Management of Life-Threatening Arrhythmias in Cats

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Introduction

Cardiac arrhythmias are the result of abnormalities in cardiac conduction. Cardiac arrhythmias are classified according to the rate of the rhythm (tachycardia vs. bradycardia) and their origin (ventricular vs. supraventricular). They are also sub-classified as premature or escape, and refined according to fine location (junctional vs, non-junctional), and behavioural (re-entrant etc.).

Whilst many arrhythmias may not result in clinical signs of illness, clinically significant arrhythmias can produce a range of clinical signs referrable not only to the abnormal cardiac rhythm itself, but also related to underlying disease. It is the aim of this short article to focus on clinically significant, life-threatening cardiac arrhythmia diagnosis and management.

Causes of Arrhythmias in the Cat

Cardiac arrhythmias can develop as a result of cardiac disease, or non-cardiac disease

Cardiac causes of arrhythmias include:

- Cardiomyopathy (dilative or hypertrophic)
- Congenital heart defects
- Endocarditis
- Myocarditis – including traumatic/trauma-related myocarditis
- Pericardial disease
- Congestive heart failure from any cause
- Cardiac neoplasia

Non-cardiac causes of arrhythmias include:

- Hyperthyroidism
- Electrolyte abnormalities
    - Calcium disorders
    - Potassium disorders
    - Magnesium disorders
- Anaemia of any cause
- Trauma
- Neoplasia – primary cardiac, secondary metastatic neoplasia, paraneoplastic syndromes
- Drug/medication-related – anaesthetic medications, toxins, cardiac medications

Clinical Signs of Life-Threatening Arrhythmias

Cats may not show any symptoms of abnormal cardiac rhythm if the rhythm does not disrupt cardiac output to a significant degree. However, as cardiac output becomes impaired as a result of the arrhythmia, clinical signs may manifest in one or more of the following:

- Depressed mentation
- Weakness
- Collapse
- Dyspnoea
- Tachypnoea
- Sudden death

Physical examination findings may include:

- Tachypnoea
- Dyspnoea
- Abnormal lung sounds
    - Rales
    - Crackles
    - Reduced lung sounds
- Cyanosis
- Hypothermia
- Hypotension
- Weak and/or irregular pulses
- Evidence of underlying cause (urethral obstruction, thyroid enlargement etc.)

Diagnosis of Arrhythmias in the Cat

The diagnosis of an arrhythmia relies on electrocardiography. Further diagnostic tests are usually required to ascertain the presence of primary or secondary heart disease, and to also evaluate the patient for the presence of underlying disease, and also co-morbidities associated with poor cardiac output resulting from the arrhythmia.

The diagnostic tests that may be applied to the cat with abnormal cardiac rhythm includes:

- ECG analysis
- Cardiac ultrasound
- TFAST ultrasound
- Complete blood count
- Serum biochemistry analysis
- Serum electrolyte analysis
- Urine analysis
- Blood gas and blood lactate
- Blood pressure
- Abdominal ultrasound

Regarding ECG analysis, it is important to note that the cat has some peculiarities when compared to the dog:

- Sinus tachycardia is common in cats, due to anxiety and heightened arousal in the veterinary setting
- A heart rate of less than 160/min on physical examination may indicate relative bradycardia, and warrants investigation
- QRS complexes in normal cats may appear inverted/negative, because cats have a wide range of mean electrical axis within the ventricular muscle
- QRS complexes in cats can be very small. No minimum height of the R wave is recognised in cats in lead II

The Arrhythmias:

As in dogs, feline arrhythmias are classified as either ventricular in origin, or supraventricular in origin. Ventricular arrhythmias are characterised by wide, bizarre QRS complexes, whereas supraventricular arrhythmias are characterised by narrow (normal) QRS complexes.

In addition to the ventricular vs. supraventricular distinction, feline (as well as canine) arrhythmias are also classified according to the heart rate produced – bradyarrhythmias or tachyarrhythmias.

Bradyarrhythmias:

Sinus Bradycardia

Sinus bradycardia is a regular rhythm of normal appearance that originates from the sino-atrial node, but occurs at a rate that is inappropriately slow. Sinus bradycardia is abnormal in cats unless they are well acclimated to the practice, and the presence of a serious underlying disorder should be investigated.

