ELECTROCARDIOGRAPHIC INTERPRETATION OF CARDIAC ABNORMALITIES IN DOGS

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Electrocardiography (ECG) has been used in veterinary medicine to diagnose primary cardiac problems as well as systemic diseases which affect the heart secondarily. It provides a basis for accurate diagnosis of certain functional abnormalities of heart. Different conditions that can be diagnosed with the help of ECG are summarized below.

I. Evaluating cardiac diseases

a)Evaluation of anatomic cardiac changes
b)Evaluation of arrhythmias
c)Evaluation of therapy
d)Electrolyte disturbances
e)Evaluation of progress of the disease

II. Differentiating of non specific diseases that cause weakness, fatigue, fever, lethargy, collapse, seizures

III. Monitoring before, during and after surgery IV. Evaluation of critically ill patient's ego trauma cases

Abnormalities of Impulse Formation

These can be further categorized as follows:

Supraventricular

Atrial

- Sinus arrest
- Atrial premature complexes (APCs)
- Atrial bigeminy, trigeminy
- Atrial tachycardia
- Atrial flutter
- Atrial fibrillation

Atrioventricular Junction

- AV junctional premature complexes
- A V junctional tachycardia
- A V junctional escape rhythm (secondary arrhythmia)

Ventricular

- Ventricular premature complexes (VPCs or PVCs)
- Ventricular bigeminy, trigeminy
- Ventricular tachycardia
- Ventricular flutter
- Ventricular fibrillation
- Ventricular asystole
- Ventricular escape rhythm (secondary arrhythmia)

Sinus Arrest

In this type of conduction disturbance, there is a long pause following a normal complex. The length of the pause may be more than twice the length between two normal beats (two times normal P-P interval). In addition, no P waves are apparent and usually a ventricular or junctional escape beat follows. Sinus arrest is frequently caused by high parasympathetic tone due to one or many factors, such as surgical stimulation, impingement upon the vagus nerve (neoplasia), respiratory disease (frequently seen in brachycephalic breed dogs), drug toxicity, or electrolyte imbalance. Disease of the sinus node itself, "sick sinus", can also occur and result in periods of sinus arrest progressing to complete inactivity.

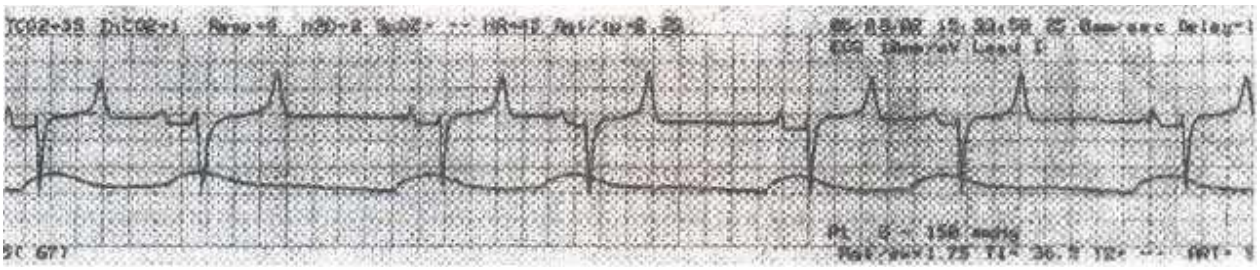
Atrial Premature Complexes

APCs (sometimes Jess accurately called atrial premature contractions), are not an uncommon finding in veterinary patients. They are an example of an ectopic rhythm- the impulse originates in the atrial tissue rather than from the sinus node. On examination of the ECG, one would note an abnormally shaped P wave that is premature, and may be "buried" or superimposed on the preceding T wave. The QRS complex that follows the abnormal P wave may be premature and slightly narrowed. Occasionally after very early APCs, QRS configuration is abnormal, due to refractoriness of part of the normal conduction pathway. APCs may or may not be clinically significant. If they occur infrequently, after exercise (as frequently seen in the horse), in the absence of any other cardiac disease or pathological arrhythmia, treatment is usually unnecessary. If, however, APCs occur frequently at rest, cause exercise intolerance, or occur in conjunction with other cardiac disturbances (such as atrial tachycardia, atrial fibrillation, or atrial flutter), then treatment is indicated.

