APPROACH TO ECGS:
THERAPY FOR ACUTE & CHRONIC ARRHYTHMIAS
JEREMY ORR DVM, DVSC, DACVIM (CARDIOLOGY)

ECG BASICS

▸ Definition:
   ▸ A recording of the electrical potential differences which occur between locations of the body surface as a result of depolarization and repolarization of the heart

▸ Utility:
   ▸ Gold standard for diagnosis of arrhythmias
   ▸ Information on rate, conduction, anatomic orientation of the heart and chamber enlargement

CLINICAL INDICATIONS FOR AN ECG

▸ Arrhythmias
▸ Cardiac monitoring (anesthesia, etc.)
▸ Cardiac chamber enlargement
▸ Drug effect/toxicities
▸ Myocardial disease
▸ Pericardial disease
▸ Electrolyte imbalances
▸ Acid-base abnormalities
▸ Syncope
▸ Systemic diseases
ECG ACQUISITION

- Patient positioned in right lateral recumbency
- Leads placed on appropriate limbs (LA, RA, LL, RL + chest leads if available)
- Use alcohol or conduction gel
- Avoid leads coming in contact = artifact
- Avoid panting, stabilize limbs = artifact

ECG WAVES

QRS COMPLEX LABELING
ECG DEFLECTIONS – HOW ARE THEY REGISTERED

FRONTAL PLANE DIAGRAM & LEADS

TRANSVERSE (HORIZONTAL) PLANE LEADS
MEAN ELECTRICAL AXIS (MEA)

- Use frontal plane leads only
- Isoelectric method
- Greatest net deflection method
- Allows determination of the direction of the average wavefront during depolarization
CHAMBER ENLARGEMENT CRITERIA

<table>
<thead>
<tr>
<th>Chambers:</th>
<th>ECG Parameter:</th>
<th>Body Wt</th>
<th>Canine</th>
<th>Feline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Atrium</td>
<td>PII duration</td>
<td>&gt;0.04 s</td>
<td>&gt;0.04 s</td>
<td></td>
</tr>
<tr>
<td>Left Atrium</td>
<td>PII amplitude</td>
<td>&gt;0.4 mv</td>
<td>&gt;0.2 mv</td>
<td></td>
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<td>RII amplitude</td>
<td>&gt;0.4 mg</td>
<td>&gt;0.3 mg</td>
<td></td>
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<tr>
<td>Right Atrium</td>
<td>Pattern of S, SII, &amp; III MEA points to Rt Vent</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Right Atrium</td>
<td>S in V3</td>
<td>&gt;0.7 mv</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Atrium</td>
<td>S in V3 &gt; R in V3</td>
<td>Present</td>
<td></td>
<td></td>
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RHYTHM DIAGNOSIS: SINUS RHYTHM

» What are the criteria for a normal sinus rhythm?
» P wave preceding every QRS complex
» Constant PR interval
» P wave positive in lead II

APPROACH TO ECG INTERPRETATION

» Quality of ECG?
» Artifact can limit our ability to make a diagnosis
» Artifact can be misleading and can sometimes be identified as a “real” arrhythmia
» Can be identified by:
  » It occurs between end of the QRS and first half of the T wave of that QRS
  » It fails to alter the underlying rhythm
  » It fails to be followed by a depolarization (T) wave
  » If there is a depolarization wave there must be a repolarization wave
APPROACH TO ECG INTERPRETATION

- Identify the paper speed
  - Common speeds: 50 mm/sec, 25 mm/sec
  - 50 mm/sec can help with morphology of complexes as they are “more spread out”
- Note the lead markings
  - More than one lead is ideal if possible for rhythm analysis
- Determine the heart rate
  - At 50 mm/sec each small box is 0.02 sec, at 25 mm/sec each is 0.04 sec
  - Pen Rule
    - At 50 mm/sec = 3 seconds
    - At 25 mm/sec = 6 seconds
APPROACH TO ECG INTERPRETATION

