REVIEW ARTICLE

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 cholinergic medications such as reversal agents and

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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.

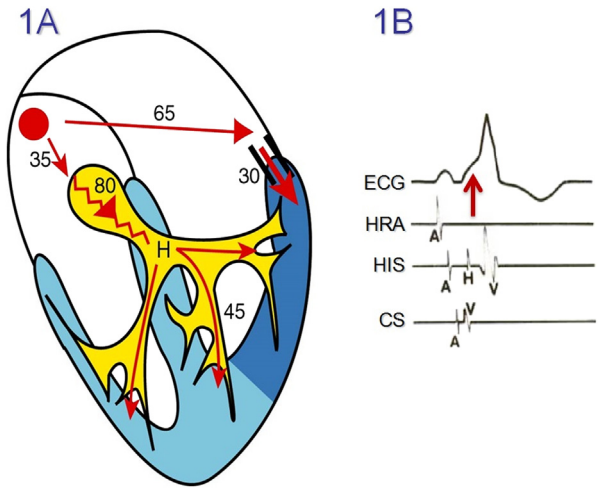

Fig 1. Illustration of ventricular activation during sinus rhythm in a patient having a Wolff-Parkinson-White (WPW) electrocardiogram. Panel A. In addition to the atrioventricular (AV) node and His-Purkinje system, a patient with the WPW pattern has a connection between the atria and ventricles called 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 ischemia, hypertrophy, or pericarditis. A delta wave may mask

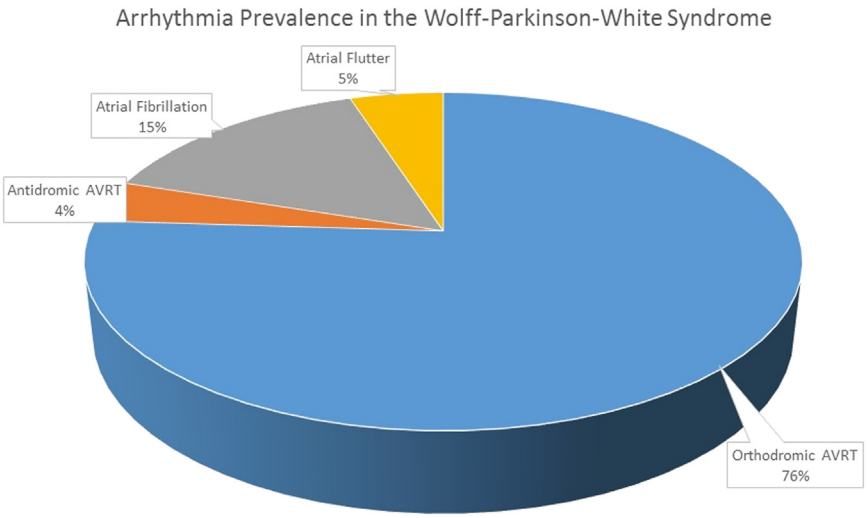

Fig 2. The prevalence of different arrhythmias in the Wolff-Parkinson-White Syndrome. AVRT, atrioventricular re-entrant tachycardia.

or mimic Q-waves from a previous infarct. It also may be mistaken for a premature ventricular contraction if only intermittent, or may be mistaken for ventricular bigeminy when 2:1 block occurs in the AP during sinus rhythm. (4) Some APs may only conduct in a retrograde manner and can participate in re-entrant arrhythmias. In this case, the ECG during sinus rhythm may be normal and does not show any characteristics of a WPW pattern. These pathways are termed "concealed".[27]

CLINICAL PRESENTATIONS

Arrhythmias

Patients with the WPW syndrome can be put into 3 categories in terms of arrhythmia type (Fig 2). Eighty percent of arrhythmias are atrioventricular re-entrant tachycardias (AVRT) divided into orthodromic and antidromic conduction; while 20% of patients present with atrial fibrillation or flutter.[28]

ORTHODROMIC ATRIOVENTRICULAR RE-ENTRANT TACHYCARDIA (OAVRT)

OAVRTs are the most common type (76%) of arrhythmia encountered in patients with the WPW syndrome.[29] OAVRTs are a re-entrant tachycardia in which a closed loop of conduction is formed and continues until block occurs in the tachycardia circuit. A simple inciting event, such as an atrial premature beat, can cause normal conduction down the AVN to the ventricles, back up to the atria via retrograde conduction through the AP, then again down the AVN anterograde. (Fig 3).

OAVRTs display a narrow QRS complex on ECG because atrioventricular conduction is via the AVN. Therefore, ventricular depolarization and re-polarization are normal, and there is no pre-excitation. During OAVRT, the QRS may be wide if the patient has a pre-existing or rate-related bundle-branch block, which is known as "aberrant conduction." Aberrancy may be present due to a high heart rate or occasionally due to ischemia, hyperkalemia, or anti-arrhythmic medications.[30,31] Therefore, diagnosis of an OAVRT must be made cautiously. A wide QRS with a fast rate does not rule out OAVRT, and absence of a delta wave does not necessarily mean the absence of an AP.

ANTIDROMIC ATRIOVENTRICULAR RE-ENTRANT TACHYCARDIA (AAVRT)

AAVRT, as the name implies, is also a re-entrant tachycardia, but depolarization impulse travels in the opposite direction of OAVRT. These arrhythmias make up 4% of the total arrhythmias in the WPW syndrome.[32] Atrioventricular conduction is antegrade down the AP to activate the ventricles, travels retrograde up the normal AVN conduction system to the atria, then

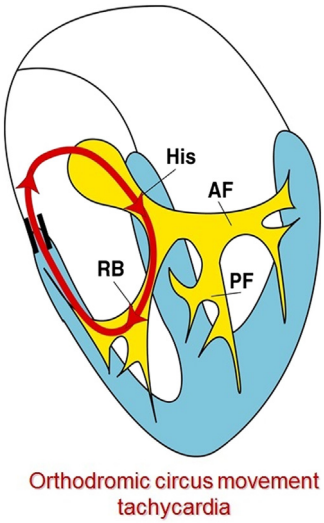

Fig 3. Orthodromic circus movement tachycardia. AF, anterior fascicle of the left bundle branch; PF, posterior fascicle of the left bundle branch; RB, right bundle branch.

