Cardiovascular Therapeutics for Advanced Practitioners Mini Series

Session Two: Treatment of arrhythmias: when and how to treat arrhythmias

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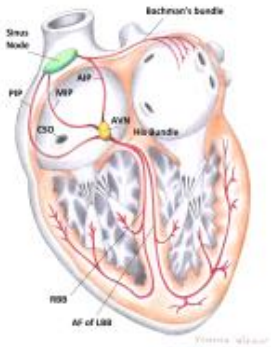
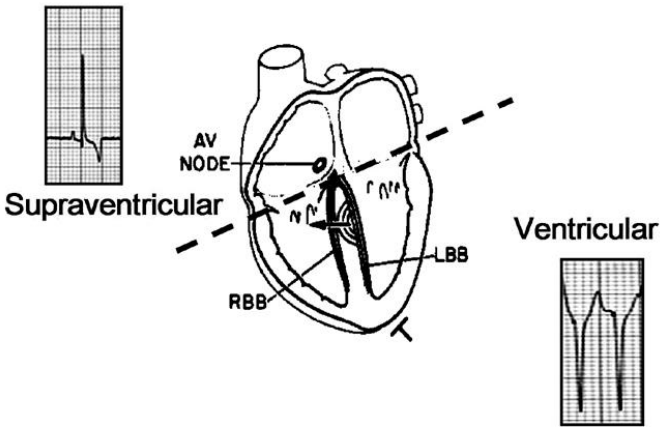


TACHYARRHYTHMIAS

Tachyarrhythmias are sometimes differentiated as to whether the QRS complexes are narrow (i.e., normal width) or wide. Narrow QRS complexes typically indicated that atrioventricular conduction occurs via the AV node. These types of tachycardias are grouped according to location of impulse formation as “supraventricular”, or literally “above the ventricle”. So any rhythm which is initiated above the ventricle and uses the AV node for conduction is a supraventricular rhythm. Rhythms which are wide are thought to occur below the AV node as they are unable to utilize the specialized conduction tissue and thus must travel muscle cell to muscle cell and result in a wide complex. These types of tachycardias are also grouped according to location of impulse formation as “ventricular”, or literally “within the ventricle”.



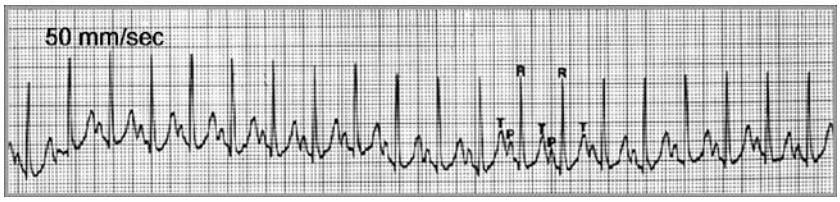
Caveat some supraventricular impulses can be conducted with a bundle branch block or with a fascicular block and may therefore not look ‘supraventricular’. This is the *exception* however, and generally supraventricular rhythms will look > 90% like ‘normal’ while ventricular rhythms will look < 90% like ‘normal’.

SUPRAVENTRICULAR TACHYARRHYTHMIAS

a. Sinus tachycardia

1. All the criteria for sinus rhythm are met, however the HR is too fast.

2. This is usually a physiologic response to something external and an underlying etiology can be identified.
   a. Sympathetic nervous system stimulation
      1. Fear
      2. Excitement
      3. Pain
      4. Fever
      5. Hyperthyroidism
      6. Hypovolemia
      7. Cardiac tamponade
      8. Heart failure
      9. Hypoxia
      10. Anemia
   b. Drugs
      1. Catecholamines
      2. Atropine
      3. Terbutaline
      4. Aminophylline
      5. Theophylline

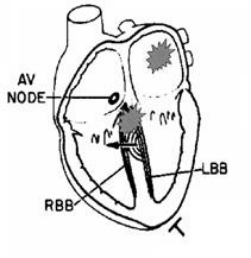


Lead II ECG from a dog who was hit by a car and bleeding. The heart rate is 270 bpm. A P, QRS, and T wave are identified for each complex and numerous physiologic causes of tachycardia are identified clinically (Pain, fear, hypovolemia, anemia, hypoxia, excitement).

