RECOGNITION OF COMMON ARRHYTHMIAS – THEIR CAUSES AND TREATMENT OPTIONS

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OBJECTIVES FOR PRESENTATION

- Patient Examination
- Specific Diagnostics
- Electrocardiography Setup
- Electrocardiography Basics
- Electrophysiology Basics
- Identification and Treatment of
  - Sinus Dysrhythmias
  - Atrial Dysrhythmias
  - AV Nodal Dysrhythmias
  - Ventricular Dysrhythmias
  - Miscellaneous

PATIENT EXAMINATION

Identifying the at risk population

- Lung Sounds
  - Crackles, wheezes
- Abdominal Palpation
  - GDV, Spleenic Mass
- Jugular Pulses/Distension
- Femoral Pulses
  - Weak vs. Bounding
- Heart Sounds
  - Murmur
  - Gallop
  - Arrhythmia
SPECIFIC DIAGNOSTICS

- Complete Blood Count
  - Anemia
- Biochemistry Panel (Electrolytes)
  - K⁺
  - Ca²⁺
- Thoracic Radiographs
  - Cardiomegaly
  - Pulmonary edema
- Blood Pressure
  - Hypotension
- Echocardiogram

SPECIFIC DIAGNOSTICS

- Biochemistry Panel (Electrolytes)
  - K⁺
  - Ca²⁺

ELECTROCARDIOGRAM SETUP

- Proper Positioning
  - Right lateral recumbency
**ELECTROCARDIOGRAM SETUP**

- Lead Placement
  - Black – left front limb
  - White – right front limb
  - Red – left rear limb
  - Green – right rear limb

**ELECTROCARDIOGRAPHY BASICS**

- ECG Alphabet

**ELECTROCARDIOGRAPHY BASICS**

- Heart Rate
  - Average
  
  \[
  \text{Heart rate} = 110 \text{ bpm}
  \]

- 6 seconds

- 25.0 mm/s
Electrocardiography Basics

- Heart Rate
  - Instantaneous
    - 25 mm/s = 1500 / # small boxes
    - 50 mm/s = 3000 / # small boxes
  Heart rate = 1500/13 = 115 bpm

Electrocardiography Basics

- Sinus
  - Positive P wave Lead II
  - HR = 45 – 220 bpm
- SA Nodal Impulse
  - Silent on surface ECG

Electrophysiology Basics

- Action Potential
  - Phase 0 – Na⁺ enters
  - Phase 1 – Na⁺ Ch close
  - Phase II – Ca²⁺ enters
  - Phase III – K⁺ efflux
  - Phase IV – Na⁺/Ca²⁺ enter
### ELECTROCARDIOGRAPHY BASICS

<table>
<thead>
<tr>
<th>Class</th>
<th>Mechanism</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Na channel blocker</td>
<td>Lidocaine, Mexilitine, Procainamide</td>
</tr>
<tr>
<td>II</td>
<td>Beta blocker</td>
<td>Atenolol, Esmolol</td>
</tr>
<tr>
<td>III</td>
<td>K channel blocker</td>
<td>Amiodarone, Sotalol</td>
</tr>
<tr>
<td>IV</td>
<td>Ca channel blocker</td>
<td>Diltiazem</td>
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</tbody>
</table>

### SINUS DYSRHYTHMIAS

- **Sinus Bradycardia**
  - Heart Rate = 23 bpm
  - Generally the result of a physiologic predominance of the parasympathetic nervous system.
    - Opioid drugs
    - Gastrointestinal disease
    - Respiratory disease
    - Increased intracranial pressure
    - Asphyxiation (closed pop-off valve)
    - Alpha-2 agonists
  - Therapy warranted if patient is hemodynamically compromised
SINUS DYSRHYTHMIAS

