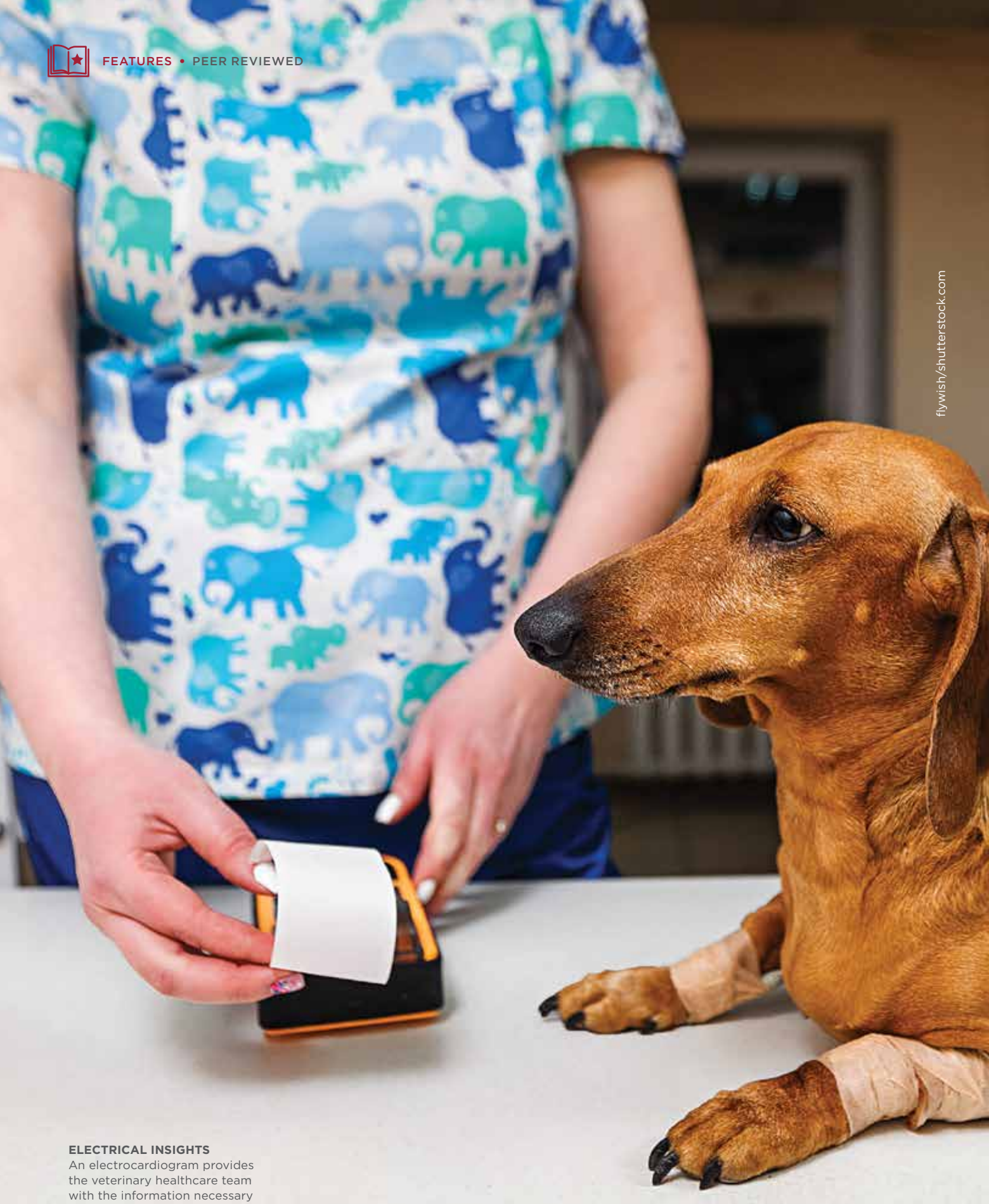

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ELECTRICAL INSIGHTS
An electrocardiogram provides the veterinary healthcare team with the information necessary to calculate a patient's heart rate and heart rhythm.