Once heart rate is known, classify rhythm as:
- Tachyarrhythmia
- Bradyarrhythmia
- Normal rate with "weird" stuff

By separating rhythms based on rate, it will help guide you to the proper diagnosis and therefore proper management of the arrhythmia

Normal heart rates:
- Dog: 60-180 bpm
- Cat: 140-240 bpm

APPROACH TO ECG INTERPRETATION

After determining heart rate, scan the whole strip
- Assess R-R intervals - regular? any pattern?
- Assess P-P intervals - regular? any pattern?
- Is there a P for every QRS-T?
- Is there a QRS-T for each P? Are they associated (related)?
- Is there an intermittent AV conduction disturbance present?
- If premature complexes are present, are they more similar in morphology to the sinus complexes? Is there an associated premature P wave?

APPROACH TO ECG INTERPRETATION

Determine the MEA
- Utility: identify RV enlargement, bundle branch block
- I prefer the greatest net deflection method (=lead with the tallest R wave)
- Measure complexes
  - May suggest underlying chamber enlargement
  - More sensitive diagnostic methods exist
    - Thoracic radiographs, echocardiography
BRIEF STATEMENTS REGARDING ARRHYTHMIAS

- Not all arrhythmias occur in patients with primary cardiac disease
- Not all arrhythmias require therapy
- Treatments considered for patients with hemodynamic compromise (lethargy, weakness, syncope, hypotension)
- Anti-arrhythmics can be pro-arrhythmic
- An improvement in the ECG rhythm does not always equate to clinical improvement or reducing the risk for sudden death

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<tr>
<th>RHYTHM</th>
<th>HEART RATE</th>
<th>P WAVE</th>
<th>ASSOCIATED DISORDERS</th>
</tr>
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<tbody>
<tr>
<td>Slow</td>
<td>Yes</td>
<td>No</td>
<td>Sinus Bradycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2nd Degree Heart Block</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td></td>
<td>Atrioventricular Rhythm</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hyperkalemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Persistent Atrial Standstill</td>
</tr>
<tr>
<td>Regular</td>
<td>No</td>
<td>Yes</td>
<td>Normal Sinus Rhythm</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Atrial Flutter</td>
</tr>
<tr>
<td>Fast</td>
<td>Yes</td>
<td>No</td>
<td>Sinus Tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Supraventricular Tachycardia</td>
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BRADYARRHYTHMIAS

- Defined as a heart rhythm with a rate below the normal range for that particular species
- Most involve either the sinoatrial node or atrioventricular node
- Determination of origin of the bradyarrhythmia will guide if therapeutic intervention is required

SINUS BRADYCARDIA

- Most often a physiologic response to systemic disturbance
- Increased vagal tone due to primary respiratory, GI, CNS disease
  - Hypothermia, hypothyroidism, hypoglycemia
- Drugs
- Electrolyte abnormalities

SINUS ARREST/SINUS BLOCK

- Defined as an intermittent absence of P waves
- Pauses may be terminated by an escape beat
  - QRS without a P that often has a wide and bizarre shape that follows a pause - don't mistake for a VPC
- May be an early sign of SA node disease (sick sinus syndrome)
**ATRIAL STANDSTILL**

- Defined as an absence of P waves with a normal to slowed ventricular rate
- Junctional or ventricular escape beats are often noted
- Temporary: due to hyperkalemia
- Permanent: due to an atrial myopathy where atrial myocytes are replaced (including conduction tissue)

**SICK SINUS SYNDROME**

- A 'catch-all' term used to describe ECGs with the following features:
  - Sinus bradycardia
  - Periods of sinus arrest
  - AV block
  - Bursts of SVT
- Most common in older small breed dogs - Westies, Schnauzers, Cocker Spaniels
- Affected dogs present for syncope and exercise intolerance
SICK SINUS SYNDROME

