
Continuing Education

Electrocardiograms and Arrhythmias: An Introduction

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This is the second in a series of four continuing education articles addressing patient care and management. This text is designed to present the technologist with a basic understanding of the principles and workings of the EKG and its applications in the diagnosing and monitoring of arrhythmias. Upon completion of this article, the technologist should be able to: 1) explain the principles of an EKG and the heart's conduction system; 2) identify a normal rhythm strip; and 3) recognize abnormal strips that may influence patient care during a nuclear procedure.

The electrocardiogram (EKG), first introduced over eighty years ago, is the first diagnostic test ordered when a cardiac problem is suspected. Although the EKG often gives a clear and irrefutable answer in the portrayal of arrhythmias (a disturbance of the normal cardiac rhythm), it is much less specific in cardiac disease. An EKG may be normal, even in the presence of heart disease, if the underlying problem does not affect the electrical activity of the heart. Conversely, there may be an arrhythmia with no underlying cardiac problem present (due to drugs, i.e., digitalis, cocaine). Although not perfect, the EKG remains a valuable diagnostic test, especially when used in conjunction with a thorough clinical evaluation.

Originally, only physicians were expected to have the expertise needed to interpret EKGs. With the advent of coronary care units (CCUs) in the early 1960s, interpretation of rhythm strips became one of the nurses' primary responsibilities. Today, the use of monitors is no longer restricted to CCUs, demonstrating routine use in areas throughout a hospital, including nuclear medicine. It thus becomes important that the basic principles of EKGs be understood, as well as their unique applications. This continuing education article is designed to present the reader with an elementary understanding of the electrical system of the heart and the more common arrhythmias that are likely to be encountered.

PRINCIPLES OF EKG

An EKG is a graphic representation of the electrical im-

pulses generated by the heart during a cardiac cycle. To make a recording of this activity, there must be an electrical circuit between the heart and the EKG machine. This is accomplished by placing electrodes on two or more body sites. As the electrodes are moved, different views of the heart's electrical impulse are observed, with each varying view labeled a lead.

Three standard limb leads have been employed for more than eighty years. According to Marriott, 80–90% accuracy in diagnosis can be achieved with the inspection of these three leads alone (1). The 12-lead EKG however, consists of six limb leads (combinations of electrodes on the extremities), and six chest leads corresponding to six sites on the chest. Leads I, II, and III are considered to be the standard limb leads. Lead I records the difference in potential between the left arm (LA) and the right arm (RA). Lead II records the electrical potential between RA and the left leg (LL), and lead III reflects the difference between LA and LL. The right leg (RL) electrode is an inactive ground for all leads (2).

There are three "augmented" limb leads: aVR, aVL, aVF, where a is defined as augmented, V is vector unipolar, R is right arm, L is left arm, and F represents foot or leg. These augmented leads measure the electrical potential between the center of the heart and the right arm (aVR), the left arm (aVL), and the left leg (aVF). The six standard chest or "precordial" leads (V1, V2, V3, V4, V5, and V6) are recorded by placing electrodes at six different positions on the chest, surrounding the heart.

ELECTRICAL CONDUCTION SYSTEM

Before attempting to interpret a monitor strip, it is important that one understand the electrical conduction system of the heart and relate it to the heart's mechanical action since each electrical event precipitates a mechanical event.

Conduction System

Figure 1 shows the heart's conduction system. The myocardium consists of special conduction cells which, when stimulated sequentially, cause the heart to contract and pump blood. Each cell is capable of initiating, as well as carrying an impulse. The conduction system consists of the sino-atrial

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(SA) node, the atrio-ventricular (AV) node, the bundle of His, bundle branches, and the Purkinje fibers. Certain specialized cells, such as those in the SA node, the AV node, and the His-Purkinje system are able to discharge spontaneously. This property, known as automaticity, creates the potential for these cells to depolarize (activate) the rest of the heart. Normally, the SA node rules as the pacemaker since it spontaneously discharges faster than the other latent pacemakers. The SA node normally fires at a rate of 60–100 impulses per minute. In later stages of the conduction system, the intrinsic rate becomes slower. If, for example, the SA node fails for any reason then the AV node, with an intrinsic rate of 40–60, is able to take over as pacemaker. Should this also fail, the ventricles could then take over at a rate of 20–40, which may or may not be compatible with sustaining life.

PQRST Waves

Figure 2 shows PQRST waves. The SA node is located high in the right atrium, near the entrance of the superior vena cava, and just above the tricuspid valve. It is the heart's dominant pacemaker, firing 60–100 times per minute. The impulse spreads from the SA node to the right atrium (via the internodal pathways) and to the left atrium (via Bachmann's bundle), in a ripple-like pattern. This atrial activation, or depolarization, immediately precedes atrial contraction and is represented on the EKG strip as a P-wave.

Conduction slows as the impulse reaches the AV node, which serves as a kind of ventricular gatekeeper, policing the number of impulses that may be allowed to pass through. This conduction delay allows time for the blood to flow from the atria to the ventricles. As the impulse leaves the AV node, conduction again speeds up, and the impulse is carried through the bundle of His and down the right and left bundle branches. These branches supply the inner shell (endocardium) of their respective ventricles with a profusely branching network called the Purkinje fibers. The time it takes for the impulse to get from the SA node to this point is displayed on

the EKG by the P-R interval, normally measuring 0.12–0.20 sec. The Purkinje fibers allow both ventricles to be depolarized almost simultaneously by spreading the impulse rapidly throughout the ventricular epicardium. Ventricular depolarization occurs just before the ventricles contract, expelling blood forward into the pulmonary artery and aorta. This action is represented on the EKG strip by the QRS complex, which has a normal duration of 0.06–0.10 sec. The T-wave follows the QRS complex, and is simply the repolarization, or recovery phase, of the ventricles. The atria also have a repolarization phase, but it is not usually seen on the EKG, being obscured by the QRS complex.