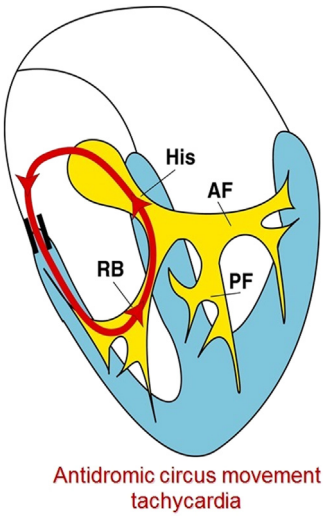

Fig 4. Antidromic circus movement tachycardia. AF, anterior fascicle of the left bundle branch; PF, posterior fascicle of the left bundle branch; RB, right bundle branch.

back down the AP again. The cycle continues until block occurs somewhere in the circuit, and is termed "antidromic" because conduction of the impulse in the AVN is opposite the normal direction (Fig 4). The rate of the tachycardia is nearly the same as in OAVRT because decremental conduction at the AVN applies to either direction. However, if a second AP exists, conduction from atria down to the ventricles can occur via 1 AP and back to the atria via the other AP. Heart rates using this mechanism can reach 250 beats per minute.[32] On ECG, AAVRTs appear as a regular wide-complex tachycardia and, unfortunately, cannot be distinguished from ventricular tachycardia.[30]

ATRIAL FIBRILLATION AND FLUTTER

One out of every 5 patients with the WPW syndrome experiences atrial fibrillation and/or flutter.[28] In contrast to re-entrant tachycardias, atrial fibrillation and flutter are not based on atrioventricular circular electrical loops. (Atrial flutter is a circular loop in the atrium only.) During the rapid atrial rate in atrial flutter and fibrillation, the impulses from the atria are conducted down to the ventricles via the AP. The ventricular rate then will be determined by the refractory period of the AP. These rhythms are especially dangerous as ventricular rates can be up to 300 beats per minute. This is a life-threatening situation.[28]

QRS wave morphology is variable during atrial fibrillation and can be wide and/or narrow and is dependent upon the anterograde AP refractory period.[33] Both cardiac rhythm and pulse during the arrhythmia are irregular. Different degrees of pre-excited QRS complexes usually are present, and become more evident with shorter AP refractory periods. This is because during short AP refractory periods, preferential conduction is over the AP (wide QRS); while in the case of a long AP refractory period, most impulses conduct down the AVN (narrow QRS).

All APs are not created the same with regard to arrhythmia potential. Patients who have malignant arrhythmias are more likely to have shorter AP refractory periods, more often have multiple APs, and are more likely to have AVRT-inducible, sustained pre-excited atrial fibrillation.[34] In fact, the risk of having a malignant arrhythmia in a patient with WPW syndrome can be independently predicted based on a short AP refractory period and AVRT-triggered, sustained atrial fibrillation.[34]

PREOPERATIVE CONSIDERATION

A critical step for the perioperative care team is to obtain a thorough cardiac history from the patient, such as a feeling of heart racing, palpitations, syncope, dyspnea, angina, or dizziness, as these may be the only clues to the clinician of the existence of the WPW syndrome. Some traits are suggestive that a patient may experience symptoms from WPW. These include[14,35,36] young age (especially in the first year of life), male gender (twice the prevalence of females), presence of multiple APs (13%[37] prevalence in pattern patients), or short AP refractory period. A patient with a WPW pattern usually will be recognizable on a 12-lead ECG. However, perioperative clinicians should consider history taking as the most important piece of information to make diagnosis of the WPW syndrome, as preoperative ECGs may not always display WPW features.

CHOICE OF ANESTHETIC

Choice of anesthetic and medications to be used during procedures has been studied in patients with WPW. It was thought initially that many common anesthetics, such as propofol,[38] benzodiazepenes,[39] fentanyl and analogs,[40,41] and isoflurane,[42] sevoflurane,[43] or desflurane[44] might alter electrical mapping of conduction pathways, which would be important while attempting pathway ablation. While some of these medications may affect conduction times and pathway refractoriness to varying extents, none was clinically limiting for ablation application.

Studies from the early 1970s advocated using anesthetics with minimal circulatory disturbance, nitrous oxide-narcotic techniques[45] or a deep inhalation agent,[46] to avoid increasing blood catecholamine levels. Light planes of anesthesia can cause episodes of stress and tachycardia in the patient and be pro-arrhythmic. In the late 1970s, anesthetics for WPW focused on neurolept anesthesia and avoiding drugs with negative inotropic effects on the heart.[47]

Recent literature suggested that the use of regional anesthetic techniques to avoid sympathetic stimulation would be beneficial in WPW patients. In this manner, laryngoscopy and the use of muscle relaxant reversal agents may be avoided, both of which create a permissive environment for arrhythmia. Some reports suggest a 20% incidence of supraventricular tachycardia during induction of general anesthesia in WPW patients and ventricular fibrillation in 10% of patients.[48,49] With the more frequent use of laryngeal mask airways and non-depolarizing muscle relaxants that avoid histamine release, sympathetic activity, or limiting reversal medication, anesthetic differences may become less apparent. It should be noted that arrhythmia potential remains increased compared to baseline while a

patient is anesthetized, regardless of regional or general technique.[15] If a regional technique is considered near a vascular bed, clinicians might consider not using epinephrine with local anesthetic as reports of even submaximal doses of epinephrine absorbed into the systemic circulation during oral surgical procedures caused marked elevations of plasma epinephrine.[50,51]

Finding a medium balance between extreme sympathetic and vagal tone should be a perioperative goal, as both can be pro-arrhythmic. Increase in vagal tone causes AVN slowing, directing conduction down the AP.[15–18] Increase in sympathetic tone promotes impulse generation and tachycardia, which the AP can propagate far better than the AVN. For example, neostigmine should be avoided if a patient has baseline atrial fibrillation and WPW, as it can cause rapid ventricular response via the AP.[52] In contrast, atropine accelerates conduction and shortens refractoriness in the AP, and meperidine should be used cautiously as well for this reason.[53]

Adequate preloading should be considered to decrease the use of sympathomimetics. Phenylephrine should be used as the vasopressor of choice whenever possible, as it limits tachycardia and has been shown to actually terminate supraventricular tachycardia in a patient with WPW.[54] Anxiolytics are helpful in patients who may hyperventilate due to stress, as this would enhance AP conduction.[55] Table 1 provides a summary of commonly used anesthetics and medications that clinicians may use perioperatively in relation to the WPW syndrome.

MANAGEMENT OF THE WPW SYNDROME

Perioperative clinicians must be well-informed about treatment modalities for patients with the WPW syndrome. It is reassuring to know that patients having only the WPW pattern on ECG have an extremely low rate of sudden death due to malignant arrhythmia.[36] A preoperative cardiology consult should still be obtained, however, as patients may not spontaneously report their experience of rapid heart rates. The cardiologist may recommend noninvasive methods to determine the AP anterograde refractory period and arrhythmia potential, such as exercise testing, evaluating an intermittent WPW pattern on Holter monitoring, and testing for presence or absence of conduction over the AP following procainamide injection. Typically, no therapy is necessary preoperatively for patients with WPW pattern, including catheter ablation.