- Diagnosis can be aided with:
  - Holter monitor - determine etiology of syncope
  - Atropine response test: 0.04 mg/kg IV/IM
- Treatment:
  - Definitive treatment is pacemaker therapy
  - Palliative therapy can be considered - response variable and short-lived
    - Terbutaline 2.5 mg/dog q. 12 hrs
    - Theophylline 10-20 mg/kg q. 12 hrs
    - Anti-cholinergics: Hyoscyamine 0.003-0.006 mg/kg PO q. 8 hrs

AV NODAL ORIGIN

- 1st degree AV block
  - Defined as a prolonged PR interval (> 0.13 seconds in the dog, > 0.09 seconds in the cat)
  - PR interval represents conduction time from SA node through AV node, Bundle of His, bundle branches and Purkinje fibers
  - Patients are asymptomatic
  - Typically associated with increased vagal tone
  - Can be associated with AV fibrosis due to aging
  - Can be early manifestation of hyperkalemia
1ST DEGREE AV BLOCK

- No treatment indicated but ensure no electrolyte abnormalities
- If no identifiable cause for the AV block is noted (enhanced vagal tone, drug effect) may wish to periodically re-evaluate to ensure no progression to higher grades of AV block

AV NODE ARRHYTHMIAS

- 2nd degree AV block - Mobitz type I
  - Presence of P waves not followed by QRS complexes
  - QRS morphology normal
  - PR interval progressively lengthens prior to block and then shortens immediately after block
  - Patients are usually asymptomatic
  - Generally associated with high vagal tone; common in patients during GA/sedation
  - Resolves with atropine administration
AV NODAL ARRHYTHMIAS

▸ 2nd degree AV block type II
  ▸ P waves not followed by QRS complexes
  ▸ Can have multiple P waves not followed by QRS (→ high grade AV block)
  ▸ PR interval is constant
  ▸ QRS complexes may be normal or wide
  ▸ Concern that may progress to 3rd degree AV block
  ▸ Consider pacemaker therapy if high grade block and patient is symptomatic

AV NODAL ARRHYTHMIAS

▸ 3rd degree AV block
  ▸ Complete AV dissociation
  ▸ P waves unassociated with QRS with faster atrial rate (P-P)
  ▸ Slow ventricular escape rhythm
  ▸ Cause unknown - fibrosis, inflammatory, Lyme disease, Chagas disease, neoplasia, congenital form in Pugs
  ▸ Patients are symptomatic - syncope, weakness, lethargy
  ▸ Cats is often incidental as escape rate does not cause observable signs
3RD DEGREE AV BLOCK

- Ventricular rate typically < 60 bpm in the dog, 100-130 bpm in the cat
- Rate dependent on where in the conduction system the escape rhythm originates
  - Peri-AV node, bundle branches, Purkinje fibers

BUNDLE BRANCH BLOCK

- Defined as delayed conduction through the bundle branches
- Right bundle branch block is common and considered non-pathologic
  - Can be noted with increased vagal tone, anesthesia, following right heart catheterization or may be incidental finding
- Left bundle branch block is uncommon and concern is that it may represent a precursor to higher grades of heart block (3rd degree AV block)
TACHYARRHYTHMIAS

- Defined as a heart rhythm with a rate above the normal range for that particular species
- Originates from either the SA node, supraventricular region (atria, AV node/junction) or the ventricle
- Determining origin can help understand the cause of the arrhythmia and the need for intervention

SINUS TACHYCARDIA

- Typically a secondary response to some systemic disturbance
  - Hypotension, pain, sepsis, fever, hyperthyroidism, cardiac tamponade, drug effects
  - Typically not associated with primary cardiac disease
  - However can be seen in patients with CHF
- Treatment is aimed at the underlying cause
SUPRAVENTRICULAR PREMATURE CONTRACTIONS (SVPC)