3. Specific treatment is not indicated for sinus tachycardia. If an underlying etiology is present, this should be addressed (e.g. pain, hypovolemia)

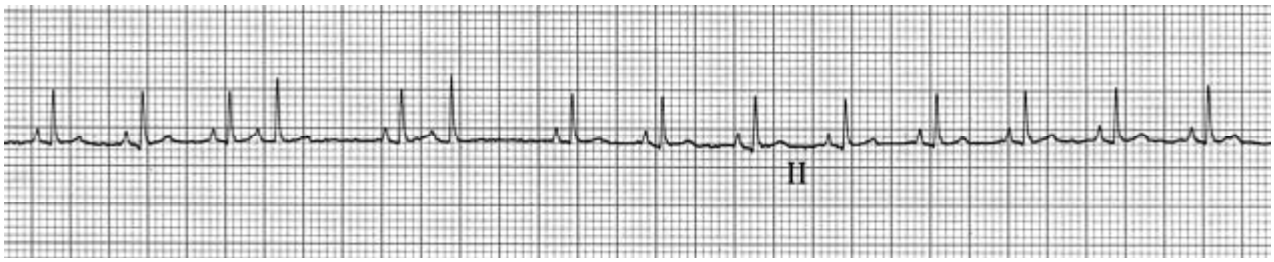
b. Supraventricular premature depolarizations (atrial, junctional)

1. Early depolarizations originating from an abnormal site anywhere above the ventricles ("supra-ventricular"). This could include the atrial myocardium or junctional tissue (proximal AV bundle, AV node, bundle of His).



2. These are single premature (faster than the SA node) beats. Typically, a premature beat will fire faster than a rate of about 160 bpm (so a coupling interval faster than 375 msec).

3. A QRS complex is present that is >90% like the normal QRS complex in all leads (because the ventricular depolarization still uses the bundle of His, bundle branches and Purkinje fibers). A P wave may or may not be present and if present, may or may not be normal in configuration and timing, depending on the site of origin of the ectopic b



Lead II ECG (25 mm/sec, standard sensitivity) from a 19 yr Mc DSH. The 4th and 6th beats are premature supraventricular complexes. These beats appear >90% like the normal sinus beats and occur prematurely. A P wave is not readily evident for these beats. The T wave of the previous normal beat has a slightly different morphology which may represent a superimposed P wave.

4. Since many times it may not be possible to distinguish a premature atrial beat from a premature junctional beat, the term **premature supraventricular beat** is preferred.

5. *Why might a P wave not be present in association with a premature supraventricular beat?*

    1. It may be buried in the previous T wave if the beat is very premature.

2. If the premature beat originates in the junctional tissue, it may not retrogradely penetrate the atria.

3. If the premature beat originates in the junctional tissue and does retrogradely penetrate the atria, this may occur coincident with the ventricular depolarization, thus burying the P wave in the QRS complex.

6. Single premature supraventricular depolarizations do **not** need to be treated. This arrhythmia is uncommon in normal dogs and therefore its recognition should prompt an investigation into the possibility of cardiac disease. Also remember that non-cardiac disease such as electrolyte abnormalities, ischemia, drug toxicities can also lead to development of arrhythmias such as these.

c. Supraventricular tachycardia (atrial, junctional)

1. Supraventricular tachycardia (SVT) is usually defined as 3 or more consecutive premature supraventricular complexes in a row.

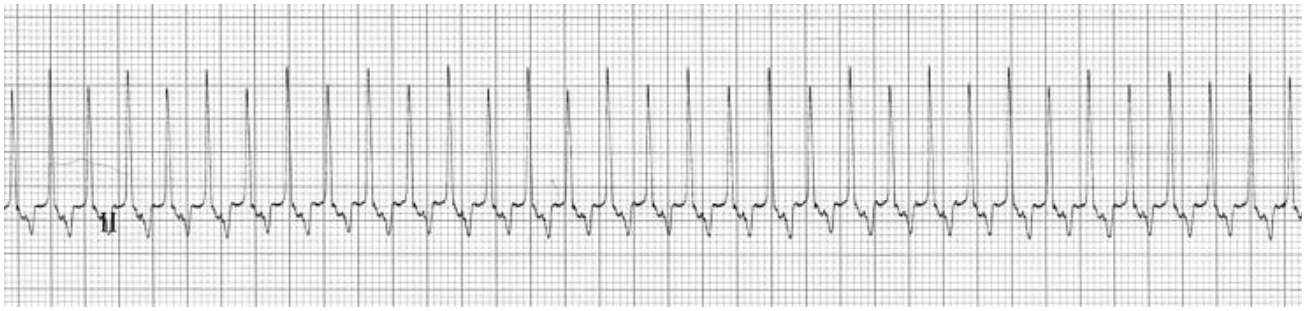
2. As discussed above for premature supraventricular depolarizations, the origin of the SVT may be atrial or junctional. The QRS configuration should be >90% like the normal QRS in all leads and a P wave may or may not be present, may or may not be normal in configuration (positive or negative) and may occur before, simultaneous with or after the QRS complex.

3. Reentry seems to be the most common mechanism and produces a regular tachycardia. These have a rapid onset and rapid termination.

4. Automatic SVTs also occur and these may be irregular (e.g. they "warm into and out of" the arrhythmia, the rate slowly increases and slowly decreases).