- Sinus Bradycardia
  - Therapy
    - Remove underlying imbalance whenever possible
    - Anticholinergic drugs
      - Atropine (0.01 – 0.04 mg/kg IV)
      - Glycopyrrolate (0.005 – 0.01 mg/kg IV)
    - Temporary cardiac pacing
    - Alpha-2 agonists (Medetomidine, Dexmedetomidine, Xylazine)
      - Atipamezole (same volume as alpha-2 used IM, IV)
      - Yohimbine (0.11 mg/kg IV, 0.25-0.5 mg/kg IM, SC)

- Sinus Arrhythmia
  - Normal physiologic response in dogs associated with changes in intrathoracic pressure (HR with inspiration & with expiration).
  - Physiologic response is present in cats, but adrenergic response in clinic setting makes this a rare finding.
  - Characterized by varying R-R intervals in a classic regularly irregular rhythm pattern
  - No therapy is generally required for this rhythm
Sinus Dysrhythmias

- Sinus Tachycardia
  - Heart Rate = 200 bpm

Sinus Dysrhythmias

- Sinus Tachycardia
  - Generally the result of a physiologic predominance of the sympathetic nervous system
  - Pain
  - Hypovolemia
  - Anemia
  - Congestive heart failure
  - Iatrogenic
  - Pheochromocytoma

- Sinus tachycardia may be a compensatory mechanism, therefore therapy should be carefully considered in all cases
- Cardiac Output = Heart Rate X Stroke Volume

Sinus Dysrhythmias

- Sinus Tachycardia
  - Therapy
    - Remove underlying imbalance whenever possible
    - Beta-blocking drugs
      - Esmolol (0.05-0.1 mg/kg IV slowly, then CRI 50-200 mcg/kg/min)
        - Myocardial depression, decreased cardiac output, bradycardia
    - Calcium Channel blocking drugs
      - Diltiazem (0.25 mg/kg IV slowly)
        - Hypotension, myocardial depression, bradycardia, AV block
SINUS DYSRHYTHMIAS

- Sick Sinus Syndrome

- Complex arrhythmia characterized by sinus node dysfunction with or without AV nodal involvement
- Classically described as “Brady-Tachy Syndrome”
- Usually requires a Holter monitor for definitive diagnosis
- High risk breeds
  - Miniature Schnauzer
  - Cocker Spaniel
  - West Highland White Terriers
  - Dachshund

- Therapy
  - Permanent or temporary cardiac pacing usually required
  - Anticholinergic medications
    - Unreliable response
    - May exacerbate tachyarrhythmias
Atrial (Supraventricular) Dysrhythmias

- Atrial Tachycardia (Supraventricular)
  
  Instantaneous HR = 300 bpm
  Abrupt stop with overdrive suppression

- Atrial Tachycardia (Supraventricular)
  Abrupt start & HR = 215 bpm

Atrial (Supraventricular) Dysrhythmias

- Atrial Tachycardia (Supraventricular)
  Intermittent or continuous impulses originating from the atrial myocardium or AV node often associated with structural heart disease
  - Mechanisms include reentrant circuits or spontaneous automaticity of ectopic foci
  - Examples:
    - SA nodal reentrant tachycardia
    - Automatic atrial tachycardia
    - Atrial fibrillation
    - Atrial flutter
    - AV nodal reentrant tachycardia
    - Circus movement tachycardia
Atrial (Supraventricular) Dysrhythmias

- Atrial Tachycardia (Supraventricular)
  - Therapy
    - Vagal maneuver
    - Carotid massage
    - Ocular pressure
    - Class Ia - Procainamide (15-20 mg/kg IV, slowly 15 minutes)
    - Hypotension
    - Diltiazem IV
    - Esmolol IV
    - Therapy is often unrewarding

- Atrial Fibrillation
  - Acute Onset
    - Diltiazem IV
    - Procainamide IV
    - Lidocaine IV
    - Amiodarone IV
  - Chronic (Oral)
    - Amiodarone
    - Beta Blockers
    - Calcium Channel Blockers
    - Digoxin

Atrial fibrillation is almost always associated with structural heart disease. Extreme caution should always be taken when attempting to convert or treat this rhythm.
ATRIAL (SUPRAVENTRICULAR) DYSRHYTHMIAS