Table 1. Anesthetics and Medications in the Wolff-Parkinson-White Syndrome

Anesthetic or Medication	Effect
Volatile anesthetics	
Halothane	Pro-arrhythimc, cardiac sensitization to catecholamines, better to use alternative
Enflurane	Safe to use, no clinical effect on conduction
Isoflurane	Safe to use, no clinical effect on conduction
Sevoflurane	Safe to use, no clinical effect on conduction
Desflurane	Safe to use, no clinical effect on conduction
Nitrous oxide	Safe to use, no clinical effect on conduction
Benzodiazepines	Safe to use, no clinical effect on conduction
Opioids	Safe to use, no clinical effect on conduction
Meperidine	Induces tachycardia, better to use alternative
Muscle relaxants	
Pancuronium	Induces tachycardia, better to use alternative
Vecuronium	Safe to use, no clinical effect on conduction
Atracurium	Histamine release, better to use alternative
Cisatracurium	Safe to use, no clinical effect on conduction
Succinylcholine	Increases vagal tone, cautious use
Induction agents	
Propofol	Safe to use, no clinical effect on conduction
Etomidate	Safe to use, no clinical effect on conduction
Thiopental	Safe to use, older studies suggest cautious use
Ketamine	Sympathomimetic, safe to use, better to use alternative
Reversal agents	
Neostigmine	Increases vagal tone, cautious use
Glycopyrrolate	Induces tachycardia, cautious use
Atropine	Induces tachycardia, cautious use
Vasopressors	Generally safe to use
Phenylephrine	Safe to use, no clinical effect on conduction, has been used as an anti-arrhythmic
Ephedrine	Induces tachycardia, cautious use
Epinephrine	Induces tachycardia, cautious use
Isoproterenol	Induces tachycardia, cautious use
Anti-emetics	
Droperidol	Safe to use, may decrease arrhythmia potential
Metoclopramide	Induces tachycardia, cautious use
Regional anesthesia	Safe, caution with high spinal block due to increased vagal tone, caution with epinephrine uptake due to tachycardia

NOTE. A brief summary of anesthetics and medications that have been studied in the Wolff-Parkinson-White syndrome.

Patients with the WPW syndrome, on the other hand, usually require medical or interventional therapy to control arrhythmia propensity.

ABLATION THERAPY

Radiofrequency ablation, with a 90% to 95% success rate at the first attempt, is considered the best method for long-term, definitive therapy for the WPW syndrome.[56] Fifty percent of patients who fail their first radiofrequency ablation treatment have recurrence within the first twelve hours after the procedure, but nearly 100% of patients who undergo a second ablation have complete resolution of symptoms.[56] Complications occur at a rate of less than 4% and include significant morbidity should they occur: Acute coronary syndrome due to rate-related ischemia or catheter obstruction, transient ischemic attack from periods of decreased cardiac output with tachycardia, pneumothorax from central venous access, pericarditis from irritation of the myocardium, cardiac tamponade, bleeding, infection, perforation of atrium or ventricle, new bundle-branch block, and complete heart block from inadvertent ablation of normal conducting pathways.[56]

Surgical ablation has a nearly 100% success rate, with a mortality rate of less than 1%;[57] therefore, a patient surviving the surgery will be arrhythmia free. Surgery is reserved for malignant pathways that fail radiofrequency ablation or patients who are not candidates for the ablation therapy (ie, AP in close proximity to normal conducting pathways), or a patient who will undergo cardiac surgery for another reason; for example, for complex congenital cardiac disease.[57] Currently, most accessory AV pathways can be ablated by a catheter, and surgical ablation is the exception.

PHARMACOLOGIC THERAPY

The response to long-term medical prevention therapy in the WPW syndrome is variable. A brief review of the different regimens available for arrhythmia prevention medication follows. However, expert advice should be sought preoperatively, as some medications may worsen arrhythmias, have negative ionotropic activity, and may have central nervous system effects. Radiofrequency ablation, therefore, remains the best method for prevention therapy.

Class Ia (eg, quinidine, procainamide), class Ic (eg, flecainide, propafenone), and class III (eg, amiodarone, sotalol, dofetilide) anti-arrhythmic medications all slow AP conduction.[58] If the patient has a history of atrial fibrillation or flutter, a class II (beta-blocker) or class IV (calcium channel blocker) also should be added to class Ia and Ic medications to slow AVN conduction. Class III medications affect all myocardial tissue, so additional agents reducing AVN conduction are not necessary in this instance.[59] Importantly, class Ic drugs are contraindicated in patients who have coronary artery disease, a history of myocardial infarction, or congestive heart failure[60] because of an increased risk of sudden death due to pro-arrhythmic effects. In pregnant women with the WPW syndrome, the safest medication for chronic arrhythmia prevention is sotalol, which has a class-B rating. Flecainide has been used safely in pregnancy as well. However, it has a class C rating.[61] Table 2 summarizes the anti-arrhythmic medications and considerations with management of WPW syndrome.

The perioperative care team should avoid altering medication regimens, and medication should continue perioperatively. Since most medications used for long-term treatment are administered orally, patients should be encouraged to take usual doses on the day of surgery. Medications should be administered intraoperatively via nasogastric or orogastric tube at the usual frequency, if feasible, and routine oral medications should be resumed upon beginning oral intake.

In summary, almost all patients with symptomatic WPW are treated with radiofrequency ablation, and there is no further intervention or medication necessary preoperatively. They are cured, and no cardiology consult is needed. The select group of patients who take anti-arrhythmic medications for WPW should continue these, and no further cardiology consultation is needed. If a patient has not seen a cardiologist and has WPW pattern or syndrome, they should see a cardiologist preoperatively.

INTRAOPERATIVE MANAGEMENT

Intraoperative arrhythmia occurrence in a patient with the WPW syndrome is usually an emergent scenario that requires prompt intervention. The urgency to intervene is dependent upon the hemodynamic stability of the patient. It is important to realize that the only determinations that can be made under general anesthesia in defining stability versus instability are hypotension and presence of heart failure. Angina or altered level of consciousness obviously are masked by general anesthesia.

Table 2. Anti-arrhythmic Medications in WPW

Anti-arrhythmic Class (Vaughan Williams Classification)	Primary Mechanism	Considerations in WPW
Ia: quinidine, procainamide	block sodium and potassium channels, slowing AP conduction	Procainamide rare but excellent acute anti-arrhythmic agent
Ic: flecainide, propafenone	block sodium channels, slowing AP conduction	Used in chronic therapy but contraindicated in history of CAD, MI, CHF
II: beta-blockers	block beta adrenoceptor, slowing AVN conduction	Add class Ia or Ic agents if history of SVT
III: amiodarone, ibutilide, sotalol, dofetilide	block potassium channels, action on all myocardial tissue	Can be used in chronic therapy, excellent acute anti-arrhythmic agents
IV: calcium channel blockers	block calcium channels, slowing AVN conduction	Add class Ia or Ic agents if history of SVT

Abbreviations: AP, accessory pathway; AVN, atrioventricular node; CAD, coronary artery disease; CHF, congestive heart failure; MI, myocardial infarction; SVT, supraventricular tachycardia; WPW, Wolff-Parkinson-White.