The ST segment is measured from the end of the QRS to the beginning of the T-wave and represents early repolarization of the ventricles. Although it plays an important role in evaluating ischemia and infarction, it usually does not affect interpretation of arrhythmias, and thus will not be addressed in this text.

The entire PQRST cycle takes ~ 1 sec to complete.

PARAMETERS FOR HEART RATE DETERMINATION

Determining Heart Rate

As previously described, the detected electrical activity is monitored by an EKG machine and recorded on graph paper. This graph paper is standardized for EKGs and monitors and is equally divided into 1 mm vertical lines representative of 0.04 sec time frames, and 1 mm horizontal lines measuring voltage. The intervals between two heavy vertical lines (or one major box) is 0.20 sec. Five major boxes equal 1.0 sec, which is also equal to one inch of graph paper (Fig. 3).

The distance between two R-waves is proportional to heart rate. Ventricular rate can easily be determined by counting the number of small boxes (0.04 sec) between two consecutive R-waves and dividing into 1,500. The atrial rate may also be determined by counting the number of small boxes between

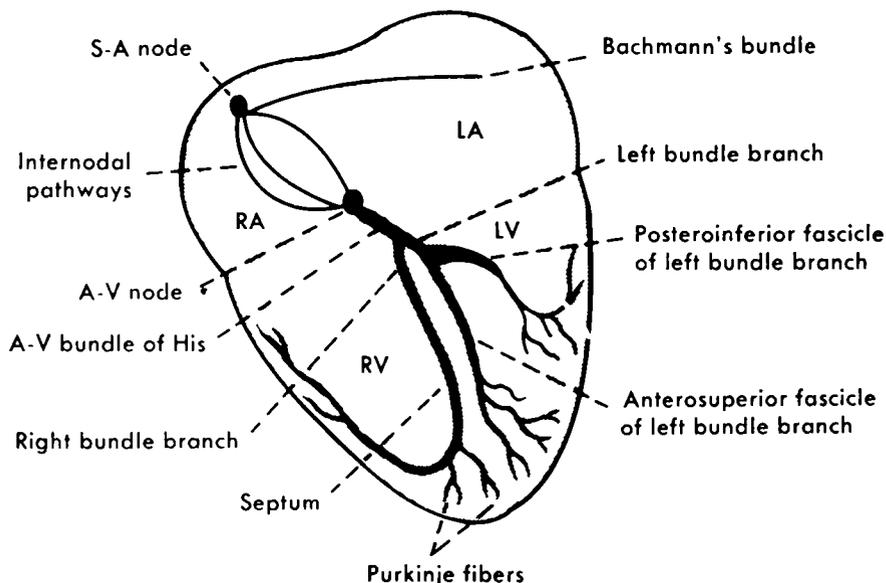


FIG. 1. The conduction system of the heart from the S-A node to the Purkinje fibers.

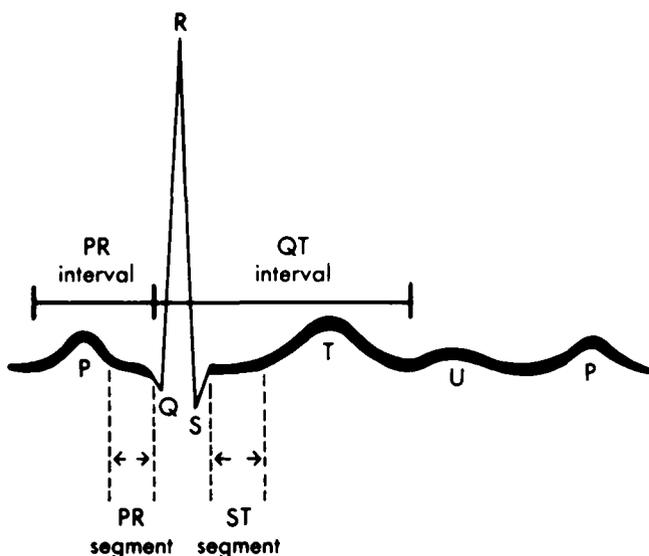


FIG. 2. Normal electrical pattern of a cardiac cycle consisting of PQRST waves.

consecutive P-waves and dividing into 1,500. An alternative method is to count the number of R-waves (or P-waves) in a six-second strip and multiplying by ten.

INTERPRETATION OF MONITOR STRIPS

Arrhythmia Analysis

An arrhythmia is a disturbance in the heart's normal rhythm. It can be benign or lethal depending on the rhythm, the patient's underlying condition, and its effect on the patient. For an individual with no cardiac disease, a moderate tachycardia is usually of no consequence. However, in patients with an acute myocardial infarction, this same arrhythmia may lower the cardiac output enough to be life threatening. Arrhythmias can be constant or intermittent and can occur because of a variety of reasons, including myocardial ischemia, drug toxicity, electrolyte imbalance, and/or sympathetic/

parasympathetic stimulation. Treatment depends not on the arrhythmia itself, but on the patient's underlying condition and clinical response to the rhythm.