- Originate from an ectopic atrial focus
- Conducts via the same ventricular conduction pathway as sinus beats therefore similar looking QRS
- Premature P wave may or may not be obvious
- Interrupts the normal P-P and R-R intervals
- Typically occurs secondary to atrial stretch/disease but can be seen in cardiac neoplasia and non-cardiac causes

SUPRAVENTRICULAR TACHYCARDIA

- 4 or more SVPCs in a row with a rate that generally is >150 bpm
- Abrupt onset and offset is common
  - How differs from a sinus tachycardia
- Often regular
- QRS morphology similar to sinus beats
- Premature P waves may be buried

ATRIAL FIBRILLATION

- Most common supraventricular arrhythmia
- Numerous small re-entrant pathways
- Rapid and disorganized atrial depolarization
- Irregular rhythm, fast, absence of P waves, QRS morphology is supraventricular in origin
ATRIAL FIBRILLATION

▸ Primary
  ▸ Also known as “lone” atrial fibrillation
  ▸ Most often noted in giant breed dogs and may not require treatment as rates are generally ‘normal’

▸ Secondary
  ▸ Typically due to severe cardiac disease (atrial enlargement)
  ▸ Can be vagally mediated
  ▸ Noted following sedation, particularly with opioids

ATRIAL FLUTTER

▸ Rare supraventricular arrhythmia
▸ Due to a large re-entrant pathway
  ▸ Typically around the tricuspid valve annulus
  ▸ Most often noted in dogs with severe tricuspid valve dysplasia
  ▸ “Flutter” waves have a saw-tooth appearance
VENTRICULAR ARRHYTHMIAS

- Represent ectopic impulses which originate from the ventricular myocardium
- SA node continues to fire independent of these ventricular ectopic impulses = AV dissociation
- Depolarizations move cell to cell, not along conduction system
- Complexes are wide, bizarre and different than the sinus complexes due to conduction delay

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<th>Ventricular Premature Beats (VPBs)</th>
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<td>The absence of a premature P does not discriminate between an SVPB and a VPC</td>
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VENTRICULAR TACHYCARDIA

- 4 or more VPCs in a row
- Can be short in duration (paroxysmal) or sustained
- Can occur suddenly and abruptly terminate
- AV dissociation is a feature
- Can precede ventricular fibrillation = terminal rhythm
### IS IT VENTRICULAR OR SUPRAVENTRICULAR IN ORIGIN?

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QRS alternans can occur - beat to beat oscillation in the amplitude of the QRS.

### "SLOW VT"

- Also referred to as an accelerated idioventricular rhythm
- Characterized by a regular run of VPCs with a rate similar to that of the normal sinus rate
- Common in hospitalized patients, post GDV, post splenectomy, etc.

### ELECTRICAL ALTERNANS

![ECG Image]
SUMMARY

- ECG indicated for patients with arrhythmia on auscultation, patients with weakness, syncope, exercise intolerance
- Not the most sensitive tool for assessing for specific cardiac chamber enlargement
- Although there are many possible cardiac arrhythmias, there are only a few common clinical ones

QUESTIONS?

APPROACH TO ECGS PART 2:

THERAPY FOR ACUTE & CHRONIC ARRHYTHMIAS
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COMMON VETERINARY ARRHYTHMIAS REQUIRING TREATMENT

- Ventricular arrhythmias
- Supraventricular tachycardias
- Bradyarrhythmias

VENTRICULAR ARRHYTHMIAS

- Second most common arrhythmia after sinus tachycardia noted in veterinary medicine
- May be a marker for underlying cardiac disease
- Can occur in patients with normal hearts
- Ventricular tachycardia is a dangerous, potentially life threatening arrhythmia due to risk for development of ventricular fibrillation
### Supraventricular Premature Beats (SVPBs) vs Ventricular Premature Beats (VPBs)

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CAUSES OF VENTRICULAR ARRHYTHMIAS

- Primary cardiac disease
  - Acquired, congenital disease
    - Common in CHF patients
  - Myocardial infarction
  - Myocarditis
  - Neoplasia
  - Pericarditis
  - Inherited ventricular arrhythmias
    - Young German Shepherd dogs

- Non-cardiac causes:
  - Hypoxia, anemia, uremia, sepsis, pancreatitis, GDV, splenic disease, DIC, endocrinopathies, liver disease, GI disease, CNS disease, electrolyte derangements, cardiotoxic drugs

HOW MANY VPCS ARE TOO MANY?