Standard American Society of Anesthesiology monitors should be placed for routine cases. The presence of the WPW syndrome alone does not warrant invasive monitoring, central venous access, or the pre-emptive placement of cardioversion pads on the patient. The perioperative clinician should use clinical judgment for each case individually. How difficult will it be to access the chest? Will the operating room table be turned? Is extra help nearby? Does the patient frequently have WPW symptoms? Will the patient be in a non-supine position? An external defibrillator should be immediately available in all circumstances, however.

If the patient is hemodynamically stable, pharmacologic treatment should be attempted. If the arrhythmia is not amenable to medical treatment; for example, medication unavailability or hemodynamic instability, synchronized direct current cardioversion should be utilized (50-to-200 Joules biphasic).[62] If synchronization is not possible, defibrillation should be immediately attempted (200-Joules biphasic).[62] Cardiopulmonary resuscitation should be initiated without delay as effective chest compressions and adequate oxygenation are keys to preventing vital organ injury before spontaneous circulation returns. Presence of hemodynamic stability is not diagnostic of arrhythmia type, and misdiagnosis based on this misconception commonly happens and is extremely dangerous.[63,64]

In order to illustrate diagnosis and intraoperative arrhythmia management, 4 case scenarios will be discussed.

CASE SCENARIO 1: PATIENT WITH A KNOWN HISTORY OF THE WPW SYNDROME PRESENTING WITH A STABLE, REGULAR, NARROW COMPLEX TACHYCARDIA

The ECG in Figure 5 shows an OAVRT, a regular, narrow complex tachycardia with no obvious pre-excitation in a patient known to have the WPW syndrome. Clinicians can block a circuit anywhere along conducting pathways; however, pharmacologic therapy should aim to block the circuit at the weakest link. In OAVRTs, the weakest link is usually the AVN, and increasing refractoriness here will terminate the tachycardia.

If the patient is hemodynamically stable and the diagnosis of OAVRT is certain, physical maneuvers may be attempted.[65] Maneuvers to increase vagal tone, such as carotid sinus massage or a Valsalva maneuver, will prolong the refractory period of the AVN and terminate the tachycardia in awake patients. Case reports have documented the effectiveness of carotid massage in treating arrhythmias even while under general anesthesia.[66] A traditional Valsalva maneuver while under general anesthesia, however, is unable to be performed. Perioperative clinicians often term persistent application of positive intra-alveolar pressure to an intubated patient a Valsalva maneuver. However, such a maneuver is very different from a true, actively performed Valsalva maneuver by an awake patient. Its effectiveness on the refractory period of the AVN is unknown when applied to an anesthetized patient.

The best first-line pharmacologic agent to be used in OAVRT is the rapid-acting, short-lasting medication adenosine.[67] It is given intravenously in doses of 6 mg, 12 mg, and then 12 mg again every 1 to 2 minutes if the arrhythmia does not break. Adenosine inhibits adenylyl cyclase, thereby reducing cyclic AMP levels, increasing potassium outflux, and, therefore, causing cell hyperpolarization and an increased refractory period at the AVN. Adenosine should be given only if emergency pacing or cardioversion equipment is available, as it causes transient complete heart block and can increase vulnerability to atrial fibrillation by indirectly decreasing atrial refractoriness (12% risk).[13] Adenosine also should be used cautiously in patients who have had a heart transplant[68] or those taking dipyridamole,[69] as these patients are extremely sensitive to the medication and can develop severe bradycardia.

The best second-line agent for OAVRT is the calcium channel blocker verapamil, which may be given intravenously in 5-mg doses every 2 to 3 minutes, for a maximum dose of 15 mg.[70] Care must be taken if a patient is hypotensive or has systolic heart failure, as verapamil can cause further hypotension or lead to decreased cardiac output and pulmonary edema.

The next choice for acute pharmacologic treatment in OAVRT is intravenous procainamide, given as 10 mg/kg over

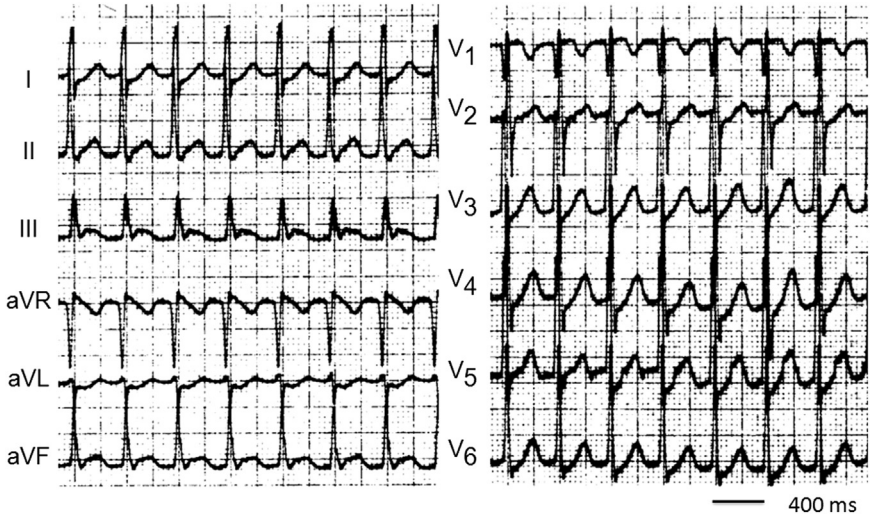

Fig 5. Regular, narrow QRS complex tachycardia.

10 minutes, up to 15 mg/kg within 30 minutes.[71] Procainamide blocks sodium channels. It prolongs refractoriness in myocardium, including the AP as well as the His-Purkinje system, but not the AVN.[72] This will reduce the rate and block retrograde impulses entering the atrium from the ventricle through the AP.

Clinical use should be judicious, as increased procainamide levels may cause adverse reactions and consequences, such as[73] hypotension, arrhythmia exacerbation, atrioventricular block, and QRS and QTc widening, ultimately ending in torsade de pointes. After the initial loading dose, a continuous infusion should be initiated at 1 mg/min-to-4 mg/minute.[73] Hypotension and QRS widening to 50% of original width are concerning signs and should be limiting factors in administration.[73]

Other second-line medications for OAVRT include intravenous beta-blockers, such as propranolol, metoprolol, or esmolol.[74] These medications prolong AVN refractoriness, resulting in block in this part of the tachycardia circuit.

Intravenous amiodarone can be considered for ongoing OAVRT arrhythmia not amenable to other medical treatments mentioned above.[75] Amiodarone prolongs refractoriness of all myocardium, including the AP and normal conducting pathways.