Each arrhythmia must be approached in a systematic manner as outlined below:

1. Determine if the rate is too slow or too fast. Are the atrial and ventricular rates the same?
2. Is the rhythm regular or irregular?
3. Are the P-waves present, and is there one for each QRS complex?
4. Is the PR interval within normal limits, and is it constant?
5. Is the QRS complex within normal limits, and is its configuration constant?

Rhythm strips of various cardiac conditions are presented along with specific points to note and suggestions for methods of treatment.

Normal Sinus Rhythm

A normal sinus rhythm (Fig. 4) is presented. Electrical and mechanical data are:

Rate: 60-100 beats per minute.

Rhythm: Regular.

P-Wave: Normal contour precedes each QRS.

PR Interval: Normal and constant (0.12-0.20 sec).

QRS: Normal and constant (0.06-0.10 sec).

Sinus Bradycardia

Data for sinus bradycardia (Fig. 5) include:

Rate: Below 60 beats per minute.

Rhythm: Regular.

P-Wave: Normal contour and precedes each QRS.

PR Interval: Normal and constant (0.12-0.20 sec).

QRS: Normal and constant (0.06-0.10 sec).

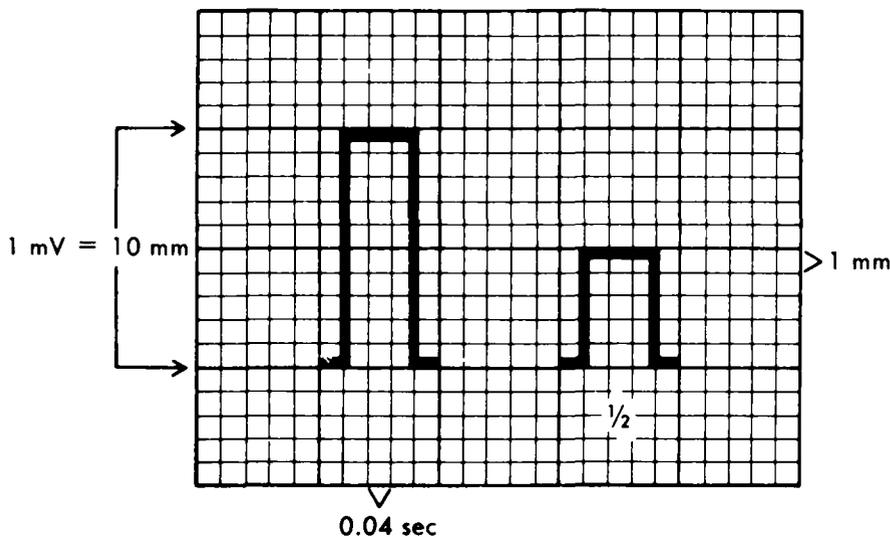


FIG. 3. Time and voltage relationship of EKG.

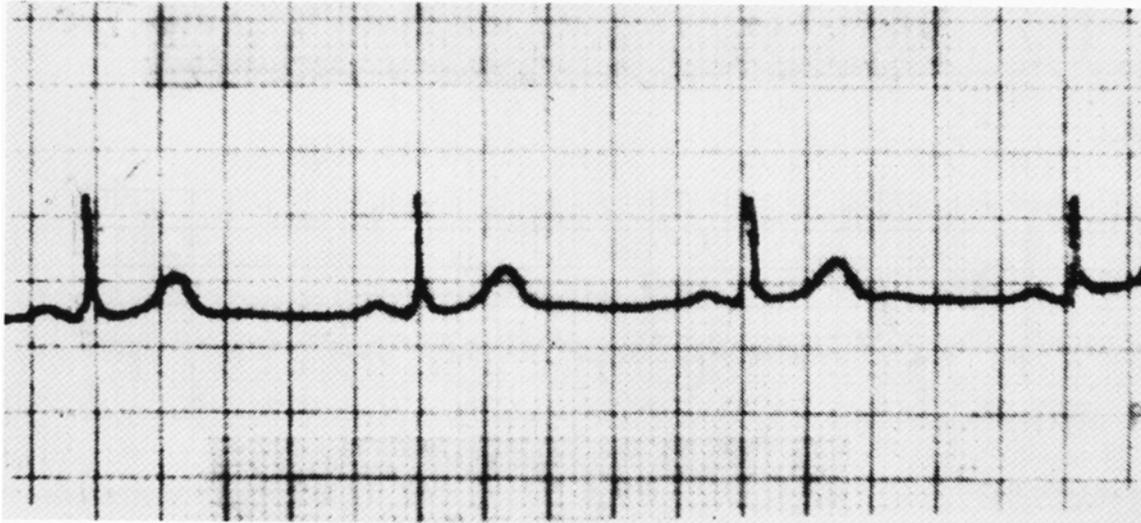


FIG. 4. Normal sinus rhythm (NSR).

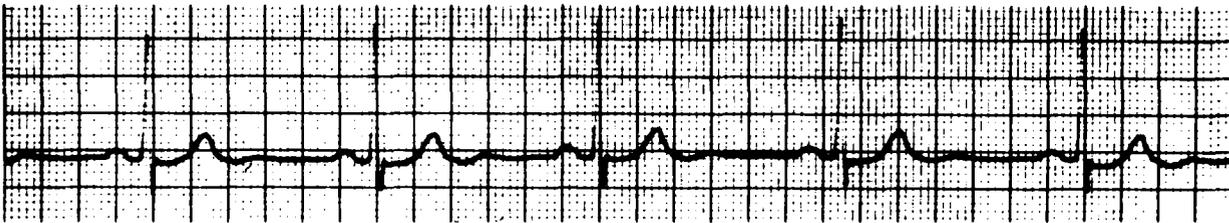


FIG. 5. Sinus bradycardia.