Common causes of sinus bradycardia in cats are listed in the table below:

• Hypothermia
• Excess vagal tone (thoracic or abdominal disease, neoplasia etc.)
• Cardiomyopathy
• Trauma
• Shock (hypovolaemia, haemorrhagic)
• Anaphylaxis
• Neurological disease (including traumatic brain injury)
• Feline dysautonomia
	• Medications
	  ○ Beta blockers
	  ○ Calcium channel blockers
	  ○ Digitalis
	  ○ Morphine
	  ○ Methadone
	  ○ Alpha-2 agonists
	• Sepsis
	• Hyperkalaemia, hypokalaemia
	• Hypercalcaemia, hypocalcaemia

Clinical signs of sinus bradycardia usually include symptoms of lethargy and depressed appetite, along with symptoms associated with underlying disease e.g. urethral obstruction and associated hyperkalaemia.

Treatment is usually unnecessary unless clinical signs are evident. The requirement for treatment also depends on the underlying cause

- Acute treatment
    - Draw blood for analysis of electrolyte levels (calcium, potassium, acid/base status) and serum drug concentrations (digoxin)
    - Atropine at 0.01-0.04 mg/kg slow IV, isoproterenol may be administered in clinically unwell patients that do not respond to atropine in the acute setting. Glycopyrrolate 0.005-0.01 mg/kg IV may be used instead of atropine. Alternatively, terbutaline 5-10 micrograms/kg SC may be used.
    - Provide a fluid bolus – this is an essential component of acute management of severe sinus bradycardia caused by drugs, electrolyte disturbances, or head trauma. The fluid of choice is lactated Ringer's solution, or 0.9% sodium chloride. Administer a bolus of 5-7 ml/kg given rapidly intravenously over 10 minutes. The fluid bolus achieves dilution of serum electrolytes, dilution of drugs, and an immediate increase in preload, afterload, and cardiac output. Titrate fluid therapy beyond the initial fluid bolus according to the response to therapy and the fluid requirements of the patient
- Pacemaker therapy is rarely required, but may be considered in cases refractory to chronic medical therapy.

Atrial Standstill

Atrial standstill occurs when the atrial myocardium is unable to depolarize. QRS complexes may be normal (narrow and tall) or abnormal (wide and bizarre) in appearance. There are no P waves present.

Hyperkalaemia is the most common cause of atrial standstill in the cat – from endocrinopathy (DKA); urinary tract rupture, acute kidney injury, sepsis, urethral obstruction.

Clinical signs are similar to those of other bradyarrhythmias, and include weakness, ataxia, stupor, coma, sudden death. Symptoms of acute congestive heart failure may be present in patients with underlying heart disease.

An ECG diagnosis is made based on the presence of a slow heart rate, regular rhythm, and ventricular escape rhythms – wither junctional (normal QRS complexes) or non-junctional (wide QRS complexes). It is important that these complexes are not suppressed with lignocaine or other anti-arrhythmic drugs, or cardiac arrest will occur. In patients with hyperkalaemia, junctional escape complexes may have tall, spiked T waves – often similar in size to QRS complexes – coupled with abnormal (wide and bizarre) QRS complexes

Treatment is directed at identifying and correcting hyperkalaemia (if present), and correcting or managing the underlying cause.

- Acute Treatment
    - Obtain blood for serum biochemistry and electrolyte analysis
    - Treat hyperkalemia with an intravenous bolus of lactated Ringer's solution (or 0.9% NaCl) at 5-7 ml/kg IV over 10 minutes, followed by a glucose bolus (7-10% solution at 5 ml/kg IV over 10 minutes) together with regular insulin 0.1 unit/kg IV. Calcium gluconate 10% given at 0.2-1.0 ml/kg given SLOW IV may be used in refractory cases, where atrial standstill persists despite treatment for hyperkalaemia.
    - In patients without hyperkalaemia, administer atropine 0.01 mg/kg slow IV or SC as a trial
    - Cautious use of intravenous fluids in patients with underlying cardiac disease is advised due to the presence of atrial disease, and the risk of development of congestive heart failure.

Sinus Arrest and Sinus Block

Sinus arrest is defined as a complete loss of SA node automaticity. Sinus block results from failure of impulses formed in the sino-atrial node to depolarize the atria, or results in a delay in atrial depolarization. Either condition may produce loss of atrial depolarization, and ventricular asystole if secondary pacemakers do not initial ventricular depolarization.

Sinus arrest in the cat is caused most commonly by atrial disease, leading to failure of the SA node to depolarize for extended periods of time. As such, it is most commonly seen in cats with marked atrial enlargement secondary to heart disease (cardiomyopathy), in patients with neoplastic infiltration of the atrial myocardium, secondary to some cardiac medications (beta-adrenergic blockers, class 1a antiarrhythmic drugs etc.), and in patients with hyperkalaemia or increased vagal tone due to concurrent disease.