Atrial Bigeminy, Trigeminy

Characterized by atrial premature complexes (APCs) alternating with normal sinuscomplexes. Similarly, one can see atrial trigeminy in which there are two APCs for every normal sinus complex. The QRS complexes for the APCs are similar to the normal sinus QRS

complexes; however the T wave of the normal complex may be abnormal due to the superimposition of the abnormal P wave on the following APC.



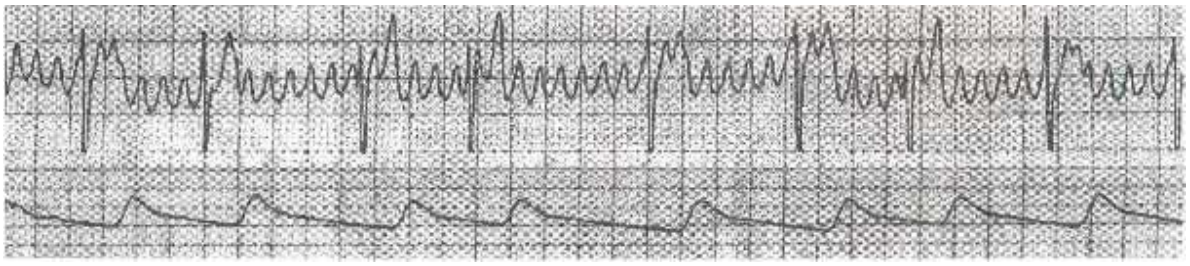
Atrial Tachycardia

Atrial tachycardia must be differentiated from sinus tachycardia, which is physiologic in nature. The heart rate is elevated above the normal range for the species, but the rhythm is typically regular. There is usually a P wave for every QRS, however, it may be abnormal in shape or fused with the previous T wave. Atrial tachycardia is another example of an ectopic rhythm typically caused by reentrant activity in the A V node. In such a case the episode of atrial tachycardia is usually initiated by an APC.

Atrial tachycardia may occur secondary to atrial enlargement or due to any number of other cardiac and non-cardiac causes.

Atrial Flutter

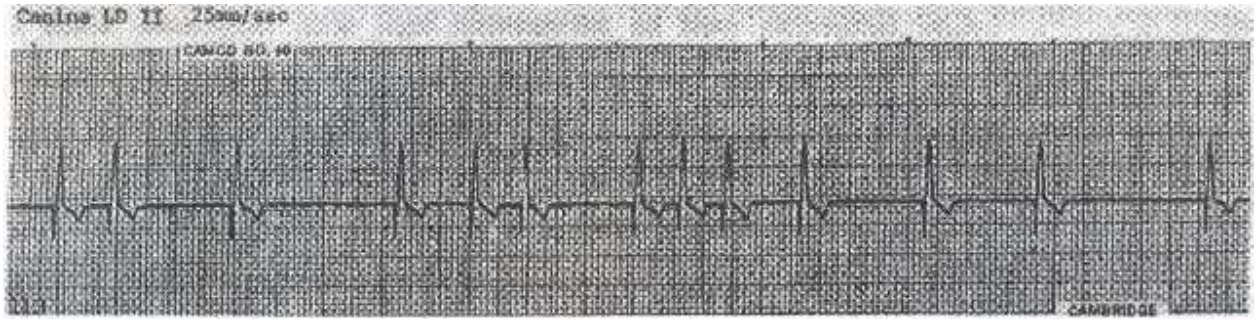
Atrial flutter is an atrial tacharrhythmia that is characterized by very rapid (atrial rate is typically greater than 300 bpm in the dog and horse), but regular heart rate, and an ECG with a typically "saw-toothed" appearance. Atrial flutter may occur secondary to atrial enlargement.