CASE SCENARIO 2: PATIENT WITH A KNOWN HISTORY OF THE WPW SYNDROME, PRESENTING WITH A STABLE, REGULAR, WIDE COMPLEX TACHYCARDIA

The ECG shown in Figure 6 demonstrates a regular, wide complex tachycardia. The differential diagnosis has been discussed previously, and, unfortunately, a 12-lead surface ECG is unable to distinguish with certainty AAVRT from ventricular tachycardia.[30] Electrophysiology studies, in which electrodes are placed along heart conduction pathways in the electrophysiology lab, would be able to distinguish the 2, but the clinician with standard intraoperative monitors would not. If the AVN is slowed similar to treatment in OAVRT, it could render any atrial impulse to preferentially travel through the AP to conduct at a ratio of 1:1 to the ventricles. The first-line OAVRT medications do not reduce the speed of AP conduction, and if the presenting rhythm is actually ventricular tachycardia and not AAVRT, the arrhythmia quickly can deteriorate to ventricular fibrillation and cardiac arrest. Therefore, the clinician must be vigilant to avoid incorrect drug selection. Atrioventricular nodal slowing agents should be avoided (eg, adenosine, beta-blockers, calcium channel blockers, digoxin).

The best medication for acute AAVRT treatment would increase refractoriness in both the AVN and the AP to limit ventricular response and avoid mistreating ventricular tachycardia. Therefore, intravenous procainamide, at the same dose as discussed earlier, 10 mg/kg over 10 minutes up to 15 mg/kg within 30 minutes, is the best treatment option. Even if procainamide does not terminate the tachycardia, it reduces the rate and improves the hemodynamic state.[71] Intravenous amiodarone, the next best option, can be administered as a load of 150 mg followed by a continuous infusion of 1 mg/min over 6 hours then 0.5 mg/min over 18 hours.[76]

Advanced cardiac life support algorithms are designed to provide appropriate responses to emergency scenarios, and the authors are cognizant of the fact that amiodarone has become the mainstay of treatment in malignant arrhythmias, independent of etiology.[77] Furthermore, procainamide may not be readily available in institutions. However, procainamide is still considered the best first-line therapy in WPW. In actuality, advanced cardiac life support guidelines have a slightly stronger recommendation score for procainamide (class IIa) over amiodarone (class IIb) in stable ventricular tachycardia.[77] Amiodarone, although a very effective medication for WPW acute arrhythmia therapy, slows AVN conduction as well as the AP. Procainamide slows the AP, preferentially diverting conduction down normal pathways. It is the best option in WPW, if available.

CASE SCENARIO 3: PATIENT WITH A KNOWN HISTORY OF THE WPW SYNDROME PRESENTING WITH A STABLE, IRREGULARLY IRREGULAR TACHYCARDIA WITH A HIGH VENTRICULAR RATE

The ECG shown in Figure 7 demonstrates a fast irregular tachycardia, most likely atrial fibrillation with rapid ventricular

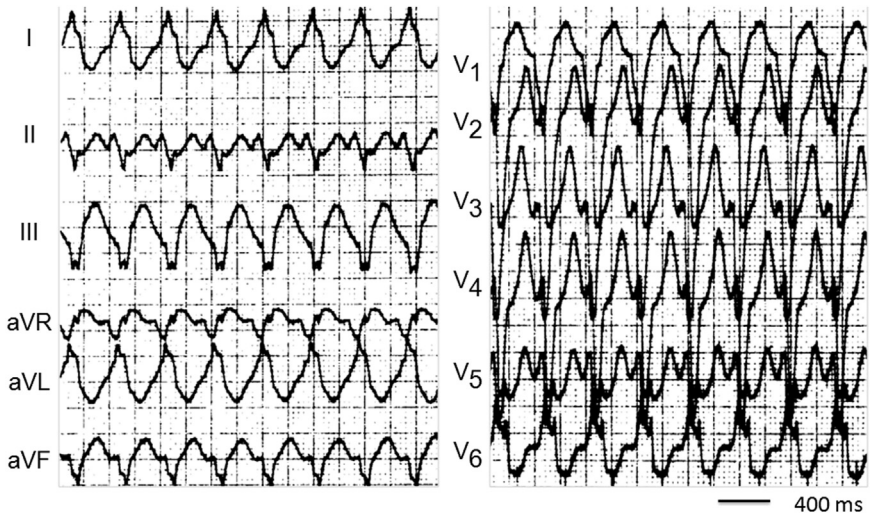

Fig 6. Regular, wide QRS complex tachycardia.

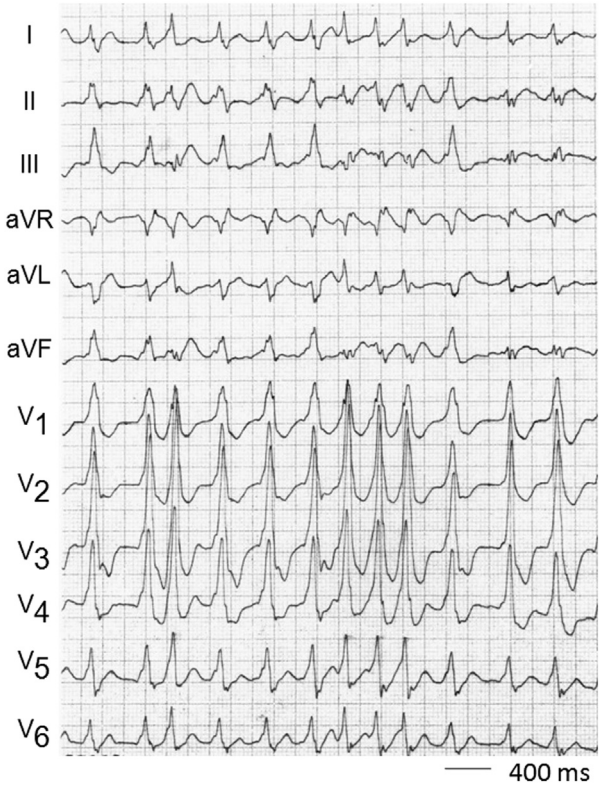

Fig 7. Irregularly irregular, varying QRS complex tachycardia.

response. The ventricular rhythm is irregular and shows different degrees of pre-excitation because of different degrees of fusion at the ventricular level between AV conduction over the AP and the AV node. As discussed earlier, atrial arrhythmias may exhibit wide or narrow complex tachycardias, and are especially dangerous because every atrial beat can be conducted at a ratio of 1:1 to the ventricles via an AP. Tachycardias at such high rates may quickly lead to ventricular fibrillation and cardiac arrest.