Clinical signs are referrable to intermittent decreases in cardiac output associated with the arrythmia, and include syncope, weakness and occasional loss of consciousness. Symptoms of congestive heart failure, and sudden death are also noted.

An ECG diagnosis is made based on the presence of sinus rhythm, containing intermittent periods of electrical inactivity lasting longer than 2 P-P intervals. Occasional junctional or non-junctional ventricular escape complexes may also be present. A syndrome of intermittent supraventricular tachycardia is observed in some patients (bradycardia-tachycardia syndrome).

Treatment involves identification of the underlying cause (electrolyte disorder, neoplasia etc.) and in patients with primary cardiac disease, anticholinergic drug therapy, +/- terbutaline and in patients in which the arrhythmia persists despite treatment of the underlying disease, pacemaker implantation is recommended.

Atrio-Ventricular Block (AV Block)

Atrio-ventricular block exists when there is a delay or complete block of atrial impulse conduction through the atrio-ventricular node

First degree AV Block

First degree AV block is characterised by a prolonged P-R interval. Causes include increased vagal tone, cardiomyopathy, electrolyte disturbances (hyperkalaemia), endocarditis (including traumatic endocarditis), beta-adrenergic blockers, calcium channel blockers, and sedative medications.

Treatment involves identification and management of the underlying cause.

Second Degree AV Block

There are three (3) types of second-degree AV block:

- Mobitz Type I (Wenckebach) - gradual increase in P-R interval until a P wave occurs without an R wave. The QRS complex is normal duration. Mobitz Type 1 AV block occurs within the AV node tissue
- Mobitz Type II - P-R interval constant but intermittent non-conducting P wave (P wave occurs without an R wave). Mobitz Type II AV block occurs within or below the Bundle of His (referred to as an infra-nodal block). Because the conduction block occurs below the His Bundle bifurcation, the QRS complexes may demonstrate abnormal morphology.
- Advanced 2nd Degree Heart Block - only one beat in a group is conducted to the ventricles - in ratios of 2:1, 3:1, 4:1 etc.

The cause of 2nd-degree AV block are similar to those causing 1st-degree AV block. Mobitz Type I AV block generally occurs secondary to increased vagal tone, or medications that depress AV node conduction, such as beta-adrenergic blockers, morphine, and calcium channel blockers. Mobitz Type II AV block is associated with organic heart disease, electrolyte abnormalities, and medications as for Mobitz Type I AV block.

Patients with 2nd-degree AV block usually present with symptoms referrable to reduced cardiac output +/- their underlying disease (if present) – with weakness, syncope and symptoms of congestive heart failure being most common.

Diagnostic evaluation should include full blood evaluation, urine analysis, echocardiogram and thoracic or abdominal imaging, depending on the suspected aetiology.

Treatment and clinical management of 2nd degree AV block involves increasing the heart rate, and management of the underlying cause

- Treatment of Mobitz Type I AV block is generally not required; monitor electrolyte and acid-base status and treat as required
- Mobitz Type II AV block is managed in the acute setting with administration of an atropine trial, glycopyrrolate, terbutaline or dobutamine administration.
- Management of symptoms of acute congestive heart failure may be managed by administration of furosemide, and benazepril as indicated by the patients' condition.
- Definitive management for long-term management is cardiac pacemaker implantation

Third Degree AV Block

Complete AV block occurs when all atrial impulses are blocked at the AV node or Bundle of His. An independent idioventricular pacemaker coordinates ventricular depolarization. As a result, the atria and ventricles are controlled by independent pacemakers.

- P waves, when present, have no fixed relationship to QRS complexes
- Ventricular rate is slow
- Ventricular escape beats are present. When ventricular escape complexes originate from the AV junction, they have normal morphology; when they originate from below the His Bundle bifurcation, they will be wide and bizarre in appearance

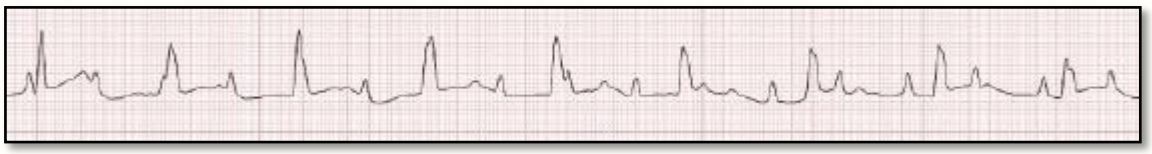


Image showing non-junctional escape rhythm at a rate of 120 beats per minute in a 15-year-old cat with HCM and 3rd degree AV block. Note the independent rates of atrial and ventricular depolarisations. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

The causes of 3rd degree AV block are similar to those for 1st and 2nd degree AV block, including cardiomyopathy, atrioventricular infarction, cardiac inflammation, sepsis and neoplasia, along with advanced heart disease secondary to inherited cardiac defects.