5. The rate for SVTs in dogs can range from 170-350 bpm.

6. Differentiation from sinus tachycardia can be difficult in some cases.



Lead II ECG (25 mm/sec, standard sensitivity) from a 16 yr Fs Husky. This is a supraventricular tachycardia (heart rate 250 bpm). P waves are evident in the ST segment confirming the origin of the tachycardia as atrial. The R wave height varies every other beat (electrical alternans) which can be seen with supraventricular tachycardias.

8. The presence or absence of clinical signs depends on the presence of absence of severe underlying myocardial disease and the rate of the SVT. SVT with rates of 250-300 bpm usually causes weakness or collapse. Perfusion is poor at these high rates because diastolic filling time is inadequate.

9. ***Treatment*** – if a dog presents for weakness or collapse, this is a medical emergency

    a. Acute therapy for SVT

        1. Physical maneuvers

            a. **Vagal maneuvers** are performed to increase vagal tone with the intent of slowing conduction through the AV node. These are often unsuccessful in veterinary medicine, possibly due to the high sympathetic tone of the patient in this situation. They are worth attempting, however and will occasionally be successful.

                1. Ocular pressure – gentle pressure on both eyes
                2. Carotid sinus massage – gentle massage of the upper neck

            b. **Precordial thump** – this is often successful for at least short term conversion of the SVT. With the dog in right lateral recumbency, the apex beat is located on the left side of the chest and thumped with the fist (strength dependent on the size of the patient). This is intended to produce a premature ventricular depolarization which may interrupt the SVT.

        2. Drugs

            a. **Calcium channel blockers intravenously** (e.g. diltiazem or verapamil).

                1. Calcium channel blockers block the slow inward calcium channel ($I_{Ca\text{-}L}$). Since this channel is present in pacemaker cells (SA node and AV node), this class of drugs often is effective for SVT by slowing conduction through the AV node. They work by prolonging the refractory period of the AV node and allowing less of the impulses coming from an SVT to traverse the AV node. If conduction is slowed enough, the depolarization will be blocked.

                2. If the SVT has a reentrant mechanism and the AV node is part of the circuit, a calcium channel blocker should be effective by slowing conduction through the AV node to the point of breaking the SVT.

                3. If the SVT originates in atrial tissue and does not use the AV node for its circuit, calcium channel blockers will not break the SVT but may still be effective by slowing the ventricular response rate (i.e. the number of atrial impulses tranversing the AV node and causing a ventricular depolarization) through the slowing of AV node conduction and prolonging its refractory period (i.e. causing AV block).

b. **Beta blockers intravenously** (e.g. esmolol)

1. Beta blockers antagonize the actions of sympathetic stimulation and therefore have negative chronotropic effects (slowing of the heart rate) and negative inotropic effects (decreased contractile force generation). Their negative chronotropic effects result in slowing of AV node conduction and prolonging the refractory period of the AV node.

2. Esmolol is short lived (seconds) and so if a response occurs, it can be given as a CRI.

3. Moderate to severe myocardial failure is a *relative* contraindication to the administration of beta blockers due to their negative inotropic effects (they decrease contractility).

b. Chronic therapy for SVT

1. **Digoxin** – digoxin has several pharmacologic effects. One of these is its effect on autonomic tone – it serves to increase vagal tone and decrease sympathetic tone. This can prove effective for treatment of SVT by prolonging conduction in the AV node.

2. **Calcium channel blockers** (diltiazem for long term use)

3. **Beta blockers** (especially atenolol for long term use)

4. **Class III antiarrhythmics** (e.g. sotalol, amiodarone) – these agents block potassium channels and thus prolong repolarization. They also block fast sodium channels and slow calcium channels as well as have anti-adrenergic properties.

d. Atrial flutter

1. Uncommon

2. Atrial flutter is a very fast SVT and the atrial rates are between 350-450 bpm. The atrial rate is so fast that some beats are blocked in the AV node because the depolarization rate exceeds the refractory period of the AV node. This produces a functional second degree AV block.

3. Treatment

a. Digoxin
b. Calcium channel blockers
c. Beta blockers

e. Atrial fibrillation (AF)

1. It is very important that you learn to recognize and feel comfortable treating atrial fibrillation, it is probably the most common arrhythmia you will see!!!!!!

2. Initiation of atrial fibrillation requires that there is a “critical mass”. Meaning, you need a large atrium to set up and sustain atrial fibrillation. This is why it is seen commonly in large breed dogs and horses and uncommonly in small breeds dogs and cats.

3. During this arrhythmia, the atria fibrillate at a rate of 500-600 bpm. Because the atrial depolarization is chaotic, organized atrial contraction does not occur.

4. These many depolarizations constantly bombard the AV node, which cannot conduct all of them to the ventricle. The pattern for which atrial depolarizations are conducted through the AV node is irregular resulting in an irregular ventricular response rate. The number of impulses that reach the ventricle is dependent on the refractory period of the AV node, its conduction characteristics and autonomic tone (high sympathetic tone will shorten the AV node refractory period and allow more impulses to get through, high vagal tone will allow fewer impulses to get through).