Atrial flutter with an irregular ventricular response and aberrant ventricular conduction pattern

Atrial Fibrillation

Atrial fibrillation is also an atrial tachyarrhythmia characterized by an irregular rhythm, an absence of P waves, and the presence of f (for fibrillation) waves. Atrial fibrillation is not an uncommon arrhythmia in veterinary patients. In race horses, or other sport horses, its onset is often associated with a sudden decrease in performance while the heart may be otherwise normal. In these horses, especially if treatment is begun soon after the onset of arrhythmia, normal sinus rhythm can often be restored with quinidine therapy. In small animals, atrial fibrillation is usually secondary to atrial enlargement.



The Ventricular rhythm is irregular and there are no P waves and a fine baseline “undulation”

AV Junctional Premature Complexes

These are similar to atrial premature complexes in that they are supraventricular and occur due to abnormal impulse formation at or near the AV junction. The impulse may spread both retrograde and antegrade from its source. P waves are typically early and may have an abnormal configuration (negative P waves may be seen in lead II). One may observe a prolonged or

shortened P-R interval depending on the distance from the origin of the impulse to the A V node.

AV Junctional Tachycardia

A V Junctional tachycardia is similar to atrial tachycardia in that it is usually a result of reentrant activity that produces an ectopic rhythm in the A V node region. The rate is increased impulse originating from an area distal to the A V Junctional tissue. These early beats do not have a preceding, corresponding P wave and the QRS complex may be abnormally wide and unusual in shape. VPCs can occur singly, in pairs (couplets), in sequence; (four or more together = ventricular tachycardia), or alternate regularly with sinus beats (v. bigeminy or trigeminy). It is normal to see occasional VPCs; however, frequent VPCs in any form may necessitate treatment.

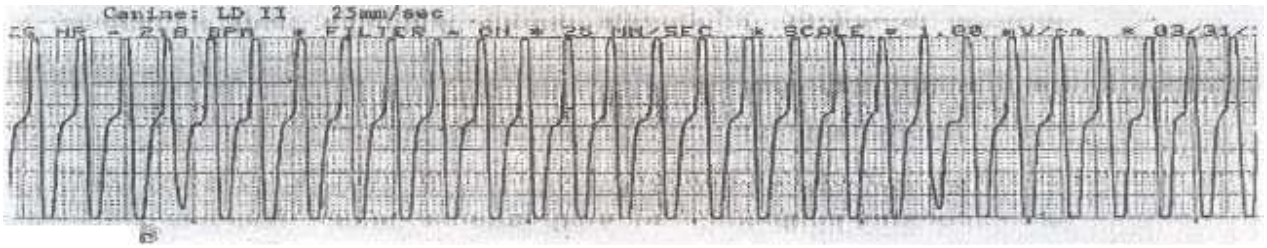
Ventricular Bigeminy

This arrhythmia consists of ventricular premature complexes (VPCs) alternating with normal sinus complexes in an "every-other-one" pattern. Necessity of treatment would depend on the presence of any other cardiac pathology and the frequency of the arrhythmia. Ventricular trigeminy can also occur. In this case, two VPCs follow each normal sinus complex.

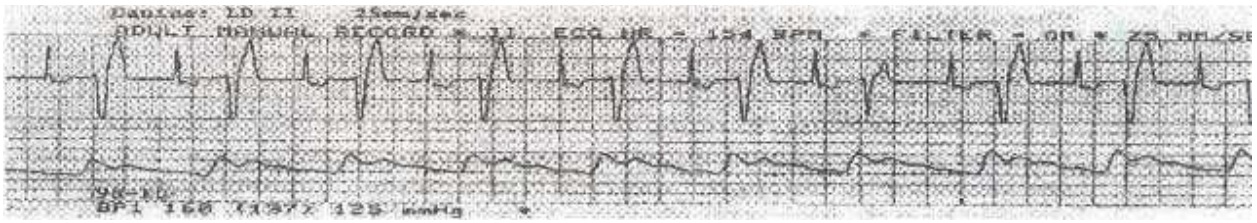
above the normal range, while the rhythm is typically regular throughout the episode. A P wave may be absent, negative, or buried in the following QRS complex.

