sudden cardiac death can occur in patients with a bypass tract when it has the ability to rapidly conduct impulses from their atrium to the ventricles. Death usually occurs in the setting of atrial fibrillation or fast atrial re-entrant rhythm, with a ventricular response not controlled by the normal filtering functions of the AV node. The ventricle is therefore stimulated at rates sufficient to cause the rhythm to degenerate to ventricular fibrillation. Sudden death in WPW patients has been estimated to occur at most in 1 per 1,000 patient-years.[4] This can even be the presenting symptom in particularly rare cases.[5,6] Sudden death as the initial manifestation of WPW is extraordinarily rare, and the very long term follow-up of asymptomatic patients is generally benign.[7] Nonetheless, with the advent of invasive electrophysiological studies, there has been a significant effort to define risk factors for malignant arrhythmias among the

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asymptomatic population with a delta wave as their only ECG abnormality.

The most potent risk factor is the presence of atrial fibrillation and reciprocating tachycardia. In patients who are resuscitated from SCD and were found to have WPW, the majority have atrial fibrillation with a rapid ventricular response. Atrial fibrillation, alone, is not very specific since 10-20% of patients with WPW syndrome suffer from paroxysmal atrial fibrillation.[8] The occurrence of syncope was also evaluated and not found to be significantly different between patients who had ventricular fibrillation and those who did not.[9] It is now appreciated, in fact, that syncope in the setting of Wolff Parkinson White *syndrome* is often due to vasovagal phenomenology occurring at the very onset of an organized re-entrant arrhythmia in the setting of a normal heart.[10] The response of delta wave behaviour to acute infusion of intravenous type I antiarrhythmia agents has also been found to be of little use for SCD risk prediction.[11] More recently, it has been suggested that septal bypass tracts and male sex may have an adverse prognostic value;[12] but since two-thirds of the patients are males, it is not clear how useful this may be.

A short RR interval at the time of atrial fibrillation, a short ventricular refractory period, and the presence of more than one bypass tract have all been found to be of prognostic value. This should not be surprising since the ventricular rate in pre-excited ventricular fibrillation is a function of the ability of sequential impulses to conduct through the bypass tract to ventricular tissue. As a result, the one test that has had the greatest study is the shortest R-to-R interval seen in pre-excited atrial fibrillation (SRR). Patients with an SRR interval of 220-250 ms, and especially patients with induced atrial fibrillation who have a SRR of <180 ms, have the worst prognosis.[4,6] Unfortunately, however, the specificity of this predictive variable in the setting of a rare phenomenon is relatively low; and short pre-excited RR intervals may be found in 17% of the patient population. Nonetheless, there are many who believe that therapy should be delivered to patients who have a SRR of <220 or even 250 ms. The implication that this necessitates the induction of atrial fibrillation in all patients who have WPW is controversial. In our own practice, if the patient with WPW is completely asymptomatic, we outline the very small risks and are disinclined to routinely offer prognostically-based invasive electrophysiological studies. This recommendation is weighted by those who may have particularly dangerous occupations where symptoms may lead to harm to others (pilots, truck drivers, etc.)

The Symptomatic Patient

Physiology

Tachyarrhythmias occur in WPW due to three reasons: The first are due to re-entrant arrhythmias. By history this implies sudden onset and offset of the tachyarrhythmia. Offset may occur either with intravenous drug infusion, vagotonic manoeuvres, or spontaneously. During the tachycardia, the conduction from atrium to ventricle occurs through the AV node in 90% of cases[13] with the result that the rhythm is usually narrow complex, although rate-related aberrancy may occur. The retrograde direction from ventricle back through atrium occurs through the accessory pathway. This pattern is called orthodromic reciprocating tachycardia. Since the AV node is an obligatory member of the re-entrant circuit, any intervention that causes transient AV nodal block will terminate the tachycardia. As such, adenosine, intravenous ß-blockers or calcium channel blockers are all potent techniques to terminate the arrhythmia.[14-16] The ability of these agents to terminate the rhythm is independent of whether there is a delta wave at rest. The common medical school teaching that all patients with WPW should never receive direct AV nodal blockers ignores this simple physiological effect.

The more rare form of re-entrant tachycardia in WPW involves the conduction of impulses, from atrium to ventricles, through the bypass tract with retrograde conduction occurring through the AV node. In this case, since the tachycardia traverses the AV node in a retrograde direction, the tachycardia is commonly called antidromic re-entrant SVT. Again, all the comments regarding termination of the rhythm noted above are present.

The accessory pathway itself can be directly interdicted with either rhythm by the intravenous infusion of any conventional antiarrhythmic agent, including intravenous procainamide, quinidine or amiodarone.