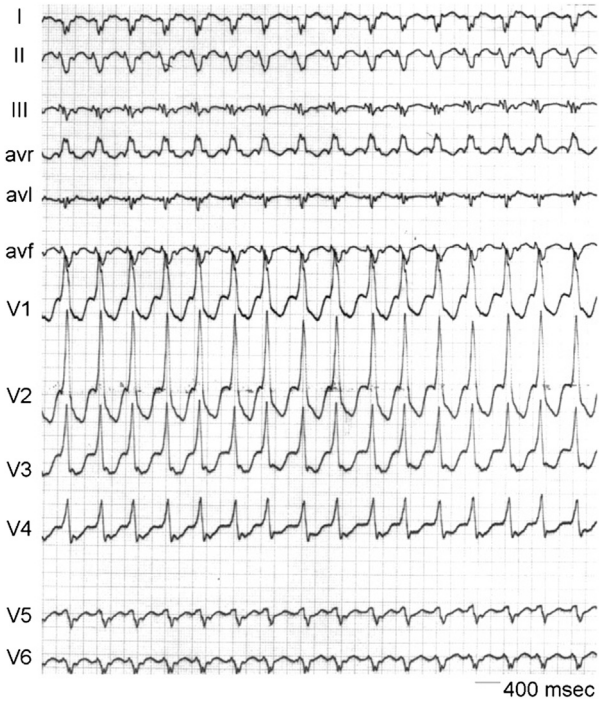

Fig 8. Regular, wide QRS complex tachycardia.

Table 3. Summary of Wolff-Parkinson-White Arrhythmias and Urgent/Emergent Therapies

Arrhythmia/ Presentation	Conduction	Electrocardiogram Features	Therapy
OAVRT	Anterograde (down) AVN, Retrograde (up) AP	Regular narrow complex tachycardia (wide if bundle-branch block exists)	1. Carotid massage, Valsalva maneuver 2. Adenosine (6 mg, 12 mg, 12 mg every 1-2 mins) 3. Verapamil 5 mg (every 2-3 minutes, maximum 15 mg) 4. Procainamide 10 mg/kg over 10 minutes, maximum 15 mg/kg in 30 minutes
AAVRT	Retrograde (up) AVN, Anterograde (down) AP	Regular wide complex tachycardia	1. Procainamide 10 mg/kg over 10 minutes, maximum 15 mg/kg in 30 minutes
AF	Anterograde (down) AVN and AP, depending upon AP refractory period	Irregularly irregular tachycardia, wide or narrow complex	2. Amiodarone 150 mg load, 1 mg/min infusion for 6 hours, 0.5 mg/min for 18 hours
Uncertain arrhythmia type			
Unstable arrhythmia			Cardioversion (50-200-Joules biphasic) or defibrillation (200-Joules biphasic), plus Cardiopulmonary resuscitation

Abbreviations: AAVRT,: antidromic atrioventricular tachycardia; AF, atrial fibrillation/flutter; AP, accessory pathway; AVN, atrioventricular node; OAVRT, orthodromic atrioventricular tachycardia.

The best medication for these arrhythmias in the WPW syndrome would once again target both the AVN and the AP to limit ventricular response from both pathways. Procainamide, at the same dose discussed earlier, is the best choice.[78] If ineffective, an intravenous amiodarone load of 150 mg followed by a continuous infusion of 1 mg/min over 6 hours then 0.5 mg/min over 18 hours can be attempted.[76]

If the patient is hemodynamically unstable, immediate electrical cardioversion should be performed.

CASE SCENARIO 4: PATIENT WITH A KNOWN HISTORY OF THE WPW SYNDROME PRESENTING WITH A STABLE, UNRECOGNIZABLE FAST ARRHYTHMIA

There may be instances when the clinician is unable to determine whether the QRS complex is wide or narrow. Figure 8 is ventricular tachycardia with a differential diagnosis including antidromic AVRT and supraventricular tachycardia with bundle-branch block. The rhythm may be a sinus tachycardia or OAVRT (as the majority of AVRTs are) with rate-related aberrancy. If the rhythm actually is AAVRT or ventricular tachycardia and incorrect treatment is given, the arrhythmia will deteriorate.[79,80] Rates may increase via further AP propagation, and hemodynamic instability may ensue with vasodilation and hypotension. When in doubt, cardioversion therapy is indicated when the patient is hemodynamically unstable. When hemodynamically tolerated, procainamide administration is the first choice for pharmacologic termination of tachycardias of unclear mechanism.

RECOMMENDATIONS

The WPW syndrome has three major arrhythmia types. Descriptions of conduction directions, features, and treatments are summarized in Table 3.

SUMMARY

The WPW syndrome is an interesting and critically important syndrome. It requires clinicians to obtain a thorough history preoperatively, understand the anatomic basis and electrophysiology, and to be familiar with the specific drugs to treat each of the different types of arrhythmia. The importance of avoiding incorrect therapies is stressed. WPW tachycardias are unpredictable and potentially life threatening; therefore, vigilance and readiness are demanded of perioperative clinicians.

REFERENCES

1. Wolff L, Parkinson J, White PD: Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. Am Heart J 5:685-691, 1930
2. Durrer D, Roos JP: Epicardial excitation of the ventricles in a patient with WPW syndrome. Circulation 35:15-21, 1967
3. Durrer D, Schoo L, Schuilenburg RM, et al: The role of premature beats in the initiation and the termination of supraventricular tachycardia in the WPW syndrome. Circulation 36:644-662, 1967
4. Lustik SJ, Wojtczak J, Chhibber AK: Wolff-Parkinson-White syndrome simulating inferior myocardial infarction in a cocaine abuser for urgent dilation and evacuation of the uterus. Anesth Analg 89: 609-612, 1999
5. Kabade SD, Sheikh S, Periyadka B: Anaesthetic management of a case of Wolff-Parkinson-White syndrome. Indian J Anaesth 55: 381-383, 2011
6. Sahu S, Karna ST, Karna A, et al: Anaesthetic management of Wolff-Parkinson-White syndrome for hysterectomy. Indian J Anaesth 55:378-380, 2011
7. Rakesh G, Renu S, Nishad PK: Patient with Wolff-Parkinson-White syndrome with intermittent pre-excitation under subarachnoid block for urological surgery. Indian J Anaesth 55:167-170, 2011
8. McGovern B, Garan H, Ruskin JN: Precipitation of cardiac arrest by verapamil in patients with Wolff-Parkinson-White syndrome. Ann Intern Med 104:791-794, 1986
9. Pediatric and Congenital Electrophysiology Society (PACES), Heart Rhythm Society (HRS), American College of Cardiology Foundation (ACCF), et al. PACES/HRS expert consensus statement on the management of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern. Heart Rhythm 9:1006-1024, 2012
10. Krahn AD, Manfreda J, Tate RB, et al: The natural history of electrocardiographic pre-excitation in men. The Manitoba Follow-up Study. Ann Intern Med 116:456-460, 1992
11. Kobza R, Toggweiler S, Dillier R, et al: Prevalence of preexcitation in a young population of male Swiss conscripts. Pacing Clin Electrophysiol 34:949-953, 2011
12. United States Census Bureau. United States Summary: 2000, Census US Profile. Available at http://www.census.gov/prod/2002pubs/c2kprof00-us.pdf. Accessed October 1, 2013.
13. Al-Khatib SM, Pritchett EL: Clinical featuers of Wolff-Parkinson-White syndrome. Am Heart J 138:403-413, 1999
14. Blomström-Lundqvist C, Scheinman MM, Aliot EM, et al: ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias–executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Supraventricular Arrhythmias). Circulation 108:1871-1909, 2003
15. Kuner J, Enescu V, Utsu F, et al: Cardiac arrhythmias during anesthesia. CHEST 52:580-587, 1967
16. Lubarsky D, Kaufman B, Turndorf H: Anesthesia unmasking benign Wolff-Parkinson-White Syndrome. Anesth Analg 68:172-174, 1989
17. Stoelting RK, Hines RL, Marschall KE: Abnormalities of cardiac conduction and cardiac rhythm. Stoelting's anesthesia and co-existing disease, Philadelphia, PA: Saunders/Elsevier, 75, 2012
18. Schoenstadt DA, Whitcher CE: Observations on the mechanism of succinyldicholine-induced cardiac arrhythmias. Anesthesiology 24: 358-362, 1963
19. Issa ZF, Miller JM, Zipes DP: Electrophysiological mechanisms of cardiac arrhythmias, clinical arrhythmology and electrophysiology. A companion to Braunwald's heart disease. Philadelphia, PA: Saunders: 2-4, 2009
20. Prystowsky EN, Page RL: Electrophysiology and autonomic influences of the human atrioventricular node. Prog Clin Biol Res 275: 259-277, 1988
21. Cain ME, Cox JL: Surgical treatment of supraventricular arrhythmias. Management of cardiac arrhythmias: The nonpharmacologic approach, Philadelphia, PA: JB Lippincott, 304, 1987
22. Gallagher JJ, Sealy WC, Kasell J: Intraoperative mapping studies in the Wolff-Parkinson-White syndrome. Pacing Clin Electrophysiol 2: 523-537, 1979

