SUMMARY

Cardiac arrhythmias are caused by disorders of action potential formation or propagation and are broadly categorized as abnormally slow rhythms (i.e., bradycardias) or abnormally rapid rhythms (i.e., tachycardias). The cardiac cellular action potential is composed of five phases determined by the activity of multiple ion channels, including the rapid sodium channel, several potassium channels, and a calcium current. Disruptions of these currents may lead to abnormal automaticity and triggered activity, which may mediate pathologic tachyarrhythmias. Reentry is the dominant mechanism of clinically significant tachyarrhythmias and requires a functional or fixed obstacle to propagation, an area of slowed conduction, and differential refractoriness for initiation and perpetuation of the arrhythmia.

Antiarrhythmic drugs are commonly divided into four broad groups using the Singh–Vaughn Williams classification. Despite its clinical utility, many antiarrhythmic drugs have multiple effects and do not fit neatly into this framework. Some, such as adenosine and digoxin, fall completely outside of it. Class I drugs slow membrane conduction by blockade of the sodium channel. Class II drugs, or β-blockers, function by blockade of the cardiac β-receptor. Class III drugs prolong repolarization and the QT interval. Class IV drugs block the slow calcium channel and are primarily active in slow-response myocytes such as the sinus and AV node.

All bradycardia is a consequence of impairment of sinus node function or AV conduction, or both. Sinus and AV nodal function is strongly influenced by autonomic tone. Parasympathetic tone dominates at rest, and significant bradycardia and second-degree AV block may be observed in normal patients due to increased parasympathetic tone, especially during sleep or athletic training. Clinical sinus node dysfunction manifests as one of several syndromes, including sinus bradycardia, chronotropic incompetence, exit block, and bradycardia-tachycardia syndrome due to sinus pauses and bradycardia when concomitant atrial arrhythmias terminate to sinus rhythm.

AV conduction disturbances may occur at the AV nodal level or infranodal level. A block at the level of the AV node tends to be indolent, characterized by gradual progression and competent subsidiary escapes that usually protect the patient from catastrophic bradycardia. This permits asymptomatic patients to be followed clinically for the development of symptoms before intervention. In contrast, second- or third-degree infranodal block at the His bundle, or more commonly at the level of the bundle branches, is potentially malignant and is often not accompanied by stable escape mechanisms. If not managed appropriately, it can cause sudden death. Clues to an infranodal level of block are Mobitz II periodicity, associated bundle branch block, worsening heart block with tachycardia or exercise, and a wide QRS escape rhythm different from the conducted QRS in the setting of a high-degree or third-degree AV block.

Tachycardias are broadly categorized as SVTs, which depend on the atrium and AV conduction system, and ventricular arrhythmias, which depend on the ventricular myocardium. Supraventricular arrhythmias are further categorized as PSVTs, which depend on AV nodal conduction, and intra-atrial arrhythmias, which depend only on atrial tissue and not on AV conduction. The PSVTs include AVNRT and AV reciprocating tachycardia related to WPW syndrome. Intra-atrial arrhythmias include organized atrial arrhythmias, such as focal atrial tachycardia, atrial flutter, macro-reentrant atrial tachycardia, and AF, a common disorganized atrial arrhythmia. Recurrent atrial flutter and AF carry a risk of thromboembolism and, based on risk stratification, should be treated with antithrombotic therapy when appropriate. Catheter ablation has an important role in the management of all supraventricular arrhythmias but remains a second-line strategy for AF, for which success rates are lower and complication rates are higher than for other supraventricular arrhythmias.

Ventricular arrhythmias include isolated ventricular premature beats; short, nonsustained runs of tachycardia; and sustained ventricular arrhythmias. Sustained VT lasts more than 30 seconds or requires intervention before then. It is classified as monomorphic if beats all share a single electrocardiographic morphology, polymorphic if the electrocardiographic morphology is variable, TdP when the morphology is variable and the arrhythmia is associated with pathologic QT prolongation, and VF when the surface ECG continuously varies without distinct QRS complexes. VT is poorly tolerated and is the major cause of cardiac arrest. Although commonly seen in the setting of ischemic heart disease, idiopathic VT may be seen in the absence of structural heart disease.

Antiarrhythmic drugs have not been effective in reducing the risk of SCD after MI. In contrast, ICDs have been shown to improve mortality rates for patients with impaired LV function after an MI and patients with heart failure and impaired LV function with or without coronary disease.

In addition to advanced structural heart disease as a cause for VT, several syndromes may result in VT in the absence of evident structural heart disease. They include the syndrome of idiopathic VT, ARVC, arrhythmogenic RV dysplasia, congenital LQTS, Brugada syndrome, and CPVT. Several of these conditions are familial, and genetic testing and family screening have important roles in their management.

SUGGESTED READINGS

Calkins H, Kuck KH, Cappato R, et al: 2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for patient selection, procedural techniques, patient management and follow-up, definitions, endpoints, and research trial design: a report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation. Developed in partnership with the European Heart Rhythm Association (EHRA), a registered branch of the European Society of Cardiology (ESC) and the European Cardiac Arrhythmia Society (ECAS); and in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), the Asia Pacific Heart Rhythm Society (APHRS), and the Society of Thoracic Surgeons (STS). Endorsed by the governing bodies of the American College of Cardiology Foundation, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, the Asia Pacific Heart Rhythm Society, and the Heart Rhythm Society, Heart Rhythm 9:632.e21–696.e21, 2012.

Fuster V, Rydén LE, Cannom DS, et al: 2011 ACCF/AHA/HRS focused updates incorporated into the ACC/AHA/ESC 2006 Guidelines for the management of patients with atrial fibrillation; a Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines developed in partnership with the European Society of Cardiology