- Breed dependent
- Various studies have revealed that normal canines can have VPCs but they are infrequent
- 9-24 VPCs / 24 hr period
- In Boxers: < 91 VPCs / 24 hr period
VPCS AND BREEDS

▸ The presence of VPCs in certain breeds should rise the index of suspicion for underlying cardiac disease
  ▸ Dilated cardiomyopathy
    ▸ Dobermans, Great Danes, Irish Wolfhounds
  ▸ Arrhythmogenic Right Ventricular Cardiomyopathy
    ▸ Boxers, Bulldogs
  ▸ Inherited Tachyarrhythmias
    ▸ Young German Shepherd dogs

VPCS AND CATS

▸ Generally the presence of VPCs in cats occurs secondary to myocardial disease
  ▸ Hypertrophic, restrictive, unclassified cardiomyopathy

102/106 cats with VPCs had myocardial disease (96%) which contrasts the frequency of primary cardiac disease as the cause for VPCs in canine patients (estimated at about 69%)

VENTRICULAR ARRHYTHMIAS – WHY TREAT?

▸ Single VPCs generally do not result in hemodynamic instability and patient is often asymptomatic

▸ Runs of VPCs (= ventricular tachycardia) is generally considered a serious and life-threatening arrhythmia that can result in hypotension, collapse, syncope and a risk for ventricular instability
  ▸ Ventricular fibrillation = terminal rhythm
WHEN TO TREAT?

- Patients who are symptomatic for their arrhythmia
- Patients with structural cardiac disease
  - DCM, severe MR, HCM, ARVC
- Hemodynamic instability
- Hypotension, collapse, syncope
- Patients at risk for ventricular fibrillation
  - High ventricular rates (>180 bpm), presence of couplets and/or triplets, multiform VPCs, R on T phenomenon

DIAGNOSTIC EVALUATION FOR PATIENTS WITH VPCS

- When VPCs are noted in a patient, minimum database diagnostics generally include:
  - CBC/chemistry/UA/T4
  - Blood Pressure
  - Thoracic radiographs
    - Assess for cardiomegaly, CHF, neoplasia
  - Abdominal & cardiac ultrasound
  - 24 hour ambulatory ECG (Holter monitor)
VENTRICULAR TACHYCARDIA - ACUTE MANAGEMENT

- For unstable patients, treatment should be initiated immediately to stabilize the patient.
- Diagnostics can be considered once the patient is stable.
- If possible, evaluation of electrolytes is helpful.
- Hypokalemia and hypomagnesemia can contribute to the development of VPCs and can alter the efficacy of anti-arrhythmic therapy.

ACUTE ER TREATMENT

- Lidocaine
  - First line treatment
  - Class IB anti-arrhythmic
    - Affects conduction velocity & refractory period
  - Rapid onset of action, short duration of action
  - Has more effect on damaged myocardial cells
  - Can hyperpolarize partially depolarized cells
  - Produces little effect on sinus rate, AV conduction

LIDOCAINE & POTASSIUM

- Effects of lidocaine depend on extra-cellular potassium concentrations
  - Hyperkalemia enhances effects
  - Hypokalemia diminishes effectiveness
- Goal K concentration: 4-5 mmol/L
- Hypokalemia should be immediately addressed in a patient with ventricular tachycardia
**ACUTE TREATMENT – LIDOCAINE**