As with other forms of AV block, clinical signs are generally referrable to the decreased in cardiac output, the severity of the underlying cause, and the rate of the ventricular escape rhythm. Typical clinical signs include weakness, syncope, collapse, seizures, congestive heart failure (tachypnoea etc.) or sudden death.

Diagnostic evaluation should include full blood evaluation, urine analysis, echocardiogram and thoracic or abdominal imaging, depending on the suspected aetiology.

Medical treatment has limited benefits for long-term management. However, emergency treatment and stabilization can be life-saving. The use of atropine, glycopyrrolate, or terbutaline in the emergency setting is preferred. Oral medication with propantheline bromide or terbutaline may be useful prior to cardiac pacing implantation.

Escape rhythms

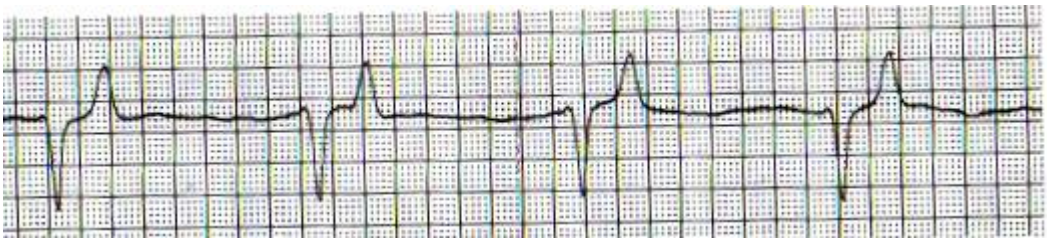
Escape rhythms arise from a physiologic (as opposed to pathologic) ectopic focus, when a superior focus (pacemaker) fails to fire. Escape rhythms originate from pacemaker cells that have a slower rate of discharge than that of the sino-atrial node, and therefore only appear when there is a sinus arrest, or sinus (sino-atrial, or atrioventricular) block. The ectopic pacemaker foci exist in the heart at one of two levels – supraventricular, or ventricular.

For junctional supraventricular escape rhythms, the QRS is generally narrow, with or without a P wave; the escape rhythm rate is 40-70 beats per minute (bpm)

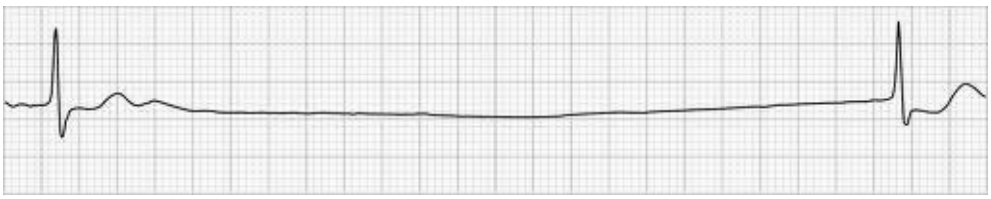
For ventricular escape rhythms, the QRS is generally wide, and resembles a VPC in appearance; the escape rhythm rate is 20-40 bpm.

Escape rhythms are generally regular. However, the ECG tracing will depend on the presence and type of rhythm disturbance that lead to the appearance of the escape rhythm in the first place

ECG of ventricular escape complexes



Non-junctional escape complexes in a patient with sinus arrest. Image: Tilley, L.P., "Essentials of Canine and Feline Electrocardiography" Tilley (Ed) LWW (1992)



Junctional escape complexes in a patient with sinus arrest. The narrow QRS complexes with T-waves suggests ventricular depolarization originates in junctional (AV node) tissue

Tachyarrhythmias

Pathological tachycardia should be suspected in the cat if the heart rate is above 240 beats per minute, in the absence of an identifiable systemic disease. Tachyarrhythmias can originate in the ventricular muscle (ventricular tachycardias) or in the supraventricular tissue (supraventricular tachycardia).

