An Approach to the Patient with a Cardiac Rhythm Disturbance
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"What This Rhythm!"
An approach to the patient with a cardiac rhythm disturbance

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Approach to the Patient with a Cardiac Rhythm Disturbance

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The patient that presents with a rhythm disturbance can present both a diagnostic and therapeutic challenge to the clinician. Once a diagnosis of a particular rhythm disturbance is made, a decision must be made as to whether to treat the abnormal rhythm or not. Once this decision is made, careful selection of therapeutic agent(s) needs to be made. These decisions are not always clear-cut. The truth is that we do not really have enough information in veterinary medicine to practice evidence-based medicine in the treatment of cardiac rhythm disturbances. Much of the information we have available to us is extrapolated from studies in human medicine - however, the majority of cardiac rhythm disturbances in humans result from myocardial infarction, and this particular type of ischemia is not what we see in our patients.

While not all cardiac arrhythmias require treatment, many arrhythmias cause symptoms such as weakness, syncope, lethargy, congestive heart failure and sudden death. There is significant debate about what arrhythmias require treatment, when to treat some arrhythmias, and which drug or drugs to use, and whether these benefit the patient. The aim of this presentation is to provide a framework on which to base sound and rational treatment of the more commonly encountered arrhythmias.

Is Treatment Indicated?

Definitive rules to determine whether a specific arrhythmia requires treatment are not available. However, affirmative answers to the following questions suggest a need for therapy

1. Is the rhythm disturbance causing clinical signs of hemodynamic compromise? Clinically significant arrhythmias cause clinical signs by reducing cardiac output. Clinical signs may be subtle, and include the following
   - Weakness
   - Syncope
   - Collapse
   - Lethargy
   - Altered blood pressure
2. Does the rhythm contribute to Congestive Heart Failure
3. Is the arrhythmia associated with Sudden Death – most arrhythmias are not associated with sudden death, however, some are, including third degree AV block, ventricular arrhythmias, tachycardias, and multiform tachyarrhythmias.
4. Does the animal have evidence of cardiac enlargement – cardiac enlargement in combination with arrhythmias increases the risk of sudden death.
Examination of the Patient

Evaluation of the patient may reveal further evidence of a clinically significant arrhythmia

- Evaluate pulse quality - strength and rhythm
- Evaluate capillary refill time
- Evaluate/measure urine output
- Perform serum biochemistry
  - BUN/creatinine/amylase/phosphorus
  - PCV/TP
  - Electrolyte abnormalities
- Review current medications - routes of elimination, possible drug interactions, metabolism, and dosage. Determine if any current medications could cause, or contribute to the genesis of an abnormal cardiac rhythm

Abnormal clinical findings in the presence of an arrhythmia support the need for therapeutic intervention.

For the remainder of this tutorial, we will focus on the interpretation of the ECG, and the management of the patient with a clinically significant arrhythmia. Where appropriate, alternative therapeutic interventions will be mentioned, and relative merits and contraindications of treatment discussed.
Evaluation of the Electrocardiogram

The ECG should be read in a systematic and logical fashion, so that the rhythm present is correctly diagnosed, and an appropriate therapeutic plan is followed. In general, the following may be used as a guide.

1. Determine the atrial rate of depolarization – If the rate is fast, a tachycardia is present. If the rate is too slow, a bradycardia is present
2. Determine the ventricular rate of depolarization - If the rate is fast, a tachycardia is present. If the rate is too slow, a bradycardia is present
3. Determine whether there is atrio-ventricular synchronization – is there a P wave for every QRS; is there a QRS for every P wave; is the relationship consistent
4. If you have ectopic beats present, determine the following
   a. What is the timing of the ectopic beat - premature vs. escape
   b. Frequency and/or duration of the ectopic rhythm
   c. What is the effect of the ectopic beat on cardiac rhythm
5. Are the P waves and QRS complexes normally configured, and are they of normal width, or are they too wide.
   a. P waves that are negative, when they should be positive may indicate that they arise from an atrial site other than the SA node.
   b. If QRS complexes are normal width, they must be using the normal intra-ventricular system for depolarization.
   c. QRS complexes that are too wide can be generated by ventricular enlargement, bundle branch block, or by an ectopic pacemaker in the bundle branch or ventricular myocardium.
      i. If a wide QRS complex is generated by bundle branch block or left ventricular hypertrophy, a P wave will be present prior to the QRS complex, and the P-R interval will be constant. If a wide QRS complex is originating from within the ventricular muscle, there will be no P wave, or P waves will not have a consistent relationship with the QRS complex
6. Is the arrhythmia causing the clinical signs, or are the patient’s symptoms due to underlying disease?
Treat any underlying disorder

- Correct hypovolaemia, dehydration
- Treat underlying valvular or myocardial insufficiency
- Correct electrolyte abnormalities
- Normalize urine output

In the following text, we will divide common cardiac dysrrhythmias into brady-arrhythmias, and tachy-arrhythmias, and consider the causes and treatment of each of these separately. However, it must be noted, that some patients may have both a brady-arrhythmia and a tachy-arrhythmia present at the same time, or may have more than one abnormal rhythm present. In these cases, it is important to examine all aspects of the ECG, including the wave-form morphology, heart rate, atrial rate and ventricular rate, establish a list of differential diagnoses for each abnormal rhythm, and decide on a treatment plan.
Brady-Arrhythmias

Sinus Bradycardia

Features and Aetiology
- Sinus bradycardia is a regular rhythm that originates from the sino-atrial node, but occurs at a rate that is inappropriately slow
- Normal sinus rhythm
- Physiologic – may occur due to hypothermia, age (young animals especially)
- Pathologic causes result in excessive vagal tone, decreasing sino-atrial node discharge rate, heart rate, and cardiac output, leading to hemodynamic embarrassment (weakness, collapse, lethargy).
  Pathologic causes include the following
  - Cardiomyopathy
  - Respiratory arrest
  - Sick sinus syndrome
  - Hypothyroidism
  - Trauma (esp. head trauma)
  - Hyperkalemia
  - Hyper- and hypo-calcaemia
  - Elevated intra-cranial pressure, CNS disease
  - Digitalis, beta adrenergic blockers, calcium channel blockers, morphine sulphate
  - Feline dysautonomia

Clinical signs
- Decreased exercise tolerance
- Seizures, weakness, lethargy

Treatment
- Treatment is usually unnecessary unless clinical signs are evident. The requirement for treatment also depends on the underlying cause
- Acute treatment
  - Draw blood for analysis of electrolyte levels (calcium, potassium, acid/base status) and serum drug concentrations (digoxin)
  - Atropine at 0.01-0.02 mg/kg slow IV, isoproterenol may be administered in clinically unwell patients that do not respond to atropine in the acute setting. Alternatively, terbutaline 5 micrograms/kg SC, or dobutamine at 5 micrograms/kg/minute constant rate infusion may be used.
  - Provide a fluid bolus – this is an essential component of acute management of severe sinus bradycardia caused by drugs, electrolyte disturbances, or head trauma. The fluid of choice is lactated Ringer’s solution, or 0.9% sodium chloride. Administer a bolus of 10-20 ml/kg given
rapidly intravenously over 10 minutes. The fluid bolus achieves dilution of serum electrolytes, dilution of drugs, and an immediate increase in preload, afterload, and cardiac output. Titrate fluid therapy beyond the initial fluid bolus according to the response to therapy and the fluid requirements of the patient.

