Review article

Cardiac electrophysiology and conduction pathway ablation

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Invasive cardiac electrophysiological (EP) testing and transcatheter ablation are new methods available for the diagnosis and treatment of complex dysrhythmias. The purpose of this review is to familiarize anaesthetists with these procedures. The information presented combines a literature review with the authors' experience. This article reviews normal cardiac conduction, tachycardia pathogenesis, principles of cardiac EP study and techniques of conduction pathway ablation. The anaesthetic considerations, including the choice of anaesthetic agent, monitoring problems, drug interactions, special methods of dysrhythmia termination in the EP lab, and complications specific to these procedures, are detailed. Balanced general anaesthesia or monitored anaesthesia care (MAC) sedation with benzodiazepines, propofol and narcotics are acceptable. Several conclusions can be drawn: transcatheter ablation is an effective treatment for many reentry tachycardias; anaesthetic assistance for this procedure will increasingly be needed; anaesthesia can easily be provided without influencing accurate EP testing; overdrive pacing is the method of choice for terminating tachydysrhythmias in the EP lab.

Les tests d'électrophysiologie (EP) invasive et l'ablation transcathétérisme cardiaque représentent des nouvelles méthodes de diagnostic et de traitement des arythmies complexes. Cet article vise à familiariser les anesthésistes avec ces interventions par l'association d'une revue de la littérature à l'expérience des auteurs. On y passe en revue la conduction cardiaque normale, la pathogénèse des tachycardies, les principes d'investigation EP et les techniques d'ablation des voies de conduction. Les considérations anesthésiques dont le choix des agents, le monitorage et ses problèmes, les interactions pharmacologiques, les principales méthodes d'arrêt des arythmie en laboratoire d'EP et les complications spécifiques à ces interventions sont abordés. L'anesthésie générale équilibrée ou la sédation monitorée avec les benzodiazépines, propofol et opiacés sont appropriées. On peut tirer plusieurs conclusions: l'ablation transcathétérisme est une traitement efficace pour plusieurs tachyarythmies de réentrée; on aura de plus en besoin de l'assistance des anesthésistes pour cette intervention; l'anesthésie elle-même n'interfère pas nécessairement avec les épreuves EP; l'entraînement électrosystolique rapide est la méthode de choix pour mettre fin aux tachydysrythmies en laboratoire d'EP.

Key words
ANAESTHESIA: cardiovascular;
HEART: arrhythmia, electrocardiography.

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Contents

Introduction
Normal cardiac conduction
Tachycardia pathogenesis
Cardiac electrophysiological study (EPS)
Influence of anaesthetic agents on cardiac conduction
Antidysrhythmic drugs
Percutaneous catheter ablation
Management of anaesthesia
Dysrhythmia termination
Complications
Summary

Invasive cardiac electrophysiological (EP) testing and transcatheter ablation techniques for treatment of reentrant dysrhythmias are in a rapid phase of development. An increasing number of centres now offers this technology to patients, and many thousands have been successfully treated.[1,2] As cardiac electrophysiologists gain experience in diagnosing and managing more seriously compromised patients, anaesthetists will be asked for assistance more frequently. Anaesthetists are asked to attend the EP lab for several purposes: to provide anaesthesia and airway management for cardioversion, to

supervise sedation and haemodynamic monitoring, and to provide general anaesthesia and invasive monitoring for selected patients undergoing transvascular ablative procedures or internal defibrillator checks.

Cardiac EP study and conduction ablation therapy present the anaesthetist with unique problems. These include the underlying cardiac disease, interaction between anaesthetic and antidysrhythmic drugs, interference by anaesthetic drugs on cardiac conduction pathway mapping, distinct monitoring requirements and special methods of dysrhythmia termination unique to the EP lab.

The purpose of this review is to provide an overview of the role of cardiac electrophysiological testing and transcatheter ablation therapy, as well as background information helpful to the safe conduct of anaesthesia in the EP lab. Normal cardiac conduction and mechanisms of tachycardia generation are reviewed. The principles of EP study are discussed and the effects of anaesthetic drugs on cardiac conduction examined. Antidysrhythmics, especially those with notable side effects, are discussed. The transcatheter ablation technique is detailed. This is followed by recommendations on the management of anaesthesia. Special methods of dysrhythmia termination in the EP lab are discussed. Important complications specific to these procedures are highlighted.

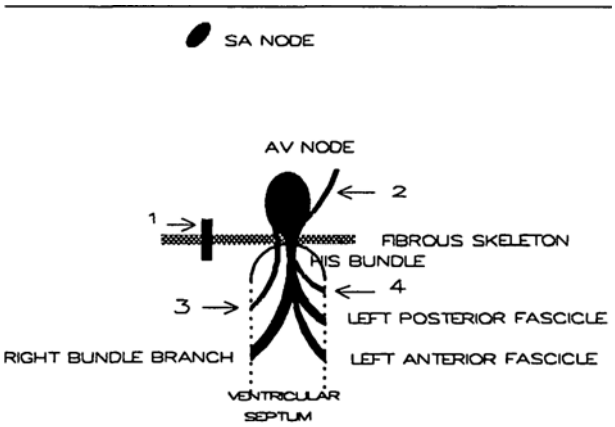

FIGURE 1 Cardiac conduction system. Possible accessory connections include: (1) atrioventricular bypass; (2) AV nodal bypass; (3) nodal-ventricular bypass; (4) His-ventricular bypass tracts.