CARDIOLOGY

Overview of Electrocardiogram Interpretation

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Electrocardiography is the most useful diagnostic technique for characterizing cardiac rhythms; however, correlating what is recorded on the tracing with the electrical activity in the heart can be confusing. Veterinary nurses should have the skills needed to identify and characterize arrhythmias, which can be seen on electrocardiograms (ECGs) of patients being monitored during anesthesia, patients presented for various emergencies, and patients with known heart disease.

The waveforms produced during an ECG recording are reflective of specific portions of the heart's electrical activity. The ECG provides information pertaining only to the electrical, not the mechanical, activity of the heart. An ECG will provide the information necessary to calculate heart rate (HR) and determine heart rhythm with the patient in any position. In the event of a suspected arrhythmia, a printed tracing simplifies interpretation.

This article presents tips for improving the accuracy of diagnosing arrhythmias and understanding when they require treatment. It begins with a brief overview of heart function, followed by a more detailed description of how that function is displayed on an ECG tracing.

HEART FUNCTION

The normal heartbeat begins with depolarization of specialized tissue called the sinoatrial node, located in the cranial right atrial wall (**FIGURE 1**). This impulse is propagated through the tissue of both atria in a wavelike pattern. The electrical activity of the atria is insulated from the ventricles by the fibrous cardiac skeleton, which forces all electrical activity to travel to the ventricles through the atrioventricular (AV) node near the intraventricular septum. After entering the AV node, the electrical impulse is conducted via specialized conduction cells through the bundle of His and then into the bundle branches, which divide into right and left bundle branches, each innervating its respective ventricle. After reaching the termination of the bundle branches, the impulse is transmitted through Purkinje fibers to the myocytes. Stimulated by the electrical impulse, the myocytes stimulate their neighboring cells and conduct the impulse, cell to cell, causing ventricular contraction.[1] These events are represented on the ECG as the waveforms. Atrial repolarization is not visible on the ECG because it is obscured by the QRS complex.

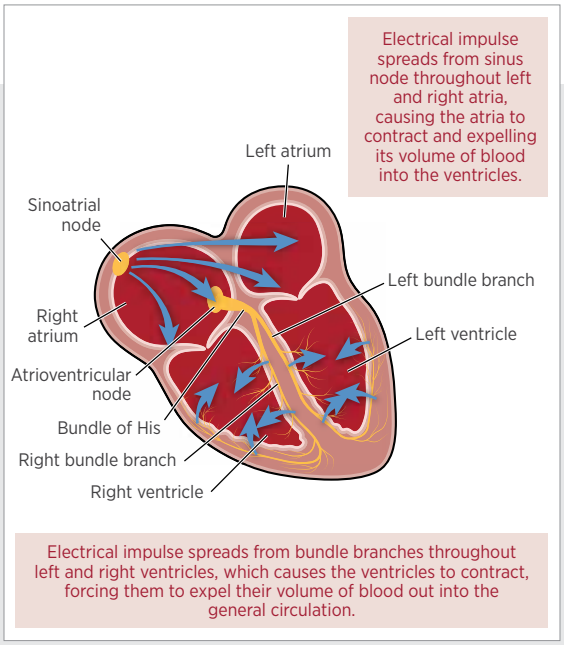

FIGURE 1. Schematic of the cardiac conduction system.

