MANUAL OF CLINICAL PROBLEMS IN CARDIOLOGY

WITH ANNOTATED KEY REFERENCES

SECOND EDITION

L. DAVID HILLIS, M.D.

BRIAN G. FIRTH, M.D., D. Phil.

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PREFACE

In this second edition of *Manual of Clinical Problems in Cardiology* we have attempted to achieve several goals. First, we have increased the number of topics from 91 to 114 so that we could introduce for discussion new areas of interest in cardiology. These include thrombolytic therapy of acute myocardial infarction, percutaneous transluminal coronary angioplasty, anthracycline-induced cardiomyopathy, systemic arterial hypertension, the digoxin-quinidine interaction, and the newest of the antiarrhythmic agents. Second, we have updated the discussions of those subjects that were covered in the first edition and, when appropriate, have lengthened them somewhat to add new information. Finally, we have extensively updated the references at the end of each discussion, and, as a result, a substantial number of them are from 1980–1983. At the same time, however, we have not deleted older references that are still worthy of note. In short, we have attempted to provide a maximal amount of up-to-date factual data and worthy reference material within the context of a manual rather than an exhaustive textbook.

We want to express our sincere thanks to several people who labored for many hours and whose help was immeasurable: Ms. Juanita Alexander and Ms. Laurie Christian for secretarial help; Ms. Sarah Hawkins for proofreading, photocopying, and helping to review the many references; and Ms. Kay Fulton for verifying the accuracy of the references.

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B. G. F.
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SINUS TACHYCARDIA, SINUS BRADYCARDIA, AND SINUS ARRHYTHMIA

Sinus tachycardia is a rhythm in which each cardiac impulse arises normally from the sinoatrial node and in which the rate exceeds 100 beats per minute. The P–R interval is usually shortened but may be unchanged or even lengthened. With rates of 100 to 130 per minute, the P waves are easily identifiable before each QRS complex. When the rate approaches 150 per minute, the P waves may be superimposed on the preceding T waves, rendering P wave identification difficult or impossible. At this rate, sinus tachycardia is often difficult to differentiate from paroxysmal supraventricular tachycardia or atrial flutter with 2:1 conduction. If such differentiation is difficult, carotid sinus massage may help to clarify which tachyarrhythmia is present. In response to this maneuver, sinus tachycardia gradually slows and then gradually accelerates after massage is discontinued. In contrast, with paroxysmal supraventricular tachycardia, carotid massage induces either no change or an abrupt reversion to normal sinus rhythm. With atrial flutter and 2:1 conduction, such massage usually acutely increases the magnitude of AV block, thus slowing the ventricular response.

Sinus tachycardia represents a response to several pathologic or physiologic phenomena, including intravascular volume depletion, fever, hypermetabolism, anxiety, and physical exertion. It can occur as a manifestation of an increased intravascular catecholamine concentration, such as occurs in pheochromocytoma. Alcohol and caffeine-containing beverages, as well as certain drugs (e.g., epinephrine and atropine), often cause sinus tachycardia. Finally, sinus tachycardia is present in about one-third of patients with acute myocardial infarction, which may signify extensive pump damage with a resultant low cardiac output.

The therapy of sinus tachycardia is directed at its underlying cause. For instance, if it is due to intravascular volume depletion, the patient should receive adequate fluid replacement. If it is due to fever, the patient's body temperature should be lowered by cooling or with antipyretic medications. No therapy should be aimed at the tachycardia itself, since it is simply a reflection of disordered homeostasis. Especially in the setting of acute myocardial infarction, the underlying cause of sinus tachycardia should be determined and corrected quickly, since persistent tachycardia augments the extent of myocardial ischemic injury.

Sinus bradycardia is a rhythm in which each cardiac impulse arises normally from the sinoatrial node and in which the rate is less than 50 to 60 per minute. In about 25 percent of healthy young men, the sinus rate is between 50 and 60 beats per minute, leading some authors to suggest that 50 per minute be used as the lower limit of normal sinus rhythm. The P waves recur regularly, and each is followed by a QRS complex. The P–R interval is often prolonged.

Sinus bradycardia is a normal occurrence in some people, especially in well-trained athletes, who may have resting heart rates as low as 40 beats per minute. It can result from vagal stimulation by any of several mechanisms, including carotid sinus pressure, the Valsalva maneuver, vomiting, and facial immersion in cold water. Increased intracranial pressure may be accompanied by sinus bradycardia. Many pharmacologic agents can induce it, including the beta-adrenergic blocking agents, verapamil, digitalis, reserpine, guanethidine, methyldopa, various pressor amines, and occasionally quinidine, procainamide, and lidocaine. Certain electrolyte imbalances, such as hyperkalemia, and both hypothermia and hypothyroidism can cause sinus bradycardia. Finally, sinus bradycardia occurs in 10 to 15 percent of patients in the setting of acute

myocardial infarction. However, its incidence in the very early phase of infarction (i.e., within 1–3 hr) is even higher, especially when the infarction involves the inferior portion of the left ventricle.

