Cardiac Arrhythmias

Reference Notes

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Heart rhythm disturbances (**arrhythmias, dysrhythmias**) can be classified based on the ventricular **heart rate** (normal, bradyarrhythmia, tachyarrhythmia); **anatomic origin** of the rhythm disturbance (sinoatrial node, atria, atrioventricular, or ventricular); or **electrophysiologic mechanism**, if evident. An overview of electrophysiologic mechanisms of arrhythmias is presented in class. These include abnormalities of **impulse formation** (enhanced automaticity, triggered activity) and various abnormalities of **impulse conduction** (including major conduction blocks such as atrioventricular (AV) block and right and left bundle branch blocks; "macro-retry" as with atrial flutter; disordered re-entry as with myocardial fibrillation; and other forms of re-entry around a focal zone of myocardial disease.) Detailed models of arrhythmogenesis are beyond the scope of this presentation.

Diagnosis

Keys to *recognizing and diagnosing cardiac arrhythmias* include an analysis of the atrial and the ventricular rates, regularity of the rhythm, identification of any patterns in irregular rhythms, P to QRS relationship, atrial waveform morphology (e.g. different than sinus node; flutter or fibrillation waves); morphology of the QRS complex (supraventricular or ventricular origin); and conduction intervals (especially duration of PR interval and duration of QRS complex). In terms of a methodological approach to rhythm diagnosis, it is recommended that one consider analysis of the ECG (EKG) as follows:

1) Identify the patient, lead(s), paper speed, calibration signals, and artifacts;
2) Decide if the ventricular rate is slow, normal, or fast for the species;
3) Identify regularity or lack thereof and search for repetitive patterns in irregular rhythms;
4) Identify P and QRS complexes and the relationship between these waveforms (P-R intervals and the distance between the QRS and following P-waves (R-P)
5) Scrutinize morphology and consistency of the P-waves, QRS complexes, and T-waves;
6) Consider the conduction intervals across the atria (P-wave duration), atrioventricular conduction system (P-R interval), ventricles (QRS duration), and repolarization (Q-T)
7) Estimate the frontal axis and analyze the amplitude and terminal orientation of the QRS;
8) Evaluate the QRS morphology for conduction disturbances, obvious bundle branch or fascicular block patterns, and for changes typical of cardiomegaly;
9) Assess the ST segment and T-wave (ST-T) for repolarization abnormalities; and
10) Interpret the ECG with consideration of the entire clinical and laboratory picture.

These are merely guidelines, but can assist with a methodical ECG analysis.

The main clinical concerns about cardiac arrhythmias are *hemodynamic* effects such as reduced blood pressure (BP) and tissue perfusion and the promotion of further *electrical instability* leading to myocardial fibrillation. Bradyarrhythmias, as third-degree atrioventricular (AV) block, can reduce cardiac output by decreasing heart rate. Supraventricular tachyarrhythmias encroach on the diastolic filling periods, reducing preload and stroke volume. In the case of atrial fibrillation (AF), atrial contribution to ventricular filling is also lost, further impairing filling. Many ventricular arrhythmias are well-tolerated, but in other patients cause hypotension from reduced diastolic filling time, loss of normal atrioventricular coordination, and decreased mechanical synchrony between the two ventricles (due to abnormal current spread). Additionally, relentless tachycardia can lead to a **tachycardia-induced cardiomyopathy**, a type of dilated cardiomyopathy that is reversible with cessation of the arrhythmia. Ventricular ectopy also predisposes to **ventricular fibrillation** (disorganized electrical activity with loss of mechanical function of the ventricles). This is a cause of sudden death in some animals.

Sinus Rhythms

Sinus rhythms are those in which the electrical impulse originates in the cells of the SA node, the normal "pacemaker" of the heart. These rhythms are characterized by a P-QRS-T relationship. The P-waves are normal in morphology (positive in lead II); there is an appropriate P-R interval indicating atrial and ventricular activities are related; and the QRS occurs once the impulse conducts from the internodal pathways through the AV node, the His-Purkinje system, and finally enters the ventricular myocardium.