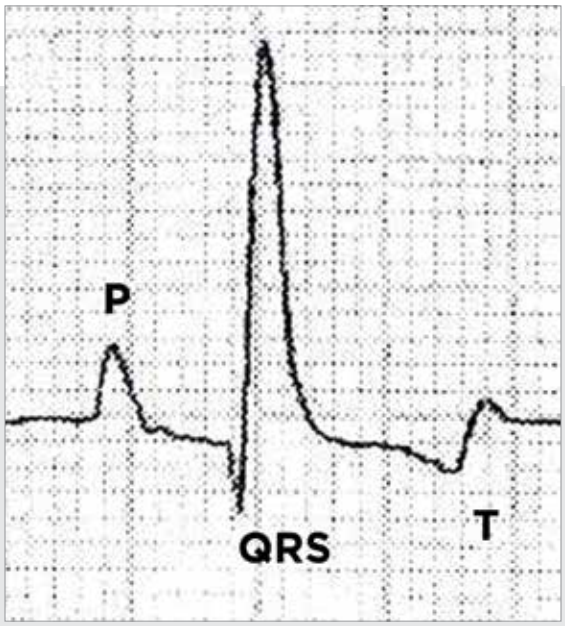

FIGURE 2. Electrocardiogram showing normal sinus rhythm in a dog. The **P wave** indicates atrial depolarization, the **QRS complex** indicates ventricular depolarization, and the **T wave** indicates ventricular repolarization. This tracing demonstrates a normal positive P wave, a negative Q wave, positive R wave, and no distinct S wave in this lead (which is considered a normal variation). The T wave of the dog may be positive, negative, or diphasic (both negative and positive) as seen here; these are all considered normal.

HEART RHYTHMS

Heartbeats that originate in the sinoatrial node, which are normally propagated to the ventricles, are termed sinus beats. Sinus beats (or sinus rhythm) are considered normal and in lead II will appear on ECG as a positive P wave, slightly negative Q wave, strongly positive R wave, and slightly negative S wave. In veterinary patients, the T wave may be positive, negative, or diphasic (i.e., both negative and positive) (**FIGURE 2**).

Any arrhythmia affecting the P wave represents changes in atrial conduction. Any arrhythmia affecting the QRS complex represents changes in ventricular conduction. Arrhythmias involving the atria are categorized as supraventricular. Arrhythmias involving the ventricles are termed ventricular.

Arrhythmias can be divided into 2 broad categories: those caused by abnormal impulse formation and those caused by abnormal conduction. Some patients will demonstrate both during the same recording. Either type of arrhythmia may be responsible for bradycardia or tachycardia.

- **Abnormal impulse formation:** A premature beat is caused by formation of an abnormal impulse and occurs early in the expected rhythm (**FIGURE 3**). Examples of abnormal impulse formation are ventricular premature complexes and atrial premature complexes. Ventricular tachycardia (**FIGURE 4**) is a series of 4 or more abnormal ventricular impulses occurring at a rapid rate. This potentially fatal rhythm should be reported to the veterinarian immediately. An escape beat is an abnormal impulse formed because of a delay in normal depolarization; escape beats arrive late in the expected rhythm. Although the escape beat is technically abnormal, it is a rescue mechanism that keeps the patient alive.
- **Abnormal conduction:** Abnormal conduction can occur at any level of the conduction system. If the sinus impulse is stopped at the AV node from reaching the ventricle, then AV block has occurred. If the impulse leaves the AV node but is blocked in the lower ventricular conduction system, then a bundle branch block has occurred.

RECORDING THE ECG

Recording an ECG involves placing electrodes on the patient on opposite sides of the heart. The electrodes are connected to a computer designed to measure the electrical activity between them. Electrodes are polarized as positive or negative. As the electrical impulse moves through the heart toward a positive

Top Left: Blamb/shutterstock.com

FIGURE 3. Heart rate (HR) calculations and premature complexes. Lead II, 25 mm/sec, 10 mm/mv. **Line T** demarcates 30 × 5–mm boxes (15 cm) or 6 seconds at this paper speed. To calculate HR, count the number of complexes within the line, which in this case is 11. Multiply 11 by 10 (60 seconds/minute divided by 6 seconds within the line = 10) to get an HR of 110 bpm. **Line I** represents calculation of an instantaneous HR. There are 8 1-mm boxes between the indicated QRS complexes. At 25 mm/sec, each box equals 40 milliseconds. Multiply 8 by 40 to arrive at 320 milliseconds between these 2 beats, then divide 60 000 by 320 to get an HR of 187 bpm. This calculation indicates that if this interval persisted, the HR would be 187 bpm. **Arrow V** points to a ventricular premature complex, and **arrow A** points to a supraventricular premature complex (commonly called an atrial premature complex).

