MANUAL OF CLINICAL PROBLEMS IN CARDIOLOGY

WITH ANNOTATED KEY REFERENCES

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PREFACE

Over the past 10 to 20 years, the proliferation of new knowledge about cardiovascular disease has been astounding. At present, the medical literature devoted exclusively to this subspecialty is formidable. In addition to a handful of major journals devoted to cardiovascular disease, there are countless other periodicals dealing with various subspecialties within the subspecialty such as catheterization and clinical electrophysiology.

In this volume we have attempted to discuss succinctly the aspects of clinical cardiology that surface most frequently. Each discussion is followed by a list of annotated references to which readers who desire a more thorough and detailed knowledge of a particular subject are encouraged to refer. Because of the rapid evolution of knowledge in many areas of clinical cardiology, we have made a special effort to include in each list of references several recent selections from the medical literature, hoping thereby to impart to the reader some feeling for the direction of current thinking about a particular clinical entity.

We express our sincere thanks for the congenial secretarial assistance of Ms. Juanita Wickersham, Ms. Belinda Lambert, and Ms. Laurie Grey.

L. D. H.
J. E. O.
J. T. W.

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RHYTHM AND CONDUCTION DISTURBANCES

NOTICE

The indications and dosages of all drugs in this Manual have been recommended in the medical literature and conform to the practices of the general medical community. The medications described do not necessarily have specific approval by the Food and Drug Administration for use in the diseases and dosages for which they are recommended. The package insert for each drug should be consulted for use and dosage as approved by the FDA. Because standards for usage change, it is advisable to keep abreast of revised recommendations, particularly those concerning new drugs.

SINUS TACHYCARDIA, SINUS BRADYCARDIA, AND SINUS ARRHYTHMIA

Sinus tachycardia is defined as a rhythm in which each cardiac impulse arises normally from the sinoatrial node and in which the rate is greater than 100 per minute. The P–R interval is usually normal but may be slightly prolonged. With rates of 100 to 130 per minute, the P waves are easily identifiable before every QRS complex. However, when the rate approaches 150 per minute, each P wave may become superimposed on the preceding T wave. Sinus tachycardia represents a physiologic response to intravascular volume depletion, fever, hypermetabolism, anxiety, or physical exertion. In addition, it may occur as a subtle manifestation of an increased intravascular concentration of catecholamines, such as occurs in pheochromocytoma. In the setting of acute myocardial infarction, sinus tachycardia often signals extensive pump damage with a resultant low forward cardiac output.

The physical examination of the patient with sinus tachycardia reveals a regular peripheral and precordial pulse at greater than 100 beats per minute. The therapy of sinus tachycardia is directed at the underlying cause of the condition. For instance, if it is due to intravascular volume depletion, the patient should be given adequate fluid replacement; or, if it is due to fever, the patient's body temperature should be lowered with cooling or antipyretic medications. No therapeutic attempt should be aimed at the sinus 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 aggressively and corrected as quickly as possible, since persistent tachycardia has been shown to augment the extent of myocardial ischemic injury during the hours to days after the onset of myocardial infarction.

Sinus bradycardia is defined as a rhythm in which each cardiac impulse arises normally from the sinoatrial node and in which the rate is less than 60 per minute. The P waves recur regularly, and each P wave is followed by a QRS complex. The P–R interval is often prolonged.

Sinus bradycardia is a normal occurrence in many persons, especially in well-trained athletes, who may have resting heart rates as low as 40 per minute. Sinus bradycardia can result from vagal stimulation by any of several mechanisms, including direct carotid sinus pressure, the Valsalva maneuver, vomiting, and facial immersion in cold water (the so-called diving reflex). Sinus bradycardia can be caused by certain pharmacologic agents, including beta-adrenergic blocking agents (blockade is most commonly accomplished with propranolol), digitalis, morphine sulfate, alpha methyldopa, and various pressor amines.

Most patients with sinus bradycardia are asymptomatic. However, sinus bradycardia occasionally causes dizziness or even true syncope, angina pectoris, or symptoms of right and left ventricular failure. If these symptoms appear, treatment should be initiated. First, any drug known to cause sinus bradycardia should be discontinued. Second, atropine sulfate, 0.5 to 1.0 mg, should be administered intravenously and can be repeated 2 to 3 times if necessary. Third, if symptomatic sinus bradycardia persists, temporary and then permanent pacing should be instituted.

