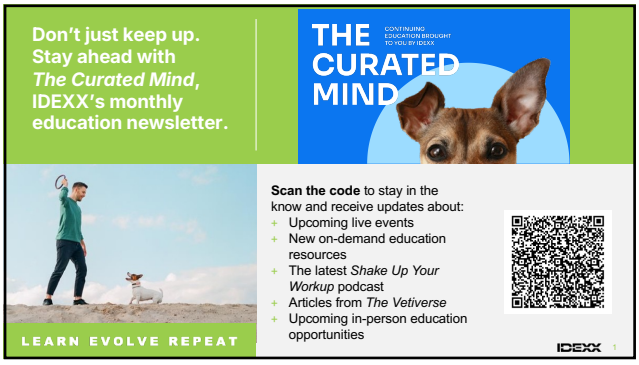
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WVC Vegas
DON'T MISS A BEAT
Augusta Pelosi, DVM,
Dipl ACVS and ACVIM (Cardiology)
WELCOME
TO Fabulous
LAS VEGAS
NEVADA

2


CONFLICT OF INTEREST DISCLOSURE
I have financial interest, arrangement or affiliation with:
IDEXX
WVC Vegas

3

OVERVIEW

PHYSIOLOGY of the ECG

OBTAINING AND UNDERSTANDING THE TRACING

NORMAL RHYTHMS

ABNORMAL RHYTHMS
- Ventricular arrhythmias
- Supraventricular arrhythmias
- Bradyarrhythmias

4

INDICATIONS

- Accurate heart rate
- Bradycardia or Tachycardia
- Irregular rhythm or sound
- Abnormalities due to electrolyte imbalance (especially K^+)
- Anesthetic monitoring

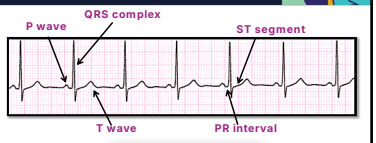
5

What is an ECG?

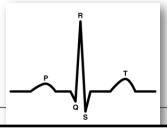
- The heart is electrically charged
- An ECG is a recording of the heart's electrical activity
- This electrical activity is measured by electrodes attached to the skin

6

WAVES/COMPLEXES:
- P wave = atria
- QRS complex = ventricles
- T wave = reset



INTERVALS/SEGMENTS:
- PQ (PR) = AV node conduction
- ST = ventricular recovery



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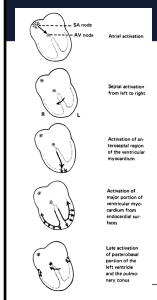
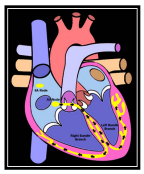
NORMAL CONDUCTION



SA node
→ atria depolarize
→ **AV node**
→ **bundle branches**
→ ventricles depolarize

→ ventricles repolarize



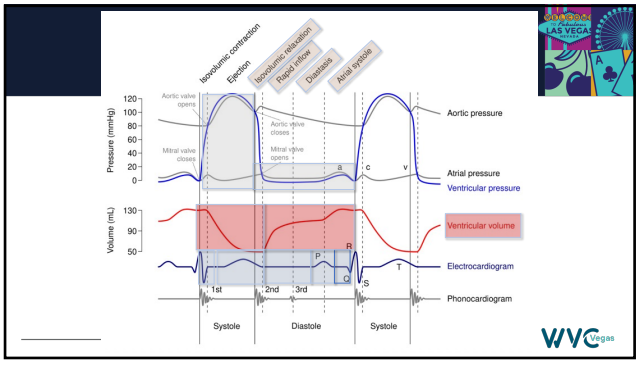
8

PHYSIOLOGY - Cardiac Pacemakers

Automatic cells in the heart

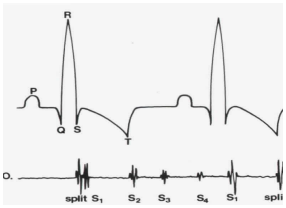
- Depolarize on their own
- Rate of depolarization affected by autonomic nervous system
  - SA node (60-180 beats/min dog) (100-240 cat)
  - AV node (40-60 beats/min dog) (80-130 cat)
  - Purkinje fibers (20-40 beats/min)
  - Bundle of HIS (20-40 beats/min)
  - Ventricular myocytes (20-40 beats/min)