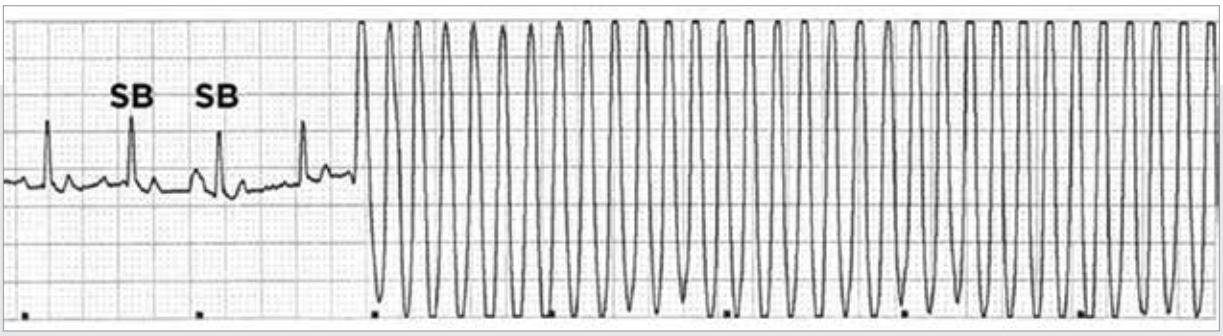

FIGURE 4. Ventricular tachycardia; lead II, 25 mm/sec, 10 mm/mv. This electrocardiogram tracing shows a sinus rhythm (indicated by **SB** for sinus beat) that suddenly changes to a rapid ventricular tachycardia. The heart rate (HR) during sinus rhythm, calculated by using the instantaneous method, is 125 bpm. The HR during ventricular tachycardia is approximately 375 bpm; this rhythm is often referred to as "R on T" phenomenon and is potentially life-threatening. This rhythm can degenerate into ventricular fibrillation or asystole without warning.

electrode, a positive deflection is traced on the ECG. The reverse is true if the impulse moves toward a negative electrode. An ECG lead is an angle created across the heart by the placement of the 2 electrodes; the tracing reflects direction and magnitude. The standard electrode arrangement creates 3 bipolar leads (**TABLE 1**). Electrocardiographic paper is graph paper. On the paper are bold lines indicating 5-mm boxes, within which are smaller grids of 1-mm boxes. When combined with paper movement speed and an electrical amplitude calibration, time and electrical voltage can be accurately calculated. The common paper speeds are 25 mm/sec and 50 mm/sec. The standard calibration is 10 mm/mv. For a complete multivariant analysis of the ECG, the patient must be in right lateral recumbency with other leads being recorded. Use of leads other than lead II is beyond the scope of this article. All changes to the ECG discussed in this article will be about lead II. Normal ECG values for waveform amplitudes, durations, and intervals are widely published.[2-4]

USING THE ECG TO CALCULATE HEART RATE

Before beginning any interpretation, first note what is termed CLAP (the amplitude **C**alibration, **L**ead

TABLE 1 Placement and Function of ECG Leads*

LEAD	POSITIVE LOCATION/COLOR	NEGATIVE LOCATION/COLOR	DIRECTION OF VECTOR ACROSS HEART
I	Right arm/white	Left arm/black	Right cranial to left cranial
II	Right arm /white	Left leg/red	Right cranial to left caudal
III	Left arm/black	Left leg/red	Left cranial to left caudal

