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Anesthesiology and Cardiac Electrophysiology Practice

Advances in practice of clinical cardiac electrophysiology (EP) began in the late 1960s with the creation and expansion of EP laboratories. Initially, the primary purpose of these labs was to serve as diagnostic centers for understanding cardiac conduction defects. The success of the EP labs spread into diagnostic studies of patients with tachyarrhythmias, both supraventricular and ventricular. The clinical EP field has now evolved into one where therapeutic techniques have displaced diagnosis as the prime focus. Therapies provided by electrophysiologists encompass two major modalities: 1) catheter-based approaches, for cure or palliation of tachyarrhythmias; and 2) device-based, for both bradyarrhythmias as well as tachyarrhythmias. More recently, EP has influenced the treatment of patients with heart failure, using unique pacing modalities such as CRT. Procedures and interventions in the cardiac EP labs are complex and involve acutely ill patients. As cardiac electrophysiologists gain experience in diagnosing and managing more seriously compromised patients, anesthesiologists are being asked for assistance more frequently. Anesthesiologists are uniquely trained to care for this complex patient population, and allow the cardiologist to focus on completing the interventional procedure successfully. Developing a robust understanding of principles of cardiac electrophysiology and mechanisms of arrhythmias, how the anesthesia drugs, autonomic tone and physiologic variables affect cardiac EP, and specific issues relating to the complex EP procedures, will aid the anesthesiologist in choosing the appropriate anesthetic techniques for complex EP procedures.

Arrhythmia mechanisms: The primary mechanisms of arrhythmogenesesis, in order of frequency and importance, are: Reentry, Abnormal automaticity and Triggered activity. Reentry mechanisms are established if circuit pathways are established between connected tissues where different regions of the myocardium have different conduction velocities and refractory times. Circuit pathways can be anatomical pathways such as those seen in WPW, or microcircuits created as a result of dynamic dispersion of refractoriness seen in the setting of AF/VT/VF. Less commonly seen, abnormal automaticity, leads to arrhythmias due to repetitive discharge from a single or few unique foci. Metabolic derangements are a cause of enhanced automaticity, and can be affected by anesthesia management and other perioperative factors. Some of these factors include increased sympathetic hyperactivity, hypoxemia, hypercarbia, acute hypokalemia, hypomagnesaemia, and changes in myocardial wall tension or ischaemia. A third, less well-defined form of arrhythmia pathogenesis is triggered activity. Triggered activity results when oscillations in the membrane potential (Early or Late afterdepolarizations), occur following an action potential and reach threshold, initiating a new depolarization. Many of the metabolic factors affecting altered automaticity are also responsible for triggered activity.

EP testing and therapy: It is important for the anesthesiologists to be familiar with the key aspects of EP procedures, and recording techniques (and parameters) in order to understand the impact of anesthetics or anesthestia techniques. An EP study is used to assess a wide range of cardiac rhythm abnormalities including assessing the function of the
SA node, AV node and His-Purkinje system. For vast majority of rhythm disorders, the EP study is essential for confirming the diagnosis and for testing response to pharmacological therapy. Reentrant arrhythmias can be intentionally triggered, their location mapped and response to therapy evaluated. EP data is typically obtained during programmed atrial and ventricular pacing while localized intracardiac electrograms (EGMs) are recorded. By recording from multiple sites in the heart, conduction time between regions of the heart can be measured. Pacing catheters are typically placed in the high right atrium, the right ventricular apex, and adjacent to the His bundle at the level of the tricuspid valve annulus. Localized depolarization of the proximal His bundle is recorded on the His bundle EGM. Location of the AV node is identified electrically by the earliest His bundle deflection. Measurement of the interval from low right atrium to His bundle (A-H interval) allows estimation AV nodal conduction delay. A pacing catheter/electrode inserted into the right ventricle is used for tachycardia stimulation, overdrive pacing or backup ventricular pacing in the event of significant bradycardia during an EP procedure. Incremental pacing and extra stimulus pacing are means of introducing premature impulses and assessing conduction and refractoriness, or for triggering reentrant rhythms. EP mapping of the site of aberrant conduction is achieved by triggering the abnormal rhythms and locating the by precisely following the sequence of impulse propagation. In case of catheter ablation, thermal scar lesions in the region of aberrant conduction are typically created by precise delivery of RF energy. Other sources of energy such as focused ultrasound (HIFU) have also been employed. Catheter ablation is successfully used for several indications- supraventricular macro-reentrant arrhythmias like atrial flutter, WPW, AVNRT; Atrial fibrillation (isolation of pulmonary veins); Ventricular tachycardias (through either endocardial or epicardial approaches; or for therapeutic ablation of AV node.