9



10

CARDIAC CYCLE: SYSTOLE



Relation to ECG

- QRS is onset of ELECTRICAL systole
- T wave represents repolarization

Relation to heart sounds

- S1 is onset of MECHANICAL systole
- S2 marks end of systole (coincides with end of T wave)

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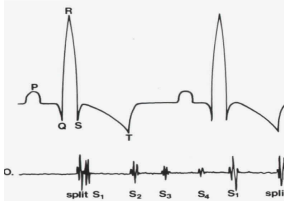
CARDIAC CYCLE: DIASTOLE



Electrical diastole (repolarization)

- Repolarization begins at end of QRS and ends as QRS complex begins (start of depolarization)

Relation to heart sounds

- Between S2 and S1
- Early filling (E wave, S3)
- Atrial kick (A wave, S4)

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WHAT ECG DETECTS

HEART CHAMBER ENLARGEMENT

Eccentric hypertrophy
- Dilation and growth of heart chambers
- Due to volume overload

Concentric hypertrophy
- Wall thickening of heart chambers
- Due to pressure overload

MEA

HEART RHYTHM

CONDUCTION DISTURBANCES

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WHAT ECG DOES NOT DETECTS

Type of Heart chamber enlargement
- Eccentric vs. Concentric hypertrophy
- Congestive Heart Failure

A Short ECG won't detect many arrhythmias
- Arrhythmias can be intermittent
- 10 minutes is <1% of the day

**** A normal ECG does not confirm nor exclude heart disease! ****

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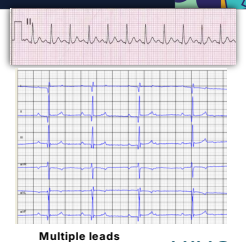
TYPES

- Single lead rhythm strip - monitor
  Anesthesia monitoring/ER
- Multiple lead (6 or 12 leads)
  'Diagnostic' ECG
- Telemetry
  ICU
- Ambulatory
  Holter/Event monitors



Multiple leads

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TECNIQUES (surface ECG)

RIGHT lateral recumbency – standardized wave appearance

Typically 4 electrodes/clips:

1. **Black (LA)**
2. White (RA)
3. **Green (RL) – ground**
4. **Red (LL)**

Alcohol or gel is applied to the skin and electrodes/clips at the point of contact

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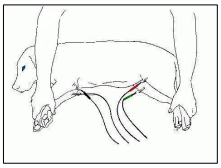
We read the newspaper in the AM (black and white)

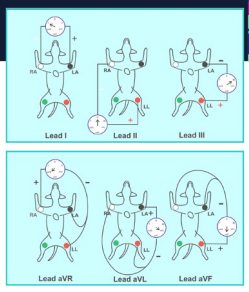


Christmas comes at the end of the year

Snow and Grass are on the ground

17

6 LEADS



LIMB LEADS

- **I** LA+ RA-
- **II** LR+ RA-
- **III** LR+ LA-

AUGMENTED LIMB LEADS

- **aVR** RA+ (summation lead III)-
- **aVL** LA+ (summation lead II)-
- **aVF** LR+ (summation lead I)-

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ECG – SYSTEMATIC INTERPRETATION

1. *Is it regular or irregular?*
2. *Determine Heart Rate*
3. *Measurements of the waves and segments*
   - P wave - width and height
   - PR interval - length
   - QRS - width and height
   - QT interval – length
   - ST segment – relative to PR interval
   - T wave - width and height
4. *Mean Electrical Axis*
5. *Rhythm determination*

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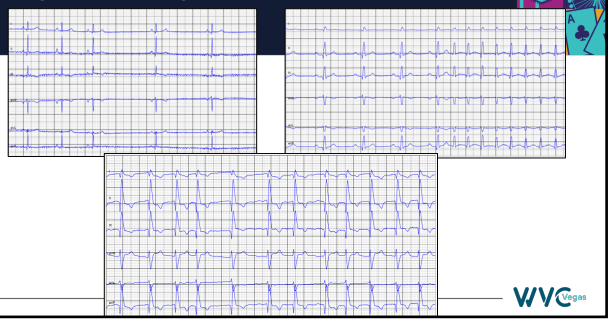
Regular vs. Irregular?