5. *Two factors predispose to the development of atrial fibrillation*.

    a. <u>Large atrial mass</u>

        1. A large atrial mass is necessary to sustain AF; large hearts fibrillate more easily than small hearts.

        2. Horses can develop AF in the absence of heart disease since their atrial mass is large normally.

        3. Giant and large breed dogs can occasionally develop AF in the absence of heart disease (“lone AF”).

        4. Small and medium sized dogs require significant cardiac disease (atrial enlargement due to pathology) to sustain AF.

        5. AF occurs in cats occasionally and only if severe atrial enlargement is present.

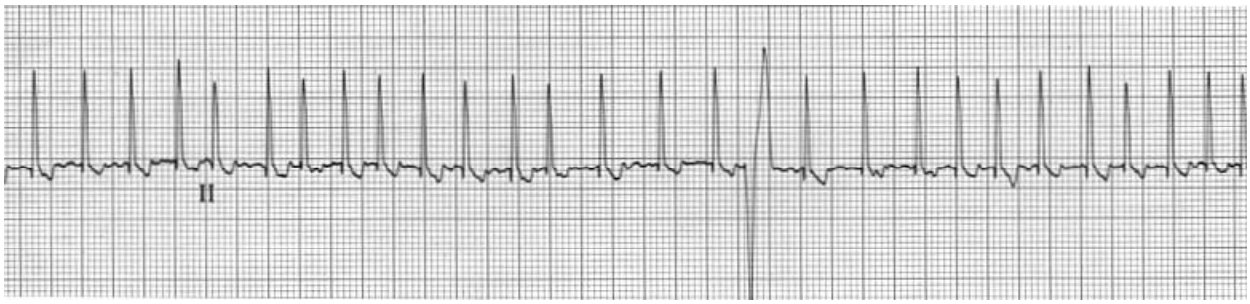
    b. <u>Elevated vagal tone</u>

        1. Both sympathetic and vagal tone shorten the refractory period in the atria and the elevation of both simultaneously may predispose to AF (e.g. pain and stress of a GDV with narcotic drugs).

        2. Vagal tone increases the heterogeneity of atrial refractoriness (allows for different refractory periods within the atria by unequal shortening of the refractory period) which can predispose to multiple reentrant wavelets (atrial fibrillation).

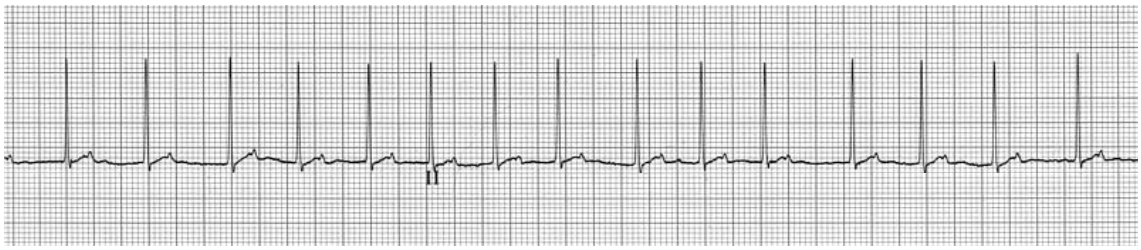
6. ECG diagnosis of AF

1. Rapid heart rate (usually greater than 200 bpm)
2. Supraventricular morphology to the QRS complexes (narrow and upright in lead II).
3. The rhythm is irregularly irregular – there is no identifiable pattern.
4. P waves cannot be identified.
5. The baseline *may* have irregular undulations (fibrillation waves).
6. There may be variation in the height of the QRS complex.
7. ****Note that when atrial fibrillation gets very fast (>250 bpm) it can be very difficult to pick out the irregularity of it. You need to run lots of ECG to be able to pick up the irregularities. It is also helpful to run the ECG at 50 mm/sec.********
8. ***If you see a rapid irregular supraventricular tachycardia without identifiable p waves, it is atrial fibrillation until proven otherwise! If you think it is an alternative diagnosis, you must prove to yourself that this is not atrial fibrillation, because it probably is!!!!***



Lead II ECG (25 mm/sec, standard sensitivity) from a 12 year old Rottweiler with dilated cardiomyopathy. The predominant rhythm is atrial fibrillation (irregularly irregular rapid supraventricular rhythm, average rate 220 bpm, no obvious P waves). One PVC is present.

b. Primary "Lone" AF
   i. In Lone AF, significant underlying cardiac disease is not present. Therefore resting sympathetic tone is not elevated and the heart rate is *usually* relatively normal (100-140 bpm in the large breed dogs in which this occurs).
   ii. The rhythm is still supraventricular, irregularly irregular and no P waves are identifiable.
   iii. There is some thought that this is a precursor to the development of dilated cardiomyopathy in the future in these dogs (e.g. Irish Wolfhound).