Sinus arrhythmia is defined as a rhythm in which, as in sinus tachycardia and sinus bradycardia, each cardiac impulse arises normally from the sinoatrial node but in which the rhythmicity of the beats varies. The variations

are usually, but not always, related to respiration, the heart rate increasing with inspiration and diminishing with expiration. Sinus arrhythmia is most common in patients with a resting bradycardia, presumably because baseline vagal influences are prominent.

Sinus arrhythmia occurs most often in children and in the elderly. It is often observed following the administration of the digitalis glycosides, during convalescence from various infectious diseases, and as a cardiovascular sign of increased intracranial pressure. In almost all patients with sinus arrhythmia, no symptoms can be attributed to the cardiac rhythm.

Sinus arrhythmia is usually of no clinical importance and therefore requires no treatment. However, if severe bradycardia is also present, dizziness or syncope may occur, in which case intravenous atropine sulfate is an appropriate therapeutic choice, with transvenous temporary pacing held in reserve.

1. Adgey AAJ, Geddes JS, Mulholland HC, et al: Incidence, significance, and management of early bradyarrhythmia complicating acute myocardial infarction. Lancet 2:1097, 1968.
Bradyarrhythmias soon after acute myocardial infarction (MI) are very common and are precursors of ventricular fibrillation.
2. Agruss NS, Rosin EY, Adolph RJ, et al: Significance of chronic sinus bradycardia in elderly people. Circulation 46:924, 1972.
Elderly patients with sinus bradycardia may have normal cardiac function.
3. Brill IC, Welch JD, Condon RJ, et al: Sinus tachycardia: Possible control with guanethidine (Ismelin). Arch Intern Med 115:674, 1965.
The control of sinus tachycardia with small doses of guanethidine is reported in 5 patients previously resistant to other measures.
4. Gifford RW, Kvale WF, Maher FT, et al: Clinical features, diagnosis, and treatment of pheochromocytoma: A review of 76 cases. Mayo Clin Proc 39:281, 1964.
Almost all the patients with pheochromocytoma in this series complained of palpitations.
5. Gregory JJ, Grace WJ: The management of sinus bradycardia, nodal rhythm, and heart block for the prevention of cardiac arrest in acute myocardial infarction. Prog Cardiovasc Dis 10:505, 1968.
Sinus bradycardia occurs in 10 to 15 percent of patients with acute MI; if symptoms arise, it should be treated with intravenous atropine or (if necessary) transvenous pacing.
6. Kirkendall WW, Liechty RD, Culp DA: Diagnosis and treatment of patients with pheochromocytoma. Arch Intern Med 115:529, 1965.
Seventeen patients with pheochromocytoma are reviewed; most had symptoms and signs of hypermetabolism.
7. Massumi RA, Mason DT, Amsterdam EA, et al: Ventricular fibrillation and tachycardia after intravenous atropine for treatment of bradycardia. N Engl J Med 287:336, 1972.
Three patients are described who developed ventricular tachycardia or ventricular fibrillation after intravenous atropine for the treatment of sinus bradycardia.
8. Redwood DR, Smith ER, Epstein SE: Coronary artery occlusion in the conscious dog: Effects of alterations in heart rate and arterial pressure on the degree of myocardial ischemia. Circulation 46:323, 1972.
In the dog with experimental coronary artery occlusion, tachycardia worsens myocardial ischemic injury.

9. Shell WE, Sobel BE: Deleterious effects of increased heart rate on infarct size in the conscious dog. Am J Cardiol 31:474, 1973.
In the dog with experimental coronary artery occlusion, augmentation of heart rate consistently increases the extent of myocardial necrosis.
10. Shillingford J, Thomas M: Treatment of bradycardia and hypotension syndrome in patients with acute myocardial infarction. Am Heart J 75:843, 1968.
In the setting of MI, bradycardia should be aggressively treated with atropine and (if necessary) isoproterenol.
11. Wenger MA, Bogchi BK, Amand BK: Experiments in India on "voluntary" control of the heart and pulse. Circulation 24:1319, 1961.
Heart rate can be slowed substantially by any maneuver that increases vagal tone.