Ventricular Premature Complexes

VPCs are an example of an ectopic rhythm



ECG ECG shows wide, high amplitude QRS complexes, no discernable S-T segment, and high amplitude T waves



ECG shows wide, high amplitude QRS complexes, no discernable S- T segment, and high amplitude T waves

P waves in this ECG are difficult to discern, but every other QRS- T complex appears normal for a canine lead II ECG. Interspersed between the normal complexes are wide, negative QRS complexes with slurred S- T segments and high amplitude T waves.

They are "coupled" to the normal complexes (P-R interval = 0.32 sec.)

Ventricular Tachycardia

Ventricular tachycardia (VT) is characterized by an elevated ventricular rate with a typically regular rhythm. QRS complexes are usually aberrant in shape and P waves are not usually present. AP wave may be present if retrograde conduction occurs and may cause a capture beat (if the ventricles are not refractory). The capture beat may then merge with an ectopic focus beat and result in a fusion beat.VT is usually the result of reentrant activity distal to the A-V junction area that leads to an ectopic rhythm.

There may be more than one ectopic focus present, which can be determined by examining the morphology of the complexes.

Unifocal (or uniform) VT arises from a single ectopic focus and produces aberrant, although similar-looking QRS complexes.

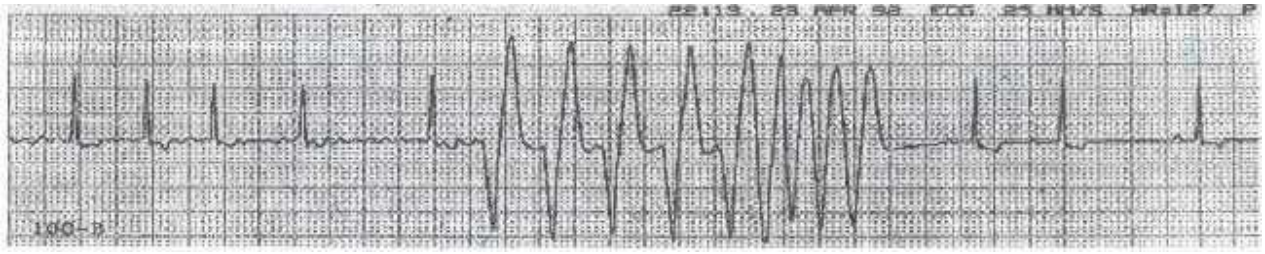
Multifocal (or multiform) VT is the result of two or more ectopic foci and produces QRS complexes attributable to each foci. Four or more· rapid QRS complexes (with or without an accompanying P wave) constitute a VT. paroxysmal VT is a short burst of ventricular tachycardia that ends spontaneously, while sustained VT is prolonged indefinitely.

?? (Obviously, the primary concern with VT is inadequate cardiac output due to decreased ventricular filling. The extent of the VT determines the course and necessity of treatment.)

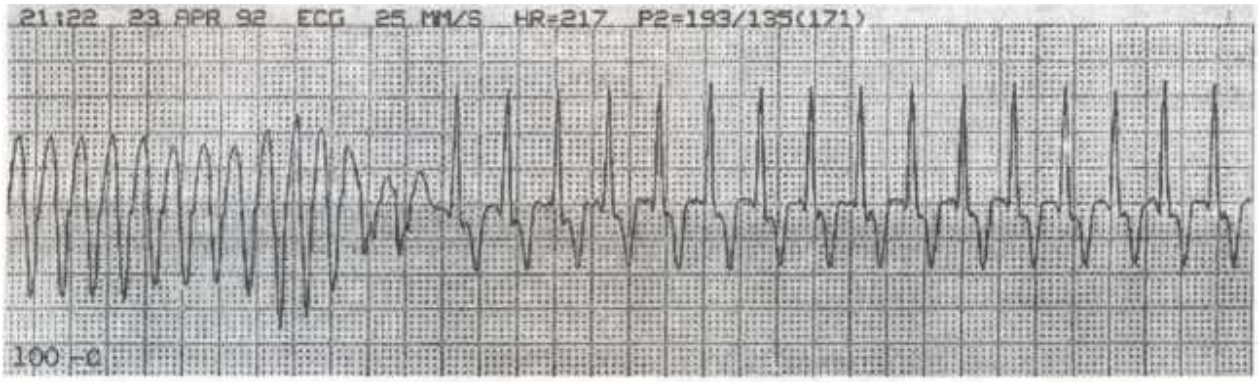
?? (Other inciting stimuli for VT under anesthesia?)