Supraventricular Tachyarrhythmias:

The most common clinically significant supraventricular tachyarrhythmias are:

1. Atrial tachycardia
2. Atrial flutter
3. Atrial fibrillation

The main difference between supraventricular tachycardia, atrial flutter, and atrial fibrillation is the rate at which the ectopic focus depolarizes. Standard definitions for atrial depolarisations in supraventricular tachycardias in cats are not described, but in dogs, the rate of pacemaker firing for supraventricular tachycardia is between 150—350 beats per minute; for atrial flutter, the rate is greater than 350 beats per minute, and for atrial fibrillation, the rate is generally greater than 500 per minute. The ventricular response-rate produced by AV nodal depolarisation in response to atrial depolarisation is variable, but typically leads to ventricular rates of 180-280 depolarisations per minute. In atrial tachycardia and atrial flutter, the ventricular rate is fairly regular; whereas in atrial fibrillation, the ventricular rate and pattern is highly variable (irregularly irregular).

The causes of supraventricular tachycardia are listed in the table below:

• Chronic obstructive pulmonary disease • Myocardial hypoxia of any cause • Shock • Sepsis • Cardiomyopathy with atrial distension • Congestive heart failure • Myocarditis (including traumatic myocarditis)	• Congenital heart defects • Electrolyte abnormalities (hypokalaemia, hyperkalaemia) • Pericardial effusion • Cardiac or metastatic neoplasia • Medications e.g. barbiturates • Systemic inflammation

Clinical signs associated with supraventricular tachycardias are related to the underlying disease, as well as reduced cardiac output, which occurs due to reduced diastolic filling time of the ventricles (in response to rapid stimulation of the AV node by rapid supraventricular depolarisations). As such, symptoms of acute congestive heart failure, weakness, lethargy and end-organ dysfunction (acute kidney injury etc.) may be present.

Diagnostic evaluation of the patient with supraventricular tachycardia involves complete blood evaluation (CBC, serum biochemistry, electrolytes), urine analysis, and diagnostic imaging. Further diagnostic tests are selected based on physical examination and diagnostic evaluation.

ECG characteristics of supraventricular tachyarrhythmias include:

- Variable, rapid atrial depolarisation rate
- The presence of P' or F waves, or complete absence of P waves (atrial fibrillation) coupled with rapid ventricular ate with normal QRS morphology
- Normal QRS complexes occurring at a rapid rate
- P in T complexes, where the P wave is embedded in the preceding T wave (seen in atrial tachycardia)
- Occasional ventricular ectopic depolarisations

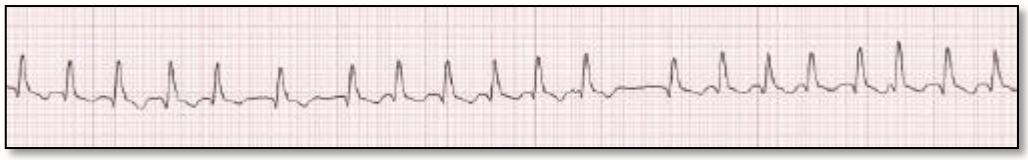


Image showing atrial fibrillation, with ventricular response rate of 240 beats per minute in a 5-year-old cat with HCM and congestive heart failure. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

Treatment of supraventricular tachycardia has the following goals

- To diagnose and manage the underlying cause
- To slow ventricular depolarisation rate, in order to improve cardiac output
- To slow the atrial depolarisation rate

Treatment recommendations are outlined below:

1. Oxygen supplementation
2. Diltiazem
    a. If heart rate >180/min with evidence of poor perfusion (hypothermia, poor pulse quality, evidence of azotemia, poor mention etc.)
    b. Diltiazem 0.1-0.4 mg/kg IV slowly over 10 minutes, followed by continuous infusion at 1-5 micrograms/kg/minute
3. Beta-adrenergic blockers
    a. May be used instead of diltiazem
    b. In cats with known systolic dysfunction, esmolol is preferred, due to its short duration of action
    c. Esmolol is used at a dose of 50-500 micrograms/kg slow IV over 1 minute, followed by a continuous infusion @25-200 micrograms/kg/minute
    d. Propranolol is used if systolic function is normal at 0.02-0.1 mg/kg IV over 5-10 minutes; or 0.1-0.2 mg/kg PO q 8-12 hrs
4. Procainamide
    a. May be used instead of diltiazem
    b. 1-2 mg/kg slow IV over 10 minutes followed by continuous infusion of 10-20 micrograms/kg/minute
    c. Procainamide can cause vasodilatation and hypotension if given too rapidly
5. Furosemide
    a. Used if congestive heart failure is present
    b. 2 mg/kg IV bolus OR
    c. Continuous infusion of 0.66-1.0 g/kg/hr
6. Glyceryl trinitrate
    a. Used if congestive heart failure is present
7. Dobutamine
    a. Used if patient is in cardiogenic shock
    b. 2.5 micrograms/kg/minute continuous infusion; titrate the dose upwards to maintain systolic arterial pressure >90 mm Hg