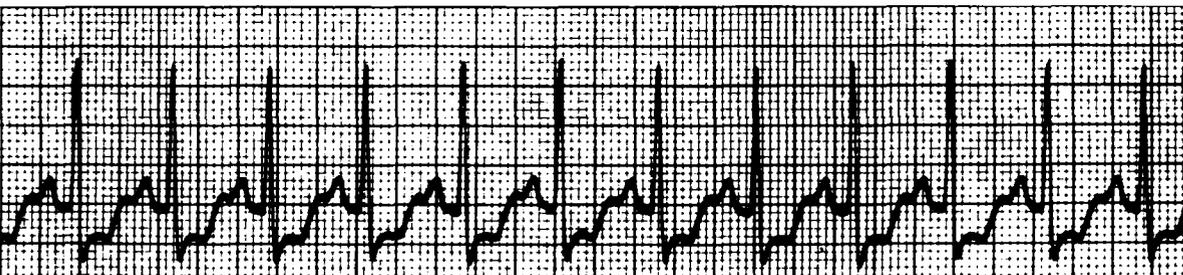


FIG. 6. Sinus tachycardia.

There is normal conduction with the SA node as pacemaker, but the rate is slow. Unless the rate is extremely slow and cardiac output is impaired, there is usually no treatment. Atropine is the drug of choice if the patient is in a compromised situation, or if arrhythmias are associated with the slow rate.

Sinus Tachycardia

Data for sinus tachycardia (Fig. 6) include:

- Rate: Above 100 beats per minute.
- Rhythm: Regular.
- P-Wave: Present and precedes each QRS. (In very rapid rates, P-wave may be hard to detect if it is buried within the preceding T-wave).
- PR Interval: Normal and constant (0.12–0.20 sec).
- QRS: Normal duration and constant configuration.

Once again there is a normal conduction with the SA node as pacemaker, but the rate is faster than normal. Treatment depends on the underlying disease and patient's tolerance of the rate. Sinus tachycardia is a frequently compensatory response by the heart to increase the cardiac output.

Sinus Arrhythmia

Data for sinus arrhythmia (Fig. 7) include:

- Rate: May vary, but usually on the slow side.
- Rhythm: Regularly irregular.
- P-Wave: Normal contour and precedes each QRS.
- QRS: Normal duration and constant configuration.

This is a phasic increase and decrease of the heart rate usually associated with inspiration and expiration, requiring no treatment.

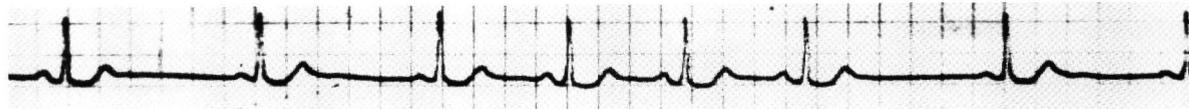


FIG. 7. Sinus arrhythmia.

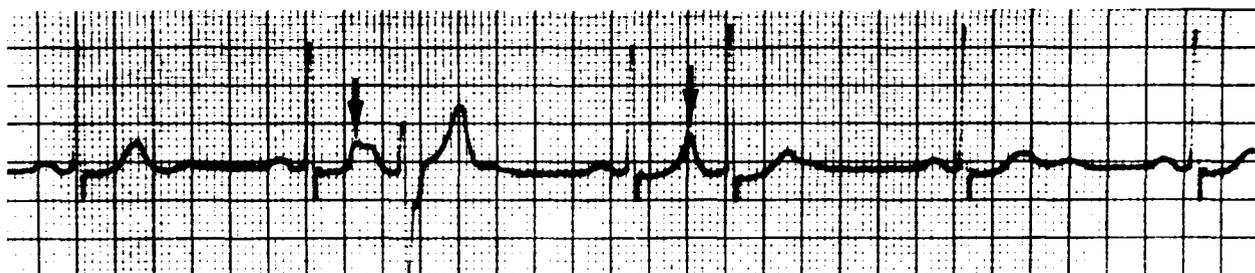


FIG. 8. Premature atrial beats (PAC).

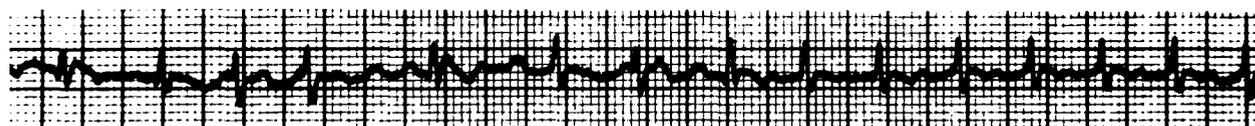


FIG. 9. Atrial fibrillation.

Atrial Premature Beats/Premature Atrial Contractions (APBs and PACs)

Data for atrial premature beats (Fig. 8) include:

- Rate: Normal, but may vary.
- Rhythm: Regular except where PAC occurs.
- P-Wave: Normal P-wave precedes each QRS. However, a P-wave associated with an early beat has a different configuration and may be hidden in the preceding T-wave.
- PR Interval: PR interval associated with PAC is usually longer than that associated with the sinus beat.
- QRS: Normal duration and constant configuration.

