# Let's Get Tachy: The Diagnosis and Treatment of Arrhythmias

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# **Objectives**

- Identify common tachyarrhythmias on ECG
- Discuss predisposition & pathophysiology of arrhythmias
- Learn how to treat arrhythmias
  - Rate versus rhythm control



"It's got a nice beat and it's easy to dance to."



#### **Supraventricular Tachycardias**

- Narrow QRS tachycardia
  - "Normal appearance"
  - Utilize ventricular conduction system
- Physiologic response
  - Sinus tachycardia
- Pathologic
  - Atrial fibrillation
  - Atrial flutter
  - Atrial tachycardia
  - AV Nodal Dependent
    - Wolff Parkinson White
    - Junctional





#### **Normal QRS Morphology**



Electrical alternans is common with SVT



#### Sinus Tachycardia



- Often confused with pathologic supraventricular tachycardias
- Fast rate with normal appearing QRS complexes
  - Dogs < 200 BPM
  - Cats < 250 BPM
- P for every QRS and QRS for every P
  - P waves may be buried in preceding T wave
- Normal PR interval
- Exhibit gradual changes in rate
  - Never abrupt rate changes
- No therapy required





# **ECG diagnosis**



#### What is this rhythm?

- A) Too fast to be a sinus tachycardia
- B) Sinus tachycardia
- C) I can't even find the p wave!
- D) Ventricular tachycardia
- E) Can I get more info?



#### **Vagal Maneuvers**

- Non-pharmacologic interventions that increase vagal stimulation to the heart
  - Slows (gradually) rate of SA nodal discharge
  - Slows AV nodal conduction
  - Little effect else where (atrial & ventricular myocardium)
- Methods
  - Carotid sinus massage
    - Pressure applied to angle of mandible
  - Breath hold
  - Diving Reflex
  - Ocular pressure





#### **Vagal Maneuvers**



Images courtesy of KE Schober



#### **Why Vagal Maneuvers are Important**



A) A sinus tachycardia again...duh!B) Is this a trick question?



What is the arrhythmia?



#### **Vagal Maneuvers**



#### What is the arrhythmia?

- A) Atrial fibrillation
- B) Atrial tachycardia
- C) Sinus tachycardia
- D) Can we talk about something besides arrhythmias?





#### Supraventricular Tachycardia



Abrupt breaks = pathologic



#### **Precordial Thump**





![](_page_11_Figure_3.jpeg)

![](_page_11_Picture_4.jpeg)

#### Pathophysiology

- Tachycardia
  - Decreases cardiac output
  - Decreases contractility
- Loss of atrial contraction
  - 20% decrease in cardiac output
  - Increase in atrial pressures
  - Increased risk of luminal thrombosis (cats)
- Sudden deterioration in clinical status
  - Perfusion abnormalities
  - CHF

![](_page_12_Figure_11.jpeg)

![](_page_12_Figure_12.jpeg)

![](_page_12_Picture_13.jpeg)

# **Predisposing Factors**

- Atrial enlargement
  - Larger atria have increased risk of development
  - Atrial chamber dilation in heart disease
  - Lone atrial fibrillation
- Atrial fibrosis
  - Promotes re-entry
    - Areas of conduction block
    - Slow conduction
  - Endothelial disruption
    - Jet lesions
    - Chamber dilation
- Electrolyte abnormalities
- Pericarditis

![](_page_13_Picture_14.jpeg)

![](_page_13_Picture_15.jpeg)

#### **Clinical Signs**

- Signs of CHF
  - Respiratory
  - Ascites
- Classic arrhythmia signs
  - Lethargy
  - Exercise intolerance
  - Syncope
  - Ptyalism
  - Agitation/anxiety

#### **Physical Exam**

- Arrhythmia
  - Extrasystole
  - Chaotic
  - Paroxysmal
  - Regular & sustained
- Femoral pulse
  - Variable pulse quality
  - Pulse deficits
  - Poor perfusion

![](_page_14_Picture_20.jpeg)

![](_page_14_Picture_21.jpeg)

#### **Atrial Tachycardia**

![](_page_15_Figure_1.jpeg)

P wave rate unaffected, decreased ventricular rate

- Atrial origin
  - P waves usually buried in proceeding T wave
  - P waves of different morphology
  - Baseline between subsequent P waves
- QRS rhythm generally regular and rapid
- Vagal maneuver slows ventricular rate
  - Easier to see P waves
  - P waves occurring at initial QRS rate
    - P waves unaffected by vagal maneuver

![](_page_15_Picture_12.jpeg)

#### **Atrial Flutter**

![](_page_16_Figure_1.jpeg)

![](_page_16_Picture_2.jpeg)