**The green electrode (if present) serves as an electrical artifact filter or "ground." It may be attached anywhere on the patient, but traditionally it is attached to the right rear leg. ECG=electrocardiogram.*

The most common rhythms of dogs are normal sinus rhythm and sinus arrhythmia, at a rate of 60 to 170 bpm.[2-4] The most common rhythm of cats is sinus rhythm at a rate of 140 to 220 bpm.[2-4]

displayed, any **A**rtifact, and **P**aper speed). Then determine the HR. This step quickly identifies the presence of bradycardia or tachycardia. Because HR is a component of cardiac output, an abnormal HR can have a deleterious effect on cardiac output. Decreased cardiac output may be noted as hypotension in the patient. Monitors may display HR for the operator, but these values should be viewed with scrutiny because the HR algorithm may incorrectly calculate HR as a result of artifact, arrhythmias, or excessively large ECG waveforms. A manual HR count is more accurate.

One way to manually calculate HR, useful for irregular rhythms, is to mark out 30 of the 5-mm boxes (a total 15 cm) on the ECG paper. This duration will equal 6 seconds at a paper speed of 25 mm/sec or 3 seconds at 50 mm/sec. To calculate the beats per minute (bpm), multiply the number of QRS complexes within the 30 boxes by 10 for a paper speed of 25 mm/sec or by 20 for 50 mm/sec. Another method, useful for rapid, or paroxysmal rhythms, is to calculate the instantaneous HR.[4] To perform this calculation, count the number of 1-mm boxes between 2 QRS complexes and then multiply the result by 40 for a paper speed of 25 mm/sec or by 20 for a paper speed of 50 mm/sec (**FIGURE 3**). The product equals the milliseconds between the 2 beats, which is then divided into 60 000, which is equal to the number of beats per minute.

STEPS FOR ASSESSING HEART RHYTHMS

1. Determine the Heart Rate

The first step is determining the HR, as described above. The rate will immediately define the presence of a bradycardia or tachycardia, thus providing focus for all subsequent interpretations of the ECG.

2. Determine the Predominant Rhythm

By assessing most beats, the predominant rhythm can be determined. The most common rhythms of dogs are normal sinus rhythm and sinus arrhythmia, at a rate of 60 to 170 bpm.[2-4] The most common rhythm of cats is sinus rhythm at a rate of 140 to 220 bpm.[2-4] Sinus rhythm is a regular rhythm displayed on ECG as a P–QRS–T wave configuration. The P wave is positive with a predominantly positive QRS complex. Sinus arrhythmia is a normal rhythm of dogs but will be irregular in a repeating pattern. Most arrhythmias are sustained long enough to be a predominant rhythm. Atrial fibrillation (**FIGURE 5**) is an exception caused by chaotic depolarization of the atria, most often associated with heart disease. Atrial fibrillation is recognized by tachycardia, lack of P waves, a positive QRS complex, and an irregular R–R interval. A wildly jagged baseline may also be noted as the fibrillation waves are recorded. It is uncommon for atrial fibrillation to return to normal sinus rhythm.

FIGURE 5. Atrial fibrillation; lead II, 25 mm/sec, 20 mm/mv. This electrocardiogram tracing is an example of atrial fibrillation. The **double arrow line** indicates 6 seconds. The average heart rate (HR) is a tachycardia of 200 bpm, there are no consistent visible P waves, and the QRS complexes are narrow and predominantly positive. These are the hallmarks of atrial fibrillation. The irregularity of the R–R interval is common with atrial fibrillation, but it should be noted that with a very rapid HR during atrial fibrillation, the R–R interval may become regular.