The SA node is the dominant pacemaker, but an irritable focus within the atria discharges an impulse for one beat and temporarily depresses the SA node. Patients are usually asymptomatic, but they may experience a palpitation or fluttering sensation. This may be due to myocardial ischemia, infection, or drugs such as digitalis, caffeine, or nicotine. Treatment is not necessary, but the patient should be observed for increasing irritability.

Atrial Fibrillation

Data for atrial fibrillation (Fig. 9) include:

- Rate: Atrial rate is above 350 beats per minute and ventricular rate is usually between 60 and 160, but it may vary.
- Rhythm: Irregular.
- P-Wave: P-waves are replaced by "f" (fibrillation) waves identified by undulations of varying contour, amplitude, and spacing.
- PR Interval: Absent.
- QRS: Normal duration and constant configuration.

An ectopic focus or foci in the atria control the pacemaking function of the heart. Because of the rapid atrial rate, only portions of the atrial muscle can respond, resulting in an ineffective atrial contraction. Ventricular response is highly irregular. The patient usually has an underlying disease state, such as rheumatic mitral stenosis, thyrotoxicosis, pericarditis, or coronary artery disease. Approximately 30% of all patients with atrial fibrillation experience systemic or pulmonary emboli due to the ineffective atrial contraction (3). Treatment includes digitalis, propranolol, and verapamil to slow the ventricular response. If a sudden onset of atrial fibrillation occurs, with rapid ventricular response resulting in acute cardiovascular decompensation, direct current (DC) conversion is the treatment of choice.

Atrial Flutter

Data for atrial flutter (Fig. 10) include:

- Rate: Atrial rate is 250–350 beats per minute, with ventricular rate depending on the degree of AV block. Atrial rate may be slower if the patient received digoxin.
- Rhythm: Atrial rhythm is regular, and ventricular rhythm may be either regular or irregular.
- P-Wave: Absent and replaced by sawtooth-like F-waves, which are regular in contour and spacing. This is usually seen in II, III, aVF, and VI leads.
- PR Interval: Absent.
- QRS: Usually normal duration and configuration.

A focus in the atria takes over as pacemaker and discharges at a rate of 250–350 beats per minute. The ventricles cannot respond to every impulse, resulting in the AV node blocking some as a safety device. If the ventricles respond only to every



FIG. 10. Atrial Flutter.



FIG. 11. Premature junctional beat (arrow).

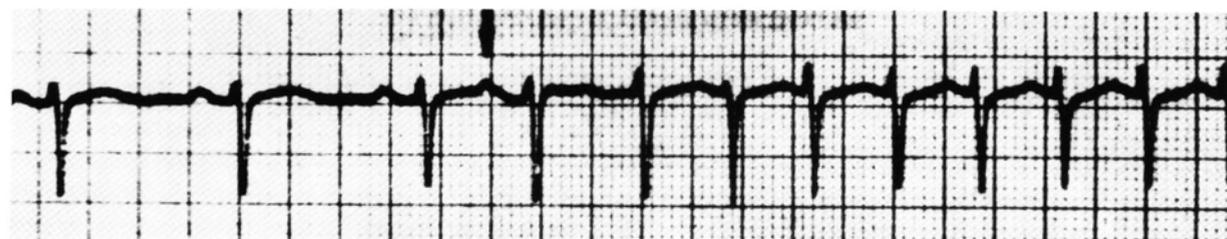


FIG. 12. Supraventricular tachycardia (starting at arrow).

other impulse, it is defined as "atrial flutter with 2:1 AV block;" or to every third, "atrial flutter with 3:1 AV block;" and if there is no set pattern of block, "atrial flutter with varying degree of AV block." In acute infarct patients, ischemia or infarct of the atria is commonly responsible for this ectopic rhythm. The patient may be asymptomatic, or experience symptoms similar to atrial fibrillation. If the ventricular rate is very rapid, congestive heart failure or shock may be present. Drugs used to slow down ventricular response include digitalis, propranolol, and verapamil. Synchronous DC conversion is the first choice of treatment whenever there is hemodynamic compromise since it promptly and effectively restores sinus rhythm. Signs of poor cardiac output include oliguria, hypotension, diaphoresis, and change in mental status.

Premature Junctional Beats

Data for premature junctional beats (Fig. 11) include:

Rate: Normal.

Rhythm: Regular except when premature beat occurs.

P-Wave: Normal contour except for that associated with premature beat, which may be inverted, buried within the QRS, or retrograde (following the QRS).

PR Interval: Normal except with premature beat that may be shorter than normal or immeasurable.

QRS: Usually normal, but it may be a little irregular if aberrant (abnormal) conduction is present.

The term junctional refers to the AV node and the bundle of His area. An impulse is discharged early from this area and stimulates the ventricles rather than the SA node. The site at which the impulse originates determines its contour and relationship to the QRS. If the patient is asymptomatic, treatment is usually not required.

Supraventricular Tachycardia (SVT)

Data for SVT (Fig. 12) include:

Rate: 150–250 beats per minute.

Rhythm: Regular except at onset and termination.

P-Wave: Difficult to identify because it is superimposed on preceding T-wave or hidden within the QRS.