PREMATURE BEATS

Premature beats, or extrasystoles, are cardiac contractions of ectopic origin that occur earlier than expected in the dominant or usual rhythm. The activating impulse may be located in the atria, AV junction, or ventricles. Such premature beats are the most common cardiac arrhythmias. They are believed to arise when an ectopic focus in the heart becomes irritable and when its rate of impulse formation exceeds that of the sinus node.

Atrial premature beats (APBs) are a common occurrence, especially in heavy consumers of alcohol or cigarettes, excess fatigue or anxiety, and a variety of infectious diseases. The patient may note "skipped beats" or "fluttering" of the heart, but may be asymptomatic. On physical examination, palpation of the peripheral pulse reveals an occasional early beat, usually followed by a compensatory pause.

The ECG reveals a premature P wave that appears somewhat abnormal. It may or may not be conducted to the ventricles, depending on its degree of prematurity. The P–R interval is usually greater than 0.12 seconds and may be abnormally prolonged. The QRS morphology following an atrial premature beat is usually normal, since the course of the impulse through the conduction system of the ventricles is normal. However, if the atrial premature beat occurs at a time when all or a part of the ventricular conduction system is still in the relative refractory period, the QRS complex may demonstrate various degrees of aberration; in fact, it may be so altered that it resembles a ventricular premature beat. The degree of aberration of ventricular activation is related to the degree of prematurity of the APB; that is, an extremely early APB is usually conducted with a great deal of aberration, since it is conducted to the ventricles at a time when they are still relatively refractory. In contrast, a later APB is likely to be conducted normally through the ventricles, since they are no longer refractory when the impulse arrives.

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

Like APBs, *A V nodal or junctional premature beats* usually cause no symptoms. Since the premature beat originates in the AV node and follows the normal pathway as it activates the ventricles, 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 premature beat. In the former circumstance, the P wave is morphologically abnormal and may appear before, simultaneous with, or after the QRS complex; in the latter instance, the P wave is normal in appearance and is positioned before, within, or imediately after the QRS complex.

Ventricular premature beats (VPBs) arise from an ectopic ventricular focus and occur earlier than the prevailing sinus beat. They have many possible causes. Various electrolyte disturbances, such as hypokalemia or hypercalcemia, are associated with ventricular ectopy. Digitalis intoxication commonly is heralded by the appearance of VPBs. Ventricular premature beats occur frequently in patients who have received sympathomimetic agents, such as dextroamphetamine and isoproterenol. They may appear in patients with all kinds of organic heart disease, and they are especially common and worrisome in patients with ischemic heart disease.

The patient with an occasional ventricular premature beat may complain of feeling a "skipped" heartbeat. On physical examination, the VPB is found to occur earlier than the expected sinus beat and is followed by an especially long pause, the so-called compensatory pause. On the ECG, the VPB has a wide, bizarre QRS morphology that differs strikingly from the normal, since the ectopic impulse takes an abnormal and longer course through the ventricles. Specifically, the QRS complex is usually of high voltage, somewhat slurred, and widened to at least 0.13 second. The T wave is oriented in a direction opposite to the QRS complex it follows. The ventricular extrasystole is followed by an abnormally long pause. The ectopic ventricular impulse usually is not conducted retrogradely to the atria; as a result, the atria are activated at the normal time by the next sinus impulse. The P wave and QRS complex of the beat following a VPB are morphologically normal, but the T wave of that beat may be inverted. Interestingly, such T wave abnormalities in the beat after a VPB almost always signify underlying cardiac disease.

Ventricular premature beats are dangerous to the patient only because they potentially can lead to ventricular tachycardia or fibrillation. The VPBs that are especially likely to do so (1) occur frequently (more often than 5-6/min) (2) appear close to the T wave of the preceding sinus beat, (3) occur in pairs, and (4) originate from more than one focus within the ventricles. If VPBs fulfill any of these criteria, they should be treated aggressively. Disturbances in serum electrolytes and acid-base balance should be corrected. If pharmacologic intervention is necessary, intravenous lidocaine is usually highly 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, quinidine (administered intramuscularly or orally) or procainamide (given intravenously, intramuscularly, or orally) can be used in an attempt to suppress them. Other, newer antiarrhythmics, such as tocainide, amiodarone, and disopyramide phosphate, are sometimes successful in controlling VPBs that cannot be suppressed with lidocaine or the older agents.