Physiologic sinus rhythms during routine exams include normal sinus rhythm (NSR) and sinus arrhythmia. **Normal sinus rhythm** is characterized by a normal heart rate, regular rhythm (varying by <10% between cycles), and consistent P-QRS relationship. **Sinus arrhythmia** is due to variation in vagal input to the sinus node and is most often observed in dogs. The sinus node discharge rate cyclically slows and speeds and the QRS-T complexes follow suit; thus, the overall ventricular rate is normal but the instantaneous heart rate (i.e., between consecutive R-waves) varies by >10%. In dogs, sinus arrhythmia is most often associated with ventilation and termed **respiratory sinus arrhythmia**. Spillover of inhibitory input as the lungs stretch at end-inspiration somehow influences vagal output to the sinus node, leading to slowing of heart rate during expiration. As respiratory inhibition decreases during expiration, the vagal input to the sinus node also starts to decline and the heart rate speeds up again. This repeats in a cyclical pattern and can be thought of to lag slightly behind the respiratory cycle (i.e. at end-inspiration the sinus node starts to slow down; at end-expiration the sinus node starts to speed up). Dogs with **respiratory disease** often show pronounced sinus arrhythmia as do some other conditions associated with high vagal tone (such as some gastrointestinal disorders). Physiologic second-degree AV block can be associated with blood pressure regulation and likely reflects varying vagal influence associated with the baroreceptor reflex.

A common feature associated with sinus arrhythmia is **wandering atrial pacemaker** in which the P-waves vary in morphology as the current originates from different portions of the SA node ("wanders") and crosses the atria in a slightly different activation sequence. Wandering atrial pacemaker is normal and is recognized by higher-amplitude P-waves in lead II during inspiration and flattening of the P-waves during expiration. The changes in P-wave morphology are usually gradual, but this rhythm can be confused with premature atrial complexes.

Sinus arrhythmia is less common in animals that are likely to be stressed during examination (i.e., cats); feline species usually have a regular rhythm (NSR) or sinus tachycardia. However, occasionally sinus arrhythmia is detected in resting cats or when blood pressure increases inappropriately during times of (psychic) stress and vagal input increases in response to baroreceptor input in an attempt to lower blood pressure (BP).

Sinus rhythm disorders include **sinus bradycardia** and **sinus tachycardia**. These are simply sinus rhythms at slower or faster discharge rates than is normal for the species. These rhythms are often physiologic, as occurs with sleep or exercise. In most cases sinus bradycardia or tachycardia are due to high vagal or sympathetic tone, respectively. Therefore, any patient with one of these rhythms should be evaluated with the likelihood of some **autonomic nervous system** influence in mind. Additionally, **drugs, anesthetics, body temperature, and endocrine** diseases (especially thyroid or adrenal) can affect sinus node rate. For example, elevated CSF pressure (which stimulates vasoconstriction to raise BP and better perfuse the brain) stimulates the baroreceptors leading to reflex sinus bradycardia ("Cushing's reflex"). Other causes of sinus bradycardia include sedatives and tranquilizers, inhalation anesthetics, hypothermia, and hypothyroidism. Counterintuitively, this rhythm is also observed in some cats with profound shock (along with hypothermia and marked hypotension). Sinus tachycardia is associated with any cause of sympathetic nervous system activation (exercise, pain, anxiety, anemia, and hypotension), as well as anticholinergic drugs (atropine/glycopyrrolate), fever, and hyperthyroidism.

