A systematic approach to the evaluation of the ECG will prevent overlooking important abnormalities. The following characteristics should be evaluated in every ECG. Familiarity with the normal parameters for the ECGs of the various species is, of course, essential for accurate interpretation.

1) Determine the heart rate
If the heart rate is regular, the number of small boxes (mm) between QRS complexes can be divided into 3,000 (at 50 mm/sec) or 1,500 (at 25 mm/sec) to find the instantaneous heart rate. The heart rhythm in animals, especially in dogs, is frequently irregular. In this circumstance the more accurate average heart rate is found by counting the number of beats in a known time interval and multiplying appropriately. Single channel ECG paper on analog recorders is usually marked by a vertical line at the top of the paper at 75 mm (1 mm = 1 small box) intervals. At a paper speed of 50 mm/sec, 75 small boxes (equivalent to 15 large boxes) represent 1.5 seconds so the heart rate per minute can be calculated by counting the number of QRS complexes in 1.5 seconds and multiplying by 40. At a paper speed of 25 mm/sec, 75 small boxes (15 large boxes) represent 3.0 seconds and the number of QRS complexes in 3.0 seconds is multiplied by 20. Many of the newer digital ECG machines calculate heart rate automatically.

2) Determine the cardiac rhythm
The heart’s rhythm is evaluated by inspection of the ECG and the findings are correlated with the physical findings. Analysis of the heart’s underlying rhythm should include the following steps.

A. What is the rhythm (including the regularity and the relationship among complexes)?
   a. Regular?
   b. Regularly irregular with a consistent and repeating pattern to the variation in the rate?
   c. Irregularly irregular where the rhythm is chaotic and there is no pattern to the irregular nature of the rhythm?
   d. Paroxysmal (which is defined as a sudden outburst)? When applied to the ECG, a paroxysm refers to a series of rapid ectopic beats, which begins and ends abruptly. The series may be as short as 3 beats or may last for minutes to hours.
   e. What is the relationship between the P and QRS complex? Is there a P wave for every QRS complex? Is there a QRS complex for every P wave? Is the duration of time between the various components (P-R interval, Q-T interval) normal? Is the duration of time between the various complexes consistent?

B. Where do the cardiac impulses originate (site of origin)? The four possible choices include:
   a. The sinoatrial (SA) node
   b. The atria
   c. The atrioventricular (AV) node/junctional
   d. The ventricles and His-Purkinje system

Impulses originating from the SA node, atria or AV node are grouped together under the heading supraventricular while impulses from the ventricles or His-Purkinje system are termed ventricular. Supraventricular beats should maintain a relatively tall, upright and narrow QRS complex because the impulse must utilize the His-Purkinje system to transmit the impulse to the ventricles. Therefore the ventricular muscle depolarizes uniformly with a set activation sequence. But when impulses arise from the ventricles or terminal branches of the His-Purkinje system they are slowly transmitted from individual myocardial cell to myocardial cell. This produces a relatively wide and bizarre QRS-T complex.

C. What are the ventricular and atrial rates?
   a. Too fast (tachycardia)
   b. Too slow (bradycardia)

D. What is the temporal relationship between any ectopic beats and the underlying heart rhythm?
   a. Premature beats are defined as ectopic beats that occur early in the sequence of normal beats, meaning the R-R interval from the preceding normal beat to the ectopic beat is shorter than the prevailing R-R interval. Premature beats are formed when the ectopic focus depolarizes more rapidly than normal, overrides the sinus node and assumes control of the heart rate for one or more beats.
   b. Escape beats are defined as ectopic beats that occur after a pause in the sequence of normal beats, meaning the R-R interval from the preceding normal beat to the ectopic beat is longer than the prevailing R-R interval.
The ectopic site assumes control of the electrical activity of the heart by default because the SA node fails to discharge or the sinus impulse is not properly conducted to the rest of the heart.

3) Calculate the mean electrical axis (MEA).
One of the most useful applications of vector principles is the calculation of the MEA for the QRS complex in the frontal plane. The MEA is the average of all the instantaneous vectors recorded during the QRS complex. Each species has a range of normal values, for example the MEA of normal dogs is between +40° and +103°. When the MEA is greater than +103° right ventricular enlargement is suggested. The mean electrical axis may be derived in two ways:

- Method 1: Using any two leads in the frontal plane, take the difference between the height of all positive QRS deflections and all negative QRS deflections in the two chosen leads. This calculates the vector for each lead. Plot the appropriate number of units, either positive or negative, on the lead axes. Draw perpendicular lines to the axes at these two points and then draw a vector from the origin of the figure to the point of intersection of these lines. The direction of this vector is the mean electrical axis.

