Anatomy, physiology, and epidemiology of arrhythmias

An arrhythmia is an abnormality of cardiac rhythm. Arrhythmias differ in their population frequency, anatomical substrate, physiological mechanism, etiology, natural history, prognostic significance, and response to treatment. As is emphasized throughout this book, it is important to gain as much information as possible about the substrate and mechanism of an arrhythmia to be able to predict the natural history and to define the prognosis and response to treatment.

Electrical anatomy of the normal heart

The diagram in Figure 1.1 shows a sketch of the electrically active parts of the normal heart. The atrial muscle and ventricular muscle are separated by insulation of the fibrous mitral and tricuspid valve rings, and normally the only connection between them is via the His bundle.

All cardiac myocytes are capable of electrical conduction and have intrinsic pacemaker activity. Each tissue has a conduction velocity and a refractory period, both of which vary with changes in heart rate and influences such as autonomic tone, circulating catecholamines, etc. The conduction velocities of various parts of the heart vary as shown in Figure 1.1.

Basic mechanisms of tachycardias

Although it is not necessary to have a deep understanding of cardiac electrophysiology to diagnose and treat a cardiac arrhythmia, some knowledge of the basics is helpful. Tachycardias are mostly caused by re-entry or abnormal automaticity. Some
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common examples are shown in Figure 1.2. A few rare types of tachycardia are probably caused by a third mechanism, triggered activity.

Figure 1.2

Many common tachycardias are caused by re-entry. This means that there is a self-propagating wave of electrical excitation which maintains the arrhythmia. The fundamental requirements for re-entry are that there should be: (1) an anatomical circuit, (2) a zone of slow conduction in the circuit, and (3) a region of unidirectional block. The best model of re-entry is an orthodromic atrioventricular (AV) re-entry, e.g. Wolff-Parkinson-White syndrome (see Chapter 13). The circuit comprises the accessory pathway, atrium, AV node, and ventricle. The slow conduction is in the AV node and functional unidirectional block can occur in the accessory pathway. Tachycardia is interrupted if one part of the circuit has a refractory period longer than the cycle length of the tachycardia. In practice this is most easily achieved by prolonging AV node refractoriness with adenosine. Tachycardia will restart only if the requirements for reinitiation are met. These include a trigger (often an atrial or ventricular premature beat) and an appropriate balance of electrical behavior of the various parts of the circuit. Re-entry tachycardias can be started and stopped by pacing and stopped by cardioversion. Other examples of re-entry include AV nodal re-entry tachycardia (see Chapter 16), atrial flutter (see Chapter 9), and some types of ventricular tachycardia (see Chapter 18).

Fewer tachycardias are caused by abnormal automaticity. The best model of automaticity is sinus rhythm. Similar to sinus rhythm, automatic (also known as ectopic) tachycardias cannot be started or stopped by pacing and cannot be interrupted by cardioversion. In the normal heart the sinus node has the highest spontaneous rate and, therefore, determines the rhythm. If the sinus node fails another part of the heart with a lower pacemaker rate, usually the AV node, will provide an escape rhythm. Sometimes an area of the heart other than the sinus node will have an abnormally high spontaneous rate and will produce an automatic (or ectopic) tachycardia, overriding the sinus node. Examples of tachycardias caused by enhanced automaticity include atrial ectopic tachycardia (a type of focal atrial tachycardia – see Chapter 7), junctional ectopic tachycardia (see Chapter 17), and some types of ventricular tachycardia (see Chapter 18).

Triggered activity is the least common tachycardia mechanism. Depolarization is caused by a trigger – either an early after-depolarization or a delayed after-depolarization. Triggered activity causes ventricular arrhythmias in long QT
syndrome, some electrolyte disturbances, and in some postoperative ventricular tachycardia with myocardial injury.

### Basic mechanisms of bradycardias

Bradycardias are due to either failure of impulse generation or failure of conduction. The most common example of failure of impulse generation is sinoatrial disease (see Chapter 30). Abnormal sinus node function may be due extrinsic effects (high vagal tone) or to depressed automaticity. Significant bradycardias are more commonly due to second- or third-degree AV block (see Chapters 28 and 29).

### Epidemiology of arrhythmias

Some arrhythmias are more common than others but there are almost no data on the population prevalence of these conditions. However, we recognize that the prevalence and spectrum of arrhythmias change with age. Faced with a new patient with an arrhythmia, our diagnosis is based mainly on the child’s age, the age of onset of arrhythmia, the history (palpitations, heart failure, syncope, etc.), and the ECG findings, but should also take into account the prevalence of different arrhythmias (in other words, a common arrhythmia is often a more likely diagnosis than a rare one).

Probably fewer than half of new tachycardias present in the first year of life. By far the most common tachycardia presenting in early infancy is orthodromic AV re-entry (see Chapter 12). Most of these infants have a normal ECG in sinus rhythm but some show ventricular pre-excitation. Other neonatal tachycardias are much less common and include atrial flutter (see Chapter 9), permanent junctional reciprocating tachycardia (see Chapter 14), atrial tachycardia (see Chapter 7), and ventricular tachycardia (see Chapters 19 and 20).

The most common tachycardia in childhood is also orthodromic AV re-entry tachycardia, although AV nodal re-entry tachycardia (see Chapter 16) becomes progressively more common after the age of 5 years. Less common tachycardias in this age group are antidromic AV re-entry (see Chapter 13), atriofascicular re-entry (see Chapter 15), ventricular tachycardias (see Chapter 18), and atrial tachycardias (see Chapter 7).

Arrhythmias presenting with palpitations include most of the common types of supraventricular tachycardia and a few cases of ventricular tachycardia. Many children with palpitations do not have an arrhythmia and a detailed first-hand history is essential before assessing the likelihood of an arrhythmia and the necessity of further investigation. Similarly very few children with chest pain have arrhythmias (or indeed any cardiac abnormality) and only a few with syncope have an arrhythmia. Again it all depends on the history.

Incessant tachycardias presenting with heart failure or apparent cardiomyopathy include focal atrial tachycardia (see Chapter 7), permanent junctional reciprocating tachycardia (see Chapter 14), incessant idiopathic infant ventricular tachycardia (see Chapter 20), and orthodromic atrioventricular re-entry tachycardia (see Chapter 15).

Arrhythmias presenting with syncope include complete AV block (see Chapter 29), atrial fibrillation in Wolff-Parkinson-White syndrome (see Chapter 13), sinoatrial disease (see Chapter 30), and ventricular tachycardia, especially in long QT syndrome (see Chapter 25), catecholaminergic ventricular tachycardia (see Chapter 26) or late after cardiac surgery (see Chapter 32). Syncope is discussed in detail in Chapter 35. Some arrhythmias are so common as to be considered as almost normal variants. They include atrial premature beats (see Chapter 11), ventricular premature beats (see Chapter 23), and transient nocturnal Wenckebach AV block (see Chapter 28).

Arrhythmias occurring early or late after cardiac surgery are specific to those situations and are considered in detail in Chapters 31 and 32, respectively.
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Key references