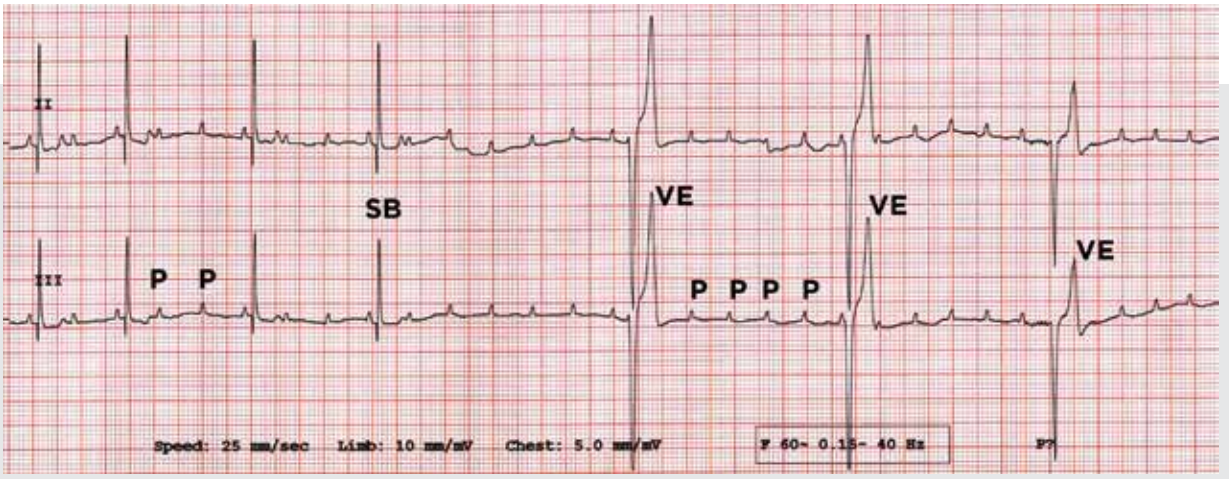

FIGURE 6. Atrioventricular (AV) block; leads II and III, 25 mm/sec, 10 mm/mv. This electrocardiogram tracing indicates both second and third degree AV block. The beginning of the tracing shows some normally conducted P waves creating a sinus beat **(SB)**. The P–Q interval is fixed at 80 ms, making this a Mobitz II second degree AV block. The heart rate (HR) at the second-degree AV block in the beginning of the trace is approximately 57–60 bpm. **P** indicates blocked P waves. Had these P waves been conducted, HR would be approximately 185 bpm. Approximately halfway through the tracing, the AV block becomes third degree (complete AV block). Blocked P waves can be seen, still at a rate of around 185 bpm. Three ventricular escape **(VE)** beats occur at a rate of 33–37 bpm according to this short tracing. These complexes are wider than they are during sinus rhythm and show the characteristic S–T segment slurring of complexes of ventricular myocardial origin. An individual P wave can be seen "buried" within the T wave of the second VE beat.

3. Determine the Anatomic Source of the Rhythm

Knowledge of the anatomic source of an arrhythmia can be used to guide therapy. Treating supraventricular arrhythmias requires different medications than those used for ventricular arrythmias. The simplest way to assess the source is to look at the width of the QRS complex.

When the arrythmia origin is supraventricular, the complexes are typically positive and narrow in lead II, shorter than 70 milliseconds.[4] A positive, narrow QRS complex is created when atrial beats move down the normal conduction tissue to depolarize the ventricle.

When the arrythmia originates in the ventricles, the complexes are wider than 80 milliseconds and show slurring of the Q–T segment; these complexes are commonly described as "wide and bizarre." These beats do not use the normal cardiac conduction system. They propagate slowly across the ventricular myocardium, cell to cell, making the wide QRS complex.

4. Determine Whether All Waves Are Present

Every sinus beat will generate P–QRS–T waveforms. The complexes formed by premature atrial and ventricular beats will lack P waves because they are abnormally formed in the atrial or ventricular tissue. Wide complexes not associated with P waves should be considered ventricular in origin until proven otherwise. Ventricular bundle branch blocks are indicated by wide complexes (>80 milliseconds)[5,6] with associated and consistent P waves. AV block will often appear as P waves with no accompanying QRS–T waveforms (**FIGURE 6**). AV block has 3 recognized forms: first, second, and third degree.