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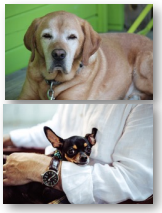
Heart Rate

Fast, slow or reasonable/appropriate
-- Dog: 70-160 bpm
-- Cat: 150-240 bpm

Prefer the term appropriate over 'normal'
(Has to be taken into clinical context)



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Giant dogs 60-140 Med-Lg dogs 70-160
Toy dogs 80-180 Puppies 70-220

Cats 100-240

Get Baseline heart rates for individuals on every visit

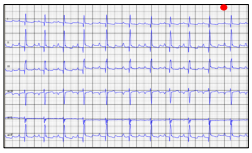
23

How to …..

At 25 mm/sec, 150mm = 6 sec

25 mm/sec : 100 bpm
50 mm/sec : 200 bpm

- 1/25 = 0.04 × 5 = 0.2 × 30 = 6
- 60/6=10
- Bic Pen Times Ten
- Accurate within 10 beats per minute



At 50 mm/sec, 300mm = 6 sec

- 1/50 = 0.02 × 5 = 0.1 × 30 = 3
- 60/3=20
- Bic Pen times Twenty
- Accurate within 20 beats per minute

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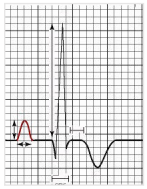
MEASUREMENT OF WAVES & SEGMENTS

P wave
SA node fires
Atrial depolarization (contraction)

Internodal tracts (shortcut to AV node)



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P wave

Normal Dog:

<0.4 mV x <0.04 sec

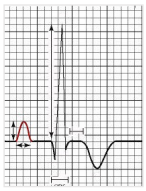
<0.5 sec in giant breeds

4 boxes tall
25 mm/sec (1-1.25 boxes wide)
50 mm/sec (2-2.5 boxes wide)

Normal Cat:

<0.2 mV x <0.04 sec

2 boxes tall



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P wave – Normal Variations

"wandering pacemaker" – increased vagal tone

Origin of impulse shifts slightly within the SA node resulting in changes in the P wave morphology (a form of sinus arrhythmia)

Usually associated with changes in vagal tone due to respiration

- Inspiration: P wave taller
- Expiration: P wave smaller

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P wave – Abnormal Variations

Wide P wave (Sometimes Notched)

- P Mitrale - LA enlargement

25 mm/sec > 1 box wide
50 mm/sec > 2 boxes wide

Tall P wave (often spiked)

- P Pulmonale - RA enlargement

Lack of P wave

- Atrial standstill

Dog > 4 boxes tall
Cat > 2 boxes tall

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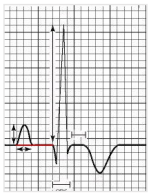
PQ Interval

Beginning of P wave to beginning of QRS
Conduction from atria to ventricles (AV node)
Establishes the ECG baseline

AV node (most of the PQ interval is here)
Bundle of HIS
Bundle branches (R&L)
Purkinje fiber network



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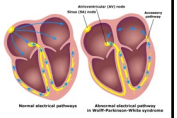
PQ interval – Abnormal Variations

Short PQ Interval (tachycardia)
- AV node is bypassed
- "Accessory pathway" (Wolff-Parkinson-White)
- Sudden onset of tachycardia in a dog



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PQ interval – Abnormal Variations

Prolonged PQ Interval (bradycardia)
- Slow conduction through abnormal AV node
- AV Blocks

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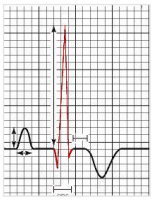
QRS Complex

QRS complex

Ventricular depolarization (systole)

Q wave 1st negative deflection
R wave 1st positive deflection
S wave 2nd negative deflection



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QRS Complex– Abnormal Variations

Tall R wave, wide QRS
- LV enlargement
- Left Bundle branch block

Deep S wave in leads I, II & III
- RV enlargement

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T wave

Ventricular repolarization (diastole)

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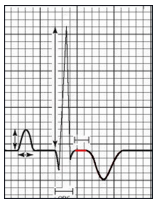
ST Segment

- Between S & T waves
- Between ventricular contraction (depolarization and ventricular relaxation (repolarization – diastole)
- Relationship with baseline



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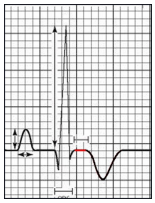
ST Segment