Normal cardiac conduction

The embryological and histopathological understanding of the cardiac conduction system is incomplete and undergoing continued investigation. Action potentials initiating cardiac depolarization begin at the sino-atrial (SA) node which is a non-discrete area of specialized cells possessing automaticity located near the junction of the superior vena cava and the right atrium. Primary pacemaker function of the heart is provided by a pacemaker complex which includes the SA node and secondary atrial pacemakers (SAP) and is referred to as a multicentric atrial pacemaker complex.[3,4] From the SA node, depolarization spreads through atrial muscle to the atrioventricular (AV) node. Cardiac muscle fibres branch and are interconnected by intercalated discs. Current is able to pass rapidly because of low resistance tight gap junctions in the regions of the intercalated discs. Preferred internodal conduction exists via atrial muscle bundles rather than via specialized tracts. These preferential routes of depolarization appear to be a product of the geometric configuration of the atrium around the openings of the superior and inferior vena cavae, foramen ovale and coronary sinus. Widespread depolarization of the ventricles from the atria is prevented by the interposed, electrically inert fibrous skeleton of the heart.

The AV node is located in the endocardium of the right atrium between the coronary sinus and the medial tricuspid valve leaflet. It contains a graduated heterogeneous group of specialized conducting cells, which are designated atrionodal (AN), nodal (N), nodal-His (NH) and His (H) bundle cells. These cells slow conduction allowing ventricular filling to occur. The histological basis for slowing of conduction in the AV node is related to the sparse intercellular connections, and thus few intercalated discs and tight gap junctions. This slowing is demonstrated on the surface ECG by the P-R interval. The atrioventricular (His) bundle emerges from the AV node and descends along the posterior border of the membranous ventricular septum. Depolarization accelerates in the His bundle. It divides almost immediately into left and right bundle branches located on each side of the septum. The left bundle divides further into anterior and posterior fascicles. Interindividual anatomical variation exists in the configuration of the bundle branches. Depolarization then proceeds swiftly to the ventricular muscle mass via the Purkinje fibres. The rapid, almost simultaneous, depolarization of ventricular muscle produces synchronized contraction and systolic ejection (Figure 1).

The action potentials of the heart are the basis of cardiac EP study. Generation of the cardiac impulse is a complex process and involves interactions between transmembrane potentials, channels in the cardiac cell membrane (sarcolemma), sodium, potassium and calcium ion gradients, and the sodium-potassium ATPase pump. Depolarization (phase 0) occurs when the transmembrane threshold potential is reached and membrane characteristics change to permit influx of extracellular sodium ions causing a less negative transmembrane potential and voltage spike. The atrial and ventricular muscle fibres and the Purkinje fibres have rapid phase 0 depolarization from fast sodium channel fluxes. However, action potentials of the SA node and AV node have gradual initial (phase

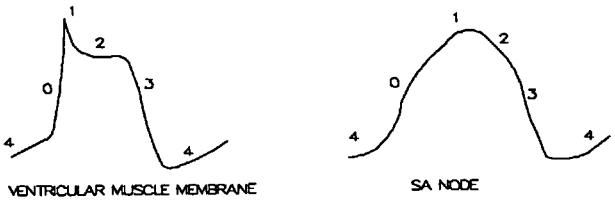

FIGURE 2 Cardiac action potentials.

0) depolarization phases dependent on slow calcium channel fluxes. Repolarization consists of a brief phase 1 followed by phase 2, the plateau phase, during which slow inward flux of calcium ions and prevention of potassium egress occurs. Phase 3 represents a return to basal transmembrane potential due to a sudden increase in potassium permeability and normalization of permeability to sodium ions. During these phases the membrane is either absolutely or relatively refractory to depolarization. Phase 4 represents the resting period between action potentials (Figure 2).[3]

Pacemaker cells, such as the SA node, gradually build up sodium ions during phase 4. As a result, they reach threshold potential spontaneously and depolarise, a property termed automaticity, which is the normal mechanism for the generation of cardiac rhythm. Usually, the cells of the SA node possess the most rapid automaticity and act as the pacemaker. However, other pacemaker cells can assume pacemaker function if they are depolarizing at a more rapid rate than the SA node or if the SA node malfunctions such as with sick sinus syndrome or conduction block.

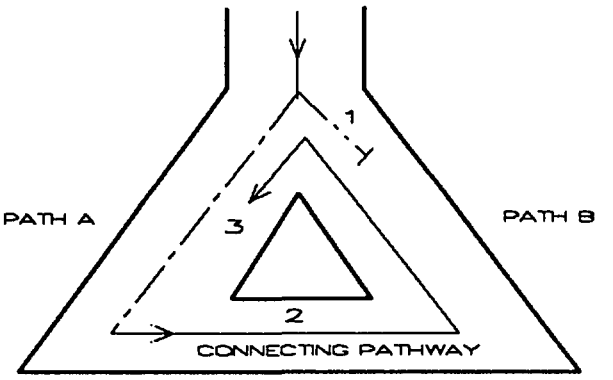

FIGURE 3 Reentry circuit. (1) Action potential finds path B refractory and path A available for depolarization; (2) conduction in path A is slow and path B recovers; (3) path B now depolarizes retrogradely and repeating reentry circuit is established.