- Chronic treatment – propantheline (Pro-banthine) bromide 3.75-7.5 mg/dog PO q 8-12 hrs, hyoscyamine (buscopan) 0.003-0.006 mg/kg PO q 8 hrs, or Terbutaline (bricanyl) 5 micrograms/kg SC; 0.1-0.2 mg/kg PO q 8-12 hrs
- Pacemaker therapy is rarely required, but may be considered in cases refractory to chronic medical therapy.

ECG showing sinus bradycardia

Atrial Standstill

Pathophysiology

• Occurs when the atrial myocardium is unable to depolarize. Due to inexcitability of atrial musculature, lack of depolarization of the atrium or specialized conduction pathways occurs causing absence of atrial contraction. No P waves are generated on the ECG.

Etiology

• Hyperkalemia is the most common cause – hypoadrenocorticism in dogs, urethral obstruction in the cat.
• Digitalis toxicity
• Cardiomyopathy (esp. dilative cardiomyopathy)
• Muscular disease - muscular dystrophy; silent atrium syndrome of English Springer Spaniels, Shi-Tzu, Old English Sheep Dogs

Clinical signs

• Weakness, ataxia
• Symptoms of congestive heart failure
• Syncope
• Sudden death

Diagnosis

• Slow heart rate, regular rhythm, and ventricular escape rhythms. Ventricular escape complexes appear as wide, bizarre QRS complexes, occurring at a low rate. It is important that these complexes are NOT suppressed with lignocaine or other anti-arrhythmic drugs, or cardiac arrest will occur.
• NO P WAVES
• In hyperkalemia, there may be spiked T waves, and wide bizarre QRS complexes

Treatment

• Obtain blood for measurement of digitalis levels. Cease administration of digitalis if the patient is currently receiving this medication. Adjust dose following stabilization if necessary.
• Treat hyperkalemia with an intravenous bolus of 0.9% NaCl at 10-20 ml/kg IV over 10 minutes, glucose 7% solution at 5 ml/kg IV over 10 minutes following sodium chloride administration, calcium gluconate 10% given at 0.2-1.0 ml/kg given SLOW IV.
• Administer atropine 0.01 mg/kg slow IV or SC as a trial in patients without hyperkalemia, or in patients with underlying cardiac disease. Patients responding to atropine may be maintained for a short period on Terbutaline or propantheline bromide until pacemaker implantation can occur.
• Cautious use of intravenous fluids in patients with underlying cardiac disease is advised due to the presence of atrial disease, and the risk of development of congestive heart failure. Intravenous fluid support should be provided at maintenance rates in patients with clinical illness.
• Pacemaker implantation may be considered for cardiac causes such as cardiomyopathy, and silent atrium syndrome etc.
ECG of Atrial Standstill

Atrial standstill – note the complete absence of P-waves (with the exception of the last complex). The presence of tall, narrow QRS complexes with associated t-waves suggests ventricular escape complexes arising from AV junctional tissue (so-called ‘junctional escape complexes’)


Atrial standstill – the latter part of this tracing shows complete absence of P-waves. The final QRS complex in this tracing is tall and narrow, suggesting it is an escape complex originating in AV junctional tissue – followed by a P-wave
Sinus Arrest and Sinus Block

Pathophysiology
- Sinus arrest is defined as a complete loss of SA node automaticity. Sinus block results from failure of impulses formed in the sino-atrial node to depolarize the atria, or results in a delay in atrial depolarization. Either condition may produce loss of atrial depolarization, and ventricular asystole if secondary pacemakers do not initiate ventricular depolarization.

Aetiology
- Respiratory pattern
- Hereditary - pugs, Dalmatians
- Increase in vagal tone
- Atrial disease - dilatation, fibrosis, cardiomyopathy, neoplastic infiltration (haemangiosarcoma), sinus node disease (sick sinus syndrome)
- Medications – digitalis, beta-adrenergic blockers, class 1a anti-arrhythmic drugs
- Hyperkalemia

Clinical signs
- Syncope, weakness, congestive heart failure
- Occasional periods of loss of consciousness
- Sudden death

Diagnosis
- Generally, sinus rhythm is the predominant cardiac rhythm, however there are periods of electrical inactivity lasting from one P-P interval (SA block), or lasting longer than 2 P-P intervals (sinus arrest)
- ECG will show the presence of junctional or ventricular escape rhythms
- Occasionally, supraventricular tachycardia will occur

Treatment
- Anticholinergic therapy – atropine, propantheline, or terbutaline may be used in the acute setting
- Intravenous fluid therapy bolus as described for sinus bradycardia if required
- Pacemaker implantation is the therapy of choice for animals with symptoms unresponsive to medical management

NB - failure of subsidiary pacemakers to initiate escape rhythms will cause sudden death.