• Atrial Flutter
  - Therapy
    - Acute Onset
      - Diltiazem IV
      - Procainamide IV
    - Chronic (Oral)
      - Amiodarone
      - Beta Blockers
      - Calcium Channel Blockers
      - Digoxin

ATRIAL (SUPRAVENTRICULAR) DYSRHYTHMIAS

• Atrial Flutter
  - Therapy
    - Acute Onset
      - Diltiazem IV
      - Procainamide IV
    - Chronic (Oral)
      - Amiodarone
      - Beta Blockers
      - Calcium Channel Blockers
      - Digoxin

AV NODAL DYSRHYTHMIAS

• First Degree AV Block

P-R = 0.2 sec
AV NODAL DYSRHYTHMIAS

- Second Degree AV Block
  - Mobitz Type 1

Progressive prolongation of the P-R interval
- P waves with no corresponding conducted QRS complex

P-R = .14 sec
P-R = .16 sec
P-R = .20 sec

AV NODAL DYSRHYTHMIAS

- First Degree AV Block & Second Degree Mobitz Type 1
  - Generally the result of a physiologic predominance of the parasympathetic nervous system.
  - Therapy warranted if patient is hemodynamically compromised
    - Same as sinus bradycardia

AV NODAL DYSRHYTHMIAS

- Second Degree AV Block
  - Mobitz Type 2

Not associated with increased vagal tone
Set P-R interval
Number of P vs R waves variable (low vs. high grade)
AV NODAL DYSRHYTHMIAS

- Third Degree AV Block
  - No PR relationship
  - Escape rhythm
  - Morphology can be variable

AV NODAL DYSRHYTHMIAS

- Third Degree AV Block
  - 3rd Degree AV Block
  - Variable Escape Morphology

AV NODAL DYSRHYTHMIAS

- High Grade Second & Third Degree AV Block
  - Therapy
    - Permanent or temporary cardiac pacing REQUIRED
    - Anticholinergic medications ineffective
    - Beta-agonists
      - Isoproterenol (dilute 1mg in 500mL in LRS or 5% dextrose 0.5-1.0 mL/min IV to effect)
**AV BLOCK AND ATROPINE RESPONSE TEST**

• Atropine Response Test
  - Give 0.04 mg/kg IV with anticipated response in 5-10 minutes
  - Response to atropine
    1. Heart rate increases to >160 bpm without further block = secondary to high vagal tone
    2. Heart rate does not increase >160 bpm and/or persistent block = AV nodal disease +/- SA nodal disease

• Treatment
  - Medical management may be considered if responds to atropine
  - Pacemaker therapy may be indicated if no response to atropine

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**VENTRICULAR DYSRHYTHMIAS**

• Single Premature Ventricular Complexes

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**VENTRICULAR DYSRHYTHMIAS**

Premature Ventricular Complexes

<table>
<thead>
<tr>
<th>Primary Cardiac Disease</th>
<th>The Usual Suspects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>GEDV</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Cardiomyopathies</td>
<td>Splenic disease</td>
</tr>
<tr>
<td>Metabolic/Endocrine Disorders</td>
<td>Hypoxia</td>
</tr>
<tr>
<td>Electrolyte (K⁺, Ca²⁺, Mg²⁺)</td>
<td>Hypovolemia</td>
</tr>
<tr>
<td>Drugs &amp; Toxins</td>
<td>Pain</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Trauma</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>Increased sympathetic</td>
</tr>
<tr>
<td>Theobromine</td>
<td></td>
</tr>
</tbody>
</table>
VENTRICULAR DYSRHYTHMIAS

• Accelerated Idioventricular Rhythm

Sinus rhythm HR = 136 bpm
Ventricular rhythm HR = 130 bpm

VENTRICULAR DYSRHYTHMIAS

• Single Premature Ventricular Complexes & Accelerated Idioventricular Rhythm
  • Therapy
    • Address underlying disease
    • Pain, stress, hypoxia, hypovolemia, GID, etc.
    • Specific therapy only needed if patient is hemodynamically affected by the underlying rhythm
    • Lidocaine (2 mg/kg IV or CRI 50-75 mcg/kg/min)
    • Procainamide (15-20 mg/kg IV slowly, 15 min or CRI 25-50 mcg/kg/min)

