REVIEW ARTICLE

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Perioperative Management of the Wolff-Parkinson-White Syndrome

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APPROPRIATE MANAGEMENT of cardiac arrhythmias is not only critical but must also be done in a timely manner. One such challenging scenario is perioperative management of the Wolff-Parkinson-White (WPW) syndrome. Incorrect treatment of the syndrome not only is ineffective but can cause quick clinical deterioration and even cardiac arrest. Therefore, perioperative clinicians must master the skill of managing such a patient. The authors provide a comprehensive review of the salient features of the WPW syndrome for perioperative clinicians, including epidemiology, anatomy, pathophysiology, and the non-pharmacologic and pharmacologic treatment modalities. The authors hope this review will enable clinicians to understand the physiology of the WPW syndrome, provide appropriate management, and avoid incorrect treatment.

The history and major milestones of what is known today as the Wolff-Parkinson-White (WPW) syndrome arise mainly from a few select articles. In the 1930s, Louis Wolff, Sir John Parkinson, and Paul Dudley White described 11 patients who had occasional episodes and electrocardiographic (ECG) findings of sinus tachycardia, a bundle-branch block QRS morphology, and a shortened PR interval.[1] Then, in 1967, during epicardial mapping at surgery in a patient with an atrial septal defect who also had WPW ECG findings, 2 atrioventricular connections were found to exist rather than one.[2] This additional accessory pathway (AP) was linked to the WPW syndrome. Later that year, the mechanism for a WPW tachycardia, how it can be initiated and terminated by programmed stimulation of the heart, and how APs can be localized via intracardiac mapping were demonstrated.[3] This information was the basis for the subsequent steps in the management of the WPW syndrome, including drug treatments for arrhythmia, surgical ablation, and, finally, catheter ablation of the AP.

WPW syndrome has been particularly interesting to anesthesia care providers. Almost 2 decades ago, Lustik et al reported a case of WPW with ECG features, notably Q-waves, that mimicked a myocardial infarction, an example of how the syndrome has at times been a diagnostic dilemma for anesthesia providers.[4] Recently, there were 3 case reports of WPW in young patients that concentrated on the importance of avoiding sympathetic stimulation intraoperatively and also promoted the use of regional anesthesia.[5–7] Earlier key studies, although not directly related to patient management in the perioperative period, were extremely important to understand the effects of commonly administered anesthetics and adjuvants on conductivity. However, despite these articles, a comprehensive perioperative resource guide for anesthesia providers on the subject is not available. The authors hope that the following text will provide a thorough review of the topic.

PATTERN VERSUS SYNDROME

Three characteristics on ECG are present with a WPW pattern: A short PR interval, a delta wave or pre-excitation, and a widened QRS. If these patients have symptomatic arrhythmias, they are termed to have the WPW syndrome. Appropriate medication selection for therapeutic intervention is recognized to be critically important in these patients, as inappropriate management is not only ineffective but could also lead to death.[8] Therefore, perioperative clinicians must have a clear understanding of the pathophysiology of the syndrome and be able to provide appropriate management in a timely manner.

EPIDEMIOLOGY

The WPW pattern is present in anywhere from 0.13% to 0.25% of the population[9–11] or roughly 481,000 to 925,000 of the 370 million people in the United States.[12] About 1% of those having a WPW pattern have the WPW syndrome.[9] The first presentation commonly occurs between 20 and 40 years old.[13] Although spontaneous arrhythmias occur, the risk of sudden death due to a malignant arrhythmia is estimated at 0.4% per year in patients who have the WPW syndrome.[14]

It should be noted that since the WPW pattern is only an ECG diagnosis and the general population does not routinely get ECGs, the pattern is most certainly underdiagnosed. It is unknown whether the prevalence of the WPW syndrome is greater in the patient population requiring anesthesia. However, there is an increased incidence of cardiac arrhythmias during anesthesia, general and regional, with and without pre-existing cardiac disease, with some studies suggesting rates as high as 61%.[15] General or regional anesthesia can unmask the WPW syndrome.[16] Perioperative nausea, gagging, hypothermia, sympathetic blockade via regional anesthesia, pregnancy, laparoscopic insufflation, laryngoscopy, hyperventilation, and

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cholinergic medications such as reversal agents and succinylcholine can all accentuate AP travel and/or arrhythmia propagation.[15–18] These circumstances may either influence the AP directly or affect normal conduction pathways, both of which can create a permissive environment for arrhythmia.