ECG of sinus arrest

Sinus arrest – note the complete absence of sino-atria activity, and the presence of junctional escape complexes

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Escape rhythms

**Definition**

- Escape rhythms arise from a physiologic (as opposed to pathologic) ectopic focus, when a superior focus (pacemaker) fails to fire. Escape rhythm QRS complexes generally look like ventricular premature complexes, however, escape rhythms are slow, and should not be suppressed. Escape rhythms originate from pacemaker cells that have a slower rate of discharge than that of the sino-atrial node, and therefore only appear when there is a sinus arrest, or sinus (sino-atrial, or atrioventricular) block. The ectopic pacemaker foci exist in the heart at one of two levels – supraventricular, or ventricular.
- For junctional supraventricular escape rhythms, the QRS is generally narrow, with or without a P wave; the escape rhythm rate is 40-70 beats per minute (bpm)
- For ventricular escape rhythms, the QRS is generally wide, and resembles a VPC in appearance; the escape rhythm rate is 20-40 bpm.
- Escape rhythms are generally regular, however, the ECG tracing will depend on the presence and type of rhythm disturbance that lead to the appearance of the escape rhythm in the first place

**ECG of ventricular escape complexes**

![ECG of ventricular escape complexes](https://via.placeholder.com/150)


**Junctional escape complexes in a patient with sinus arrest. The narrow QRS complexes with T-waves suggests ventricular depolarization originates in junctional (AV node) tissue**

![Junctional escape complexes](https://via.placeholder.com/150)

**Ventricular escape complexes – the wide bizarre complexes suggest the origin of these complexes is in the ventricular muscle wall, resulting in slow conduction of ventricular depolarizing impulse**

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Bundle Branch Blocks - Left and Right

**Definition**

- Bundle branch blocks are slowing’s, or interruptions of conduction involving one or more of the ventricular branches of the His bundle. This results in de-synchronization of the two ventricles. Electrocardiographically, this is seen as wide QRS complexes.
- Bundle branch blocks typically, but not always, emerge at faster heart rates.
- Left bundle branch block - polarity of QRS is positive in lead II; produces a small Q wave, then a normally oriented QRS complex, wider than normal. QRS configuration may mimic premature ventricular contraction originating in the right ventricle.
- Right bundle branch block - polarity of QRS is negative in lead II; occurs typically secondary to electrical conduction disorder, or right ventricular enlargement; rarely right bundle branch blocks may occur as a congenital disorder. Right bundle branch block produces a small R wave, and a large, wide S wave. Delayed depolarization of the right ventricle produces delayed closure of the tricuspid and pulmonic valves; producing split heart sounds (most commonly split second heart sound).
- If bundle branch block occurs with a sinus rhythm, the ECG diagnosis is straightforward, because the only abnormality is a wide QRS complex. However, if the bundle branch block occurs concurrently with a non-sinus rhythm, such as atrial fibrillation, the bundle branch block may look like ventricular tachycardia.

**Aetiology**

- Hypertrophic cardiomyopathy
- Dilative cardiomyopathy – especially boxer and Doberman
- Endocarditis
- Traumatic myocarditis
- Right BBB may be an incidental normal finding in the dog

**Clinical signs**

- Clinical signs are rare, and therefore BBB does not require specific therapy except that directed at the underlying cause.
ECG of left bundle branch block

ECG of right bundle branch block
Atrio-ventricular Block

Atrio-ventricular block exists when there is a delay or complete block of atrial impulse conduction through the atrio-ventricular node

First degree Atrio-ventricular Block

Etiology

- Increasing age – may be a normal change in certain breeds such as Cocker Spaniels and Dachshunds.
- Young puppies, and brachycephalic dog breeds may have first degree AV block.
- Cardiomyopathy
- Bacterial endocarditis
- Degenerative cardiac disease - bulldogs, dachshund, west highland white terriers
- Disorders of potassium balance – hyperkalemia, hypokalemia
- Hypothyroidism
- Vagal stimulation, vagal maneuvers
- Drugs
  - Digitalis, beta adrenergic blockers, calcium channel blockers
  - Morphine derivatives - butorphanol
  - Doxorubicin
  - Quinidine, procainamide

Characteristics

- Prolonged P-R interval in the presence of a normal sinus rhythm

First Degree AV Block – with prolonged P-R intervals
(www.kardiol.com)
Second Degree Atrio-ventricular Block

Definitions

- Mobitz Type I (Wenckebach) - gradual increase in P-R interval until a P wave occurs without an R wave. The QRS complex is normal duration. Mobitz Type 1 AV block occurs within the AV node tissue.

- Mobitz Type II - P-R interval constant but intermittent non-conducting P wave (P wave occurs without an R wave). Mobitz Type II AV block occurs within or below the Bundle of His (referred to as an infra-nodal block). Because the conduction block occurs below the His Bundle bifurcation, the QRS complexes may demonstrate abnormal morphology.

- Advanced 2nd Degree Heart Block - only one beat in a group is conducted to the ventricles - in ratios of 2:1, 3:1, 4:1 etc.

Aetiology

- See causes of First Degree Heart Block. Mobitz Type I AV block generally occurs secondary to increased vagal tone, or medications that depress AV node conduction, such as beta-adrenergic blockers, morphine, and calcium channel blockers. Mobitz Type II AV block is associated with organic heart disease, electrolyte abnormalities, and medications as for Mobitz Type I AV block.

Clinical Presentation

- Exercise intolerance, weakness, syncope

- Symptoms of congestive heart failure

Treatment and Clinical Management

- Treatment of Mobitz Type I AV block is generally not required; monitor electrolyte and acid-base status and treat as required.

- Mobitz Type II AV block is managed in the acute setting with administration of an atropine trial, glycopyrrolate, terbutaline or dobutamine administration. Management of symptoms of acute congestive heart failure may be managed by administration of furosemide, and benazepril as indicated by the patients’ condition. Definitive management for long-term management is cardiac pacemaker implantation.
ECG of Second Degree AV Blocks

Mobitz Type II AV block in bigeminy. Note the intermittent non-conducting P-wave. Because the non-conducting P-wave occurs with every second atrial depolarization (bigeminy), this rhythm could also be technically classified as an advanced second-degree AV block, in a ratio of 1:1.


Mobitz Type II AV Block. Note that only occasional P-waves conduct through to a QRS complex – however, P-R intervals are constant when they do occur.

Mobitz Type I AV Block. Note the progressive lengthening of the P-R interval, until eventually a P wave occurs without a QRS complex

Mobitz Type I AV Block. Note the progressive lengthening of the P-R interval, until eventually a P wave occurs without a QRS complex
Third Degree Atrio-ventricular Block

Definition

- Complete AV block occurs when all atrial impulses are blocked at the AV node or Bundle of His. An independent idioventricular pacemaker coordinates ventricular depolarization. As a result, the atria and ventricles are controlled by independent pacemakers
- P waves, when present, have no fixed relationship to QRS complexes
- Ventricular rate is slow
- Ventricular escape beats are present. When ventricular escape complexes originate from the AV junction, they have normal morphology; when they originate from below the His Bundle bifurcation, they will be wide and bizarre in appearance

Aetiology

- See causes of First Degree Heart Block
- Cardiomyopathy
- Atrioventricular infarction, inflammation, neoplasia
- Congenital - aortic stenosis, ventricular septal defects, congenital AV block

Clinical Features

- The magnitude and severity of clinical signs depends primarily on the underlying cause, and the rate of the ventricular escape rhythm
- Exercise intolerance, weakness, syncope, seizures, or congestive heart failure
- Sudden death

Treatment

- Medical treatment has limited benefit for long-term management. However, emergency treatment and stabilization can be life-saving. The use of atropine, Glycopyrrolate, or Terbutaline in the emergency setting is preferred. Oral medication with propantheline bromide or Terbutaline may be useful prior to cardiac pacing implantation.