>0.2mV between baseline and ST

CAUSES:
- Hypothermia
- Hypokalemia
- Digitalis toxicity
- Bundle branch block
- Myocardial infarction
  - Rare in dogs - hypothyroidism
  - Can be seen in feline HCM



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QT Interval

- Beginning of QRS to end of T wave ventricular depolarization & repolarization
- Pulse generated
- Drug-inducted QT changes

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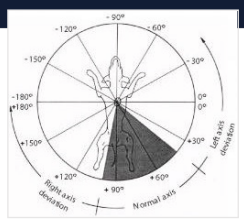
MEAN ELECTRICAL AXIS (MEA)

- when a wavefront spreads toward an electrode, the largest possible deflection will occur
- When a wavefront spreads perpendicular to a lead, the smallest or no deflection occurs
- ECG shows the sum of all wavefronts relative to the lead being used to measure (MEA)

ISOELECTRIC LEAD

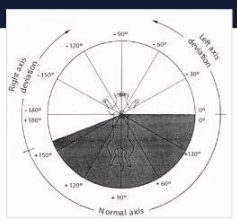
- lead with the smallest deflection
- Perpendicular to the MEA

40



Normal Canine MEA

40-110°



Normal Feline MEA

0-160°

41

The normal MEA is 40° to 100° in the dog

- Lead II is most perpendicular to the normal MEA
  - largest deflections
  - best for measurements
- aVL is most often the isoelectric lead. Approximates MEA in normal dogs

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Estimating MEA

- Find the isoelectric lead
  - NOT the lead with smallest deflections
  - Lead with smallest NET DEFLECTION
- MEA is perpendicular to that, in the direction of net deflection

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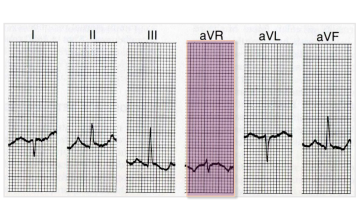

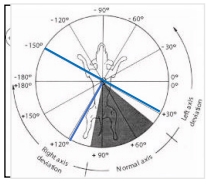

MEA = +120°
Right Axis Shift

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Calculating MEA

1. Calculate the net deflection in lead I
   Graph on "x axis"
2. Calculate net deflection in lead aVF
   Graph on "y axis"
3. Draw the vector between the two (MEA)

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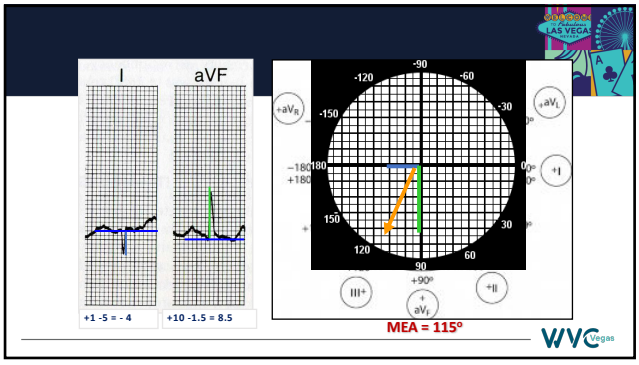

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MEA – Abnormal Variations

RIGHT AXIS SHIFT

- Right ventricular enlargement
  - RV hypertrophy or dilation
- Right bundle branch block

LEFT AXIS SHIFT

- HCM in cats
- Hyperkalemia

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ECG – Helpful Hints

- Always in right lateral recumbency
- Patient on a towel or rubber mat
- Metal tables are more problematic
- Limbs perpendicular to body
- Place a towel between the legs
- Place leads below the elbow and knee
- No one moves while the ECG is being recorded
- Enhance lead contact with gel or alcohol

Alcohol is FLAMMABLE!!