CARDIAC ANATOMY AND ELECTROPHYSIOLOGY

Atrial and ventricular myocardial tissues, although mechanically connected, are well-separated electrically. In a normal human heart, depolarization starts at a sinus nodal complex in the right atrium and propagates across the atrium. The true initiation point is dependent on a few factors, such as autonomic tone and membrane potentials.[19] The P-wave on ECG represents atrial depolarization. Depolarization impulse then moves from atrium to ventricle by way of the atrioventricular node (AVN) and the His-Purkinje system (the PR interval). Then, depolarization occurs across the ventricles, presenting the QRS complex. This specialized conducting pathway is the only electrical connection between the atria and ventricles, as the valve annuli are excellent insulators that prevent impulse from being conducted through them. Patients with WPW pattern or syndrome have an AP in addition to the normal atrioventricular conduction system (Fig 1A). The AP is likely a tissue remnant left over from embryologic formation of the heart.[17] Since 2 parallel pathways exist with different conduction speeds, as opposed to the single normal pathway, depolarization impulses from the atria can reach the ventricles via 1 or both pathways. This results in 3 features of abnormal electrical conduction (Fig 1B): (1) The PR interval is short, less than 0.12 seconds. Conduction over the AP is much faster than AVN conduction, leading to a shorter PR interval.[20] (2) Pre-excitation, or the delta wave. Earlier activation of the ventricles via the AP rather than through the AVN and His-Purkinje system leads to earlier ventricular depolarization at the AP connection site, creating a fusion between early (via the AP) and late (via the AVN) ventricular depolarization. This fusion, or pre-excitation, is seen as the delta wave on ECG (Figs 1A and 1B). (3) A widened QRS-wave, as a result of pre-excitation.

The time interval between atrial activation (the P-wave) and the initiation of ventricular activation (the delta wave) depends upon the location of the AP. If the atrial end of the AP is closer to the sinus node (a right-sided location), the P-delta interval will be shorter and the delta wave larger than those APs farther from the sinus node (at a left-sided location). The locations of APs are quite heterogenous. The frequencies of the ventricular insertion sites of the APs across the atrioventricular groove are 46% to 60% in the left ventricle free wall, 25% in the posteroseptal myocardium, 13% to 21% in the right ventricle free wall, and 2% in the anteroseptal myocardium.[21,22]

An important safety feature of the normal AVN is termed "decremental conduction". This refers to the ability of the AVN to reduce the speed of conduction until some but not all of atrial depolarization impulses reach ventricular tissue.[23] It serves as a protective mechanism, reducing the speed of conduction in the AVN as heart rate increases, and is more apparent at faster heart rates. This is why patients can experience atrial fibrillation, with atrial beats around 500 beats per minute, but rarely have ventricular response at such a high rate. In contrast, the AP does not display decremental conduction and can conduct extremely rapidly at a ratio as high as 1:1 from the atria to the ventricles. Decremental conduction also may serve a physiologic function, as it allows for maximal atrial contraction filling of the ventricles at fast heart rates.

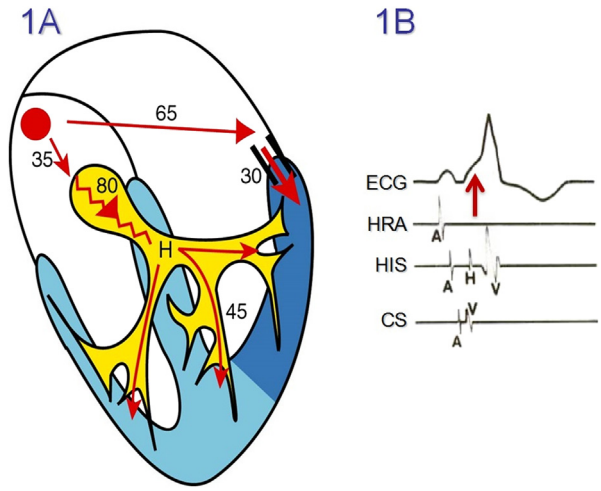

Fig 1. Illustration of ventricular activation during sinus rhythm in a patient having a Wolff-Parkinson-White (WPW) electrocardiogram. Panel A. In addition to the atrioventricular (AV) node and His-Purkinje system, a patient with the WPW pattern has a connection between the atria and ventricles called an "accessory pathway" (AP). In the drawing, the AP is dark blue. Ventricular activation is the result of activation by 2 wave fronts called "fusion of ventricular activation". Due to more rapid conduction over the AP, initial activation of the ventricle occurs over the AP, leading to the delta wave on the electrocardiogram (indicated by red arrow in panel B). Fig 1A illustrates how in, relation to the location of the AP, the different conduction time intervals (the numbers are in milliseconds) determine the pattern of ventricular activation. Note the more rapid conduction over the AP as compared to the slower conduction in the AV node of the AV node-His-Purkinje pathway. Fig 1B. An illustration of why the electrocardiogram has a delta wave (indicated by the red arrow), and also the activation times of atrium and ventricle during sinus rhythm using catheter recordings from the high right atrium (HRA), the His bundle region and the coronary sinus (CS). When these electrical potentials are summed, pre-excitation becomes evident on the electrocardiogram.

It is important to realize that certain factors can affect the degree of pre-excitation evident on ECG. Therefore, the absence of a delta wave or the degree of delta slurring does not signify the absence or severity of WPW pathology, respectively. For example: (1) AP conduction is influenced by the duration of the AP refractory period and autonomic tone.[24] AP conduction may be intermittent or absent at faster heart rates due to the length of the refractory period of the AP and faster AVN conduction, such as when sympathetic tone is high, eg, a patient who is exercising or is extremely anxious. (2) If an AP is located in the lateral left atrium, and about 50% are, the impulse generated by the sinoatrial node may reach the AP long after it reaches the AVN. Therefore, pre-excitation may not be apparent.[25] (3) Abnormal activation of the myocardium leads to abnormal repolarization, which is seen as abnormal ST- and T-waves on ECG. Pre-excitation may be interpreted as other cardiac pathology,[26] such as