Third Degree AV Block – Note the loss of a fixed relationship between P-waves and QRS complexes. The QRS complexes are wide and abnormal in appearance, indicating origin outside of the AV nodes and Bundles of His. These are called “escape complexes”

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Sick Sinus Syndrome – A Short Note

Sick Sinus Syndrome is a term used to describe primary conduction abnormalities that produce the following:

- Erratic, inconsistent SA node depolarization
- Inadequate subsidiary escape rhythms
- Clinical signs of bradycardia

Rhythms that are typically produced in sick sinus syndrome include:

- Sinus bradycardia
- SA block
- Intermittent SA arrest
- Inconsistent junctional or ventricular escape complexes
- Variable heart rate, frequent “pauses” in conduction, producing a slowing of the heart rate
- Occasionally, animals will have APC’s or VPC’s present intermittently, or periods of supraventricular tachycardia

The condition occurs most commonly in:

- Miniature Schnauzer
- West highland White Terrier
- American Cocker Spaniels
Tachyarrhythmias

Supraventricular Tachyarrhythmias

**Definition**

- The primary abnormality in supraventricular tachyarrhythmias is the presence of premature depolarization that originates from an abnormal site somewhere above the ventricles. Early beats may arise from ectopic foci within the atria or atrio-ventricular junctional region, proximal AV node, and Bundle of His. The premature depolarization’s may be singular, as in an isolated atrial premature contraction (supraventricular premature depolarization), or may be repetitive as in supraventricular tachycardia, atrial flutter and atrial fibrillation. The primary difference between supraventricular tachycardia, atrial flutter, and atrial fibrillation is the rate at which the ectopic focus depolarizes. The rate of pacemaker firing for supraventricular tachycardia in the dog is between 150—350 beats per minute; for atrial flutter, the rate is greater than 350 beats per minute, and for atrial fibrillation, the rate is generally greater than 500 per minute.

**Aetiology**

- Chronic obstructive pulmonary disease
- Tissue hypoxia, myocardial hypoxia
- Chronic valvular heart disease
- Dilated cardiomyopathy
- Hypokalemia, hyperkalemia
- Congenital - accessory AV pathway (pre-excitation syndrome), PDA, AV valve insufficiency
- Pre-excitation syndromes (accessory pathways)
- Pericardial effusion
- Neoplasia - haemangiosarcoma, heart base tumor
- Drugs - digitalis, anesthetic drugs (esp. barbiturates)
- Congenital heart defects
- Gastric dilatation-volvulus
- Cardiac trauma
- HCM (cats), intermediate and dilative cardiomyopathy (cats)
- Myocardial ischemia, trauma
Classification of Supraventricular Tachyarrhythmias

Atrial origin SVT’s
- Sinus nodal re-entrant tachycardia
- Automatic atrial tachycardia
- Intra-atrial re-entrant tachycardia
- Atrial flutter
- Atrial fibrillation

Junctional origin SVT’s
- Common AV nodal re-entrant tachycardia
- Orthodromic atrio-ventricular reciprocating tachycardia
- Automatic junctional tachycardia

Electrocardiography Features of Supraventricular Tachyarrhythmias

1. Identify the presence of narrow QRS complexes occurring at a rapid rate
2. Determine whether the QRS complexes occur in a regular, regularly irregular, or irregularly irregular fashion
   a. Atrial fibrillation - irregularly irregular
   b. Variable AV block - produces regularly irregular QRS rate
   c. Most other Supraventricular tachyarrhythmias have a regular pattern
3. Identify P’ waves. P’ waves are atrial depolarization’s that do not originate in the sino-atrial node, and generally have a slightly different, inverted, or more variable morphology to normal P waves
4. Examine ALL ECG leads
5. Right and left “arm” electrodes can be placed along the sternal borders of the chest wall while monitoring lead I to better demonstrate atrial activity
6. The absence of P’ waves during a regular SVT rhythm is suggestive of typical atroioventricular nodal re-entrant tachycardia, during which atrial and ventricular activation occur together
7. If the P’ wave occurs closer to the preceding QRS complex than to the subsequent QRS complex, so that the R-P’ interval is less than or equal to 50% of the R-R interval, the SVT is classified as “short”; if the R-P’ interval is greater than 50% of the R-R interval, the SVT is classified as “long”. Long SVT’s are typically atrial SVT’s, whereas short SVT’s are typically junctional

NOTE: in atrial fibrillation, no P waves are present, and the baseline may have small irregular undulations called f-waves
1. **Supraventricular Premature Depolarization’s (SPD’s)**
   a. Definition – Premature depolarization may occur when an ectopic site depolarizes at a rate that is faster than the sinus node. SPD’s most commonly arise from diseased left atrial myocardium, but can arise from the right atrium, or AV nodal region.
   b. ECG Findings – characterized by a normally appearing QRS complex that occurs too early i.e. the R-R interval is shorter than normal. The premature QRS complex may or may not have a P wave associated with it. When present, the P wave may have normal appearance, or may be shorter than normal, or may be inverted.
   c. Compensatory Pauses with SPD’s – Following a SPD, there may be a pause before the next P-QRS complex appears. On the ECG, the interval between the last normal sinus beat and the premature complex will be less than normal (i.e. the beat is premature). The interval between the premature beat and the next normal sinus beat may be normal (termed a non-compensatory pause) or may be longer than the normal R-R interval (termed a fully compensatory pause). The reason for the difference in compensatory pause depends on the location of the ectopic site causing the SPD, and whether the sinus node is refractory to stimulation by the premature beat or not.
   d. Bundle Branch Blocks and SPD’s – Occasionally, an SPD can produce a wide bizarre QRS complex. This occurs when the premature depolarization reaches the bundle branches before one of them has the time to repolarize, or when one of the bundle branches has only partially repolarized from the previous normal sinus beat. This situation results in a functional bundle branch block. If this occurs regularly, or if P waves are not clearly visible, distinguishing an SPD from a VPD may be difficult.