Lead II ECG (25 mm/sec, standard sensitivity) from a 7 yr Irish Wolfhound with a structurally normal heart. Again, the rhythm is irregularly irregular, supraventricular and no P waves are present. The average rate is normal (130 bpm). This is lone AF.

7. *Hemodynamic consequences of AF*

    a. The hemodynamic consequences of Lone AF are minimal since underlying heart disease is not present and the rate is usually slow.

    b. Since atrial contraction contributes about 20-25% to the cardiac output ("atrial kick"), the loss of atrial contraction in horses with AF and no underlying heart disease is usually only evident when exercised. Decreased performance, usually in racehorses, is the typical chief complaint, while no signs are evident at rest.

    c. The loss of atrial contraction due to the development of AF in a dog with serious heart disease can be very detrimental.

        1. Stroke volume and cardiac output decline and end diastolic pressure increases.
        2. Clinical deterioration occurs, possibly with the development of congestive heart failure
        3. The sustained increase in heart rate can induce further myocardial systolic dysfunction over several weeks (tachycardiomyopathy).

8. *Clinical features of atrial fibrillation*

    a. Erratic cardiac rhythm and varying intensity of the heart sounds ("tennis shoes in a dryer").

    b. Pulse deficits and variation in pulse quality (the strength of the pulse depends on the preceding diastolic interval which varies due to the irregular heart rate). Pulse deficits are common and a heart rate based off pulse rate is inaccurate.

    c. It is difficult to count a heart rate by auscultation in AF due to the usual rapid and irregular nature of the arrhythmia. It is always best to determine heart rate off an ECG with AF.

9. *Treatment of atrial fibrillation*

    a. <u>Conversion to sinus rhythm ('rhythm control')</u>

        1. This is often not a realistic goal. The presence of severe underlying heart disease usually makes these dogs refractory to successful conversion.

        2. The need for conversion in Lone AF is controversial. The ventricular response rate is slow in these dogs (or can be controlled with medication if it is rapid) and they are asymptomatic, making the need for conversion debatable. In human medicine, rate control has been shown to be as good as rhythm control.

        3. Conversion is also difficult in long standing atrial fibrillation. "Atrial fibrillation begets atrial fibrillation" meaning the longer standing the AF, the more likely you are to sustain this rhythm. Many electrical changes occur within the atria during AF which preclude the ability to convert. In general, it is less likely that conversion will occur if you have been in AF for longer than 3

weeks. The less time you have been in AF, the more likely conversion will be possible.

4. Conversion can be attempted with medical therapy (amiodarone) or with electrical current (DC conversion).

b. Control of heart rate ('rate control') with medical therapy is usually the treatment used in dogs and cats. Drugs that slow AV node conduction are used to slow the ventricular response rate to AF. These drugs increase the AV node refractory period and therefore allow less impulses to traverse the AV node.

1. Digoxin

a. Increases vagal tone thereby increasing the refractory period of the AV node and slowing conduction. This decreases the number of depolarizations that are conducted to the ventricle.
b. This is usually used first because it is a weak positive inotrope (as opposed to the remaining drugs which are negative inotropes) and therefore will not depress contractility.
c. It usually only slows the HR 10-30 bpm and therefore often a second drug is necessary to slow the heart rate further.

2. Calcium channel blockers

a. Slow conduction through the AV node (see for SVT)
b. Diltiazem

3. Beta blockers

1. Decreases sympathetic tone resulting in slowed conduction through the AV node (see SVT)
2. Atenolol and propranolol are the most commonly used beta blockers.
3. Again, caution is used with beta blockers when myocardial failure is present. These are not contraindicated but must be started low and tapered to a dose that can be tolerated by the patient.

NOTE: therapy efficacy should ideally be assessed by a 24 hour Holter monitor. A recent study has shown an increased survival in AF dogs with a 24-hour mean heart rate < 125 bpm (Pedro et al, JVIM 2017). Therefore in a 'rate control' approach one should aim for a ventricular response rate <125 bpm (mean HR in 24 h).

VENTRICULAR TACHYARRHYTHMIAS

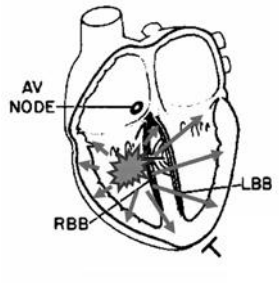
a. Ventricular premature depolarizations (single, bigeminy, trigeminy etc)

1. **VPCs** (ventricular premature contractions), **VPDs** (ventricular premature depolarizations), **PVCs** (premature ventricular contractions) = one and the same

2. *ECG characteristics*

   a. Premature (earlier than the sinus beat)

   b. Wide and bizarre QRS (<90% like the normal beat). This is because the VPC originates below the bundle of His and therefore cannot take advantage of the specialized conduction system. It therefore must depolarize the ventricles muscle cell to muscle cell. This is relatively slow and produces a wide bizarre complex. QRS complexes can be positive in lead II or negative, depending on the site of origin.