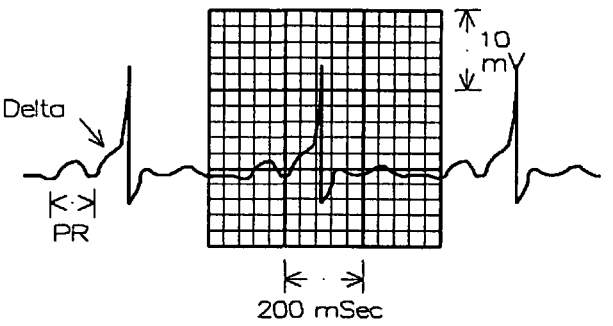

FIGURE 4 Wolff-Parkinson-White syndrome. Standard Lead II, sinus rhythm. Note short PR, widened QRS and delta wave.

Tachycardia pathogenesis

Reentry and increased automaticity are the two basic mechanisms by which tachycardias are generated; of these, reentry is the more common. Connected pathways with differing conduction velocities and refractory times are required to produce a reentry circuit. In the presence of these conditions tachycardia can be initiated by a premature impulse. If the premature depolarization enters the circuit when one pathway is able to propagate the impulse and the other pathway (initially refractory) is able to conduct retrogradely, a self-perpetuating "circus movement" may be initiated (Figure 3). As part of its loop a reentrant circuit may include an accessory atrioventricular pathway, the divisions of a bundle branch, the terminal Purkinje fibres or be within the AV node itself.

Patients with pre-excitation syndrome are a subset who are especially prone to reentry tachycardia. These syndromes are characterized by congenital anomalous muscular connections through the fibrous skeleton which link the atria to the ventricles and allow direct conduction. Pre-excitation via the Bundle of Kent is named Wolff-Parkinson-White (WPW) syndrome. Paroxysmal supraventricular tachycardia (PSVT) in these patients is due to anterograde (antidromic) or retrograde (orthodromic) reentry through accessory pathways.[5] The polarity of the delta wave on the surface electrocardiogram (ECG) as well as behaviour during EP study help to locate the accessory pathway (Figure 4).[6] Atrial fibrillation (AF) can be life-threatening for patients with WPW. Extremely rapid ventricular rate incompatible with adequate cardiac output can occur if there is one-to-one conduction through the accessory pathway to the ventricle.[7] Ventricular fibrillation may even occur. Patients with WPW may have a normal surface ECG but have a pathway capable of orthodromic SVT; this is called a "concealed" accessory pathway. It is not uncommon for patients with pre-excitation to have multiple accessory connections. The electrophysiological mechanism of reentry in WPW is well understood and transcatheter ablation is curative.[8]

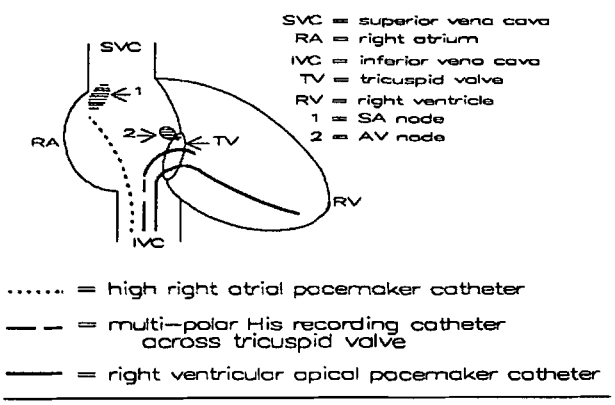

FIGURE 5 EPS catheter locations.

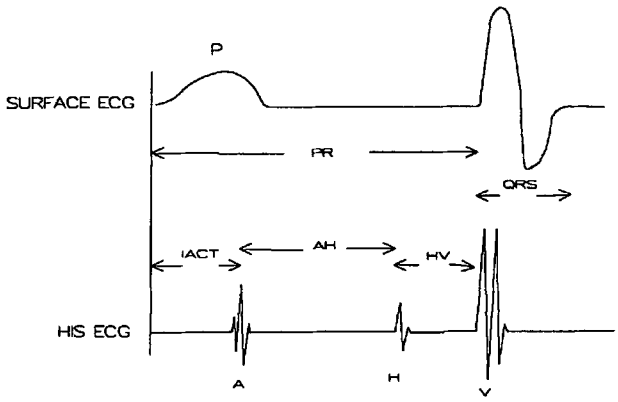

FIGURE 6 HIS bundle electrogram. IACT = intraatrial conduction time; AH = atrial-His conduction time; HV = His-ventricular conduction time.