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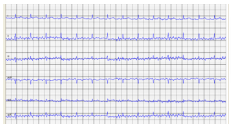
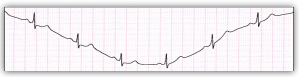
MINIMIZING ARTIFACT

- Lots of alcohol (or gel)
- Avoid metal on metal contact between the clips/electrodes
- Reduce motion
- Choose a quiet, comfortable environment



purr/shiver artifact



wandering baseline artifact

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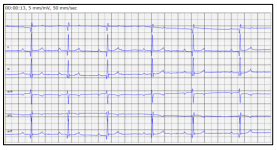
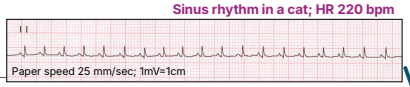
NORMAL PHYSIOLOGIC RHYTHMS

NORMAL SINUS RHYTHM (NSR)

- Regular and consistent R-R interval
- 'Reasonable' heart rate
- P, QRS and T waves in each complex
- P wave for every QRS complex



Sinus rhythm in a cat; HR 220 bpm



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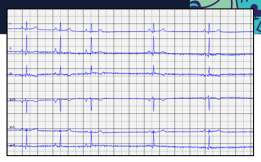
SINUS ARRHYTHMIA



- 'Regularly irregular' R-R interval
- P, QRS and T waves in each complex
- P wave for every QRS complex
- Reasonable heart rate, may be a little slow
- Speeds up with inhale, slows with exhale (vagal tone variance, in a regular cycle)
- Secondary to high vagal tone
- Normal in dogs
- Variable P wave – wandering pacemaker
- Heart rate less than 200

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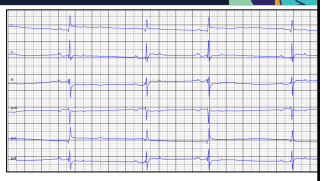
SINUS BRADYCARDIA



- P wave for every QRS complex
- Regular and consistent R-R interval
- Low heart rate (within reason)
- Typically seen with high vagal tone
- No treatment is necessary; only warranted in case it is associated with clinical signs

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SINUS TACHYCARDIA

- Regular and consistent R-R interval
- P wave for every QRS complex
- Elevated heart rate (within reason)

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ABNORMAL RHYTHMS

Atrial arrhythmias (supraventricular)
- Atrial premature complexes (APCs)
- Atrial tachycardia
- Atrial fibrillation

Ventricular arrhythmias
- Ventricular premature complexes (VPCs)
- Idioventricular rhythm (IVR)
- Ventricular tachycardia
- Ventricular fibrillation

Bradyarrhythmias
- AV block
  - 1st degree
  - 2nd degree
    - Mobitz type I
    - Mobitz type II
  - 3rd degree
- Sinus arrest
  Atrial Standstill
- Sick Sinus Syndrome

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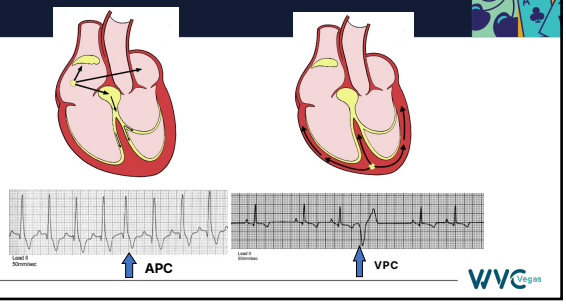
SUPRAVENTRICULAR vs. VENTRICULAR BEATS



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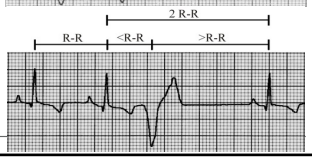
VPC and APC Pauses

APC:
NON-COMPENSATORY PAUSE

VPC:
COMPENSATORY PAUSE



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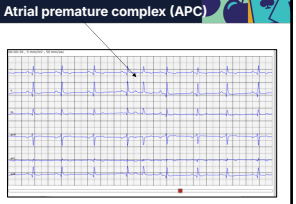
SUPRAVENTRICULAR ARRHYTHMIAS



ATRIAL PREMATURE COMPLEXES (APC)

- Premature beat (occurs early)
- Normal QRS morphology
- +/- P wave
  - Often hidden in the previous complex or T wave
- Can be confused with sinus arrhythmia

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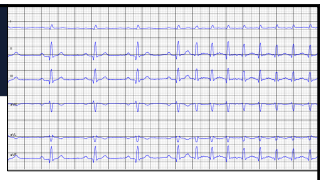
ATRIAL TACHYCARDIA



- Run of APCs
- QRS complexes normal to narrow
- Very rapid rate (often > 200)
- Not a common arrhythmia
- Associated with atrial disease *or* systemic disease

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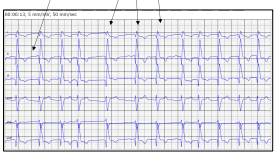
ATRIAL FIBRILLATION (A FIB)