Although most patients with sinus bradycardia are asymptomatic, it can occasionally cause dizziness or even syncope, angina pectoris, or symptoms of biventricular congestive heart failure. If these symptoms appear, treatment should be initiated. First, any of the drugs known to cause sinus bradycardia should be discontinued. Second, atropine sulfate, 0.5 to 1.0 mg, should be administered intravenously and repeated 2 to 3 times if necessary. Third, if symptomatic sinus bradycardia persists, temporary and then permanent pacing should be instituted.

Sinus arrhythmia is a rhythm in which each cardiac impulse arises normally from the sinoatrial node but in which the rhythmicity of the beats varies, so that the P–P interval varies by more than 0.16 seconds. Most commonly, this change in sinus rate is related to respiration: the heart rate increases gradually during inspiration and decreases with expiration. These fluctuations in vagal tone are caused by reflex changes in the pulmonary and systemic vascular systems. Sinus arrhythmia is most common in patients with a resting bradycardia, presumably because baseline vagal influences are prominent. In these patients, the rhythm usually becomes regular when the rate is increased with exercise or atropine.

Sinus arrhythmia occurs most often in children and in the elderly. It may be seen following digitalis administration, during convalescence from various infectious diseases, and as a cardiovascular sign of increased intracranial pressure. It is common after an acute inferior myocardial infarction. In almost all patients with sinus arrhythmia, no symptoms can be attributed to the rhythm. At times, sinus arrhythmia can be difficult to distinguish from sinoatrial block or an ectopic atrial rhythm.

Since sinus arrhythmia is usually of no clinical importance, it requires no treatment. However, if severe bradycardia is also present, dizziness or syncope can occur, and intravenous atropine is the appropriate therapeutic agent, with transvenous temporary pacing held in reserve.

Sinus Tachycardia

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   In this study, sinus bradycardia appeared to be a prelude to cardiac standstill or ventricular fibrillation.
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    In this group of 46 patients with persistent sinus bradycardia, sick sinus syndrome was eventually diagnosed in almost all.
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    Normal standards are provided for resting heart rate from among 432 healthy adults, ages 16 to 70.
13. Dighton DH. Sinus bradycardia: Autonomic influences and clinical assessment. Br Heart J 1974; 36:791–7.
    Some patients with sinus bradycardia have poor autonomic responses, whereas in others the bradycardia is simply a marked physiologic phenomenon.

PREMATURE BEATS

Premature beats, or extrasystoles, are cardiac contractions of ectopic origin that occur earlier than expected in the usual rhythm. They are the most common of the cardiac arrhythmias. The activating impulse may be located in the atria, AV junction, or ventricles.

Atrial premature beats (APBs) are extremely common, even in individuals without organic heart disease. They occur with even greater frequency in persons who abuse alcohol or cigarettes, in persons with excessive fatigue or anxiety, and in persons with a variety of infectious diseases. Finally, their incidence is increased in patients with atrial disease or atrial enlargement of any kind, including mitral valve disease and cor pulmonale. Their occurrence is not increased in individuals with coronary artery disease. Although the patient may note "skipped beats" or "fluttering" of the heart, he is usually asymptomatic. On physical examination, palpation of the peripheral pulse reveals an occasional early beat.

Since the premature impulse originates from an ectopic focus in the atria, the normal sequence of atrial activation is altered, so that the P wave not only appears early but is also abnormal in configuration. It may or may not be con-

ducted to the ventricles, depending on the degree of prematurity. The P–R interval of the premature beat may remain unchanged, become shorter, or lengthen. The QRS complex following an APB is usually morphologically normal, since the course of the impulse through the ventricular conduction system is normal. However, if the APB occurs at a time when the ventricular conduction system is partially refractory, the QRS complex may demonstrate various degrees of aberration; in fact, it may be so altered as to resemble a ventricular premature beat. The degree of aberration of ventricular activation is related to the degree of prematurity of the APB: an extremely early APB is usually conducted with a great deal of aberration, whereas a later APB is likely to be conducted normally. As the ectopic atrial impulse propagates and depolarizes the sinoatrial node, it resets the sinus cycle; as a result, the cycle length after the APB is similar to the basic cycle length. In contrast to ventricular premature beats, therefore, the pause following an APB is usually not fully compensatory.

Atrial premature beats usually require no therapy. If suppression is desired, quinidine or propranolol is usually efficacious.