Another, less common, mechanism of tachycardia pathogenesis is altered automaticity. Increased automaticity leads to cardiac dysrhythmias due to repetitive firing from a single abnormal focus. Metabolic factors are the most common cause of enhanced automaticity. These include increased circulating catecholamines, hypoxaemia, hypercarbia, acute hypokalaemia, hypomagnesaemia, changes in myocardial wall tension and myocardial ischaemia.[5]

A third, but less well deliniated, form of dysrhythmia pathogenesis is triggered automaticity. Triggered automaticity has some of the characteristics of both reentry and enhanced automaticity. Triggered automaticity results when oscillations in the transmembrane potential occur following an action potential (afterdepolarizations), reach threshold, and themselves initiate depolarization. Many of the metabolic factors which are thought to facilitate altered automaticity are implicated in triggered automaticity. The importance of triggered automaticity is under investigation by electrophysiologists.[5,7]

Cardiac electrophysiological study (EPS)

The EPS can be used to assess a wide array of cardiac rhythm disturbances. The function of the SA node, AV node and His-Purkinje system can be characterized. Electrophysiological study is invaluable for confirming the diagnosis and for selecting and testing response to pharmacological therapy.[9] It is an important tool for the identification of tachycardia mechanism in those patients surviving sudden cardiac death (SCD) not associated with myocardial infarction.[10] In addition, reentrant dysrhythmias can be intentionally triggered, their location mapped and response to therapy evaluated.[11] Information is obtained by programmed atrial and ventricular pacing during which localized intracardiac electrograms are recorded and analyzed. Computerization has made storage, retrieval and display of the generated data manageable. By recording from multiple locations in the heart, conduction time from one location to another can be measured. Transvenous pacing catheters are placed in the high right atrium, the right ventricular apex, and adjacent to the His bundle at the level of the tricuspid valve ring (Figure 5). The location of the AV node is identified electrically by the earliest His bundle deflection. The His bundle electrogram (HBE) records the localized depolarization of the proximal His bundle (Figure 6). Measurement of the interval from low right atrium to His bundle (A-H interval) allows approximation of the delay in conduction over the AV node. The temporary transvenous right ventricular pacing lead is tested for capture prior to conduction mapping. Its purpose is dysrhythmia stimulation, overdrive pacing and backup ventricular pacing in the event of symptomatic bradycardia. Incremental pacing and "extra stimulus" pacing are ways of introducing premature impulses and assessing conduction and refractoriness. They are also a means of triggering reentrant rhythms. Electrophysiological mapping of abnormal conduction is accomplished by triggering the offending dysrhythmia and precisely following the sequence of impulse propagation to find the locus of aberrant conduction.[12]

Influence of anaesthetic agents on cardiac conduction

Many drugs commonly administered by anaesthetists influence cardiac conduction. Inhalational anaesthetics, intravenous induction agents, neuromuscular relaxants, opioids and anticholinergics may all interfere with EPS. Mechanisms by which anaesthetic drugs influence conduction include direct electrophysiological effects, neurally mediated changes in autonomic nervous system tone, and indirect acid-base and electrolyte changes occurring during spontaneous and controlled ventilation.[13,14] The ideal anaesthetic for EPS should not alter intrinsic pacemaker function, impulse propagation, refractoriness or autonomic tone. Nor should it prevent necessary triggering

of reentrant arrhythmias. In addition, anaesthesia should be rapidly reversible with minimal delay in emergence. Most anaesthetic agents have not undergone scrutiny in the context of EPS and, therefore, agents must be selected by cautious extrapolation from animal and laboratory experiments. A retrospective analysis of the anaesthetic management of 181 patients undergoing surgical cryoablation of accessory conducting pathways concluded that the majority of these patients can be managed with standard balanced anaesthesia methods.[15] It is yet to be determined if this same conclusion holds for the more subtle percutaneous mapping and ablation methods.

Inhalational anaesthetics alter cardiac conduction by a variety of mechanisms. All of the commonly used volatile agents (halothane, isoflurane and enflurane) enhance automaticity of secondary atrial pacemakers (SAP's) relative to the SA node thus accounting for the occurrence of ectopic atrial rhythm disorders and wandering atrial pacemakers.[16,17] There are varying effects on the AV node and His-Purkinje system.[18] In general, the volatile anaesthetics prolong the QT interval and cause dose-dependent reductions in myocardial contractile force.[19,23] Much of the laboratory investigation of dysrhythmogenicity of inhalational anaesthesia has been performed using either ischaemic cardiac canine models or examining thresholds to norepinephrine or epinephrine induced dysrhythmias.[20-22]

Halothane is particularly noted for its profound ability to depress cardiac impulse formation in the SA node resulting in junctional and wandering atrial pacemaker rhythms.[23] In addition, halothane decreases central release of catecholamines which leads to vagal predominance with sinus bradycardia and junctional rhythm. There is also a reduction of automaticity (phase four depolarization) in the AV node and the His-Purkinje system.[24-26] This effect of halothane, in conjunction with sensitizing the heart to catecholamines, may facilitate AV nodal reentry and may be the mechanism which makes halothane apt to produce tachydysrhythmias.[27-29]