2. **Supraventricular Tachycardia (SVT)**
   a. Definition - Supraventricular tachycardia is defined as repetitive SPD’s occurring at a rate between 150 and 350 depolarization’s per minute. SVT can originate in atrial myocardium, or AV junctional tissue. Supraventricular tachycardia typically has a regular rhythm (caused by an ectopic focus that has a re-entry circuit associated with it), but can be an irregular rhythm if the ectopic pacemaker is a persistently discharging automatic focus.
   b. ECG Findings – SVT is characterized by a rapid atrial rate occurring at 150-350 beats per minute. P waves typically have variable amplitude and appearance, and may be inverted in appearance. QRS complexes are usually narrow and upright in lead II. As with SPD’s, occasionally there is a co-existing functional bundle branch block, that can make it difficult to distinguish SVT from ventricular tachycardia.
   c. Clinical Features – patients presenting with SVT generally have symptoms of low cardiac output, poor tissue perfusion, and may also present with symptoms of congestive heart failure. Echocardiography reveals low end-diastolic volume and diameter due to inadequate ventricular filling secondary to rapid atrial and ventricular rate.
d. SVT caused by Pre-excitation (Accessory Pathways) – Under normal circumstances, the atria and ventricles are electrically isolated from each other, with the only structures permitting electrical activity between the atria and ventricles being the specialized conducting tissue of the AV node and His Bundle. Accessory pathways between the atria and ventricles can exist (such as congenital aberrant myocardium), that permit an accessory electrical pathway to communicate between the atria and ventricles. The electrical impulse that traverses this bypass from atria to ventricles reaches the ventricle before the cardiac impulse that traverses the AV junction reaches the ventricles, resulting in early depolarization of the ventricle. On an ECG, this type of ventricular pre-excitation results in a shorter P-R interval, and a slurred upstroke in the QRS complex (called a delta wave). The remainder of the QRS complex is hybrid, and results from both ventricular pre-excitation and ventricular depolarization that originates from normal conduction pathways. Accessory pathways can cause supraventricular tachycardia if they conduct a ventricular depolarization retrograde through the accessory pathway to the atrium, and cause a premature atrial depolarization. On an ECG, the P-R interval will be shorter, the P-P interval will be shortened, and delta waves may or may not be present. Typically abnormal P waves, including inverted P waves will be present whenever retrograde depolarization from ventricle to atria occurs.

3. Atrial Flutter
   a. Definition – Atrial Flutter may be best defined as very rapid supraventricular tachycardia. When the supraventricular atrial rate at which the refractory period of the AV node is longer than the P-P interval of the SVT. At this stage, a functional AV block occurs, because the AV node cannot transmit all of the SVT impulses transmitted to the AV node. Generally, at this time, the SVT rhythm is termed atrial flutter.
   b. ECG Features and Diagnosis – P waves are either directly discernible, or visible as a saw-tooth baseline. The conduction rate to the ventricles is variable.

4. Atrial Fibrillation
   a. Definition – atrial fibrillation is defined as atrial depolarization’s occurring at a rate of greater than 350-500 per minute. The depolarization process is self-propagating, due to the constant regional repolarization of cells within the atrial myocardium, allowing successive depolarization cycles. These depolarization’s continually bombard the AV junctional tissue. The AV node transmits impulses to the ventricle depending on the state of depolarization/repolarization the nodal tissue is in. This feature contributes to the irregular irregularity in ventricular rhythm associated with atrial fibrillation.
   b. ECG Features – P waves are replaced by small amplitude oscillations referred to as f-waves. QRS complexes vary in amplitude. QRS complexes are generally normal in appearance, but maybe wide and bizarre in configuration if pre-excitation or bundle branch blocks are present. R-R interval is continuously variable.
   c. Clinical Features – Atrial fibrillation most commonly occurs secondary to serious underlying heart disease. The most common underlying diseases are dilative cardiomyopathy, and severe mitral
regurgitation. Large atrial surface area is required for the development and propagation of atrial fibrillation. Decreasing atrial surface area has been shown to terminate atrial fibrillation in experimental studies. Dogs and cats with atrial fibrillation show symptoms of congestive heart failure, and have high sympathetic tone, rapid heart rates, variable pulse quality, and an irregular heart rhythm. Cardiac output is decreased due to ineffective atrial contractions, and decreasing ventricular preload.

*ECG of Atrial Flutter*

![ECG of Atrial Flutter](image)


*ECG of Atrial Fibrillation*

![ECG of Atrial Fibrillation](image)

Treatment of Supraventricular Tachyarrhythmias

The treatment of supraventricular tachyarrhythmias is controversial, and at times can be challenging. The following discussion details the most common approach to the management of supraventricular tachyarrhythmias.

Treatment of supraventricular tachycardia

Once an ECG diagnosis of a supraventricular tachyarrhythmia has been made, the following approach to treatment should be used.

1. Attempt to determine if the SVT is short or long using the guidelines under “ECG Features”. Typically, short SVT’s are junctional, whereas long SVT’s are of atrial origin. This may help in determining what we can expect if treatment is successful – that is, the potential to control the arrhythmia, versus controlling the ventricular response rate.
   a. Junctional tachyarrhythmias may be successfully treated with single agent therapy directed at any essential component of the arrhythmia circuit
   b. Atrial tachyarrhythmias may require dual therapy, with one drug used to slow atrioventricular conduction, and the second drug used in an attempt to terminate the atrial tachyarrhythmia itself
2. Vagal maneuver - carotid sinus massage, gag reflex, ocular pressure
   a. If the arrhythmia stops abruptly, then it is probably AV nodal re-entrant tachycardia, orthodromic AV reciprocating tachycardia, or sinus nodal re-entrant tachycardia
   b. Many SVT’s, including those above will not terminate in response to a vagal maneuver.
   c. Complications of vagal maneuvers include ventricular fibrillation and death
   d. Vagal maneuvers are frequently unsuccessful, however a forceful precordial thump to the left apex beat is successful in many cases – at least for a short period of time (mechanical defibrillation)
3. Begin Treatment for acute congestive heart failure (if present)
   a. Provide oxygen supplementation by placement of nasal oxygen catheter, or oxygen cage
   b. Furosemide 2-4 mg/kg IV q 6-8 hrs
   c. Nitroglycerine spray, ointment or patches to reduce preload
   d. Withhold administration of vasodilators such as ACE inhibitors and hydralazine and Nitroprusside until therapy of the SVT has been established, and the need for these agents has been clinically determined
4. Begin specific treatment for the arrhythmia
   a. Diltiazem – Diltiazem is a calcium-channel blocker, and is considered the drug of choice for the management of supraventricular tachyarrhythmias in all patients except those with
advanced congestive heart failure – these patients may be more sensitive to the negative inotropic and mild vasodilator effects of diltiazem. In these patients, a combination of digoxin and diltiazem may provide adequate control of the arrhythmia or ventricular response rate, or alternatively, procainamide may be used in this setting