The third rhythm at presentation for WPW may be atrial fibrillation. In the setting of a patient capable of conducting through the accessory pathway, from atrium to ventricle (i.e., having a delta wave present in sinus rhythm), the manifestations will be that of an irregular tachycardia with wide-complex beats generally seen and occasional sudden narrowing of beats when pathways refractoriness is reached at a time when the AV node can accommodate an impulse. In this situation, the pre-excited (wide complex) ventricular rate is an indirect measure of pathway refractory properties. In this rhythm, use of direct AV nodal blocking agents may potentially enhance the ventricular rate and should be avoided. This is particularly so with agents that shorten atrial refractoriness, such as intravenous digoxin and adenosine.

Concealed Conduction

Approximately 50% of patients with WPW syndrome have no delta wave present on the ECG. This phenomenon is referred to as concealed conduction, i.e., the accessory pathway is only capable of conducting from ventricle back to atrium. In this situation, the presence of WPW syndrome may only be inferred noninvasively and requires invasive procedures to prove. The suggestion that a tachycardia is mediated by a concealed pathway is based on the occurrence of a sudden-onset/sudden-offset clinical history with a discrete P-wave seen during the tachycardia after the QRS and according to some studies (but not all) the presence of beat-to-beat alterations (alternans phenomena) in QRS amplitude.[17] As well, one should consider the degeneration of an organized bypass-tract-mediated re-entrant tachycardia to atrial fibrillation in young patients who present with paroxysmal atrial fibrillation. Historically, one should seek out evidence that the arrhythmia began as a regular rhythm that then degenerates commonly to atrial fibrillation or, ideally, attempt to document the onset of the atrial fibrillation. In rare cases we have performed diagnostic electrophysiological studies for young patients with paroxysmal atrial fibrillation in whom this phenomenon is suspected.

Localization of accessory pathway

In patients who have an overt pathway present, a delta wave will always be present. In these situations, it is useful to try to locate where the accessory pathway is located via a non-invasive analysis of the 12-lead scalar ECG. This is of use with respect to strategizing the approach to an ablation procedure. (See table.)

TABLE 1

Common delta wave patterns in WPW	
Pattern	Implication
Positivie delta wave in II, III, AVF Q-wave in V_1, V_2, V_3	Right free wall, all venous ablation procedure, Ebstein's anomoly R/O.
Negative delta wave, i.e. Q-wave in I, AVL, and positive delta wave in V_1-V_6	Left free wall; access to left side for ablation via retrograde aorta or transeptal
Q-wave in II, III, AVF (i.e. negative delta wave) Q-wave in V_1, positive delta wave in V_3 and either positive delta wave or negative delta wave (Q) in V_2	Posteroseptal; 75% accessible via right side

Therapeutic Choices

There is no consensus on optimal long-term therapy for WPW syndrome. All antiarrhythmia drugs have a role in the prevention of a tachycardia recurrence. Since these patients generally have no structural heart disease, the type IC agents are particularly effective.[16,18] Class IA drugs were previously agents of choice but may be less potent than the type IC agents. Amiodarone is also highly effective through its multiple electrophysiological effects. Sotalol[16] is also highly effective and has the added effect of providing concomittant ß-blocking efficacy should tachycardia recurrence take place. The more fundamental question to determine at the outset is whether drug therapy is to be used preparatory to an elective admission for an ablative procedure or be used in a long-term role. An intermediate approach of intermittent drug therapy

only upon the occurrence of tachycardia events can be used theoretically but has had no significant study.

Ablative Therapy

Drug treatment is highly effective in the treatment of tachycardia but carries with it the risk of potential side effects of the various agents, the burden of long-term drug treatment especially in young, otherwise-healthy persons, and the significant cost and at times limitations imposed on individuals, particular in those with high-risk jobs. As well, there is an often overlooked psychological dimension to patients who may be fearful of significant travel time abroad or disabled due to the fear of recurrence more than the physiological sequellae of recurrence. Certainly, all patients with WPW have to be aware of the fact that catheter ablation techniques are available and very effective. Our own bias is to recommend ablation for any patient who is about to contemplate the requirement for long-term drug-suppressant therapy beyond the use of simple agents such as digoxin. As well, women in child-bearing years, or any patient who has required at least one emergency room admission for tachycardia termination, should be considered for an ablation procedure.

The first successful surgical ablation of an accessory pathway was performed in 1968.[19] However, despite its success in specialized centres of expertise, generally surgical approaches to the accessory pathway are of historical interest only. Radiofrequency catheter ablation is the modality of choice. The overall success rate has been reported to range from 83-99%[20] and is to a degree operator-dependent. Most experienced laboratories can anticipate 90% success with a 5.5-10% recurrence rate and a significant complication rate of 1.8-4%.[20] In patients with left-sided pathways, complications that must be mentioned include the risk of arterial embolism or tamponade (mostly in relation to trans-septal approach). Other complications that occur may include bleeding, pseudo-aneurysm, arterial thrombosis, embolysm, fistula formation, pneumo-thorax, myocardial infarction, and inadvertent AV block (in septal pathways). The risk of death is probably exceedingly low and in the range of 0.1%.[16] Overall, the success and complication rates mainly depend on accessory tract location and operator experience. We generally suggest that there is a 1-2% risk of significant procedural morbidity and a very low rate, never quoted, for procedural mortality and stroke.