Enflurane does not interfere with cardiac impulse conduction to the same degree as halothane and, in general, the heart rate remains constant. However, enflurane slows AV conduction and the QT interval is markedly lengthened. The myocardium is not sensitized to the effects of catecholamines and, overall, there is a reduced tendency to dysrhythmias.[30,31]

Heart rate increases with isoflurane. Conduction of impulses through the His-Purkinje system is slowed. However, isoflurane does not depress conduction within the atrioventricular node.[23,32,33] The newer inhalational anaesthetics, sevoflurane and desflurane appear to have cardiovascular effects similar to the other volatile agents.[34]

Theoretically, sympathetic activation by nitrous oxide may, in the presence of myocardial sensitization by halogenated agents, set the stage for dysrhythmias. This effect, when examined in clinical studies, exists but only to a small degree.[35-37]

Neuromuscular relaxants affect cardiac conduction at several levels. They may influence autonomic tone through ganglionic stimulation or blockade, act directly at sympathetic nerve terminals or, through histamine release, cause vasodilatation and reflex tachycardia.[38] The acetylcholine (ACh)-like activity of neuromuscular relaxants may cause varied effects at sites of ACh neurotransmission other than at the neuromuscular junction, e.g., at autonomic ganglia and parasympathetic nerve terminals. Succinylcholine causes both brady-and tachydysrhythmias. Pancuronium is vagolytic at the postganglionic nerve terminal and thus increases heart rate.[23] In addition, pancuronium releases norepinephrine at cardiac sympathetic nerve terminals. Vecuronium and atracurium may be associated with bradycardia, particularly if used in combination with other vagotonic drugs such as the potent opioids and propofol.[39,40] The newer, short- and intermediate-acting, nondepolarizing muscle relaxants, mivacurium and rocuronium, are reported to be essentially free of cardiovascular side effects.

Opioids are well known for their central vagotonic effect and resultant bradycardia, especially if given rapidly in high doses.[41-44] Opioids affect cardiac calcium and potassium ion channels to prolong the action potential which supports evidence of opioid antidysrhythmic activity similar to class 3 antiarrhythmic agents.[45-47] During opioid-based anaesthesia the QT interval is prolonged,[48-50] but it has not been shown whether these effects are mediated by direct membrane-specific actions of opioids or via opioid receptors in the heart.

Propofol is widely used not only as an intravenous induction agent but also as a sole anaesthetic agent.[51,52] Occasionally, propofol can cause bradycardia or tachycardia.[53-56] However, the preponderance of literature suggests that there is little change of heart rate with propofol.[57,58] Propofol, in a dose-dependent manner, enhances epinephrine-induced arrhythmias in dogs.[59] Hypotension with propofol is thought to be mediated by inhibition of the sympathetic nervous system with impaired baroreflex regulatory mechanisms.[60]

Benzodiazepines produce qualitatively similar effects but vary in their speed of onset and duration of action. All reduce blood pressure by decreasing peripheral vascular resistance and contractility in a dose-dependent manner, leading to reflex tachycardia. There are no known specific side-effects of benzodiazepines on cardiac conduction.[23]

Phenothiazines have a direct negative inotropic effect

and also dilate the peripheral vasculature. They possess type 1A-like antidysrhythmic properties resulting in P-R and Q-T interval prolongation.[23]

In summary, most, if not all, anaesthetic drugs influence cardiac electrophysiological properties.

Antidysrhythmic drugs

In the context of EPS and cardiac conduction pathway ablation the anaesthetist must be aware of the therapeutic indications and potential side effects of antidysrhythmic drugs. Especially important are amiodarone, sotalol, procainamide, quinidine and propafenone.[61]

Antidysrhythmic drugs have been classified on the basis of their cellular mechanism of action by Vaughan Williams (Table).[62]

Amiodarone is primarily used for the oral maintenance therapy of ventricular tachycardia that is not suppressed by other drugs at repeat EP testing. It is also used to prevent recurrent paroxysmal atrial fibrillation or SVT and is currently under investigation for post-infarction dysrhythmia prophylaxis. It causes a unique spectrum of side effects which include potentially fatal pulmonary fibrosis, thyroid function abnormalities and hepatic dysfunction. Amiodarone interferes with thyroid function at multiple sites, decreasing production of thyroxine and interfering with peripheral conversion of T_3 to T_4. This effect most commonly manifests itself as hypothyroidism. Abnormalities in liver function are identified at first by an increasing aspartate aminotransferase (AST, SGOT). Skin photosensitivity is very common with blue discolouration in some. Corneal microdeposits occur in all patients and occasionally interfere with vision. Peripheral neuropathy can occur. These patients should be followed with serial chest radiographs and possibly with thyroid and liver function tests.[63-66]

Sotalol is used in the primary therapy of both VT and SVT. Sotalol, although a class 3 drug, possesses beta-blocking properties with the associated problems of negative inotropism and exacerbation of bronchospasm. Sotalol also causes QT prolongation and thus predisposes to polymorphic ventricular tachycardia (Torsades de Pointes).[67] The QT interval should be carefully monitored in these patients.[68]