Ventricular Flutter

This is a tacharrhythmia characterized by a high ventricular rate with a regular rhythm. An ECG shows large sine wave-like complexes that oscillate in a regular pattern. P waves are not present, and the QRS complex is indistinguishable from the T wave. Ventricular flutter, if untreated, may precede ventricular fibrillation. In severe cardiac or systemic disease states, ventricular tachycardia can progress to ventricular flutter, then to ventricular fibrillation. These final two arrhythmias are severe and require· immediate attention.



A period of atrial flutter with an irregular ventricular response is punctuated by a short run (5 complexes) of unifocal ventricular tachycardia and then a period of ventricular flutter. Following that, sinus rhythm is restored but atrial flutter returns for a short period (T waves are small but clearly visible during sinus rhythm).



Ventricular flutter is followed by supraventricular tachycardia.

VentricularFibrillation

This ventricular tachyarrhythmia is characterized an irregular pattern of high or low-amplitude waves that cannot be differentiated into QRS complexes or T waves. In cardiac arrest, it may be preceded by ventricular flutter. This is a serious arrhythmia which requires immediate treatment. Often, electrical defibrillation is indicated.

Ventricular Asystole

Ventricular asystole is indicative of cardiac arrest. It requires immediate attempts at resuscitation, with a poor prognosis at that. It is characterized by the absence of electrical activity for a length of time with intermittent ventricular complexes of abnormal configuration. This arrhythmia often indicates the imminent demise of the patient.

Abnormalities of Impulse Conduction

These can be categorized as follows:
Sino-atrial Block
Persistent Atrial Standstill ("Silent Atrium", Hyperkalaemia)
A-V Block
- First-Degree
- Second-Degree
- Third-Degree (complete heart block)

A-V Dissociation

In this type of conduction disturbance, the impulse is blocked as it exits the SA node and no conduction reaches the cardiac tissue to produce any deflections in the ECG. This results in pauses in the ECG that are equal to multiples of the P-P interval. This differs from sinus arrest in that an impulse is generated, but not propagated to the myocardium.

Persistent Atrial Standstill

This condition occurs secondary to fibrosis of the atrium (secondary to chronic mitral regurgitation or other ongoing pathology). Fibrosis prevents proper conduction and so vel1tricular contraction occurs in the form of escape beats, and thus the heart rate is slow. ECG examination usually reveals an absence of P waves and may show a widening of the QRS complex. Persistent atr1al standstill, or "silent atrium", should be differentiated from atrial standstill caused by hyperkalaemia (secondary to renal failure, ruptured bladder, other electrolyte imbalances, or excessive IV K + administration). Hyperkalaemia alters atrial trans-membrane resting potential, and the atria become in excitable at very high plasma K + levels.

First-Degree AV Block

Characterized by a delay of the impulse in the A V node region. Heart rate is usually normal and rhythm may be regular or irregular. There is a P for every QRS complex and the most notable finding is a prolonged but constant P-R interval.

Second-Degree A V Block

Second-degree A V block is more than just a delayed impulse at the A V node. The impulse is delayed and blocked resulting in both conducted and non-conducted P waves on the ECG. This translates into some P waves that are followed by a QRS complex and some P waves that occur without a subsequent QRS complex. Second-degree A-V block can result from high vagal tone, electrolyte imbalances (hyperkalaemia), pharmacologic effects (opiates in dogs), or A-V nodal disease. Second-degree A-V block with only occasional non-conducted complexes does not require treatment. If pauses in ventricular activity are frequent or of long enough duration to result in significant decreases in blood pressure or cardiac output, treatment is necessary. There are two types of second-degree A V block that are differentiated by the character of the P-R interval:

- Mobitz type I (or Wenkebach): The P-R interval gradually lengthens until a QRS-T is dropped, thus the P-R interval is variable. This type is a common finding in the normal horse (and can also occur as a result of digitalis treatment or electrolyte imbalance). It is abnormal in cattle.
- Mobitz type II: The P-R interval is constant before and after the dropped beat.