- Very common arrhythmia
- Chaotic, disorganized atrial activity

- Three classic criteria:
  1. No P waves (fibrillation waves)
  2. 'Irregularly irregular' known as Tennis shoes in a dryer
  3. Rapid rate

- Often associated with significant underlying structural heart disease

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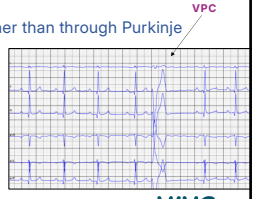
VENTRICULAR ARRHYTHMIAS

VENTRICULAR PREMATURE COMPLEX (VPC)



- Depolarization wave through myocardium rather than through Purkinje network on affected side
- Abnormal QRS morphology (wide and bizarre)
- Premature beat (occurs early)
- No P waves
- Very common

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- Caused by systemic or cardiac disease
- VPCs can be persistent or intermittent
- Multiform VPCs are more serious - Multifocal areas of LV pathology

VPCs vs. ESCAPE BEATS

- VPCs are like escape beats as they originate from the ventricular myocardium
- VPCs are abnormal due to primary LV pathology or secondary to metabolic disease
- Escape beats are the normal life saving response to a failure of cardiac pacemaker cells

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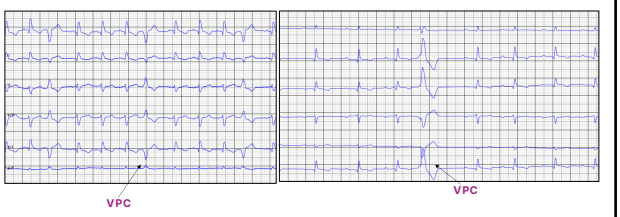

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VENTRICULAR TACHYCARDIA

- Run of VPCs
- Wide and bizarre QRS complexes
- Very rapid rate (often > 200)
- Potentially life threatening
- Associated with heart disease or systemic disease

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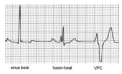
IDIOVENTRICULAR RHYTHM (IVR)



HR = ~150bpm

- 'Slow v-tach'
- Run of VPCs
  - Not tachycardic
  - HR >160-180
- Typically not hemodynamically significant
- May begin and end with a **fusion beat**
- Most commonly associated with non-cardiac disease (GDV, Splenectomy, Sepsis, Trauma)

64

Tuesday, 10 yo, FS, Greyhound

Presented for a dental

No clinical signs

Auscultation:

- Tachycardia
- No murmur

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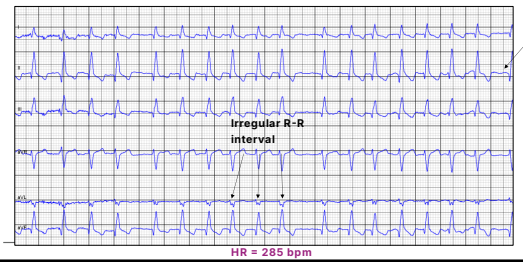
ATRIAL FIBRILLATION



66

Which drug would you select?

1. Lidocaine
2. Sotalol
3. Diltiazem
4. Atenolol

Acute treatment of SVT
0.05 – 0.1 mg/kg IV administered over 2 minutes; this dose may be repeated up to 2 times with 5 minutes between doses
CRI: 2 – 6 micrograms/kg/MINUTE IV CRI has been described when the arrhythmia recurs before oral dosing is feasible.

Chronic treatment of SVT:
0.5-1.5 mg/kg PO every 8 hours and titrated upward to a maximum of 2.5 mg/kg PO every 8 hours.

The nondihydropyridines (eg, diltiazem) act by blocking inward movement of calcium via L-type calcium channels, which both prolongs the refractory period and slows nodal conduction (negative dromotropy).