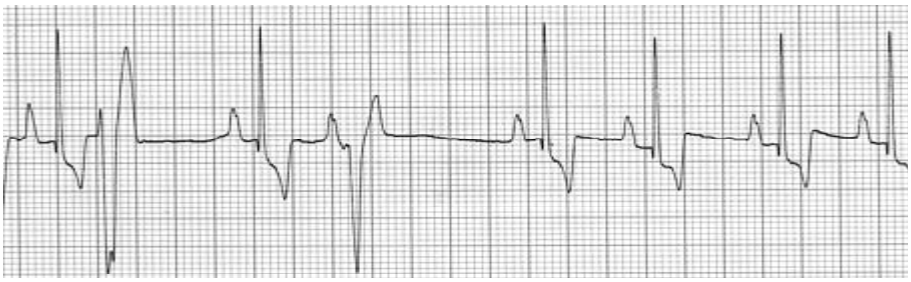
   c. No related P wave

   d. Large bizarre T wave. Because depolarization is abnormal, repolarization is also abnormal.

3. *VPCs can occur in patterns*

   a. Ventricular bigeminy – every other beat is a VPC
   b. Ventricular trigeminy – every 3rd beat is a VPC

4. *R on T phenomonon* – if the VPC fires rapidly and occurs on the downslope of the T wave of the sinus beat it is called "R on T". The downslope of the T wave is the vulnerable period and depolarization during this time may predispose to ventricular fibrillation.



Lead II ECG (25 mm/sec, standard sensitivity) from a 1yr F Retriever with dilated cardiomyopathy. The 2nd and 4th complex are premature ventricular contractions. The 2nd PVC occurs just after the sinus node depolarizes (P wave in front of PVC. Other abnormalities on this trace include wide and tall, notched P waves and ST segment depression.

   a. Can be seen in normal animals in *low* numbers (less than 50 per 24 hour period on a Holter monitor)

b. Very commonly in association with non-cardiac disease. Possibly due to myocardial ischemia, electrolye abnormalities or other factors surrounding the underlying disease.

1. Post trauma (e.g. traumatic myocarditis)
2. Post surgery
3. Gastric dilatation and volvulus
4. Splenic disease
5. Other systemic diseases

c. Anesthetic causes

d. Primary myocardial diseases (especially Doberman pinschers with DCM and Boxers with ARVC or boxer cardiomyopathy)¨

e. Myocarditis (rare)

6. Treatment

a. Clinical signs do not occur with single VPCs.

b. Dogs with VPCs secondary to systemic disease often do not need to be treated and the arrhythmias tend to be self limiting if the underlying disease is addressed.

c. Dogs with single VPCs and underlying myocardial disease may or may not need to be treated. Usually this indicates the need to further investigate the arrhythmia (e.g. with Holter analysis) to determine if more serious arrhythmias are occurring at other times.

d. Since dogs with VPCs do not show clinical signs associated with only single premature beats, alleviation of clinical signs is not a reasonable reason to treat. In this instance, the reason to treat is to attempt to prevent sudden death. However our ability to predict the likelihood of sudden death associated with VPCs is poor.

e. It appears that in dogs with VPCs and no underlying heart disease, the number of VPCs is not predictive of the risk of sudden death and that risk appears to be low.

f. In the presence of myocardial disease, the presence of VPCs may indicate an increased risk of sudden death or they may simply be a marker for the severity of the disease (e.g. Doberman pinschers with dilated cardiomyopathy).

g. The presence of VPCs in Boxers with familial ventricular arrhythmias may indicate an increased risk of sudden death.

h. If deemed necessary, the treatment is the same for ventricular tachycardias.

b. Ventricular tachycardia (VT)

1. A ventricular couplet is two consecutive VPCs, a ventricular triplet is 3 consecutive VPCs and ventricular tachycardia is >3 consecutive VPCs.

2. Ventricular tachycardia is sustained if it lasts >30 seconds and nonsustained if it lasts <30 seconds.

3. <u>Ventricular tachycardia vs. accelerated idioventricular rhythm</u>

a. A ventricular rhythm that is less than approximately 140-170 bpm is not truly a tachycardia and is therefore best termed an accelerated idioventricular rhythm (this is a fancy term for 'slow ventricular tachycardia' but is an appropriate name because a ventricular myocyte is now firing as a pacemaker cell at an accelerated rate).

b. A true ventricular tachycardia is a ventricular rhythm that is truly tachycardic (rate >170 bpm).