Ventricular Tachycardia

Ventricular tachycardia is defined as 3 or more ventricular premature complexes (VPCs) in a row. Ventricular tachycardia may be intermittent (paroxysmal) or sustained. Typically, QRS complexes are wide and bizarre in appearance; with the heart rate being rapid.

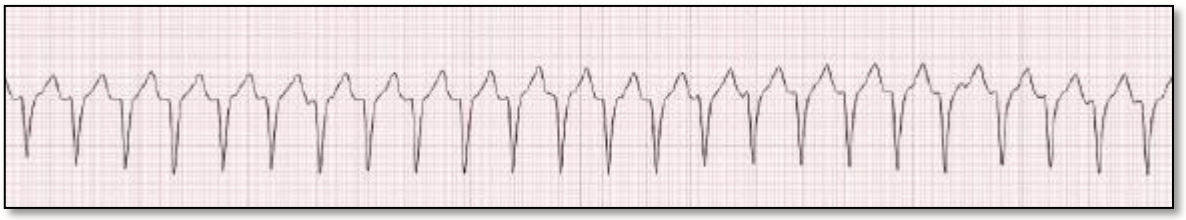


Image showing ventricular tachycardia at a rate of 240 depolarisations per minute in a 14-year-old cat with HCM and congestive heart failure. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

The causes of ventricular tachycardia are listed in the table below:

• Cardiomyopathy
• Traumatic myocarditis
• Shock
• Sepsis
• Systemic inflammatory disease
• High sympathetic tone
• Anaemia
	• Electrolyte disorders
	  ○ Hypokalaemia
	  ○ Hypomagnesaemia
	• Metabolic acidosis
	• Pain
	• Medications – barbiturates, sympathomimetic agents etc.

Clinical signs associated with ventricular tachycardia include those associated with underlying disease, as well as with reduced cardiac output, owing to reduced diastolic filling time due to tachycardia, and include lethargy, collapse, signs of acute heart failure, or sudden death.

The diagnostic approach to the patient with ventricular tachycardia is aimed at diagnosing potential predisposing causes, as well as cardiac assessment, and include complete blood count, serum biochemistry, electrolyte analysis, urine analysis and diagnostic imaging as appropriate.

Treatment of ventricular tachycardia is recommended if the ventricular rate is in excess of 220-240 beats per minute, and associated with symptoms of perfusion abnormalities, and cat involves:

1. Oxygen supplementation
2. Diagnosis and management of underlying disease or predisposing condition e.g. correct electrolyte disorders, provide analgesia, fluid resuscitation, transfusion therapy etc.
3. Antiarrhythmic therapy is attempted using either of the following
    a. Procainamide 3-8 mg/kg slow IV over 10 minutes
    b. Propranolol 0.25-0.5 mg/cat slow IV over 10 minutes
    c. Esmolol 50-500 micrograms/kg IV over 1 minute; followed by continuous infusion @ 25-200 micrograms/kg/minute
    d. Lidocaine 0.25 mg/kg initial dose – increasing to 1.0 mg/kg if required; administered slowly IV followed by continuous infusion @ 10-40 micrograms/kg/min

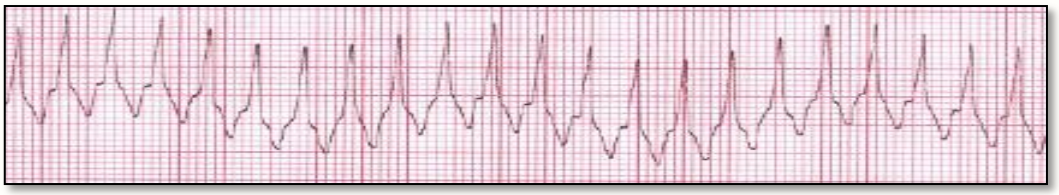


Image of rapid ventricular tachycardia in a cat with arrhythmogenic right ventricular cardiomyopathy – an uncommon primary myocardial disease of cats, in which right ventricular myocyte death occurs, with replacement by fatty deposits and fibrous tissue. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

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