Tird-Degree A V Block (Complete Heart Block)

In this case, the impulse is completely blocked at the A V node region. Therefore, the P wave and QRS complex are dissociated. In awake patients, this rhythm usually indicates disease of the A-V node. If a ventricular escape rhythm is present, the patient may have adequate cardiac output to maintain consciousness. This arrhythmia often necessitates treatment. A pacemaker can be placed. In anesthetized patients, this arrhythmia can result from surgical stimulation (e.g.? cardiac reflex), from hyperkalaemia or, in the horse, after a sudden increase in arterial blood pressure following vasopressor therapy. An adequate ventricular escape rhythm is rarely present during anesthesia and aggressive therapy is necessary.

A-V Dissociation

This is a descriptive term for independent atrial and ventricular rhythms. A-V dissociation is technically not an arrhythmia itself, but is present in conjunction with other arrhythmias such as ventricular tachycardia and in third degree A-V block (completer A-V dissociation). As the atrial rhythm slows, an ectopic focus distal to it may take over and another rhythm develops. Typically, the ECG would show P waves with little to no relation to one another (except in retrograde conduction - see ventricular tachycardia).

Abnormalities of Impulse Formation and Conduction

Prexication
- Wolf-parkinson-White syndrome

Parasystole
Other complex rhythms
- Electrical Alternans

Pre-Excitation

Arrhythmias due to pre-excitation occur when "an impulse originating in the atrium activates the ventricles earlier that would be expected if the impulse traversed the normal route from the AV node to the His bundle", via an accessory pathway.

Wolf-Parkinson-White Syndrome

This arrhythmia is denoted by ventricular pre-excitation with episodes of paroxysmal supra-

ventricular tachycardia. The P-R interval is shortened (the impulse reaches the ventricles without going through the A V node) due to the activation of an alternate, shorter pathway through the myocardium. Again, this often due to re-entry of the impulse back into the atria (reciprocal re-entry). The “detour” of the impulse may cause an aberrant QRS configuration. This syndrome may occur in patients with valvular abnormalities, cardiomyopathy, or otherwise normal heart function.

Para systole

Para systole is a type of tachyarrhythmia that is caused when two pacemakers independently cause excitation. The second focus of excitation can occur in any area of the heart and is evident because it is not over ridden by the primary pacemaker. The myocardium must be non-refractory (has already, recovered from the previous impulse; i.e, is excitable) to the impulse in a particular area for Para systole to occur. Electrocardiographic features include: Variable P-P intervals and fusion beats.

This is technically not an arrhythmia, but occurs secondary to some underlying pathology. Most commonly it is associated with pericardia! effusion, but can also occur secondary to pleural effusion and atrial tachycardia. In the instance of pericardial effusion, the heart tends to move within the fluid with each contraction. "When the notion of the heart within (a) pericardia! effusion is increased markedly and, in particular, when this is accompanied by tachycardia (also secondary to some underlying pathology), the heart may not have returned to its previo~s position Py the time the next cardiac cycle commences. With each depolarization the heart is in a slightly different position, and the electrocardiographic QRS complex also varies with alternating heights of R waves on the electorcardiogram, i.e., electrical alternans." Because electrical alternans is a "secondary electrocardiographic finlling, it is necessary to treat the underlying cause (pericardiocentesis is often indicated).

Electrical Altternans

a)Congestive heart failure

- Digoxin 0.03 mg/Kg for 48 hours in divided doses orally .
- Diuretics: Furosemide 2-4 mg/kg bwt 6-8 hr interval .
- Salt free diet
- In refractory cases, vasodilator e.g. captopril 0.5-2.0 mg/kg bwt 8-12 h interval orally.

b)Supra-ventricular premature beats and, atrial fibrillations.

- Use digoxin. But it is contraindicated in atrioventricular block and ventricular premature beats.

c)Ventricular premature beats and ventricular tachycardia

- Quinidine sulphate 3 gr tab

dose: 3-10 mg/lb bwt every 6-8 h orally

- Propranolol 10-40mg tid orally
- Lidocaine (2% without epinephrine)

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