#### **Atrial Fibrillation**

![](_page_17_Figure_1.jpeg)

- Supraventricular origin
  - Normal QRS complexes
- (+/-) tachycardia
- <u>Marked irregularity</u> between subsequent QRS complexes!!
- No P waves
- Baseline undulations
  - f waves
- Generally sustained

![](_page_17_Picture_10.jpeg)

![](_page_17_Picture_11.jpeg)

#### **Atrial Fibrillation**

- Most common type of SVT
  - Seen predominately with heart disease
    - DCM
    - Mitral valve disease
  - May occur in normal heart
    - Lone AF
      - Giant breeds of dogs, horses
  - May also be triggered by pericarditis

![](_page_18_Picture_9.jpeg)

#### **Atrial Fibrillation**

![](_page_19_Figure_1.jpeg)

![](_page_19_Picture_2.jpeg)

![](_page_19_Picture_3.jpeg)

#### **Wolff-Parkinson White Syndrome**

- SVT due to re-entry across an accessory pathway
- Accessory pathway
  - Aberrant myocardium that connects atria to ventricles
  - Impulse travel bidirectionally

Accessory Pathway

Congenital abnormality

![](_page_20_Figure_7.jpeg)

![](_page_20_Picture_8.jpeg)

#### **Wolff-Parkinson White Syndrome**

• What abnormality would indicate the presence of an accessory pathway?

- A) Prolonged P wave duration
- B) Abnormally short PR interval
- C) Lack of PR interval
- D) Second degree AV block

![](_page_21_Figure_6.jpeg)

![](_page_21_Picture_7.jpeg)

#### **Wolff-Parkinson White Syndrome**

![](_page_22_Figure_1.jpeg)

- Ventricular pre-excitation
  - Abnormally short PR interval

![](_page_22_Picture_4.jpeg)

![](_page_22_Picture_5.jpeg)

#### **SVT Guidelines for Therapy**

- Sudden death unlikely
- Heart rates
  - Rates > 180 BPM for 3 weeks results in cardiomyopathy and heart failure
    - Refractory CHF
  - Average rate > 120 BPM associated with decreased survival times
- Hemodynamic significance
  - Most SVT's associated with underlying heart disease
  - Detriment to cardiac function
  - Likely associated clinical signs
    - Exercise intolerance
    - Syncope
    - Progressive CHF

![](_page_23_Picture_13.jpeg)

# **SVT Therapy**

- Rate control
  - Medical therapy aimed at blocking AV nodal conduction
    - Slowing the ventricular rate
    - Arrhythmia persists
  - Benefits
    - Easy to accomplish & maintain
      - Atrial fibrillation
      - Atrial flutter
    - Well tolerated medications
    - Inexpensive
  - Negatives
    - Loss of atrial contraction
      - Decreases cardiac output
      - Clot potential
    - Difficult
      - A tachycardia

![](_page_24_Picture_17.jpeg)

![](_page_24_Picture_18.jpeg)

# Which anti-arrhythmic used to treat SVTs also decreases contractility?

- A) Atenolol
- B) Lidocaine
- C) Sotalol
- D) Digoxin
- E) Diltiazem
- F) More than one of these

![](_page_25_Picture_7.jpeg)

![](_page_25_Picture_8.jpeg)

#### **Medical Treatment – Rate Control**

- Digoxin
  - Dosing
    - <20 kgs: 0.03 mg/kg
    - >20 kgs: 0.22 x lean meters squared body weight
  - Toxicity
    - GI signs
- Class 2 antiarrhythmics
  - Atenolol (1 mg/kg PO BID)
  - Metoprolol
  - Propranolol
  - Esmolol
- Class 4 antiarrhythmics
  - Diltiazem (2-4 mg/kg PO BID\*)
    - Extended release

![](_page_26_Figure_15.jpeg)

![](_page_26_Picture_16.jpeg)

#### **Medical Treatment – Rhythm Control**

- Restore sinus rhythm
  - Abolish arrhythmia
- Benefits
  - SA nodal control of heart rate
  - Restore atrial contraction
    - Improved cardiac output
  - Avoid the need of lifelong medication
- Negatives
  - Expensive
  - Redevelopment of arrhythmia
    - Heart disease
    - Chronicity
      - Electrical remodeling

![](_page_27_Picture_14.jpeg)

![](_page_27_Picture_15.jpeg)

# **Medical Therapy - Rhythm Control**

- Diltiazem
  - AV Nodal dependent tachycardia
- Class 3 (K+ channel blocker)
  - Sotalol
    - Concurrent beta-blockade
  - Amiodarone
    - Ventricular and atrial efficacy
- Class 1C (Na+ channel)
  - Flecainide
  - Propafenone