23. Patterson E, Scherlag BJ: Decremental conduction in the posterior and anterior AV nodal inputs. J Interv Card Electrophysiol 7: 137-148, 2002

24. Klein GJ, Gulamhusein SS: Intermittent preexcitation in the Wolff-Parkinson-White syndrome. Am J Cardiol 52:292-296, 1983

25. Reddy GV, Schamroth L: The localization of bypass tracts in the Wolff-Parkinson-White syndrome from the surface electrocardiogram. Am Heart J 113:984-993, 1987

26. Wang K, Asinger R, Hodges M: Electrocardiograms of Wolff-Parkinson-White syndrome simulating other conditions. Am Heart J 132:152-155, 1996

27. Miller JM: Therapy of Wolff-Parkinson-White syndrome and concealed bypass tracts: Part I. J Cardiovasc Electrophysiol 7: 85-93, 1996

28. Campbell RW, Smith RA, Gallagher JJ, et al: Atrial fibrillation in the pre-excitation syndrome. Am J Cardiol 40:514-520, 1977

29. Yee R, Klein GJ, Sharma AD, et al: Tachycardia associated with accessory atrioventricular pathways. Cardiac Electrophysiology, Philadelphia, PA: Saunders, 463, 1990

30. Miller JM, Hsia HH, Rothman SA, et al: Ventricular tachycardia versus supraventricular tachycardia with aberration: Electrocardiographic distinctions. Cardiac Electrophysiology from Cell to Bedside, Philadelphia, PA: Saunders, 696, 2000

31. Wellens HJ: Electrophysiology: Ventricular tachycardia: Diagnosis of broad QRS complex tachycardia. Heart 86:579-585, 2001

32. Bardy GH, Packer DL, German LD, et al: Preexcited reciprocating tachycardia in patients with Wolff-Parkinson-White syndrome: Incidence and mechanisms. Circulation 70:377-391, 1984

33. Wellens HJ, Durrer D: Wolff-Parkinson-White syndrome and atrial fibrillation. Relation between refractory period of the accessory pathway and ventricular rate during atrial fibrillation. Am J Cardiol 34: 777-782, 1974

34. Pappone C, Vicedomini G, Manguso F, et al: Risk of malignant arrhythmia in initially symptomatic patients with Wolff-Parkinson-White Syndrome: Results of a prospective long-term electrophysiology follow-up study. Circulation 125:661-668, 2012

35. Pappone C, Santinelli V: Should catheter ablation be performed in asymptomatic patients with Wolff-Parkinson-White syndrome? Catheter ablation should be performed in asymptomatic patients with Wolff-Parkinson-White syndrome. Circulation 112:2207-2215, 2005

36. Wellens HJ: Should catheter ablation be performed in asymptomatic patients with Wolff-Parkinson-White syndrome? When to perform catheter ablation in asymptomatic patients with a Wolff-Parkinson-White electrocardiogram. Circulation 112:2201-2207, 2005

37. Colavita PG, Packer DL, Pressley JC, et al: Frequency, diagnosis and clinical characteristics of patients with multiple accessory atrioventricular pathways. Am J Cardiol 59:601-606, 1987

38. Sharpe MD, Dobkowski WB, Murkin JM, et al: Propofol has no direct effect on sinoatrial node function or on normal atrioventricular and accessory pathway conduction in Wolff-Parkinson-White syndrome during alfentanil/midazolam anesthesia. Anesthesiology 82: 888-895, 1995

39. Sharpe MD, Dobkowski WB, Murkin JM, et al: Alfentanil-midazolam anaesthesia has no electrophysiological effects upon the normal conduction system or accessory pathways in patients with Wolff-Parkinson-White syndrome. Can J Anaesth 39:816-821, 1992

40. Gómez-Arnau J, Márquez-Montes J, Avello F: Fentanyl and droperidol effects on the refractoriness of the accessory pathway in the Wolff-Parkinson-White syndrome. Anesthesiology 58:307-313, 1983

41. Sharpe MD, Dobkowski WB, Murkin JM, et al: The electrophysiologic effects of volatile anesthetics and sufentanil on the normal atrioventricular conduction system and accessory pathways in Wolff-Parkinson-White syndrome. Anesthesiology 80:63-70, 1994

42. Lavoie J, Walsh EP, Burrows FA, et al: Effects of propofol or isoflurane anesthesia on cardiac conduction in children undergoing radiofrequency catheter ablation for tachydysrhythmias. Anesthesiology 82:884-887, 1995

43. Sharpe MD, Cuillerier DJ, Lee JK, et al: Sevoflurane has no effect on sinoatrial node function or on normal atrioventricular and accessory pathway conduction in Wolff-Parkinson-White syndrome during alfentanil/midazolam anesthesia. Anesthesiology 90:60-65, 1999