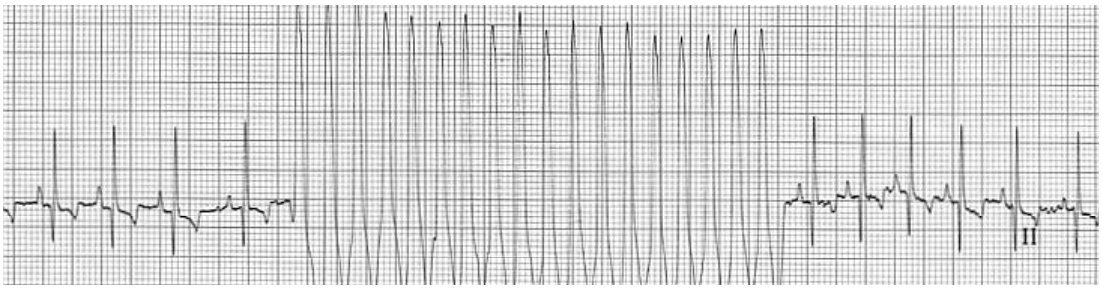
c. Accelerated idioventricular rhythms are seen commonly in hospitalized patients in association with non-cardiac diseases (e.g. GDV surgery, splenic disease, pancreatitis, prostatitis, post trauma and neurologic disease). These are often benign, the rate is slow and they often do not require therapy.

4. *ECG diagnosis of ventricular tachycardia*

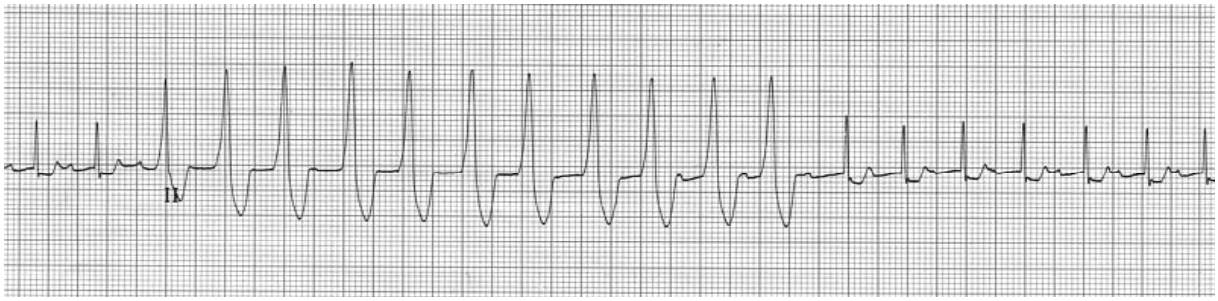
a. The morphology of the QRS complex is wide and bizarre as it is for VPCs. The shape and orientation depends on the origin of the ectopic depolarization and the conduction pathway.

b. They can be monomorphic (one morphology) or polymorphic (several different morphologies). The latter might imply multifocal origins for the arrhythmias, however different pathways may also produce multiform complexes.

c. Most VTs are regular however they may be irregular.



Lead II ECG (25 mm/sec, standard sensitivity) from a 5yr Fs Boxer. Paroxysmal ventricular tachycardia is present. The intrinsic rate of the VT is very rapid (370 bpm). The VT morphology is upright or similar to a left bundle branch block. This is common in boxers with cardiomyopathy.



Lead II ECG (25 mm/sec, half sensitivity 5mm/mV) from a 2 yr M Pitbull with renal failure. The sinus rhythm is interrupted by an accelerated idioventricular rhythm. Although the abnormal beats are ventricular in origin, the rate is relatively slow and similar to the sinus rate (approx 150 bpm). This is a benign rhythm that does not require therapy. 1st degree AV block is present during sinus rhythm.

5. *Clinical signs of VT*

a. If the rate of the VT is slow, no clinical signs may be present.

b. If the rate of the VT is rapid (greater than 250-300 bpm) signs of poor cardiac output may be present or syncope may occur.

c. Clinical signs will be influenced by the degree of underlying heart disease.

6. *Treatment of VT*

a. <u>The goals of treatment</u>

1. To improve hemodynamics
2. To prevent sudden death

b. <u>Making the decision to treat or not to treat</u>

1. Is this arrhythmia causing hemodynamic compromise?

a. A healthy heart can tolerate much abuse whereas a diseased heart cannot. Therefore the presence or absence of underlying cardiac disease is an important factor in this decision.
b. Evaluate peripheral pulse quality, mucous membrane color, attitude of the patient and arterial blood pressure.
c. Sustained VT at a high rate is more likely to cause hemodynamic compromise than VT at slower rates.