- Loading dose of 2 mg/kg (dogs) - can be repeated and dose titrated based upon ECG findings
- Dose > 8 mg/kg (cumulative) generally results in CNS signs
- CRI dose in dogs: 50-80 mcg/kg/min
- Cats are sensitive to the CNS effects of lidocaine
  - Dose is 0.25-1 mg/kg IV SLOWLY
  - CRI dose is 10-20 mcg/kg/min

**OTHER TREATMENT OPTIONS FOR VENTRICULAR TACHYCARDIA**

- Procainamide
  - Generally considered second line of treatment, especially if lidocaine fails
  - Class IA anti-arrhythmic with both supraventricular and ventricular effects
  - Extracellular potassium concentrations important
  - Mild cardiac depression, vasodilation occurs when given IV

**PROCAINAMIDE**

- Dosing:
  - Dogs: 6-8 mg/kg IV over 5-10 minutes; CRI dose is 25-40 mcg/kg/min
  - Cats: 1-2 mg/kg IV over 5-10 minutes; CRI dose is 10-20 mcg/kg/min
- Can be used as first line treatment if unsure if tachyarrhythmia represents a ventricular or supraventricular tachycardia
HYPOMAGNESEMIA

- If hypomagnesemia is noted or ventricular arrhythmia is refractory, can consider magnesium infusion
- Dosed at 30 mg/kg diluted 1:1 with saline given IV over 5-10 minutes

BETA BLOCKERS

- Prefer IV administration over oral in the acute setting due to rapid onset of action
- Exhibit more potent negative inotropic and chronotropic effects compared to the class I anti-arrhythmics
- Caution in patients with fulminant CHF or systolic dysfunction
- Contraindicated in patients with bradyarrhythmias, AV block

BETA BLOCKERS

- Effect on heart rate and rhythm is by inhibiting myocardial effects of the sympathetic nervous system
- Can decrease automaticity
- Can restore a more homogenous pattern of repolarization
- Useful in both supraventricular and ventricular tachycardias
**PROPRANOLOL**

- Non-selective beta blocker, available in IV
- Longer duration of effect
- Dose: 0.02-0.1 mg/kg IV slowly
- Not ideal option given longer duration of effect, lack of beta-selective effects
  - Caution in patients with primary respiratory disease

**ESMOLOL**

- Ultra short acting beta-1 blocker (selective)
- Short half life (2 minutes) = CRI administration
- Can be given as a loading dose for conversion or can be placed on CRI without loading dose
  - Avoid loading dose in patients with systolic dysfunction and/or CHF
- Canine: loading dose 200-500 mcg/kg IV over 1-2 minutes; CRI is 25-200 mcg/kg/min
- Feline: loading dose 200-500 mcg/kg IV over 1-2 minutes; CRI is 25-200 mcg/kg/min

**SOTALOL IN THE ACUTE ER SETTING**

- Oral class III anti-arrhythmic with beta blocker and potassium blocker effects
- Can be initiated in patients receiving an IV class I anti-arrhythmic for combination effect when no other IV options are available
- Often initiated while in hospital to help transition to chronic oral anti-arrhythmic therapy
- Generally well tolerated in veterinary species but dose may require adjustment in those with systolic dysfunction/CHF
  - Start low dose in these patients (0.5 mg/kg PO q. 12 hrs)
"NEWER" OPTIONS FOR REFRACTORY VENTRICULAR TACHYCARDIA

- Amiodarone
  - Class III anti-arrhythmic
  - Limited use to date in veterinary medicine
  - Side effects are common
  - Contraindicated in patients with AV block, bradyarrhythmias
- Nexterone: 2-5 mg/kg IV over 30-60 minutes while monitoring BP and heart rhythm closely

WHAT IF A PATIENT FAILS ANTI-ARRHYTHMIC THERAPY

- If patient with ventricular tachycardia is refractory to appropriate anti-arrhythmic therapy (and rhythm diagnosis is correct) with normal potassium and magnesium concentrations, then:
  - Electrical cardioversion - general anesthesia required