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![](_page_28_Figure_12.jpeg)

![](_page_28_Picture_13.jpeg)

#### **Rhythm Control**

![](_page_29_Figure_1.jpeg)

![](_page_29_Picture_2.jpeg)

![](_page_29_Picture_3.jpeg)

Atrial fibrillation

![](_page_29_Picture_5.jpeg)

![](_page_29_Picture_6.jpeg)

![](_page_29_Picture_7.jpeg)

## Ablation

- Radiofrequency necrosis of arrhythmia site
  - Identification with catheter mapping
- Permanent cure
  - Wolff Parkinson White
  - Atrial tachycardia
  - Atrial flutter

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![](_page_30_Picture_8.jpeg)

![](_page_30_Picture_9.jpeg)

#### **Ventricular arrhythmias**

![](_page_31_Picture_1.jpeg)

![](_page_31_Picture_2.jpeg)

#### What are ventricular arrhythmias?

• Abnormal, rapid, heart beats that originate in the bottom chamber of the heart

![](_page_32_Figure_2.jpeg)

![](_page_32_Picture_3.jpeg)

#### **Ventricular arrhythmias TIP**

Abnormal Electrical Conduction due to Ventricular Ectopic Foci

Remember that morphology of the ventricular complex is the between a supraventricular ectopic focus and a ventricular

• A supraventricular focus almost invariably results in a norm

![](_page_33_Figure_4.jpeg)

![](_page_33_Figure_5.jpeg)

• A ventricular focus almost invariably results in a wide, bizarre, complex associated with ventricular depolarization and repolarization.

![](_page_33_Figure_7.jpeg)

![](_page_33_Picture_8.jpeg)

#### What causes ventricular arrhythmias?

- A. Can be found in normal dogs
- B. Primary cardiac disease
- C. Drug effects
- D. Neoplasia
- E. Changes in autonomic tone

e. hypoxia, anemia, uremia, GDV, pancreatiis, electrolyte disturbances, metabolic, infectious conditions, trauma b. Dilated cardiomyopathy, degenerative valve disease, congenital disease

 c. digitalis, catecholamines, anesthetic agents (ketamine, barbiturates)

F. All of the above

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#### **Ventricular arrhythmias TIP**

- Anything and everything may result in VPCs in dogs !
- In cats, VPCs are almost always (96%) secondary to underlying heart disease.

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![](_page_35_Picture_4.jpeg)

- Ventricular premature complexes (VPC) are named by the pattern they appear in.
- Most VPCs are single and by themselves.

![](_page_36_Figure_3.jpeg)

Isolated VPC

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#### **Uniform v. multifocal VPCs**

# \* mynymyt

**Multifocal VPCs**: from 2 or more foci within the ventricular wall

![](_page_37_Picture_3.jpeg)

![](_page_38_Figure_1.jpeg)

#### Ventricular bigeminy

![](_page_38_Figure_3.jpeg)

#### Ventricular trigeminy

![](_page_38_Picture_5.jpeg)

![](_page_39_Figure_1.jpeg)

![](_page_39_Figure_2.jpeg)

#### Ventricular couplet

#### Ventricular triplet

![](_page_39_Picture_5.jpeg)

![](_page_40_Figure_1.jpeg)

Ventricular tachycardia !!!

![](_page_40_Picture_3.jpeg)

J Vet Cardiol. 2017 Oct;19(5):455-461. doi: 10.1016/j.jvc.2017.08.003. Epub 2017 Sep 19.

Sudden cardiac death in a dog during Holter recording-R on T phenomenon.  $\underline{\text{Gunasekaran } T^1, \underline{\text{Sanders } RA^2}.}$ 

#### **R-on-T phenomenon**

![](_page_41_Figure_3.jpeg)

![](_page_41_Picture_4.jpeg)

#### **Ventricular fibrillation**

![](_page_42_Figure_1.jpeg)

![](_page_42_Picture_2.jpeg)

#### Severity of ventricular arrhythmias

Grade	Description
1	Single VPCs
2	Ventricular bigeminy, trigeminy
3	Couplets, triplets
4	R-on-T, multifocal ventricular arrhythmias, ventricular tachycardia

![](_page_43_Picture_2.jpeg)

## Accelerated idioventricular rhythm

- Cause: unknown
  - Seen with dogs that are acutely ill from an array of systemic disorders
- Control of the cardiac rhythm is intermittently exchanged between a ventricular focus and the SA node
- Ventricular beats occur in long paroxysms with a rate no more than ~10% higher than the normal rhythm – NO TACHYCARDIA!
  - Beats are wide, bizzare, biphasic, not premature
- Treatment: not required
  - Resolves spontaneously 2-3 days
  - Is not hemodynamically significant
  - Impossible to suppress