Like APBs, *AV nodal or junctional premature beats* usually cause no symptoms. Since the premature beat originates in the AV node and follows the normal pathway of ventricular activation, the QRS complex usually is morphologically normal. The P wave may appear abnormal in position and configuration, representing either retrograde activation of the atria by the nodal impulse or normal sinus activation of the atria in close temporal proximity to the activation of the ventricles by the junctional depolarization. In the former circumstance, the P wave is morphologically abnormal and may appear before, simultaneously with, or after the QRS complex; in the latter instance, the P wave is normal in appearance and is positioned before, within, or immediately after the QRS complex.

In most instances, AV junctional premature beats function as an "escape" rhythm in patients with sinus arrhythmia, sinus bradycardia, sinus arrest, or high-degree AV block. Occasionally, they may occur following the pause after cessation of a supraventricular tachyarrhythmia or an atrial or ventricular premature beat. Thus, AV junctional escape beats are usually a secondary phenomenon, and they carry the same clinical implications as those of the underlying primary rhythm disturbance. For example, drugs, such as digitalis or propranolol, that suppress the sinoatrial node or impair AV conduction may be accompanied by AV junctional escape beats.

Ventricular premature beats (VPBs) arise from an ectopic ventricular focus and occur earlier than the prevailing sinus beat. Infrequent VPBs occur even in young and apparently healthy persons, in which case they are often the result of fatigue, anxiety, or overindulgence in tobacco, coffee, tea, or alcohol. In these healthy individuals, the frequency of VPBs may increase or decrease in response to exercise. More frequently, the appearance of VPBs is associated with several disease entities. Various electrolyte disturbances, such as hypokalemia or hypercalcemia, are associated with VPBs. Digitalis intoxication commonly is heralded by their appearance. They occur frequently in patients who have received sympathomimetic agents, such as dextroamphetamine or isoproterenol. They can appear in patients with many kinds of organic heart disease, including valvular and primary myocardial dysfunction. Finally, they are especially common and worrisome in those with ischemic heart disease.

The patient with an occasional VPB may complain of palpitations, and rarely the patient may actually note chest discomfort induced by a VPB. On physical examination, the VPB occurs earlier than the expected sinus beat and is usually followed by a long pause, the so-called compensatory pause. On the ECG, the VPB has a wide, bizarre QRS morphology that differs strikingly from nor-

mal, since the ectopic impulse takes an abnormal and longer course through the ventricles. The QRS complex is usually of high voltage, is somewhat slurred, and is widened to at least 0.13 second. The T wave is oriented in a direction opposite to its QRS complex. Those VPBs that arise from the same focus have a constant coupling interval; that is, the interval between the VPB and the preceding beat of the basic rhythm is reproducible. In most instances, the VPB is followed by the abnormally long compensatory pause. The P wave and QRS complex of the beat following a VPB are morphologically normal, but the T wave of that beat may be abnormal.

The prognostic significance of VPBs depends mainly on the patient population in which they occur. In those without demonstrable underlying heart disease, VPBs do not adversely influence mortality. In contrast, VPBs are associated with an increased risk of sudden death in individuals with any kind of organic heart disease, especially those ischemic in origin. Both in the setting of acute myocardial infarction and in the postinfarction period, frequent or complex VPBs are accompanied by a greatly enhanced likelihood of ventricular tachycardia and fibrillation. In these patients, therefore, antiarrhythmic medications should be administered in an attempt to suppress ventricular ectopic activity. Those VPBs that are especially ominous include (1) those that occur frequently (more than 5–10/min), (2) those that occur close to the T wave of the preceding sinus beat, (3) those that occur in pairs, and (4) those that originate from more than one focus within the ventricles.

Those VPBs that fulfill any of these criteria should be treated aggressively. Disturbances in serum electrolytes and acid-base balance should be corrected. Acutely, intravenous lidocaine is usually effective in abolishing VPBs: a bolus of 50 to 100 mg is followed by a continuous infusion of 1 to 4 mg per minute. If lidocaine is unsuccessful, intravenous procainamide or bretylium can be used in an attempt to suppress them. Chronically, VPB suppression can be accomplished with oral quinidine, procainamide, propranolol, or any of several new antiarrhythmics, including tocainide, amiodarone, and disopyramide.

It is often advantageous to differentiate VPBs from ventricular parasystole, since they differ substantially both in pathophysiology and prognosis. Although VPBs are often a manifestation of digitalis intoxication, parasystole does not occur in this setting. VPBs should be treated aggressively in the setting of myocardial ischemia or infarction, since they are often the precursor of ventricular tachycardia or fibrillation. In contrast, ventricular parasystole is a more benign rhythm, even in the setting of ischemic injury.