2. Is this arrhythmia likely to degenerate into ventricular fibrillation, which will cause sudden death?

a. Again the presence or absence of underlying cardiac disease is important since a healthy heart can tolerate more than a diseased heart.
b. The faster the VT, the more likely that a beat will fall within the vulnerable period and induce ventricular fibrillation (R on T phenomenon).
c. Polymorphic VT is thought to be more dangerous than monomorphic VT
d. Certain patient groups appear to have an increased risk of sudden death associated with ventricular tachycardia

1. Doberman pinchers with dilated cardiomyopathy
2. Boxers with ARVC (formerly known as Boxer cardiomyopathy)
3. Dogs with severe subaortic stenosis
4. German shepherds with inherited ventricular arrhythmias
5. Cats with hypertrophic cardiomyopathy

c. <u>Therapy of VT</u>

1. Acute therapy of VT

a. Lidocaine is the initial drug of choice

i. Class IB antiarrhythmic (blocks fast sodium channels)
ii. Only available for intravenous use

iii. Given as a bolus or as a CRI
iv. Reasons for lack of efficacy
   a. Hypokalemia
   b. Incorrect diagnosis (lidocaine is ineffective for supraventricular arrhythmias)
   c. Inappropriately low dose
   d. Rarely a VT may not respond to lidocaine
   e. Idioventricular rhythms also do not respond well to lidocaine administration. The slower the VT, the less likely it is to respond. Another reason to not treat slow VT
   f. Note that 'response' may be simply a slowing of the VT and not breaking the VT
v. Toxicity (neurologic signs at high doses, cats are more sensitive)

b. Procainamide can be tried if lidocaine is ineffective

c. Beta blockers can be tried

d. Magnesium may be effective in some cases.

e. Be certain that the patient is hydrated, acid base status is normal and electrolyte concentrations are normal.

2. *Chronic therapy of VT*

a. Class I

1. Mexilitine is the most commonly used oral drug in this class for ventricular arrhythmias. A combination of mexilitine and atenolol or sotalol has been shown to be effective for the reduction of ventricular ectopy in boxers with ARVC (boxer cardiomyopathy).

b. Class II agents (beta blockers)

1. These can be mildly effective for ventricular arrhythmias but are usually used in combination with other drugs. They are contraindicated in moderate to severe myocardial failure due to their negative inotropic effects
2. Atenolol is the most commonly used beta blocker.

c. Class III agents (prolong the action potential)

1. Sotalol has been shown to be effective for the reduction of ventricular tachycardia in Boxers with ARVC. Since sotalol also has some beta blocking properties it must be used with caution in dogs with myocardial failure.
2. Amiodarone is very effective for refractory arrhythmias, however may have significant side effects (e.g. hepatotoxicity, hypothyroidism)

THERAPEUTIC CONSIDERATIONS FOR TACHYARRHYTHMIAS

The decision to treat an animal specifically for an arrhythmia is a controversial topic.

A. The need for antiarrhythmic treatment should first be determined.

1. Presence or absence of clinical signs.

2. Are there electrolyte or acid-base abnormalities that can be corrected?

3. Is myocardial function normal or abnormal?

    a. A structurally normal heart can tolerate arrhythmias better than a diseased heart.
    b. Most antiarrhythmics have negative inotropic actions which may cause decompensation in a patient with severe myocardial failure

4. Is it likely that the arrhythmia will degenerate into a fatal arrhythmia?

    a. If only we knew!!
    b. The **faster** the arrhythmia, the more dangerous.
    c. **Polymorphism** is thought to be more malignant than monomorphism.
    d. **Repetitive forms** are much much more dangerous than single abnormal beats.
    e. **Ventricular arrhythmias** are more likely to cause a fatal arrhythmia than supraventricular ones.
    f. In certain **breeds with certain diseases** (e.g. boxer ARVC, dobermans with DCM, cats with HCM, dogs with severe SAS), arrhythmias are thought to be more dangerous than in other breeds and diseases.
    g. <u>Arrhythmias that are most likely not dangerous and do not require treatment</u>:

        Single supraventricular complexes
        Single ventricular complexes (ideally frequency and complexity of VPCs should be assessed with a 24 hour Holter monitor!)

    h. <u>Arrhythmias that are probably not dangerous and usually do not require treatment:</u>

        Accelerated idioventricular rhythms

    i. <u>Arrhythmias that are most likely dangerous and require treatment</u>:

        Rapid nonsustained or sustained ventricular tachycardias
        Rapid nonsustained or sustained supraventricular tachycardias

B. Can the animal be appropriately monitored during therapy?

With any form of antiarrhythmic therapy, regular recheck intervals and monitoring is needed. The reason for this is not only to ensure efficacy of treatment, but to ensure that the arrhythmia has not gotten worse on therapy. Remember that any antiarrhythmic agent can also be proarrhythmic. Rechecks are usually performed via both echocardiographic evaluation and Holter evaluation. Holters are performed in order to obtain a longer duration of monitoring.

Flow charts below summarise the approach to Narrow-QRS and Wide-QRS tachycardias. These are part of the BSAVA Manual of Canine and Feline Emergency and Critical Care, 3rd Edition; Chapter 6 Cardiovascular Emergencies; Jose Novo Matos & Nuala Summerfield