The exact benefit of catheter ablation on patient-perceived, health-related quality of life has been quantitated.[21] We have found that the health-perceived quality-of-life measures on generic scales of patients who have SVT is significantly high, commensurate with that degree of quality-of-life impairment seen in patients with myocardial infarction. Six months post-ablation, the health-perceived quality of life improves to the levels seen within the normal population.[21] It is important for patients undergoing ablation to appreciate that the goal of ablative therapy is interruption of actual bypass tract's ability to conduct impulses and not the abolition of all ectopic beats that have engendered the tachyarrhythmia.

Conclusions

Although the asymptomatic delta wave is associated with an extremely low risk of sudden cardiac death, it is not zero. Nonetheless, the issues of treatment in WPW syndrome largely are issues related to symptom burden and quality of life. For the person who is asymptomatic, the benefit of intervention should be weighed against the risks involved, and these should be evaluated with the informed patient. There are no good guidelines to predict prognosis beyond invasive procedures during which the shortest RR interval in induced atrial fibrillation would be determined. It is still unclear that, in evaluating every person with a delta wave, the risks outweighed the small potential benefits to be gained.

For the patient who has symptoms, the threshold to moving on to an ablation procedure becomes lower as the ease, success rate, complication rate, and availability of this procedure all improve. The symptomatic patient will generally benefit from an ablative procedure with a reasonable risk:benefit ratio, with a treatment aimed to improve quality of life.

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San Diego, California, USA
(Society of Thoracic Surgeons)
Tel.: 312-644-6610

Abstracts of Interest

Accessory atrioventricular pathways with successful ablation of antegrade and retrograde conduction at different sites

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Catheter ablation may eliminate the antegrade and retrograde accessory pathway conduction at closely adjacent but anatomically discrete sites. However, mechanisms of this discrepancy, electrophysiologic characteristics and possible anatomy of these pathways are not available. The purposes of this study were to investigate the electrophysiologic and anatomic characteristics of the accessory pathways in which antegrade and retrograde conduction was successfully eliminated by radiofrequency ablation at different sites. Thirty-eight (10.9%) patients (19 males and 19 females, mean 37 ± 15 years) with separate ablation sites for antegrade and retrograde conduction were designated as Group 1, and the other 310 patients (215 males and 95 females, mean 47 ± 10 years) were designated as Group II.

Results: The patients with right-side free wall pathways had the highest incidence (18.6%) of separate ablation sites. The anatomic distance between antegrade and retrograde directions (13 ± 4 vs 8 ± 4 mm, $P<0.01$) and incidence of conduction impairment in one direction after successful ablation of another direction (15% vs 78%, $P<0.05$) differed significantly between left and right free wall pathways. A cutoff value of < 100 msec for Δ AP1:1 and Δ APERP of left free wall pathways could predict successful ablation at the same site with a high positive predictive value (94.0% and 94.1%, respectively). However, the posterior/ anterior anatomic relation for antegrade/retrograde accessory pathways was similar in any of the accessory pathway locations.

Conclusions: This study showed that anatomic and functional dissociation of the accessory pathway into an antegrade and a retrograde component was possible and each component could be selectively ablated.

Excerpted from *PACE*, 1996;19:674.

Multiple and multifiber accessory pathways in retrograde direction: potential mechanism of atrial fibrillation in the Wolff-Parkinson-White syndrome

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In the Wolff-Parkinson-White syndrome (WPW), it is well experienced that successful ablation of the accessory pathways (AP) can reduce the incidence of atrial fibrillation (AF), although there are no differences identifiable in atrial vulnerability. We tested the hypothesis that the existence of ventriculoatrial (VA) conduction over the multiple (MP) or multifiber (MF) AV AP in retrograde direction could be causative of AF initiation in WPW. According to the response to radiofrequency catheter ablation, APs were classified into single (S), MP and MF (M) AP in 250 WPWs (A:107, B:61, concealed:82). MF AP was defined as an AP requiring multiple RF energy deliveries at supra- or infravalvular sites ≥ 1 cm apart along the AV ring associated with stepwise modifications in preexcitation and/or retrograde activation pattern. MP AP was defined following standard criteria.

Results: S, MP and MF APs were identified in 221, 16 and 13 WPWs, respectively. Incidences of clinical (C) AF and initiated (I) AF during V pacing or AV reentrant tachycardia (AVRT) were significantly higher in M than S AP, in spite of the same incidence of AVRT between the 2 groups as below (* $p<0.001$).

	C- & I-AVRT	C-AF	I-AF
M AP (29)	25 (86%)	18 (62%)*	12 (31%)*
S AP (221)	206 (93%)	51 (23%)*	32 (14%)*

Conclusion: It is suggested that the existence of retrograde conduction over the MP or MF AP in retrograde direction might be a potential mechanism of AF initiation in WPW.

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