Diltiazem is typically associated with weak negative inotropic effects

67

Tucker, 12 yo MN Labrador

Presented for lethargy and weakness
Auscultation:
- Tachycardia
- No murmur

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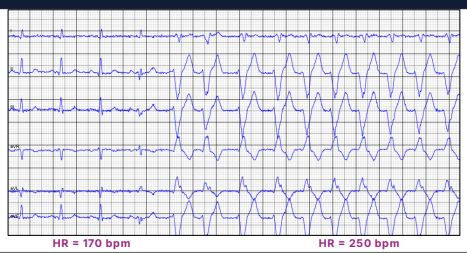
VENTRICULAR TACHYCARDIA



HR = 170 bpm HR = 250 bpm

69

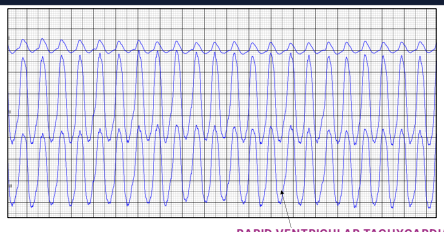
ZEUS, 8 MN BOXER

Presented for syncope

Auscultation:

- Tachycardia
- No murmur

70



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How would you treat this patient?

ACUTE vs CHRONIC

LIDOCAINE bolus and CRI

Start infusion at 17 – 50 micrograms/kg/*MINUTE* (1 – 3 mg/kg/*HOUR*) IV followed by bolus 1 – 2 mg/kg IV

Lidocaine acts by binding to inactive fast sodium channels, which inhibits recovery after repolarization. Class IB agents demonstrate rapid rates of attachment to and dissociation from sodium channels in ventricular conducting tissue more so than in atrial tissue.

SOTALOL

Sotalol 1.5 – 3.5 mg/kg PO twice daily either alone (typical) or in combination with mexiletine (less common) 5 – 8 mg/kg PO every 8 hours

Sotalol is a nonselective beta blocker and class III antiarrhythmic agent (potassium channel blocker). The beta-blocking activity of sotalol, which causes negative inotropic and chronotropic effects, is ≈30% that of propranolol.

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BRADYARRHYTHMIAS

AV blocks P waves are 'blocked' with variable conduction at the AV node
- Most commonly secondary to idiopathic fibrosis
- Tends to be an age-related change
- 3 types AV bocks

1st degree AV block
2nd degree AV block
3rd degree AV block

Sinus arrest
Atrial standstill
Sick Sinus Syndrome

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FIRST DEGREE AV BLOCK

- Prolongation of the P-R interval
- Every P wave gets through
- May be normal or due to high vagal to
- Due to increased vagal tone
- Non-pathogenic
- No treatment required

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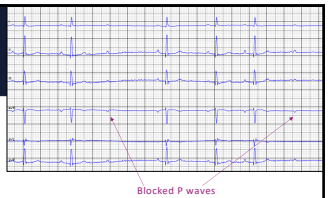
SECOND DEGREE AV BLOCK



- Some P waves get through, some don't
- Can be associated with primary AV node disease *or* from high vagal tone
- Commonly seen with anesthesia
- Occasionally symptomatic (syncope)

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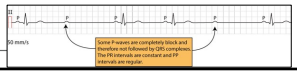
Mobitz Type I

- Progressive prolongation of the PR interval before a blocked P wave
- Also known as Wenckebach phenomenon
- Typically associated with high vagal tone/athletes

Mobitz type II

- Sporadically occurring blocked P waves
- High vagal tone or AV node fibrosis



Images from: https://ecgwaves.com/second-degree-av-block-ii-2nd-2/

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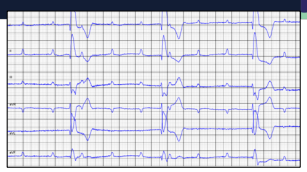
THIRD DEGREE AV BLOCK

Blocked P waves

Ventricular escape beat



- No relationship between P waves and QRS
- P waves have their own rate (faster), determined by the normal SA node
- QRS has its own rate (slower), determined by the automaticity of the fastest remaining functioning pacemaker

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- Escape rhythm – life saving mechanism of the ventricles **NOT** a VPC!
- The fastest functioning pacemaker in the heart takes over, by default
- The closer to the AV node, the more the escape beat will resemble normal QRS
- The closer to the ventricle, the more wide and bizarre the QRS will appear
- **Escape rhythm** – pacemaker other than SA node takes over, because SA node fails to fire

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Often symptomatic: Syncope, weakness

Treatment
- pacemaker
- EMERGENCY - Pacemaker often required

Prognosis
- Cats – without anesthesia, potentially very good
- Dogs – eventual asystole is likely, if no pacemaker implanted