VENTRICULAR DYSRHYTHMIAS

• Paroxysmal Ventricular Tachycardia

Paroxysmal ventricular tachycardia
Instantaneous HR = 300 bpm
VENTRICULAR DYSRHYTHMIAS

- Non-sustained Ventricular Tachycardia

Non-sustained ventricular tachycardia
Instantaneous HR = 375 bpm

VENTRICULAR DYSRHYTHMIAS

- Sustained Ventricular Tachycardia

Sustained ventricular tachycardia
HR = 375 bpm

VENTRICULAR DYSRHYTHMIAS

- Sustained Ventricular Tachycardia

- Boxers with Arrhythmogenic Right Ventricular Cardiomyopathy
- German Shepherds with inherited sudden cardiac death
- Therapy
  - Hemodynamically devastating rhythm
  - Potential for rapid decompensation and development of ventricular fibrillation
  - Lidocaine (2 mg/kg IV or CRI 50-75 mcg/kg/min)
  - Procainamide (15-20 mg/kg IV slowly, 15 min or CRI 25-50 mcg/kg/min)
VENTRICULAR DYSRHYTHMIAS

- Treatment Failure
  - Hypokalemia
    1. Hyperpolarization
    2. Prolong repolarization
      Increases the dispersion of refractoriness

- Increased number of Na⁺ channels available
decreases efficacy of class I drugs

CONSEQUENCES OF ARRHYTHMIAS

- Hemodynamic
  - Decreased cardiac function
  - Drop in blood pressure
  - Reduced tissue perfusion
  - Limited exercise capacity
  - Syncope

  Cardiac Output (CO) = Heart rate X Stroke volume

  ↑HR – decreased diastolic filling time
  ↓HR – adequate filling with decreased ejection

  Blood pressure = CO X Systemic Vascular Resistance

CONSEQUENCES OF ARRHYTHMIAS

- Electrical Instability
  - Myocardial fibrillation
  - Asystole
  - Sudden cardiac death
VENTRICULAR DYSRHYTHMIAS

• Ventricular Fibrillation

Torsade de Pointes

VENTRICULAR DYSRHYTHMIAS

• Ventricular Fibrillation

VENTRICULAR DYSRHYTHMIAS

• Ventricular Fibrillation
  • Therapy
    • Electrical Defibrillation (2-10 J/kg transthoracic, 0.2-1 J/kg internal)
    • Magnesium (0.2 mEq/kg IV)
**MISCELLANEOUS**

- Hyperkalemia
- Urethral Obstruction
- Ruptured Bladder
- Addison's Disease
- Iatrogenic
- Accelerated Junctional Rhythm
- Feline under anesthesia
- Bundle Branch Block
- Atrial Standstill

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**MISCELLANEOUS**

- Hyperkalemia

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**MISCELLANEOUS**

- Hyperkalemia Therapy
**MISCELLANEOUS**

- Accelerated Junctional Rhythm

  - Accelerated ventricular/junctional escape compared with sinus rate

  - Atrial Rate = 115 bpm
  - Ventricular Rate = 136 bpm

**MISCELLANEOUS**

- Accelerated Junctional Rhythm Therapy
  - No therapy required
  - Rhythm will resolve with cessation of anesthesia

**ATRIAL STANDSTILL**

- Atrial Myocarditis
  - Ventricular escape
  - Wide complexes
  - Regular rhythm
  - Slow
  - P waves never return

- Hyperkalemia
  - Sino-ventricular rhythm
  - Usually slow
  - May show R-R variation
  - QRS complexes wide
  - P waves appear with resolution of electrolyte imbalances
Atrial Standstill

Right Bundle Branch Block

Site of right bundle branch block

Lead III

Slow

Rapid

Left Bundle Branch Block

Site of left bundle branch block

Lead III

Slow

Rapid
QUESTIONS

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