- **First degree AV block** is recognized as a prolonged P–Q interval, but all P waves do have associated QRS–T complexes.
- **Second degree AV block** shows intermittent blocking of P waves and some normal complexes. The P–QRS interval in second degree block may be progressively elongated (Mobitz I), can be normal, or can have fixed elongation (Mobitz II) before atrial conduction is blocked.
- **Third degree AV block** is a total dissociation of P waves from the QRS–T complexes. Patients with third degree AV block rely on a rhythm of ventricular escape beats to maintain cardiac output; these patients are bradycardic (30 to 60 bpm) and often syncopal.

MANAGING ARRHYTHMIAS

A detailed description of arrhythmia therapy is beyond the scope of this article; however, some general information is warranted. The decision of whether an

Emergency treatment of ventricular arrhythmias almost exclusively involves use of lidocaine. Chronic management of ventricular arrhythmias involves sotalol, procainamide, atenolol, and mexiletine.

arrythmia warrants treatment depends on the hemodynamic consequences, the likelihood of an increase in severity, and the risks posed by medication.

- **Hemodynamic consequences:** Perhaps the most important consideration for determining when to treat an arrhythmia is its hemodynamic consequence. The simplest way to assess hemodynamic consequence is by measuring the patient's blood pressure (BP). Accelerated idioventricular rhythm is a ventricular rhythm commonly seen after abdominal surgery, especially splenectomy. The ventricular rate is only 10% faster than the underlying sinus rhythm,[7] and the patient is largely normotensive. No therapy is required; this rhythm self-resolves as the patient heals from surgery. If the arrhythmia is lowering BP, it can be presumed that cardiac output is depressed, and therapy is indicated, especially when monitoring anesthesia. Sinus bradycardia that follows use of α-2 agonists (e.g., dexmedetomidine) is common, but BP is usually normal initially; hence, no therapy is needed. However, if bradycardia persists as hypotension over time, increasing HR is indicated because of the hypotension.
- **Likelihood of severity increase:** The next consideration is whether the arrhythmia is likely to increase in severity. Ventricular tachycardia can quickly degenerate into fatal ventricular fibrillation; thus, emergency therapy with lidocaine is indicated.
- **Risk from medication:** The final consideration is the risk posed by therapy. All antiarrhythmic medications can be proarrhythmic. Atropine, given during anesthesia for bradycardia, often causes a transient second degree AV block, which has minimal effect on BP and, because it tends to be transient, requires no intervention. Another example is the use of digoxin to control supraventricular arrhythmias. Although digoxin slows AV nodal conduction and reduces ventricular response to arrhythmias such as atrial fibrillation, it also is arrhythmogenic for the ventricle.

In brief, treating supraventricular arrhythmias involves the use of β-adrenergic–blocking drugs, calcium channel–blocking drugs, or digoxin. Emergency treatment of ventricular arrhythmias almost exclusively involves use of lidocaine. Chronic management of ventricular arrhythmias involves sotalol, procainamide, atenolol, and mexiletine. The choice of medications depends on multiple factors, including arrhythmia frequency, cardiac function, or successful treatment of other diseases. **TVN**

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Edward is the senior cardiology veterinary technician at Southwest Florida Veterinary Specialists. He has worked in veterinary medicine since 1976. He is a charter member of the Academy of Internal Medicine for Veterinary Technicians and served on its executive board representing cardiology for 12 years. Previously, he spent 18 years at the University of Missouri in the cardiology service and 3.5 years at Ross University School of Veterinary Medicine in the anesthesia service. He presents internationally on veterinary cardiology topics. He has a passion for teaching, is the editor of *Cardiology for Veterinary Technicians and Nurses,* and has written over a dozen peer-reviewed articles.

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