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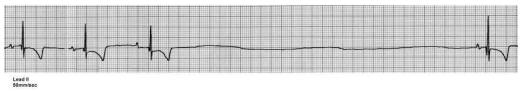
SINUS ARREST



- SA node fails to depolarize
  - Depression of normal automaticity
- Definition: pause > 2 R-R intervals
- Causes: vagal tone, injury to SA node, hyperkalemia, drug induced
- May cause clinical signs if extended period of asystole

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ATRIAL STANDSTILL

Atrial tissue no longer conducts impulse to the AV node
- Ventricles controlled by a junctional or ventricular escape rhythm

Two main causes
- Severe hyperkalemia (sinoventricular rhythm)
- Idiopathic destruction of the atrial myocardium

- Idiopathic disease: English Springer Spaniels
- Hyperkalemic disease: Hypoadrenocorticism

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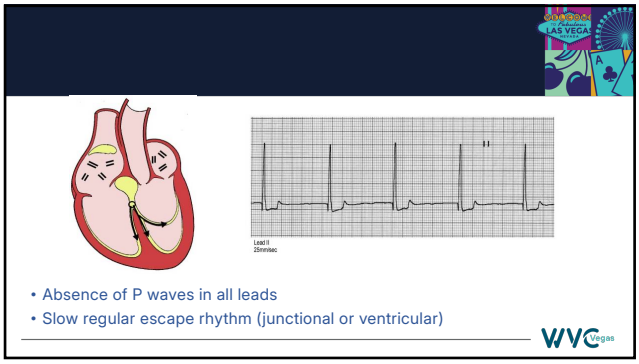
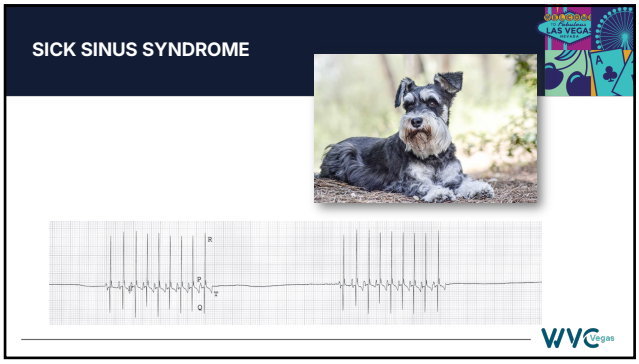

• Absence of P waves in all leads
• Slow regular escape rhythm (junctional or ventricular)

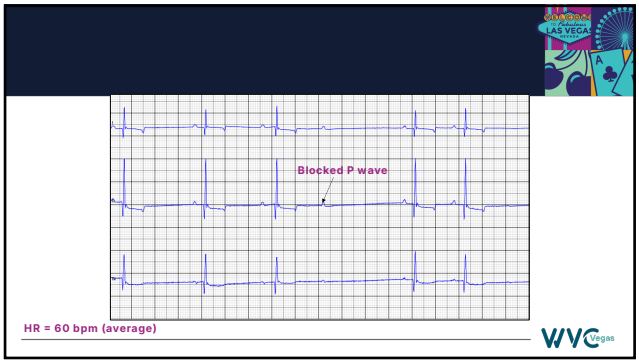
84


SICK SINUS SYNDROME

85


DOLLY, 6 MO, FI SHIH TZU
Presented for spay
Auscultation:
• Bradycardia and irregular rhythm
• No murmur
Pre-op ECG performed

86



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Should we anesthetize this patient?

YES or NO?

Which test should we consider prior to anesthesia?

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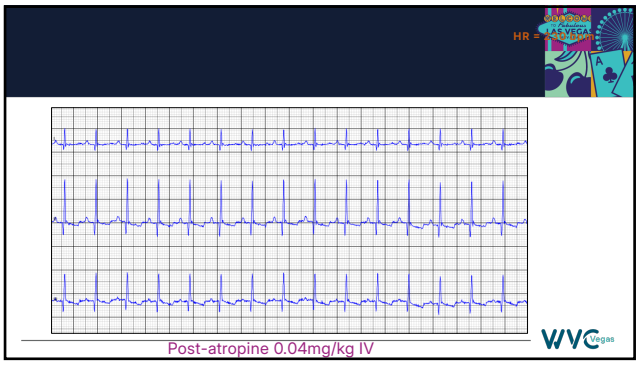

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THANK YOU

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IDEXX 91

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