In ventricular parasystole, there is an ectopic ventricular pacemaker that activates the ventricles independently of the basic rhythm. As a result, the coupling intervals vary, and the longer interectopic intervals are whole-number multiples of the shortest one. Finally, since a parasystolic focus is independent of the basic cardiac rhythm, it can discharge its impulse at approximately the same time that the basic rhythm impulse arrives at the ventricles. Therefore, the ventricles are depolarized by two activation fronts, resulting in a fusion beat. Although such fusion beats are not unique to ventricular parasystole, they are most commonly seen in association with it.

Ventricular parasystole is relatively uncommon. It can occur in patients with or without underlying organic heart disease. Since it does not portend the same ominous prognosis as VPBs, it does not require as aggressive a therapeutic approach.

Atrial Premature Beats

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Ventricular Premature Beats

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3. Jelinek MV, Lown B. Exercise stress testing for exposure of cardiac arrhythmias. Prog Cardiovasc Dis 1974; 16:497–522.
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Ventricular Parasystole

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24. Moe GK, Jalife J, Mueller WJ, Moe B. A mathematical model of parasystole and its application to clinical arrhythmias. Circulation 1977; 56:968–79.
Many electrocardiographic patterns attributed to a reentrant "extrasystolic" rhythm may actually represent the modulated activity of a parasystolic focus.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

Paroxysmal supraventricular tachycardia (PSVT) occurs in persons of all ages, including infants, children, and adults. It is often seen in otherwise healthy

young adults, but it also occurs in patients with rheumatic, atherosclerotic, hypertensive, or thyrotoxic heart disease. It has been described in 2 to 8 percent of patients with acute myocardial infarction. It is the most common tachyarrhythmia in individuals with the Wolff-Parkinson-White syndrome. In some patients, PSVT appears in relation to emotional stress, mental or physical fatigue, or excessive use of tobacco, coffee, or alcoholic beverages. Other precipitating factors include deep inspiration, hyperventilation, physical exertion, changes in position, and swallowing.

Episodes of PSVT usually have a sudden onset and termination. In persons with otherwise normal hearts, symptoms are usually mild and include palpitations or an uneasy feeling in the chest. Some patients describe precordial pain, weakness, dizziness, nausea, vomiting, and even syncope. In patients with atherosclerotic heart disease, episodes of PSVT may be accompanied by angina or even myocardial infarction. In those with rheumatic or hypertensive heart disease, symptoms of pulmonary and peripheral venous congestion may arise, and an occasional patient may develop evidence of vascular collapse during a sustained episode. Such collapse is especially likely to occur when the heart rate exceeds 200 beats per minute.

Electrocardiographically, PSVT has the following characteristics. First, the P waves are morphologically different from the sinus P waves. In most instances, they are small and difficult to identify, since they are often superimposed on the preceding T waves. Second, the atrial rate is between 150 and 250 per minute, with an average of 180 to 200 per minute. Third, a QRS complex follows each P wave; although it usually resembles that of the sinus beats, it may appear different because of aberrant ventricular conduction. Fourth, PSVT is a regular rhythm, although one sometimes observes a gradual increase in rate at the beginning of an episode (the so-called warm-up period). Finally, as with sinus tachycardia, S–T segment depression and T wave alterations may occur during episodes of PSVT, and they may persist for hours or even days after its conversion to sinus rhythm.

Paroxysmal supraventricular tachycardia can have one of four electrophysiologic mechanisms. Most commonly, it is due to a reentry circuit within the AV node utilizing dual AV nodal pathways. Such AV nodal reentry accounts for 60 percent of cases of PSVT. Second, PSVT can occur by a reentry mechanism in which the normal AV pathway is used for antegrade conduction and an AV bypass tract is employed for retrograde conduction. This electrophysiologic mechanism is responsible for about 30 percent of cases of PSVT. Lastly, PSVT can be produced by reentry within (1) the sinoatrial node or (2) intra-atrial tissues, together accounting for less than 10 percent of cases.

The conversion of PSVT to sinus rhythm can be accomplished in several ways. A number of so-called vagal maneuvers are designed to stimulate the vagus nerve, thereby inducing a reversion to sinus rhythm, including carotid sinus massage, the Valsalva maneuver, direct pressure on the eyeballs, and the so-called diving reflex (facial immersion in cold water for 5–10 sec). If these maneuvers are unsuccessful in causing a reversion, pharmacologic conversion is usually accomplished with intravenous verapamil, 5 to 10 mg by bolus injection, which is successful in converting approximately 90 percent of PSVTs to sinus rhythm. If intravenous verapamil is not available, one of the following pharmacologic agents can be administered: propranolol (1 mg intravenously every 3–5 min to a total dose of 0.1 mg/kg of body weight), digoxin (0.5–1.0 mg by bolus injection), edrophonium (10 mg by bolus injection), or lidocaine (50–100 mg by bolus injection).

If pharmacologic conversion is unsuccessful, electrical means can be employed. Rapid atrial pacing is used in a manner similar to that described under