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1. Damato AN: Diphenylhydantoin: Pharmacological and clinical use. Prog Cardiovasc Dis 12:1, 1969.
A review of diphenylhydantoin's place as an antiarrhythmic agent, especially in the control of digitalis-induced ventricular arrhythmias.
2. Engel TR, Meister SG, Frankl WS: The "R on T" phenomenon: An update and critical review. Ann Intern Med 88:221, 1978.
Although early observations suggested that R-on-T was likely to initiate

sustained ventricular tachyarrhythmias, more recent evidence suggests that R-on-T is not a critical determinant of primary ventricular fibrillation and sudden death.

3. Gaughan CE, Lown B, Lanegan J, et al: Acute oral testing for determining antiarrhythmic drug efficacy: 1. Quinidine. Am J Cardiol 38:677, 1976.
   A new technique is described for determining expeditiously if a specific antiarrhythmic agent is effective against VPBs.
4. Gibson D, Sowton E: The use of beta-adrenergic receptor blocking drugs in dysrhythmias. Prog Cardiovasc Dis 12:16, 1969.
   A large clinical series reporting that beta blockade is efficacious in reverting those dysrhythmias precipitated by exercise or emotion, those associated with the Wolff-Parkinson-White syndrome, and those ectopic atrial or ventricular dysrhythmias due to digitalis toxicity.
5. Kennedy HL, Underhill SJ: Frequent or complex ventricular ectopy in apparently healthy subjects. Am J Cardiol 38:141, 1976.
   A clinical syndrome of frequent complex ventricular ectopy can occur in apparently healthy people.
6. Kisten AD: Problems in the differentiation of ventricular arrhythmia from supraventricular arrhythmia with abnormal QRS. Prog Cardiovasc Dis 9:1, 1966.
   Some tips on differentiating ventricular and supraventricular tachycardias.
7. Lown B, Calvert AF, Armington R, et al: Monitoring for serious arrhythmias and high risk of sudden death. Circulation 52 (Suppl III):189, 1975.
   A classification of VPBs based on their frequency, multiformity, repetitive pattern, and degree of prematurity appears to be helpful in identifying patients at high risk of sudden death.
8. Marriott HJL, Sandler IA: Criteria, old and new, for differentiating between ectopic ventricular beats and aberrant ventricular conduction in the presence of atrial fibrillation. Prog Cardiovasc Dis 9:18, 1966.
   The title is self-explanatory.
9. Mason DT, Spann JF, Zelis R, et al: The clinical pharmacology and therapeutic applications of the antiarrhythmic drugs. Clin Pharmacol Ther 11:460, 1970.
   Excellent discussion of pharmacologic principles of antiarrhythmic therapy.
10. Rosenbaum MB, Chiale PA, Halpern MS, et al: Clinical efficacy of amiodarone as an antiarrhythmic agent. Am J Cardiol 38:934, 1976.
    Most patients with recurrent atrial fibrillation or flutter, repetitive supraventricular tachycardia, and ventricular tachycardia have suppression of their arrhythmia with daily oral administration of amiodarone.
11. Ruberman W, Weinblatt E, Frank CW, et al: Ventricular premature beats and mortality in men with coronary heart disease. Circulation 52 (Suppl III): 199, 1975.
    Among a large group of men with documented coronary disease, mortality was higher among those with VPBs than among those without.
12. Winkle RA, Meffin PJ, Harrison DC: Long-term tocainide therapy for ventricular arrhythmias. Circulation 57:1008, 1978.
    Long-term tocainide therapy is successful in suppressing VPBs and ventricular tachycardia in some patients who are refractory to other antiarrhythmic agents.
13. Woosley RL, McDevitt DG, Nies AS, et al: Suppression of ventricular ectopic depolarizations by tocainide. Circulation 56:980, 1977.

Tocainide, an oral lidocaine-like agent, is shown to be effective in suppressing VPBs when given at 8 to 12-hour intervals.