PR Interval: Usually constant if P-waves can be identified.

QRS: May be normal or widened if Wolff Parkinson-White syndrome (WPW) or aberrant conduction is present.

Paroxymal atrial tachycardia (PAT) or paroxymal junctional tachycardia (PJT) is characterized by rapid regular atrial or junctional tachycardias of sudden onset and termination with rates of 150–250 beats per minute. Because rapid rates sometime obscure the P-waves, making it impossible to determine the origin of the impulse, the rhythm is termed SVT (meaning it originates somewhere other than the ventricles). Clinical presentation, EKG characteristics, and treatment for both conditions are virtually the same. Onset of the rhythm is abrupt, usually preceded by a PAC that conducts with a prolonged

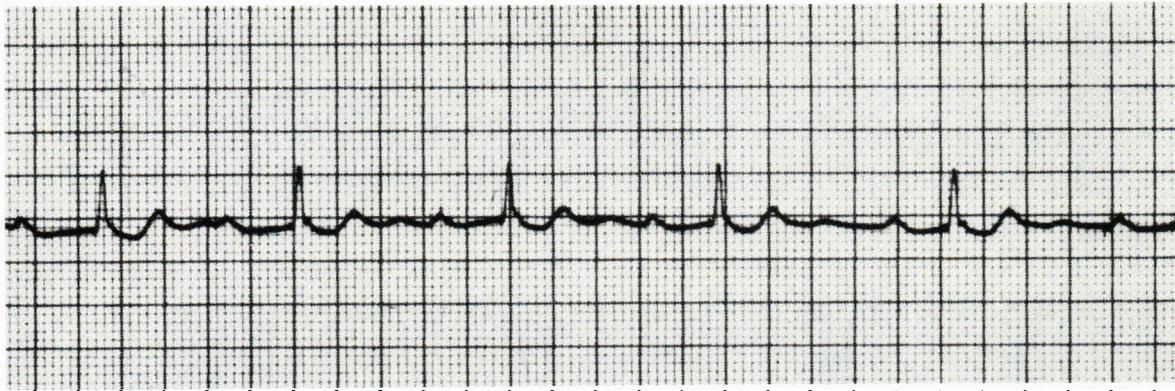


FIG. 13. First degree AV block.

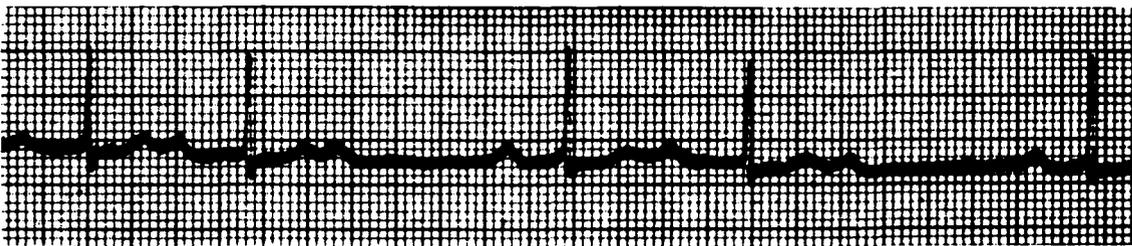


FIG. 14. Second degree AV block Type I (Wenckebach).

PR interval. Abrupt termination is sometimes followed by a brief period of asystole (no electrical activity).

This condition may occur at any age and can be unassociated with underlying heart disease. However, when organic heart disease is present, it is usually coronary artery disease. Thyrotoxicosis is also frequently associated with SVT. This arrhythmia may be related to specific inciting causes such as overexcitement, emotional stimuli, drinking coffee, or smoking. It may occur during stress testing and can be particularly alarming especially if aberrant (abnormal) conduction is present. As many as 20–30% of patients with PAT or PJT have an accessory pathway (pre-excitations or WPW syndrome) that conducts only in a retrograde direction. In WPW, there is an accelerated pathway that allows part of the ventricle to be activated before it should be by an impulse descending through the normal A-V conduction system. The classical WPW pattern consists of a short P-R interval and a widened QRS complex with a slurred initial component (*I*). The clinical importance of WPW is a predisposition to tachycardias.

Symptoms that frequently occur include palpitations, anxiety, angina, heart failure or shock, depending on duration and the rate arrhythmia, and presence of heart disease. SVT may cause syncope because of rapid ventricular rate, reduced cardiac output, and cerebral circulation.

Treatment depends on the clinical situation and how well rhythm is tolerated, as well as the presence of associated diseases. Treatment may simply be sedation, rest, and reassurance. Carotid sinus massage may slow or terminate the rhythm. Drug therapy includes tensilon, verapamil, digitalis, and iderol. If vagal maneuvers and drug therapy are ineffective, or if cardiac decomposition occurs, synchronized DC shock should be employed.

First Degree AV Block

Data for first degree AV block (Fig. 13) include:

Rate: Normal.

Rhythm: Regular.

P-Wave: Normal contour and precedes each QRS.

PR Interval: Prolonged > 0.20 sec.

QRS: Normal duration and constant configuration.

The atrial impulse is delayed as it travels through the AV node or His bundle, manifested by prolonged PR interval. Once it passes this region, it is conducted normally through the ventricles. It may result from ischemia of the AV node, or it may be drug induced (i.e., lidocaine, digitalis, quinidine, or procainamide). Treatment consists of drug reassessment (should drugs be the cause) or continued monitoring since this condition may precede more advanced degrees of block.