44. Schaffer MS, Snyder AM, Morrison JE: An assessment of desflurane for use during cardiac electrophysiological study and radiofrequency ablation of supraventricular dysrhythmias in children. Pediatr Anaesth 10:155-159, 2000

45. Katz J, Kadis L: Anesthesia and uncommon diseases. Philadelphia, PA: WB Saunders, 217-219, 1973.

46. Kumazawa T: Wolff-Parkinson-White syndrome and anesthesia. Jpn J Anesthesiol 19:68-73, 1970

47. Van der Starre PJA: Wolff-Parkinson-White syndrome during anesthesia. Anesthesiology 48:369-372, 1978

48. Oh JK, Holmes DR, Hayes DL, et al: Cardiac arrhythmias in patients with surgical repair of Ebstein's anomaly. J Am Coll Cardiol 6: 1351-1357, 1985

49. Rossi L, Thiene G: Mild Ebstein's anomaly associated with supraventricular tachycardia in sudden death: Clinical morphologic features in three patients. Am J Cardiol 53:332-334, 1984

50. Dionne RA, Goldstein DS, Wirdek PR: Effects of diazepam premedication and epinephrine-containing local anesthetics on cardiovascular and plasma catecholamine responses to oral surgery. Anesth Analg 63:640, 1984

51. Troullos ES, Goldstein DS, Hargreaves KM, et al: Plasma epinephrine levels and cardiovascular response to high administered doses of epinephrine contained in local anesthetics. Anesth Prog 34: 10, 1987

52. Kadoya T, Seto A, Aoyama K, et al: Development of rapid atrial fibrillation with a wide QRS complex after neostigmine in a patient with intermittent Wolff-Parkinson-White syndrome. Br J Anaesth 85: 815-818, 1999

53. Morady F, Kadish AH, Schmaltz S, et al: Effects of resting vagal tone on accessory atrioventricular connections. Circulation 81:86-90, 1990

54. Jacobson L, Turnquist K, Masley S: Wolff-Parkinson-White syndrome. Termination of paroxysmal supraventricular tachycardia with phenylephrine. Anaesthesia 40:657-660, 1985

55. Chen CC, Chen SA, Tai CT, et al: Hyperventilation facilitates induction of supraventricular tachycardia: A novel method and the possible mechanism. J Cardiovasc Electrophysiol 12:1242-1246, 2001

56. Jackman WM, Wang XZ, Friday KJ, et al: Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. N Engl J Med 324:1605-1611, 1991

57. Johnson DC, Nunn GR, Richards DA, et al: Surgical therapy for supraventricular tachycardia, a potentially curable disorder. J Thorac Cardiovasc Surg 93:913-918, 1987

58. Wellens HJ: The wide QRS tachycardia. Ann Intern Med 104: 879, 1986

59. Wellens HJ, Lie KI, Bär FW, et al: Effect of amiodarone in the Wolff-Parkinson-White syndrome. Am J Cardiol 38:189-194, 1976

60. Echt DS, Liebson PR, Mitchell LB, et al: Mortality and morbidity in patients receiving encainide, flecainide, or placebo. The Cardiac Arrhythmia Suppression Trial. N Engl J Med 324:781-788, 1991

61. Gray M, D'Amato L: Medically complex pregnancy: A case report illustrating CNM/MD collaborative management. Journal of Midwifery & Women's Health 6:552-557, 2000

62. Gupta AK, Thakur RK, Wide QRS: complex tachycardias. Med Clin North Am 85:245-266, 2001

63. Akhtar M, Shenasa M, Jazayeri M, et al: Wide QRS complex tachycardia. Reappraisal of a common clinical problem. Ann Intern Med 109:905-912, 1988

64. Morady F, Baerman JM, DiCarlo LA Jr, et al: A prevalent misconception regarding wide-complex tachycardias. JAMA 254: 2790-2792, 1985

65. Mehta D, Wafa S, Ward DE, et al: Relative efficacy of various physical maneuvers in the termination of junctional tachycardia. Lancet 1:1181-1185, 1988

66. Tyagi A, Sethi AK, Agarwal V, et al: Rate-dependent left bundle branch block during anaesthesia. Anesth Intensive Care 32:715-718, 2004

67. DiMarco JP, Sellers TD, Lerman BB, et al: Diagnostic and therapeutic use of adenosine in patients with supraventricular tachyarrhythmias. J Am Coll Cardiol 6:417-425, 1985

68. Ellenbogen KA, Thames MD, DiMarco JP, et al: Electrophysiological effects of adenosine in the transplanted human heart: Evidence of supersensitivity. Circulation 81:821-828, 1990

69. Lerman BB, Wesley RC, Belardinelli L: Electrophysiologic effects of dipyridamole on atrioventricular nodal conduction and supraventricular tachycardia: Role of endogenous adenosine. Circulation 80:1536-1543, 1989

70. Rinkenberger RL, Prystowsky EN, Heger JJ, et al: Effects of intravenous and chronic oral verapamil administration in patients with supraventricular tachyarrhythmias. Circulation 62:996-1010, 1980

71. Mandel WJ, Laks MM, Obayashi K, et al: The Wolff-Parkinson-White syndrome: Pharmacologic effects of procainamide. Am Heart J 90:744-754, 1975

72. Wellens HJ, Durrer D: Effect of procainamide, quinidine, and ajmaline in the Wolff-Parkinson-White syndrome. Circulation 50:114-120, 1974

73. Lexi-Comp Online, Procainamide: Drug Information, Hudson, Ohio: Lexi-Comp, Inc.; July 2013. www.merckmanuals.com/professional/lexicomp/procainamide.html.

74. Kowey PR, Friehling TD, Marinchak RA: Electrophysiology of beta blockers in supraventricular arrhythmias. Am J Cardiol 60: 32D-38D, 1987

75. Gomes JA, Kang PS, Hariman RJ, et al: Electrophysiologic effects and mechanisms of termination of supraventricular tachycardia by intravenous amiodarone. Am Heart J 107:214-221, 1984

76. Kappenberger LJ, Fromer MA, Steinbrunn W, et al: Efficacy of amiodarone in the Wolff-Parkinson-White syndrome with rapid ventricular response via accessory pathway during atrial fibrillation. Am J Cardiol 54:330-335, 1984

77. Neumar RW, Otto CW, Link MS, et al: Part 8: Adult Advanced Cardiovascular Life Support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 122:S729-S767, 2010

78. Fuster V, Ryden LE, Cannom DS, et al: ACC/AHA/ESC 2006 Guidelines for the Management of Patients With Atrial Fibrillation A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation). J Am Coll Cardiol 48:854-906, 2006

79. Stewart RB, Bardy GH, Greene HL: Wide complex tachycardia: Misdiagnosis and outcome after emergent therapy. Ann Intern Med 104:766-771, 1986

80. Dancy M, Camm AJ, Ward D: Misdiagnosis of chronic recurrent ventricular tachycardia. Lancet 2:320-323, 1985