CHRONIC MANAGEMENT OF VENTRICULAR ARRHYTHMIAS

- Sotalol
  - Canine dose: 0.5-2 mg/kg PO q. 12 hrs; feline dose: 10 mg PO q. 12 hrs
- Mexiletine
  - Class IB anti-arrhythmic; Canine dose: 4-8 mg/kg PO q. 8-12 hrs
  - GI effects are common
- Atenolol
  - Class II anti-arrhythmic (beta blocker)
  - Caution in patients with systolic dysfunction and/or CHF
  - Doses: 0.25-2 mg/kg PO q. 12-24 hrs
- Consider combination approach (class I and class II anti-arrhythmic) particularly for those with significant arrhythmias
CHRONIC MANAGEMENT OF VENTRICULAR ARRHYTHMIAS

- Amiodarone
  - Class III anti-arrhythmic
  - Side effects are common: hepatic, pulmonary fibrosis, thyrotoxicity, GI signs, neutropenia, thrombocytopenia
  - Efficacy against ventricular and supraventricular arrhythmias
  - Dose: 10 mg/kg PO q. 24 hrs x 7 days, then 5 mg/kg PO q. 24 hrs thereafter
  - Recommend CBC/chemistry/T4 routinely in patients receiving amiodarone

CHRONIC MANAGEMENT OF VENTRICULAR ARRHYTHMIAS

- Omega fatty acids
  - Anti-arrhythmic properties have been noted in people and Boxer dogs
  - Dose noted in study was 780 mg/day EPA and 497 mg/day DHA

CHRONIC MANAGEMENT OF VENTRICULAR ARRHYTHMIAS

- Monitoring:
  - In hospital ECG for ~5 minutes is specific for evidence of a tachyarrhythmia but is insensitive for monitoring response to therapy
  - Arrhythmias are intermittent
  - Ideal to perform a Holter monitor 2-3 weeks after starting oral therapy
  - Goal is > 85% reduction in VPC number and reduction in complexity
SUPRAVENTRICULAR TACHYCARDIA (SVT)

▸ Although a serious arrhythmia, generally not considered life threatening

▸ Some patients who present with sustained SVT will be minimally symptomatic

▸ Alternatively may present for signs of hypoperfusion:
  ▸ GI signs
  ▸ Weakness, lethargy
  ▸ Collapse

SVT CAUSES

▸ Generally identified in patients with pre-existing structural cardiac disease

▸ Chronic valvular degeneration, dilated cardiomyopathy, cardiac neoplasia

▸ Can be noted in young animals due to the presence of “accessory pathways”
  ▸ Similar to Wolff-Parkinson-White Syndrome in humans

▸ Can be associated with non-cardiac causes as well
SVT

▸ Results in reduction in cardiac output
▸ Identifying a premature P wave (AV association) during tachycardia is diagnostic for SVT
▸ Ocular pressure, carotid sinus massage, Valsalva maneuver (difficult in our patients!)
▸ Phenylephrine
   ▸ Alpha agonist which results in reflex bradycardia
   ▸ 0.04 mg/kg IV for both dogs and cats

SVT TREATMENT

▸ Why treat?
   ▸ Determined by heart rate, clinical signs, blood pressure
   ▸ Pre-existing heart disease/CHF

SVT TREATMENT

▸ Acute treatment
   ▸ Diltiazem is first line therapy
     ▹ Class IV anti-arrhythmic
     ▹ Dosed at 0.1-0.25 mg/kg IV slowly; CRI 1-5 mcg/kg/min
   ▸ Could consider procainamide as dosed for ventricular arrhythmias
**SVT TREATMENT**

- If refractory to diltiazem and procainamide, consider IV beta blocker
  - Esmolol preferred
  - As dosed for ventricular arrhythmias
  - Caution in patients with systolic dysfunction and/or CHF