14. Zito RA, Reid PR: Lidocaine kinetics predicted by indocyanine green clearance. N Engl J Med 298:1160, 1978.
In patients in whom lidocaine clearance may be reduced (i.e., those with hepatic congestion), indocyanine green clearance can predict lidocaine dosage requirements.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

Paroxysmal supraventricular tachycardia has an atrial rate of 150 to 250 beats per minute. This rhythm disturbance typically has a sudden onset and termination and is characterized by a series of premature P waves that are morphologically different from those that represent sinus node activity. It is more common in female than in male patients and is frequently noted in anxious young people, in the physically fatigued, and in patients who consume relatively large quantities of coffee, tea, or alcohol, or smoke to excess. It occasionally develops in patients with myocardial ischemia and acute myocardial infarction. Paroxysmal supraventricular tachycardia occurs in some patients with myocardial disease during systemic arterial hypoxia and in some with serious mitral valve disease. This rhythm disturbance may be noted in patients with the Wolff-Parkinson-White syndrome.

Paroxysmal supraventricular tachycardia that results in symptoms (e.g., angina, shortness of breath, and/or syncope) requires treatment. Some patients are so concerned over palpitations that therapy is mandated. Several different maneuvers designed to cause vagal stimulation may convert paroxysmal supraventricular tachycardia to sinus rhythm. In particular, it may be converted by carotid sinus massage, pressure on the eyeballs, the Valsalva maneuver, and/or the diving reflex (facial immersion in cold water for a few seconds). In addition to vagal stimulation, rapid atrial pacing or elevation of systemic arterial pressure may convert paroxysmal supraventricular tachycardia to sinus rhythm. Sedation, withdrawal from excessive consumption of coffee, tobacco, and alcohol, and proper rest may also correct the arrhythmia.

The pharmacologic conversion of paroxysmal supraventricular tachycardia may be accomplished in some patients with a bolus intravenous injection of lidocaine. If this is unsuccessful, intravenous edrophonium (10 mg by bolus injection) may effect conversion to sinus rhythm. Digitalis is also efficacious in this rhythm disturbance, and propranolol, also effective, has been gaining popularity. Recent studies from Europe have demonstrated the marked success with which intravenous verapamil converts this tachyarrhythmia to sinus rhythm. Once paroxysmal supraventricular tachycardia is converted to sinus rhythm, digitalis or propranolol may be used as maintenance therapy to prevent recurrences.

1. Marriott HJL: Differential diagnosis of supraventricular and ventricular tachycardia. Geriatrics 25:91, 1970.
Electrocardiographic clues for distinguishing these rhythm disturbances are presented.

2. Sandler IA, Marriott HJL: The differential morphology of anomalous ventricular complexes of RBBB-type in lead VI: Ventricular ectopy versus aberration. Circulation 31:551, 1965.
In this article, 100 unselected tracings showing ventricular ectopic beats of right bundle branch block form in lead V1, and 100 unselected examples of RBBB in V1 were analyzed for clues that aid in distinguishing aberrancy from ventricular ectopy.
3. Lown B, Wyatt NF, Levine HD: Paroxysmal atrial tachycardia with block. Circulation 21:129, 1960.
A detailed review of this entity.
4. Morgan WL, Breneman GM: Atrial tachycardia with block treated with digitalis. Circulation 25:787, 1962.
Fifteen patients with this problem treated in this manner are presented, and their clinical courses are reviewed in detail.
5. Wood P: Polyuria in paroxysmal tachycardia and paroxysmal atrial flutter and fibrillation. Br Heart J 25:273, 1963.
An observation that polyuria may occur with and following the cessation of paroxysmal tachycardia.
6. Goldreyer B, Gallagher JJ, Damato AN: The electrophysiologic demonstration of atrial ectopic tachycardia in man. Am Heart J 85:205, 1973.
Three patients in whom intracardiac electrograms and programmed atrial stimulation sequences suggested atrial tachycardia to be the result of a rapidly firing automatic atrial pacemaker are described.
7. Pittman DE, Makar JS, Kooros KS, et al: Rapid atrial stimulation: Successful method of conversion of atrial flutter and atrial tachycardia. Am J Cardiol 32:700, 1973.
In 20 or 23 patients receiving rapid atrial stimulation, either the atrial tachyarrhythmia was converted to sinus rhythm or the flutter tachycardia terminated with resultant atrial fibrillation.
8. Williams DO, Davidson PH: Long-term treatment of refractory supraventricular tachycardia by patient controlled atrial pacing. Br Heart J 36:336, 1974.
Two patients in whom this approach was successful are presented.
9. Wildenthal K, Leshin, SJ, Atkins JM, et al: The diving reflex used to treat paroxysmal atrial tachycardia. Lancet 1:12, 1975.
The demonstration that brief facial immersion in a container of cold water will terminate many supraventricular tachyarrhythmias.
10. Bellet S: Diagnostic features and management of supraventricular arrhythmias. Prog Cardiovasc Dis 8:483, 1966.
A complete review of supraventricular arrhythmias.
11. Han J: The mechanism of paroxysmal atrial tachycardia: Sustained reciprocation. Am J Cardiol 26:329, 1970.
A review of this subject.
12. Bigger JT, Goldreyer BN: The mechanism of supraventricular tachycardia. Circulation 42:673, 1970.
Recurrent episodes of supraventricular tachycardia were analyzed in 6 patients in order to study mechanisms by which this arrhythmia is initiated and sustained.
13. Bigger JT: Management of cardiac arrhythmias with antiarrhythmic drugs. In JT Willerson, CA Sanders (Eds), Clinical Cardiology. New York, Grune & Stratton, 1977.
A thorough review of the antiarrhythmic actions of various pharmacologic agents and a review of mechanisms of arrhythmic development.