Second Degree AV Block: Type I (Wenckebach)

Data for this condition (Fig. 14) include:

Rate: Atrial rate is normal; however, the ventricular rate is slower than the atrial because the atrial stimuli do not always cause a ventricular response.

Rhythm: Atrial regular. Ventricular irregular.

P-Wave: Normal contour, but it may be hidden in preceding T-wave as the PR interval lengthens.

PR Interval: Progressively lengthens.

QRS: Normal in contour and configuration.

Each atrial pulse is delayed for an increasingly longer time period—as it travels through the AV node and His bundle un-

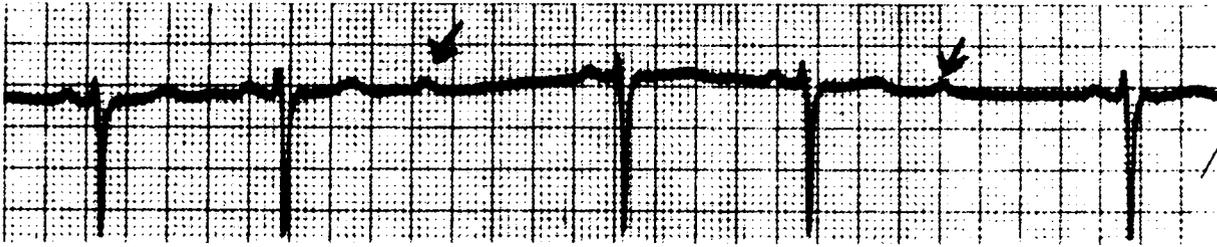


FIG. 15. Second degree AV block Type II (arrows).



FIG. 16. Complete heart block.

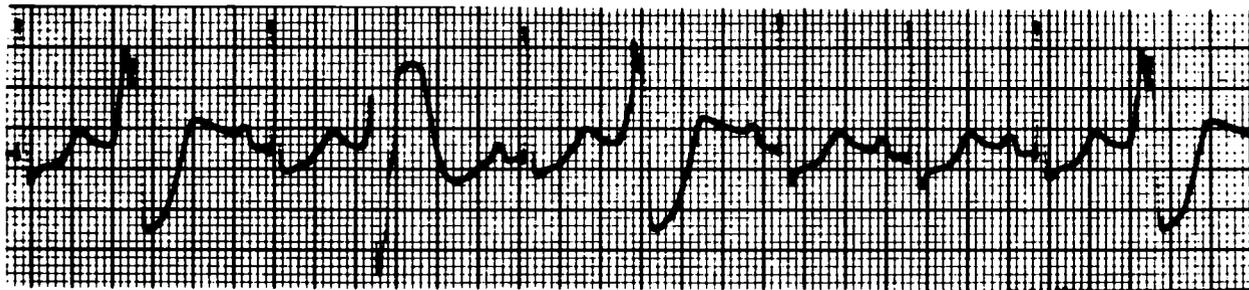


FIG. 17. Multifocal premature ventricular contractions (PVCs, VPBs).

til, finally, an impulse is blocked completely. There is a P-wave, but it is not followed by a QRS complex. This cycle then repeats itself. This is a more advanced degree of block and is commonly seen in inferior infarctions. However, it may even be caused by those drugs mentioned with first degree block. The patient is usually asymptomatic, but continued monitoring is necessary.

Second Degree AV Block: Type II

Data for this condition (Fig. 15) include:

Rate: Atrial rate is regular, and ventricular rate is slower than atrial.

Rhythm: Atrial rhythm is regular, but ventricular rhythm varies, depending upon the degree of the block.

P-Wave: Normal but is not always followed by QRS complex.

PR Interval: Normal or it may be prolonged.

QRS: Usually widened.

The atria are being paced normally by the SA node. Not all impulses, however, are conducted through the AV node to stimulate the ventricles. This is a more serious block than type I and is frequently followed by complete heart block, especially in the anteroseptal MI. Depending on the ventricular response, the patient may be asymptomatic or may show signs of decompensation. Treatment depends upon the patient's response, and may consist of atropine or isuprel to increase the rate. Insertion of a pacing wire may also be required.

Complete Heart Block

Data for this condition (Fig. 16) include:

Rate: Atrial rate is usually 2 to 3 times faster than the ventricular rate.

Rhythm: Atrial and ventricular rhythms are both regular, but they are independent of each other.

P-Wave: Normal contour, but there is no fixed relationship to the QRS.

PR Interval: Constantly changing.

QRS: Configuration depends on the site of the impulse formation. If the impulse arises high enough in the AV node, the QRS may be normal. If, however, the impulse originates lower in the conduction system, the QRS will be wide and bizarre.

The atria and ventricles each have their own pacemaker, with its own rate and beat, completely independent from each other. No atrial impulses are conducted to the ventricles. Patients may be asymptomatic as in the case of a chronic, stable complete block. In the setting of an acute myocardia infarction this condition may be life threatening, requiring immediate insertion of a pacing wire. Isuprel and atropine are used to increase the ventricular rate until this procedure is completed.

Premature Ventricular Contractions/Ventricular Premature Beats (PVCs and VPBs)

Data for this condition (Fig. 17) include:

Rate: Usually normal but may vary.

Rhythm: Regular except when interrupted by the premature beat.