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#### Accelerated idioventricular rhythm

![](_page_45_Figure_1.jpeg)

![](_page_45_Picture_2.jpeg)

#### **Patient presentation**

- Predisposed canine breeds: Boxer dogs, Doberman Pinschers
- Predisposed feline breeds: cats with hypertrophic cardiomyopathy
- History
  - Depends on severity of arrhythmia
  - Varies from normal at home to sudden death
    - Syncope
    - Weakness
    - Lethargy
    - Exercise intolerance
- Physical exam
  - Irregular rhythm
  - Tachycardia
  - Asynchronous pulses
  - Prolonged CRT

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![](_page_46_Picture_16.jpeg)

![](_page_46_Picture_17.jpeg)

![](_page_46_Picture_18.jpeg)

## Diagnostic work up

- May or may not proceed treatment
- General practice work up should include:
  - <u>A thorough physical exam!</u>
  - CBC, Chemistry, UA, T4
  - ECG
  - Chest radiographs
- Cardiac work up may include:
  - <u>A thorough physical exam!</u>
  - Echocardiogram
  - ECG (6 or 10 lead)
  - Telemetry or Holter monitor
  - +/- Abdominal ultrasound

![](_page_47_Picture_13.jpeg)

![](_page_47_Picture_14.jpeg)

#### **Severity of ventricular arrhythmias**

Grade	Description		
1	Single VPCs	Investigate!	
2	Ventricular bigeminy, trigeminy		
3	Couplets, triplets	Controversial, in my	
4	R-on-T, multifocal ventricular arrhythmias, ventricular tachycardia	Treat!	

Justification to treat is usually to prevent progression to more serious arrhythmias such as ventricular tachycardia and ventricular fibrillation, which may result in sudden death.

![](_page_48_Picture_3.jpeg)

#### Treatment

- Intravenous options
  - Lidocaine
    - Should stock in GP!
  - Procainamide
  - Magnesium sulfate
  - Nexterone (Amiodarone)

![](_page_49_Picture_7.jpeg)

![](_page_49_Picture_8.jpeg)

#### Lidocaine

- Mechanism of action: class Ib sodium channel blocker
- Dosage
  - Canine: 2 mg/kg IV slowly up to three times (6 mg/kg)
    - If effective start a CRI at 50 mcg/kg/min (25-75 mcg/kg/min)
  - Feline: 0.25 mg/kg IV slowly up to 2 mg/kg
- Side effects
  - Acutely: GI upset (vomiting) give slowly over 1 minute
  - If toxic dose reach: neurologic (seizures)
- Dosing trick (2% lidocaine or 20 mg/mL)
  - IF a patient weighs 33 kgs (and we are giving 2 mg/kg) they need 3.3 mls
  - IF a patient weighs 15 kgs (and we are giving 2 mg/kg) they need 1.5 mLs

![](_page_50_Picture_12.jpeg)

![](_page_50_Picture_13.jpeg)

#### What if IV medications don't work?

![](_page_51_Picture_1.jpeg)

![](_page_51_Picture_2.jpeg)

**Electrical cardioversion** 

![](_page_51_Picture_4.jpeg)

# **Oral options**

- Class I antiarrhythmics
  - Mexiletine
- Class III antiarrhythmics
  - Sotalol
  - Amidoarone

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![](_page_52_Picture_7.jpeg)

#### Sotalol

- Dose (canine) 1-2 mg/kg PO BID, feline 1-2 mg/kg BID or 10 mg PO BID/cat
- Side effects
  - Negative inotropic and chronotropic effect
    - \*BE CAUTIOUS IN PATIENTS WITH SYSTOLIC DYSFUNCTION OR HEART FAILURE
  - Proarrhythmic

#### Mexiletine

- Dose (canine) 5-7 mg/kg TID, feline ???
- Side effects
  - Anorexia, vomiting, tremors (neurologic signs), hepatic toxicity, urine dribbling

#### Amiodarone

- Dose (canine) 12-15 mg/kg SID x 14 days; then 5-7 mg/kg SID; feline ???
- Side effects
  - Increase in liver enzymes, thyroid dysfunction, GI signs, neutropenia, pro-arrhythmic

![](_page_53_Figure_14.jpeg)

![](_page_53_Figure_15.jpeg)

![](_page_53_Picture_16.jpeg)

![](_page_53_Picture_17.jpeg)

#### Remember...

When treating arrhythmias...

- Take a deep breath!
- Assess severity and risk factors
- Formulate a diagnostic and treatment plan
- Re-evaluate as needed

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#### **Questions?**

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