I. Diltiazem dose: 0.05 - 0.25 mg/kg IV over 5-10 minutes
II. Slows atrioventricular conduction, and prolongs atrioventricular refractoriness
III. Decreases ventricular response to rapid atrial tachyarrhythmias
IV. Terminates junctional tachyarrhythmias (except automatic junctional tachycardia)

b. Propranolol – is a beta-adrenergic blocking agent, which has activity against both beta-1 and beta-2 adrenergic receptors. Beta-blockers have negative chronotropic and inotropic activity, and must be used with caution in patients with severe congestive heart failure. As with diltiazem, beta-blockers may be used in combination with digoxin in the setting of acute congestive heart failure.
   I. 0.02 mg/kg slow IV doses up to a total dose of 0.1 mg/kg
   II. Beta-adrenergic blocking agent

c. Esmolol – is a short-acting beta-adrenergic blocking agent that is specific for beta-1 receptors i.e. cardiac specific
   I. 0.05-0.1 mg/kg boluses q 5 minutes up to a maximum of 0.5 mg/kg.
   II. If initial boluses are effective, Esmolol can be administered as a CRI at a dose of 25-200 micrograms/kg/min
   III. Esmolol is a potent vasodilator and negative inotrope – begin at the lowest effective dose and increase dose to effect

d. Digoxin – Digoxin increases vagal tone to AV junctional tissue. In the presence of severe myocardial failure, digoxin may be the drug of choice for the management of supraventricular tachyarrhythmias
   I. 0.01-0.02 mg/kg. Give ¼ of this dose intravenously q 30 minutes, up to 4 times.
   II. Digoxin is the only positive inotrope that is a negative chronotrope

e. Procainamide -
   I. Class Ia antiarrhythmic
   II. 6-8 mg/kg IV over 3 minutes; 6-20 mg/kg IM
   III. Prolongs refractory periods of atrial and ventricular myocardium, and accessory pathways, and retrograde atrio-ventricular nodal pathways
   IV. May be useful in terminating atrial tachyarrhythmias (once drugs to slow AV nodal conduction have been administered) and atrio-ventricular reciprocating tachycardia
   V. Impairs myocardial contractility to a lesser degree than diltiazem and propranolol, except at higher doses, which increases the margin of safety in the presence of left ventricular and myocardial failure
5. Continue therapy for congestive heart failure
   a. Furosemide at 1-4 mg/kg PO q 8-12 hrs
   b. Begin therapy with an ACE inhibitor such as benazepril 0.25-0.5 mg/kg PO q 24 hrs once
      the patient is stable
   c. Obtain a diagnostic ultrasound of the heart to determine the presence and severity of the
      underlying cardiac pathology
   d. Begin oral therapy for the ongoing management of the SVT as follows

**Chronic therapy of SVT's**

I. Treat congestive heart failure

II. Digoxin
   a. 0.005-0.01 mg/kg PO q 12 hrs (dog)
   b. 0.0312 mg/cat PO q 24-48 hrs (cat)
   c. NB use diltiazem in cats with HCM, NOT digoxin
   d. The ventricular response rate is frequently not sufficiently slowed with digoxin alone,
      necessitating the addition of either diltiazem or atenolol

III. Diltiazem
   a. 0.5-1.5 mg/kg PO q 8 hrs (dog)
   b. 7.5 mg PO q 8 hrs (cat)

IV. Atenolol
   a. 0.25-1.0 mg/kg PO q 12-24 hrs (dog)
   b. 12.5 mg PO q 12-24 hrs (cat)

V. Procainamide (gut and pro-arrhythmic effects preclude chronic use)
   a. 10-20 mg/kg PO q 4-6 hrs (dog)
   b. 3-8 mg/kg PO q 6-8 hrs (cat)

VI. Propafenone, flecainide (class 1c antiarrhythmics); amiodarone, sotalol (class III antiarrhythmics) may
    be useful, however veterinary experience with these drugs is limited

VII. Radio frequency ablation of ectopic focus
Ventricular Tachyarrhythmias

Pathophysiology of Ventricular Tachyarrhythmias

When ventricular myocardium is diseased or damaged, a region of the myocardium may develop the ability to depolarize automatically, thereby establishing a new or ectopic pacemaker. If the ectopic pacemaker site depolarizes at a rate faster than the sinus node, it can dominate the cardiac rhythm. Myocardial damage of any cause can produce re-entry circuits, abnormal automaticity, or triggered activity within the ventricular myocardium. Examples of the types of myocardial injury that may result in ventricular tachyarrhythmias include the following

1. Gastric dilatation-volvulus causes myocardial ischemia through reduction in venous return to the heart, and reduced coronary blood flow
2. Anesthetic agents may induce vagolytic activity, and enhanced sympathetic nervous system stimulation, decreasing myocardial refractoriness and predisposing the myocardium to spontaneous depolarization
3. Traumatic myocarditis following chest trauma results in microvascular hemorrhage within the myocardium, leading to myocardial ischemia
4. Neoplastic infiltration e.g. haemangiosarcoma may result in disruption to microvascular blood flow within the myocardium
5. Dilated Cardiomyopathy with elevated preload decreases coronary blood flow during diastole, resulting in myocardial hypoxia and ischemia
6. Patients in sepsis may develop ventricular dysrrhythmias without any evidence of underlying cardiac disease

Ventricular extra-systoles, ventricular premature contractions, ventricular premature depolarization’s are generated by an ectopic focus located within the ventricular tissue, distal to the bifurcation of the Bundle of His. This results in the wave of depolarization spreading from cell to cell, rather than spreading through the normal fast-conducting tissue. This causes the ECG to show wide bizarre QRS complexes, without an associated P wave, and having a large associated T wave, unless the ectopic focus originates close to the atrioventricular node. Differential diagnoses for ventricular tachyarrhythmias include cardiomegaly, idioventricular rhythm within the bundle branches, left bundle-branch block with supraventricular tachycardia, and ventricular escape complexes
Electrocardiographic Features of Ventricular Tachyarrhythmias

A ventricular premature beat (VPD) is characterized by the premature appearance of a wider-than-normal QRS complex, with an accompanying T wave that is opposite in polarity to the QRS complex, and that is larger than normal. There is no associated P wave. Because most VPD’s do not travel retrograde through the AV node to depolarize the atrium, SA nodal discharge occurs at its normal rate. This results in a "compensatory pause" following a VPD, until the next sinus wave is triggered by the SA node.