14. Barold SS, Coumel P: Mechanism of atrioventricular junctional tachycardia. Role of re-entry and concealed accessory bypass tracts. Am J Cardiol 39:97, 1977.
A thorough review of this subject.
15. Selzer A, Kelly JJ, Johnson RB, et al: Immediate and long-term results of electrical cardioversion of arrhythmias. Prog Cardiovasc Dis 9:90, 1966.
A clinical review of the results of cardioversion in 189 patients.

ATRIAL FLUTTER

Atrial flutter is a supraventricular tachycardia with an atrial rate of 250 to 350 per minute. In an occasional elderly patient or a patient who is taking quinidine, the atrial rate may be slower than 250 per minute (i.e., around 220–240/min). It is usually associated with second degree AV block, usually 2 : 1, 3 : 1, or 4 : 1. In contrast to paroxysmal supraventricular tachycardia, atrial flutter ordinarily occurs in persons *with* underlying organic heart disease, most commonly mitral valve disease, primary myocardial disease, pericardial disease, or thyrotoxicosis. It occasionally occurs in the setting of acute myocardial infarction. As a general rule, atrial flutter does not occur as a manifestation of digitalis intoxication.

The patient with atrial flutter may complain of palpitations. Because of a reduced forward cardiac output, the patient may note generalized fatigue and weakness, coolness of the skin, and dizziness. If there is underlying heart disease, the patient may show symptoms of left ventricular failure (dyspnea, orthopnea, and paroxysmal nocturnal dyspnea), inadequate coronary perfusion (angina pectoris and/or myocardial infarction), and/or inadequate cerebral perfusion (dizziness, syncope, and/or focal neurologic symptoms and signs in patients with underlying cerebrovascular disease). On physical examination the peripheral and precordial pulses are found to correspond to the ventricular rate. With 2 : 1 AV block, the ventricular rate is around 150; with 3 : 1 block, around 100; and with 4 : 1 block, around 75. If the AV block is variable, the resultant ventricular response is 75 to 150 per minute and irregular. The jugular venous pulse may demonstrate regular flutter waves at 250 to 350 per minute. Otherwise, the general physical examination reveals the findings typical of the underlying cardiac disease that is present.

On the ECG, the atrial flutter waves appear regularly at a rate of 250 to 350 per minute. As a result, the baseline of the standard ECG often assumes a sawtooth pattern. The P waves are usually oriented negatively in the inferior leads (II, III, and aVF), indicating that atrial electrical activity arises in the caudal portion of the atria. The P–R interval is often slightly prolonged (but may be difficult to measure). The QRS complexes are usually normal in duration and configuration, occurring at a rate of 75 to 150 per minute. In atrial flutter with 2 : 1 block, alternate P waves may be obscured by the QRS complexes.

As mentioned under Paroxysmal Supraventricular Tachycardia, carotid sinus pressure in the patient with atrial flutter will acutely increase the degree of AV block, resulting in the appearance of several flutter waves in succession without a QRS complex. As carotid sinus pressure is released, the severity of AV block lessens and returns to its baseline level.