P-Wave: Normal contour and precedes each QRS except with

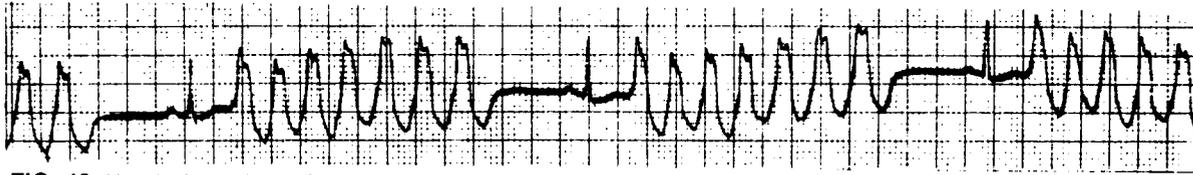


FIG. 18. Ventricular tachycardia.



FIG. 19. Ventricular fibrillation.

the premature beat. In which case, the P-wave will be absent.
PR Interval: Normal duration but absent with premature beat.

QRS: Normal duration except when premature beat occurs, then the QRS becomes wide and bizarre, with the T-wave usually opposite in direction to the QRS.

A focus within the ventricles becomes irritable and discharges an impulse, causing a ventricular contraction, temporarily depressing the SA node. The beat occurs early, and because there is no atrial stimulation there is no P-wave. The QRS becomes wide and bizarre since it is not following normal conduction pathways. The QRS is followed by a short pause (compensatory) until the SA node discharges its next impulse. This irritability is usually caused by ischemia. However, it may also be caused by electrolyte imbalance or drug toxicity. Patients may be asymptomatic or may complain of a "thumping," fluttering, or skipping sensation. Because a PVC may occur before the ventricle has had a chance to fill completely, the resulting contraction is less effective hemodynamically than with a normal beat. If these beats are numerous, they may burden an already damaged heart.

PVCs are treated if they number more than five per minute, if there is run of two or more, should they be multifocal (arising from more than one focus), or if they occur on the upswing or apex of the preceding T-wave (the vulnerable period in which ventricular tachycardia may occur). It is common practice to treat these PVCs only in the presence of a diseased ventricle.

Treatment consists of anti-arrhythmic drugs such as lidocaine, procainamide, and more recently, bretylol. PVCs may be a precursor of ventricular tachycardia or fibrillation in the acute infarct patient and should be monitored carefully. PVCs may occur in an identifiable pattern; every second (bigeminy), every third (trigeminy), or every fourth (quadrigeminy) beat after a normally conducted beat.

Ventricular Tachycardia

Data for this condition (Fig. 18) include:

Rate: Ventricular is 150–200 beats per minute.
Rhythm: Regular or slightly irregular.
P-Wave: Obscured by the QRS complex.

PR Interval: Undeterminable.

QRS: Wide (> 0.12 sec) and has a bizarre configuration. The T-wave is usually opposite in direction to the QRS.

Ventricular tachycardia is defined as a run of three or more consecutive PVCs. It occurs when an irritable focus in the ventricle takes over at a faster rate than the SA node and establishes itself as the pacemaker. Untreated, it may last seconds or weeks and possibly lead to congestive failure, shock, or perhaps death. Mental confusion, convulsions, or unconsciousness reflect decreased blood and oxygen supply to the brain, and immediate treatment is required. Lidocaine is the usual drug of choice, and DC counter shock is employed immediately should cardiac decompensation and circulatory insufficiency become evident.

A rapid ventricular tachycardia (over 210 beats per minute) is called ventricular flutter and is even more dangerous than tachycardia. This condition leads to ventricular fibrillation within a minute or less of its onset.

Ventricular Fibrillation

Data for this condition (Fig. 19) include:

Rate: Ventricular 400–600 beats per minute.
Rhythm: Grossly irregular.
P-Wave: Absent.
PR Interval: Absent.
QRS: Replaced by undulations of varying contour, amplitude, and spacing.

An ectopic focus (or foci) within the ventricles discharges impulses at a rate too rapid for the ventricles to respond, resulting in an ineffective ventricular contraction. The ventricles merely quiver, disallowing any effective cardiac output. Because there is no cardiac output, faintness, convulsions, and death occur rapidly if immediate treatment is not instituted. The only effective treatment consists of immediate DC cardio-conversion with CPR employed between countershocks to maintain blood circulation.

This arrhythmia occurs frequently as the terminal event in a variety of diseases. It may also be seen in cardiac pacing, during cardiac catheterization, anesthesia, or accidentally by improperly grounded electrical equipment. It commonly oc-

curs as the first sign of coronary artery disease; primary ventricular fibrillation, cardiac arrest, and clinical sudden death. When it occurs in a favorable setting (i.e., in a witnessed arrest), ventricular fibrillation can be successfully treated with electrical defibrillation without subsequent evidence of myocardial infarction.

It may occur in the early hours following an acute myocardial infarction. In CCUs, successful defibrillations in the acute infarct patient is usually successful and life saving.

CONCLUSION

The preceding text has been designed to assist the technologist who desires to become proficient in an understanding of patient cardiac monitoring. In-depth texts are available that deal with more complicated arrhythmias as well as interpretation of full twelve-lead cardiograms (4-7).

A final word of caution—when in doubt as to whether an arrhythmia is life threatening, evaluate the patient's clinical

response and stability (i.e., verify whether the patient is conscious and talking coherently with no complaints of distress). A loss monitor lead or in some cases electrical interference may mimic ventricular fibrillation or asyctole.

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