If a ventricular premature depolarization originates from the right ventricle, the QRS complexes will be upright in leads I, II, III and aVf, and look identical to those produced by a left bundle branch block. Ventricular premature depolarization’s originating from the left ventricle produces negative orientation of QRS complexes in leads I, II, III and aVf. These complexes may look identical to those generated by a right bundle branch block.

Ventricular tachycardia - is a series of 3 or more ventricular extra-systoles occurring at a high rate; and may be continuous (sustained) or intermittent (paroxysmal)

Aetiology
- Rare in the normal dog or cat
- Common post-trauma, following surgery associated with gastric dilatation-volvulus complex
- Commonly associated with systemic disease e.g. sepsis, splenic neoplasia
- Anesthetic agents e.g. barbiturates
- Primary myocardial disease e.g. dilated cardiomyopathy

Clinical signs
- May be absent - echocardiography studies have shown that patients with ventricular tachycardia may be asymptomatic if they have adequate cardiac output.
- Weakness, syncope
- Sudden death
- NOTE: the sound of the premature beat is frequently softer than normal

Diagnosis
- ECG - see below. ECG diagnosis may be challenging in cases of continuous ventricular tachycardia of septal origin, which tends to produce narrow QRS complexes resembling supraventricular tachycardia

Treatment

- The requirement for treatment of ventricular tachyarrhythmias is dependent on the setting in which VPD’s or ventricular tachycardia are identified.
- The principle reason to treat ventricular tachycardias is to improve hemodynamic stability of the patient (pulse quality, relieve symptoms of poor cardiac output etc.), and to prevent sudden death.
- Dogs with ventricular arrhythmias fall into 2 groups:
  - Dogs with severe underlying cardiac disease - these dogs are at risk of sudden death. The presence of severe underlying cardiac disease is the greatest risk factor for sudden death, regardless of ventricular firing rate, or the form of the ventricular dysrhythmias. Usually, the ventricular dysrhythmia does not result in a significant alteration in hemodynamics. Examples of dogs with ventricular tachycardia and severe underlying heart disease are as follows:
    - Doberman, Boxer with dilated cardiomyopathy
    - Severe sub-aortic stenosis
    - German shepherds with inherited ventricular arrhythmias
    - Cats with hypertrophic cardiomyopathy
  - Dogs with no underlying cardiac disease, but with moderate disease or trauma to other body systems - these dogs are also at risk of sudden death, although the risk is considered low to medium, and can have hemodynamic embarrassment resulting from the ventricular arrhythmia. Treatment should first be directed at the underlying disease process. If this treatment does not result in termination of the arrhythmia, or a reduction in the severity of the arrhythmia, treatment for the arrhythmia should be considered. Treatment should be initiated if the arrhythmia is sustained, at a rapid rate (generally greater than 160 bpm) and if the abnormal rhythm is compromising the cardiovascular system. Note that these patients may also have an accelerated idioventricular rhythm. This is a benign arrhythmia, and usually subsides without the need for treatment.
- Treatment of ventricular dysrhythmias is indicated if one of the following is present:
  - There is hemodynamic compromise to the patient
  - R on T phenomenon, where depolarization of the ventricle occurs during the T wave. This phenomenon can induce ventricular fibrillation
  - Rapid ventricular rate
  - Ventricular fibrillation
- Dogs under anesthesia, or with severe systemic disease - these dogs potentially may develop ventricular fibrillation.
NOTE 1: electrical defibrillation is warranted for ventricular flutter or ventricular fibrillation

NOTE 2: The Lown and Wolf (1971) scale for the identification and classification of ventricular arrhythmias has never been studied in veterinary medicine with respect to predicting death. Ventricular premature depolarization’s in human medicine in this scale relate to patients with coronary arteriosclerosis; the incidence of ventricular premature depolarization’s, their number and form etc. and their relationship to death in other forms of cardiac disease such as those we more commonly see in veterinary medicine, is not known.

**Suggested Treatment Protocol for Ventricular Tachycardia in the Dog**

1. Reduce Sympathetic Tone
   a. Ensure the patient has adequate intravascular volume support – provide and intravenous fluid bolus if indicated, with 10-20 ml/kg LRS given over 10 minutes, or dextran 70 3 ml/kg IV over 10 minutes. Continue fluid support to maintain normal cardiac parameters as needed
   b. Analgesia - provide analgesia if required using opiate analgesia. Avoid morphine if possible as morphine has vagolytic activity and may produce tachyarrhythmias such as ventricular or (more commonly) supraventricular tachycardia
2. Check electrolyte status of the patient – measure sodium, potassium, and chloride +/- magnesium. Correct as necessary. Therapy with antiarrhythmic medications is frequently ineffective if electrolyte balance is not normal
3. Check acid-base status of the patient and correct as indicated using oxygen supplementation, ventilation therapy, intravenous fluid therapy as above etc.
4. Administer lignocaine at 2 mg/kg IV as a bolus, and repeat up to 3 times over 5 minutes if necessary. Following conversion, place the patient on a CRI of lignocaine at 50ug/kg/min. Signs of acute toxicity include seizuring – control with diazepam 0.5-2 mg/kg IV as needed
5. Procainamide 0.2-0.5 micrograms/kg/minute IV OR 2-15 mg/kg IM. Monitor response. Continue IM dose at 4-6 hr intervals if treatment is successful
6. Esmolol 500 ug/kg slow IV bolus may be used if no symptoms of myocardial failure exist
7. Magnesium sulphate 30mg/kg slow IV, following by 30 mg/kg IV infusion over 12-24 hrs if a response is seen
8. Cardioversion – to be performed by trained personnel only – Administer narcotic analgesia with fentanyl, or administer short acting general anesthesia with propofol IV followed by 1 J/kg transthoracic shock, up to 3 times in short succession. Follow conversion with lignocaine.
9. Long-term oral treatment may be attempted with a class 1 anti-arrhythmic agent such as mexiletine (5-8 mg/kg PO q 8 hrs), +/- atenolol (0.3-0.6 mg/kg PO q 12 hrs)
Ventricular Fibrillation

- Ventricular fibrillation is a disorganized chaotic sequence of ventricular depolarization’s involving complete de-synchronization of ventricular electrical activity. Clinically, this produces circulatory collapse and death.