Anesthetic techniques and cardiac electrophysiology: It is commonly assumed that many drugs administered by anesthesiologists influence cardiac conduction and myocardial refractoriness. However, data on this subject are limited and the exact influences anesthetics have on cardiac EP in the clinical setting are not entirely evident. Inhalational anesthetics, intravenous agents, neuromuscular blockers, opioids and anticholinergics may all interfere with cardiac EP parameters under certain conditions. Mechanisms by which anesthetic drugs influence conduction include direct myocardial effects, neurally mediated changes in autonomic nervous system tone, and indirectly through changes in acid-base or electrolyte changes occurring during spontaneous and controlled ventilation. The ideal anesthetic should not alter intrinsic pacemaker function, impulse propagation, refractoriness or autonomic tone. It should not suppress the ability to identify aberrant pathways during attempts to trigger and simulate the reentrant arrhythmia. Additionally, anesthesia should be quickly reversible allowing rapid emergence in most instances. Most anesthetic agents have not undergone a comprehensive study in the context of clinical EP study or for ablation procedures. Thus, anesthesia drugs and techniques should be chosen by measured extrapolation from animal and laboratory investigations. A large retrospective study patients undergoing open surgical cryoablation of accessory conducting pathways suggested that for majority of these patients a balanced anesthesia technique can provide adequate conditions for identifying aberrant pathways without significantly altering electrophysiology. Several small clinical prospective investigations have tried to address similar questions regarding various anesthetics during interventional EP and ablation procedures. However, detailed evaluations in patients undergoing AF/VT ablation are not available.
Inhalational anesthetics alter cardiac conduction by a variety of mechanisms. All of the commonly used volatile agents enhance automaticity of secondary atrial pacemakers relative to the SA node accounting for the occurrence of ectopic atrial rhythms and wandering atrial pacemakers. Inhalational anesthetics also demonstrate varying effects on the AV node and His-Purkinje system. Most of the volatile anesthetics prolong the QT interval and cause dose dependent reductions in myocardial contractile force. It is noteworthy that many of the laboratory investigation of arrhythmogenecity of inhalational agents have been performed using either ischemic cardiac canine models or examining thresholds to catecholamine induced arrhythmias. Although an increase in heart rate is frequently seen with isoflurane, conduction of impulses through the His-Purkinje system is slowed. AV nodal conduction is however unaffected by isoflurane. Effects of sevoflurane are similar to isoflurane. Neuromuscular relaxants influence cardiac electrophysiology by various mechanisms and at different levels in the autonomic nervous system. They modulate autonomic tone through ganglionic stimulation or blockade, act directly at sympathetic nerve terminals or, through histamine release, cause vasodilatation and reflex tachycardia. The cholinergic properties of the neuromuscular relaxants can lead to varied effects at autonomic ganglia and parasympathetic nerve terminals. For instance, succinyl choline can precipitate both brady-and tachyarrhythmias. Pancuronium is vagolytic at the postganglionic nerve terminal, increasing heart rate. In addition, pancuronium releases norepinephrine at cardiac sympathetic nerve terminals. Vecuronium may be associated with bradycardia, particularly if used in combination with other vagotonic drugs such as the potent opioids. Mivacurium and rocuronium are suggested to be mostly free of cardiovascular side effects. Opioids, especially when administered in high doses have a central vagotonic effect with resultant bradycardia. They alter cardiac calcium and potassium ion channels to prolong the action potential mimicking anti-arrhythmic activity of class III anti-arrhythmic agents. During opioid-based anesthesia the QT interval is prolonged, but it is unclear if these effects are due to direct membrane-specific actions of opioids or via opioid receptors in the heart. Propofol, another widely used intravenous agent can occasionally cause both abnormalities in heart rate response, however, in a randomized clinical study, there was no effect on AV nodal EP properties. Benzodiazepines produce qualitatively similar effects but vary in their speed of onset and duration of action. There are no known specific side-effects conduction. All reduce blood pressure by decreasing peripheral vascular resistance leading to reflex tachycardia.

Central neuraxial modulation of autonomic tone has recently been shown to be effective in management of intractable ventricular arrhythmias, and is being increasingly used in many centers. Prior animal experiments have demonstrated that spinal cord stimulation or left stellate ganglionectomy can decrease the sympathetic discharge of the cardiac ganglia and intracardiac nerve plexus, having a favorable response on sympathetically driven VT. Recent report on the successful use of selective thoracic epidural sympathetic block for controlling intractable VT, in the setting of persistent ICD shocks for VT storm, suggests that anesthesia techniques of neuraxial/stellate ganglion modulation can be effective in managing some patients with VTs. Practice guidelines proposed by ACC/ACC/HRS also support this approach as a alternative therapy in management of intractable VTs.

In addition to the consideration given to the choice of drugs, the anesthesiologist has to align his technique of sedation or general anesthesia with the needs of the interventional procedures. Certainly, there are many procedures that can be accomplished with sedation while others necessitate general anesthesia with varying levels of invasive monitoring. Another vital area in which anesthesiologist play a key role in management of these procedures is providing diagnostic imaging echocardiography for identifying pre-
procedure thrombus (and other abnormal findings) or for guiding placement/ navigation of various catheters and sheaths in the heart.

**Summary:** Cardiac EP therapies and management are one of the fastest growing fields in cardiovascular medicine with expanding indications and improving technology. The Anesthesiologists have a special role to play in the management of these challenging patients, from managing sedation, autonomic tone or providing imaging support with echocardiography. The anesthesiologist should have a working knowledge of cardiac EP and be familiar with the electrophysiological effects of anesthetic and anti-arrhythmic drugs. The environment of the EP lab, the patient's disease process and the procedure all present distinctive problems that are usually not seen in patients presenting for non-EP procedures. In addition, the anesthesiologist must be aware of procedural complications and their management. There remains an immense potential for collaborative work between clinical cardiac electrophysiologists and anesthesiologists in this rapidly evolving field.

**References:**