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**SVT CHRONIC MANAGEMENT**

- Oral diltiazem therapy 1-3 mg/kg PO q. 8 hrs
  - In dogs, could consider diltiazem XR (Dilacor) dosed at 2-4 mg/kg PO q. 12 hrs
  - Digoxin 0.003-0.005 mg/kg PO q. 12 hrs
  - Combination of diltiazem and digoxin has been shown to be most efficacious for rate control in patients with atrial fibrillation

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**SVT CHRONIC MANAGEMENT CONTINUED**

- Oral sotalol therapy 0.5-2 mg/kg PO q. 12 hrs
- Atenolol therapy 0.5-2 mg/kg PO q. 12-24 hrs
- Amiodarone therapy 10 mg/kg PO q. 24 hrs x 7 days then 5 mg/kg PO q. 24 hrs
- Holter monitoring for anti-arrhythmic efficacy
SVT CHRONIC MANAGEMENT

- In some patients, combination therapy is often required
- Caution when combining beta blocker and calcium channel blocker
- Due to combination negative inotropic and chronotropic effects
- Some patients can be “cured” of their SVT with radiofrequency catheter ablation procedure
- Cardiologist in Ohio: Dr. Kathy Wright

ATRIAL FIBRILLATION

- Electrical cardioversion is an option for rhythm restoration
  - Useful in patients with primary atrial fibrillation
  - Useful in patients with secondary atrial fibrillation who are failing medical management
- Studies have shown that people feel better in sinus rhythm and likely the same for animals
- Heart failure can be easier to manage when in sinus rhythm

3RD DEGREE AV BLOCK

- Only option is pacemaker implantation as little response with medical therapy
- Often an emergency due to slow rate, risk for sudden death
- A thorough cardiac evaluation should be performed prior to pacemaker to rule out neoplasia or infectious disease
**SICK SINUS SYNDROME**

- Rarely results in sudden death but can become critical if patient is collapsing frequently or requires anesthesia
- Definitive treatment is pacemaker therapy
- Palliative therapy can be considered - response variable and short-lived
  - Terbutaline 2.5 mg/dog PO q. 12 hrs
  - Theophylline 10-20 mg/kg PO q. 12 hrs
  - Anti-cholinergics: Hyoscyamine 0.003-0.006 mg/kg PO q. 8 hrs

**SICK SINUS SYNDROME**

- Some affected dogs will continue to collapse post pacemaker placement
  - Due to SVT
- Therapies to treat SVT are generally only instituted once pacemaker is placed as these treatments can worsen the bradyarrhythmias associated with sick sinus syndrome

**PROGNOSIS FOR PATIENTS WITH PACEMAKER THERAPY**

- Indications: sick sinus syndrome, AV block
- Prognosis is generally good
  - One study revealed 3 year survival of 65%, 5 year survival of 39%
- Complications are infrequent
  - Lead thrombosis has been reported
- Pacemaker cost: approximately $3700-4500
- In cats: Epicardial placement
TIPS FOR MANAGING PATIENTS WITH PACEMAKERS

▸ Rechecks every 6 months are generally advised with a cardiologist for pacemaker interrogation
▸ When placed transvenously, patient should NEVER wear a neck lead
  ▸ Blood should NEVER be drawn from a jugular vein
▸ Caution when using cautery
▸ Should have heart rate adjusted for any anesthetic procedures
  ▸ Antibiotics for any “dirty” procedure or wound

SUMMARY

▸ Treatment may not always been indicated for every arrhythmia, however a thorough assessment for underlying causes is indicated
▸ If patient is not responding to anti-arrhythmics ensure rhythm diagnosis is correct
▸ Anti-arrhythmics can be pro-arrhythmic
▸ Ability to reduce risk for sudden death is controversial with anti-arrhythmics
▸ Rapid ventricular tachycardia is a life threatening arrhythmia and should be treated immediately

QUESTIONS?