Procainamide and *quinidine* are both class 1A drugs. They are used to treat both VT and SVT, sometimes in combination with mexilitene. Procainamide causes a positive antinuclear antibody (ANA) in about one-third of chronic users with a lupus-like syndrome occurring in 15 to 20%. Quinidine causes frequent nausea and diarrhoea and may cause thrombocytopenia and reversible bone marrow depression. Both drugs prolong the QT interval and may predispose to Torsade de Pointes. Given intravenously both can cause profound hypotension by virtue of vasodilation and should be administered slowly.[69]

TABLE Classification of antidysrhythmic drugs

Class	1	Rapid sodium channel blockers
	1A	Slow conduction, prolong refractoriness e.g., quinidine, procainamide, disopyramide
	1B	Minor conduction slowing, no effect or shortens refractory period e.g., lidocaine, mexilitene, tocainide
	1C	Markedly slow conduction, no effect on refractoriness e.g., propafenone, encainide, flecainide
Class	2	Beta sympathetic blockers; major direct affect on action potential e.g., propranolol, esmolol, metoprolol
Class	3	Prolong action potential, marked prolongation of refractoriness e.g., bretylium, amiodarone, sotalol
Class	4	Calcium channel blockers slow conduction and prolong refractoriness e.g., verapamil, diltiazem
Class	5	Cardiac glycosides e.g., digoxin

Propafenone is given orally to prevent both VT and SVT. It causes potent slowing of conduction especially in the Purkinje-His system. The refractory period of the AV node is mildly increased, and the refractory periods of accessory pathways are markedly prolonged. Propafenone also possesses weak beta-adrenergic blocking effects which may exacerbate asthma.[70]

Recent epidemiological studies suggest that the dysrhythmogenic (proarrhythmic) side effects of some antidysrhythmics may be an important cause of morbidity and mortality themselves.[71-74] In view of this concern about antidysrhythmic drug therapy, as well as the expense and inconvenience of life-long drug therapy, it is not surprising that considerable attention has been drawn to nonsurgical, nonpharmacological therapy for dysrhythmias.

Percutaneous catheter ablation

Cardiac electrophysiologists have evolved the EPS not only as a diagnostic but also as a therapeutic tool. Percutaneous catheter ablation is a technique of selectively interrupting cardiac conduction pathways in patients with symptomatic tachycardia refractory to drug therapy. Several adverse consequences of antidysrhythmic drug therapy exist ranging from cardiac failure, bronchospasm, and organ toxicity to proarrhythmia and death. Catheter ablation technology provides an alternative to drug therapy for patients who are either unresponsive to drug therapy or who are unable or unwilling to tolerate their side effects. It is an attractive alternative to life-long drug therapy in young patients. There are three basic applications of the catheter ablation method: ablation of supraventricular bypass tracts, AV node ablation and ablation of ventricular re-entrant pathways. Localized energy is delivered via an intracardiac catheter to the endocardium adjacent to the area of aberrant conduction. This injured

tissue becomes electrophysiologically inactive, scars and prevents recurrence. The technique has superseded open cardiac procedures for most cases.[75,76]

Transcatheter ablation for control of tachydysrhythmias was first used in 1982.[77,78] The initial energy source used DC current generated by a defibrillator and passed through a catheter electrode. The high level of uncontrolled energy caused frequent complications, including perforation and proarrhythmia. Ventricular fibrillation may occur immediately after or up to seven days after DC ablation.[79,80] Since 1986, radiofrequency (RF) energy has replaced DC shock as a more common energy source and is a safer energy source for ablation of the AV node and accessory pathways.[81] The RF energy is low-power, high-frequency alternating current which causes injury by generating heat at the electrode/tissue interface. The advantages include control of energy delivery, the creation of a smaller area of injury, and the ability to be used safely in thin-walled structures such as the coronary sinus. It is usually painless and seldom triggers malignant dysrhythmias. When DC current is used general anaesthesia is required because of the severity of pain.[82-84]

Supraventricular tachycardias amenable to catheter ablation technique include PSVT, paroxysmal atrial fibrillation (PAF), chronic AF and atrial fib-flutter.[85] In patients with pre-excitation, SVT can be abolished either by ablation of the AV node or ideally by creating a lesion in the accessory pathway itself. Injury to the AV node or His bundle, intentional or accidental, may cause complete heart block. If this occurs, a permanent ventricular pacemaker is required. When it is possible to ablate the bypass tract specifically and prevent re-entry the patient will be cured.

Another application is the specific destruction of an identifiable ventricular reentrant focus. This subset of patients presents with paroxysmal ventricular tachycardia (PVT) or paroxysmal ventricular fibrillation (PVF).

Delivering a small, precisely located, energy surge to the moving endocardium is technically difficult and often requires multiple attempts. In the patient with pre-excitation and PSVT, successful ablation is recognized by the disappearance of delta waves and lengthening of the P-R interval. In patients with AF and rapid ventricular response the goal is AV dissociation. Inability to re-trigger VT identifies success in the patient with PVT.