- Method 2: Since the line of the mean electrical axis should have half of the total forces of ventricular depolarization on either side of it, a reasonable estimate of the MEA can be obtained by finding the limb lead which is the most isoelectric (i.e. the difference between the positive and negative QRS deflections in that lead is near 0). The MEA must then be perpendicular to that lead. To determine which direction the MEA takes, look at the lead whose axis is perpendicular to the isoelectric lead. If the lead has mainly positive QRS deflections, the MEA points toward the positive pole of that lead axis, just the opposite if the lead is mostly negative. Occasionally all of the limb leads are equally isoelectric and the MEA is said to be indeterminate in the frontal plane.

4) Measure the ECG waves and intervals.
The duration and amplitudes of the waves of the ECG are important in determining whether chamber enlargement is present. When one or more of the cardiac chambers enlarge, the processes of depolarization and/or repolarization may be altered in 1) magnitude of the vectors, 2) direction of the vectors, 3) rate of activation (duration), and 4) sequence of activation. These changes are reflected in the surface electrocardiogram as alterations in 1) the amplitude in the various leads, 2) the direction of the deflections in the various leads (i.e. change in MEA), 3) the width (duration) of the waves in various leads, and 4) the development of certain abnormal patterns of activation (i.e. S waves with RVH). The duration of the various intervals is important to determine if conduction or electrolyte disturbances are present. By convention the first negative deflection, preceding a positive deflection is termed a Q wave and the first positive deflection is called the R wave. A negative deflection occurring after a positive deflection is called an S wave. A second R wave is termed an r' wave, etc.

Management of arrhythmias
Arrhythmias are clinically important because of their ability to compromise cardiac output and oxygen delivery to the body. The level of cardiac performance during an arrhythmia is dependent on the rate, site of origin, and duration of the arrhythmia, as well as the presence of underlying cardiac or systemic diseases that may adversely affect the patient. Thus, the consequences of an arrhythmia may be clinically undetectable, may produce signs of inadequate cardiac output (weakness, fainting, shock), or may lead to the complete collapse of the circulatory system and sudden death.

Depending on the underlying cause of the arrhythmia, administration of antiarrhythmic drugs may not be needed. Metabolic abnormalities (acid/base or electrolyte disturbances, hypoxia) can contribute to arrhythmia formation and should be corrected. Arrhythmias in patients with concurrent congestive heart failure will often resolve spontaneously once the heart failure is successfully treated. Finally, the clinician must be familiar with the actions and potential side effects of the antiarrhythmic drugs, and must carefully weigh the risks and benefits of treatment. Administration of antiarrhythmics is not a benign procedure. Every agent has the possibility to induce further and perhaps more dangerous arrhythmias (pro-arrhythmia).

Ventricular tachyarrhythmias (VPCs, ventricular tachycardia)

1) No therapy may be required if the VPCs are infrequent and the patient is asymptomatic. However Holter monitoring is often required to confirm the true frequency of the arrhythmia.

2) Withdraw or adjust offending drugs (digitalis) if toxicity is suspected.

3) When associated with congestive heart failure, therapy with positive inotropes under close supervision is indicated along with other measures to treat the CHF.

4) Antiarrhythmic therapy is indicated when VPCs are frequent, multifocal, or occur in rapid groups (ventricular tachycardia). The most commonly employed oral antiarrhythmics include mexiletine and sotalol. Amiodarone may be used in select cases.
5) If life threatening ventricular tachycardia develops, intravenous therapy with lidocaine or procainamide is most often used.

Supraventricular tachyarrhythmias (frequent APCs, atrial tach, atrial fib)

1) No therapy may be required if the APCs are infrequent and the patient is asymptomatic. However Holter monitoring is often required to confirm the true frequency of the arrhythmia.

2) When frequent APCs are observed in patients with congestive heart failure, digitalis or diltiazem therapy can be considered (may be a precursor for atrial fibrillation).

3) Termination of atrial tachycardia may be accomplished by vagal maneuvers, precordial (chest) thump, or control of the ventricular response rate utilizing digoxin, atenolol, diltiazem, or sotalol. The same agents may be useful for preventing recurrence.

4) Atrial fibrillation.
   a. The usual goal in patients with heart disease is to slow the ventricular response rate. This is often achieved by digitalization +/- the addition of diltiazem, atenolol or sotalol if appropriate rate control is not achieved with digoxin alone. Amiodarone may be used in select cases.
   b. Conversion to sinus rhythm is usually only attempted in patients with a reasonable probability of remaining converted (those with minimal underlying heart disease). Oral quinidine, IV procainamide, or electrical defibrillation have been employed. Intravenous administration of diltiazem, amiodarone or sotalol is occasionally effective.