Accelerated Idioventricular Rhythms

- Is a subset of ventricular tachycardia, and are called “slow ventricular tachycardia”. The ventricular rate is typically 70-160 beats per minute (bpm)
  - Occur as a response to bradycardia, and are a type of escape rhythm with a rate of 70-100 bpm
  - Occur with cardiomyopathy (esp. Boxer), shock (GDV), digitalis intoxication, and general anesthesia with a rate of 100-160 bpm
  - Treatment is usually not required

Torsades de Pointes

- Is an intermediate ventricular arrhythmia that arises from prolongation of the Q-T interval. The QRS complexes that arise are rapid in rate, and variable in amplitude due to failure of establishment of a stable re-entry circuit. Treatment involves cessation antiarrhythmic drugs, and administration of magnesium sulphate slowly intravenously
Antiarrhythmic Therapy

Phases of Cardiac Depolarization/Repolarization

Phases of cardiac cell depolarization have the nomenclature of phase 0 through to phase 4. These phases are illustrated in the diagram below.

- During phase 0, voltage-gated fast sodium channels on the cell membrane open, allowing a massive influx of sodium into the intracellular space, that raises intracellular membrane potential, and depolarizes the cell.
- During phase 1, intracellular potassium is lost to the extracellular space, to return the membrane potential to 0mV.
- Phase 2 is characterized by sodium efflux from the cell, and calcium influx into the cell.
- During phase 3, potassium efflux from the cell occurs, returning the cell membrane potential to close to the resting membrane potential.
- Phase 4 is characterized by two events – firstly, sodium: potassium trans-membrane exchange occurs to return intracellular potassium and extracellular sodium concentrations to pre-depolarization levels. Secondly, slow intracellular movement of sodium gradually raises the resting membrane potential to the threshold potential – the point at which the fast sodium channels open, and depolarization occurs again.

Trans-Membrane Ionic Shifts during Cardiac Cell Depolarization
Location of Action of Various Anti-Arrhythmic Drugs

- Calcium Channel Blockers (Diltiazem)
- Lidocaine (lignocaine)
- Flecaïnide
- Quinidine
- β-Blockers
- Amiodarone
- Sotalol

Membrane potential (mV)
- Threshold
- Membrane potential range: -100 to 0
Class 1 Antiarrhythmic Agents

- Selectively block the fast sodium channels, decreasing sodium influx during phase 0, reducing the slope of phase 0. The reduced slope of phase 0 leads to decreased conduction velocity throughout the heart. This slowed conduction velocity can interrupt a re-entrant pattern.

Class 1a
- Markedly depress phase 0 action potential
- Depress conduction of electrical impulses through the heart
- Slow repolarization by lengthening the refractory period
- Widen QRS, prolong Q-T interval
- Examples are Quinidine, procainamide
- Decrease rate of spontaneous depolarization in pacemaker fibers, prolong antegrade refractoriness in the accessory pathway, and can suppress or trigger delayed after-depolarization’s

Class 1b
- Have an affinity for binding with inactivated sodium channels, thereby acting on diseased or ischemic tissue
- Have minimal effects on sinus tissue, sino-atrial and atrio-ventricular nodes, atrial muscle, or inotropic activity
- E.g. Lidocaine, phenytoin, tocainide, mexiletine

Class 1c
- Profoundly depress phase 0
- Profoundly slow conduction velocity
- Have little effect on repolarization rate
- Depress contractility, cardiac output, systemic blood pressure
- Examples - flecainide, propafenone
Class II Antiarrhythmic Agents

- Reverse or nullify electro-physiologic and arrhythmogenic effects of Beta-adrenergic sympathetic stimulation - decrease responsiveness to catecholamine-induced tachycardia and positive inotropism
- Depress the slope of phase 4 depolarization
- Minimally raise the threshold for activation in the sino-atrial and atrio-ventricular nodal cells, depressing automaticity
- Decrease cardiac output, myocardial oxygen demand, and left ventricular work
- Current clinical uses
  - Supraventricular tachycardia, atrial premature contractions - act by slowing atrioventricular conduction and prolonging the refractory period of re-entrant pathways
  - Atrial fibrillation - will not control fibrillation, but may slow the ventricular rate. Beware in dilative cardiomyopathy, as these patients often rely on an elevated heart rate to maintain cardiac output (this is a sympathetically mediated response due to stress and poor cardiac output)
  - Ventricular arrhythmias - usually these are treated with a class I antiarrhythmic (quinidine, lignocaine, procainamide), however, if they arise from increased, or excessive sympathetic tone, they may be they may be treated with beta-adrenergic blockers; OR if they result from ischemia secondary to myocardial hypertrophy (HCM)
  - Cardiomyopathy - in hypertrophic cardiomyopathy, beta-adrenergic blockers slow sympathetically mediated elevation in heart rate, hence improving diastolic filling, reducing myocardial oxygen demand, and control ventricular arrhythmias due to ischemia
  - Cardiomyopathy - in dilative cardiomyopathy, beta-adrenergic blockers may reduce the extent of down-regulation of adrenergic receptors
  - Hypertension, thyrotoxicosis, caffeine, theobromine, theophylline toxicities
  - Digitalis toxicity - blocks digitalis-induced enhancement of central sympathetic tone

Adverse effects include negative inotropism and chronotropism that may exacerbate congestive heart failure; bronchospasm (beta-2 effect) and the induction of severe brady-arrhythmias in patients with AV conduction disturbances (heart block)
Class III Antiarrhythmic Agents

- Specifically prolong the duration of the action potential and refractory period by inhibiting the repolarizing potassium channels (prolonged phase 2 and 3)
- Tissue cannot generate a new action potential until it is repolarized, and these drugs slow or terminate a tachycardia
- Examples include amiodarone, sotalol, bretylum

Class IV Antiarrhythmic Agents

- These drugs are the calcium channel blockers, and act by inhibiting the slow inward calcium channel currents
- Dihydropyridines - act only on vasculature; examples are Amlopidine, nifedipine
- Non-dihydropyridines - have effects on sino-atrial, and atrio-ventricular tissue; examples include verapamil, diltiazem

Why do these drugs work?

- Calcium is required intra-cellularly, to be distributed and released from the sarcoplasmic reticulum during muscle contraction; with the strength on muscle contraction being proportional to the amount of calcium present.
- The depolarizing currents of the SA and AV node are carried by calcium.
- Neuron-hormonal (norepinephrine, histamine, bradykinin) mediated vasoconstriction is calcium channel dependent

- Diltiazem is the most clinically useful Class IV antiarrhythmic drug in veterinary medicine - it reduces the rate of SA node discharge, and slows conduction through the AV node - therefore it is useful in supraventricular tachycardia, atrial fibrillation (slows conduction through the AV node, decreasing the ventricular response rate) and hypertension
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