Effective ablation is further tested by attempting to re-trigger the offending dysrhythmia after administration of atropine (0.6–1.2 mg) and isoproterenol (0.5–7$\mu g \cdot min^{-1}$) by infusion. A period of observation in the EP laboratory helps to exclude delayed recovery of an ablated area. We have commonly observed arousal and awakening during light levels of anaesthesia when isoproterenol is infused and we deepen anaesthesia at this time. After successful RF destruction of the proximal AV node or offending accessory pathway the patient is monitored by telemetry for 24 hr. If the pacemaker function of the AV node is damaged a well-secured temporary pacemaker is left *in situ* before leaving the EP laboratory and the patient monitored in the cardiac care unit (CCU) until a permanent pacemaker can be inserted.

Management of anaesthesia

Preoperative preparation includes thorough patient history, physical examination, laboratory testing and optimization of medical therapy. The anaesthetist should enquire about the frequency and duration of rhythm disturbances. It is of critical importance to the safe conduct of anaesthesia that patients who decompensate during tachycardia, with symptoms of syncope, severe angina or congestive heart failure, be identified before EPS. Rapid identification of these symptoms during EPS will facilitate rapid termination of the causative rhythm. Anxiolysis by reassurance and benzodiazepine premedication minimizes excessive sympathetic tone. All non-essential drugs, especially antidysrhythmic agents are discontinued several days before EPS in order to promote SVT or VT.[86] Optimization of medical therapy generally constitutes adequate treatment of angina and congestive heart failure. Patients with sustained PVT often have severe previous myocardial damage, are at high risk for any procedure, and may also be under consideration for insertion of an automatic defibrillator.[1] Minimal preoperative laboratory tests include complete blood count (CBC), platelet count, prothombin time (PT, INR), electrolytes, urea, creatinine and ECG. Abnormalities of electrolytes, PT and platelet numbers should be identified and corrected beforehand. Patients are fasted preoperatively; those with increased risk for aspiration are given appropriate prophylaxis. Anaesthesia options are discussed with the patient.[6,87-89]

These procedures are exacting for the cardiac electrophysiologist to perform. A motionless patient is required, who, if sedated, is still able to communicate cardiopulmonary and neurological symptoms. Apart from vascular access and the RF bursts the procedure is generally not painful. Analgesia for line insertion is easily accomplished with local anaesthesia. During the 5 to 45 sec periods of RF ablation, repeated up to 15 or more times during the course of the procedure, some patients may experience brief retrosternal angina-like chest pain of mild to moderate intensity.

The EP suite is a difficult environment for the anaesthetist. It is remote from the operating room, dark, and there is limited access to the patient. There is increased potential for electrical hazard. We maintain a fully operational anaesthesia machine with all ancillary equip-

ment and drugs in the laboratory. We employ continuous ECG, combined pulse oximetry/plethysmography and noninvasive blood pressure monitoring as minimum monitors. Arterial monitoring is used for patients with haemodynamically unstable rhythm disturbances. Mapping catheters may partially occlude arterial catheter sheaths; thus a separate radial line is preferred. A defibrillator with leads attached and with the ECG displayed on the screen is employed throughout. Patients with VT or PVF are connected to a defibrillator by hard wired gel pads placed over the sternum and back.

The patient is protected from radiation exposure with thyroid and gonadal lead shields. In anticipation of a long procedure the patient may have a urinary catheter inserted and measures are instituted to prevent the insidious development of hypothermia. Patients may receive antibiotic prophylaxis with a cephalosporin and are anticoagulated with heparin if arterial or transeptal heart catheters are introduced.

We have employed both intravenous sedation and general anaesthesia. Our experience is that propofol by infusion, in the sedative to total anaesthesia dose range of (25–200 $\mu g \cdot kg^{-1} \cdot min^{-1}$), seems to have little influence on EPS and the ability to trigger either SVT or VT.[90] In those patients unable to tolerate propofol-induced hypotension we have been successful using propofol in an attenuated dose supplemented with 70% nitrous oxide. We have observed no difficulty with EPS using alfentanil for sedation or as part of total intravenous anaesthesia (TIVA) in doses of 0.25–1.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, sometimes combined with midazolam in 0.5–2 mg increments. Intravenous sedation has the advantage of allowing the patient to communicate symptoms. The anaesthetist must keep in close verbal contact with patients who are being sedated while haemodynamically unstable rhythms are triggered so that impending loss of consciousness can be identified quickly and measures instituted rapidly to provide airway and vasopressor support while the offending rhythm is terminated. Angina or presyncope signals the need for prompt return to sinus rhythm. Nitroglycerin (NTG) spray or *iv* NTG (1–3 $\mu g \cdot kg^{-1}$) by infusion is used to treat persisting angina. Coronary spasm may respond to sublingual nifedipine 10–20 mg. We prefer phenylephrine (25–100 μg bolus), because of its pure alpha agonistic properties, for initial vasopressor support.

In patients with severe haemodynamic instability or who are at increased risk for aspiration, the advantages of general anaesthesia with a secured airway outweigh the identification of symptoms. General anaesthesia can be satisfactorily accomplished using propofol 100–200 $\mu g \cdot kg^{-1} \cdot min^{-1}$, or isoflurane 0.5–1% and supplemented with nitrous oxide if necessary, with minimal interference on EPS. In patients requiring general anaesthesia, but who are at low risk for aspiration, we prefer airway management with Brain's laryngeal mask airway. Prior to transfer to the CCU or telemetry unit, patients are monitored in the post-anaesthetic care unit (PACU).

Dysrhythmia termination

All anaesthetists are familiar with the guidelines and protocols for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC) outlined by the American Heart Association (AHA) and taught in the Advanced Cardiac Life Support (ACLS) programme.[91] In addition, there are special techniques of dysrhythmia termination available in the EP laboratory.[92,93]

Patients being investigated for haemodynamically unstable rhythms such as VT or VF are provided with hard wired defibrillator gel pads over the right sternum and back so that cardioversion or defibrillation can be performed immediately without having to move fluoroscopic equipment and place defibrillator paddles manually. A machine check, to ensure that current can be delivered, is performed. In anaesthetised patients undergoing automatic implanted cardiac defibrillator (AICD) check, this circuit must be tested on the patient with low energy (e.g., 2 joules) before inducing VF to ensure that the patient can be "rescued"![94] The right ventricular apical pacing catheter must also be checked at the beginning of EPS to ensure capture in the event of symptomatic bradycardia.

Electrical pacing is available for dysrhythmia termination and is the method of choice for terminating both SVT and VT in the EP lab. In haemodynamically stable patients, introduced ventricular premature beats (VPBs) or atrial premature beats (APBs) will often convert the rhythm to sinus. If introduced APBs or VPBs fail to terminate the tachycardia then "burst" pacing is employed. In "burst" pacing a train of three to ten asynchronous paced beats is introduced at a rate faster than the tachycardia and then stopped abruptly. In either case the goal is to terminate reentry by making the circuit refractory to conduction, obviating the need for time-consuming and deleterious effects of drug therapy. For patients not responsive to these measures or who are haemodynamically unstable direct DC cardioversion is employed.

Patients being investigated and treated for PSVT, particularly those with accessory pathways, may inadvertently be triggered into AF. Atrial fibrillation is often a difficult rhythm to terminate. It may sometimes be converted to sinus rhythm with *iv* procainamide, but usually synchronized electrical cardioversion at 100–200 joules is required. Thiopentone, methohexitone and etomidate have all been used successfully for cardioversion.[95] We prefer to use propofol in a hypnotic dose of 1–1.5

$mg \cdot kg^{-1}$ for cardioversion. It has the advantages of short duration of action and salutary effects on the muscles of the airway making ventilation by bag and mask easier than if thiopentone or methohexitone are used. Propofol has become the agent of choice for cardioversion in some centres.[96] Midazolam, with flumazenil reversal, has also been reported to provide anaesthesia safely for cardioversion.[97]

Complications

In general EPS and cardiac transvascular ablation are safe. Complications of the procedure are those of heart catheterization plus those of the ablation technique. Line insertion related problem include haematomas, bleeding, phlebitis, thromboembolism, pneumothorax, pericardial tamponade, pacemaker failure, and rarely endocarditis. A large pneumothorax can be diagnosed most rapidly by fluoroscopy. The Percutaneous Cardiac Mapping and Ablation Registry (PCMAR) reported, in 1988, two deaths in 522 patients who underwent attempted AV junctional ablation with DC shock.[98] The PCMAR reported a 6.7% occurrence of procedural related deaths in those patients undergoing VT ablation in a group of patients with severe pre-existing cardiac disease. Although as yet undocumented, RF ablation appears to be considerably safer, and equally effective as DC current ablation.

The EPS study may cause cardiac ischaemia or failure during mapping of haemodynamically unstable rhythms. This may result in cardiac infarction, pulmonary oedema, hypotension, new dysrhythmia or syncope. Patients experiencing angina are rapidly converted to sinus rhythm and given nitroglycerin if angina persists. All patients have blood samples taken at the end of the procedure to measure cardiac isoenzyme levels. Catheter-induced brady- and tachydysrhythmias can degenerate into unstable rhythms leading to death. Coronary spasm has also been reported after attempted ablation.[99] Direct current, though not commonly used at our centre, is particularly noted for its proarrhythmic potential. Perforation and acute pericardial tamponade have been caused by DC shock delivered across the coronary sinus.[100]

Summary

Cardiac electrophysiological testing and conduction pathway ablation are in an era of rapid evolution with expanding indications and improving technology. The anaesthetist has a special role to play in the management of these challenging patients. The anaesthetist must have a working knowledge of normal and abnormal cardiac conduction and be familiar with antidysrhythmic drugs and their complications. The environment of the EP lab, the patient's disease process and the procedure all present unique problems. The anaesthetist is challenged to provide an anaesthetic safely which takes into account the electrophysiologist's requirements for minimal cardiac conduction interference yet provides a motionless patient who is painfree and recovers rapidly. In addition, the anaesthetist must be alert to procedural complications and their management. There is the potential for collaborative work between cardiac electrophysiologists and anaesthetists in this new and exciting field.

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