If the sinus node fails to discharge, the rhythm is termed **sinus arrest**. Strictly speaking, this is defined as an absence of sinus node discharge exceeding two normal P-P intervals. What is "normal" is easy to determine when the underlying rhythm is a regular NSR, but when there is a marked sinus arrhythmia, it can be hard to decide if the pause is abnormal or just part of the sinus arrhythmia. Short periods of sinus arrest are well tolerated, but when prolonged (e.g. >5 seconds), and if there is no escape complex to rescue the heart, weakness or syncope are more likely to occur. When sinus arrest is associated with clinical signs, the term **sick sinus syndrome** is used. This is most common in dogs (see in-class notes). Chronic, progressive, sinus node dysfunction is especially common in miniature Schnauzers, West Highland white terriers, and cocker spaniels. These patients often have abnormal conduction in other parts of the heart (e.g. atrioventricular block) and insufficient escape activity in the AV junction and ventricles indicative of diffuse conduction disease. Additionally, these dogs are prone to inappropriate sinus tachycardia and ectopic supraventricular (atrial) tachyarrhythmias.

Management of sinus rhythm disturbances is focused first on treating any underlying conditions. For example, if one encountered sinus tachycardia in a post-operative patient the first considerations would be hypotension (Rx: Fluids/Colloids), pain (Rx: opiates), increased body temperature (Rx: underlying cause), or emergence delirium (Rx: tranquilizer). Occasionally inappropriate sinus tachycardia is treated with a beta-blocker (e.g. in some cats with hyperthyroidism or in dogs with certain sympathomimetic intoxications). Sinus bradycardia can be treated in the hospital with atropine or glycopyrrolate, two drugs that block the muscarinic receptor and reduce vagal influence. In emergencies, catecholamines can be used to stimulate the beta-receptors of the SA node and increase heart rate as well as myocardial contractility. Sick sinus syndrome is often treated in practice with an oral anticholinergic drug (hycosamine) or the sympathomimetic drug (terbutaline, a beta-2 agonist or theophylline, a PDE inhibitor). However, these drugs are rarely effective in treating clinically important SSS, and the best long-term therapy is permanent transvenous pacing. Pacemaker programming is critical for optimal

system performance and long-term outcomes are very good.

Supraventricular Arrhythmias

Supraventricular rhythm disturbances are among the most common and sometimes difficult of all cardiac rhythms to diagnose and manage. Supraventricular arrhythmias are considered "**ectopic**" as these originate outside of the sinus node. The arrhythmias include premature atrial complexes, atrial tachycardia, atrial flutter, atrial fibrillation (AF), and re-entrant supraventricular tachycardia (SVT), which uses the atria as part of the circuit loop. These rhythms can be transient, recurrent, or persistent to permanent. Atrial standstill is a unique atrial rhythm disturbance characterized by inexcitable atrial myocardium.

Supraventricular arrhythmias are most often caused by structural heart diseases associated with atrial enlargement, especially in dogs and cats. Atrial dilatation can stem from some congenital heart defect or an acquired valvular, myocardial, or pericardial disease. Atrial dilatation alters atrial electrical activity, and promotes abnormal heart rhythms through abnormal impulse formation or electrical conduction. Cardiac size influences the likelihood of sustaining an atrial arrhythmia such that atrial flutter and fibrillation occur more often in larger canine breeds. In contrast, the small size of the feline atria decreases the risk of atrial fibrillation in the absence of relatively severe atrial remodeling or disease.

Supraventricular arrhythmias can occur in the absence of overt structural disease and with a normal echocardiogram. One prominent example is atrial fibrillation (AF) in this setting of a structurally normal heart; the term "**lone atrial fibrillation**" is applied. Of course, it is likely that microscopic lesions, such as myocarditis or fibrosis, or genetic or acquired cell membrane channelopathies form the basis of some of these recurrent arrhythmias. This might explain why some animals develop recurrent atrial fibrillation (AF), even after successful conversion back to NSR. However, such lesions cannot be detected clinically (or without a biopsy sample). In some canine cases, atrial arrhythmias are forerunners to cardiomyopathies, but this is not always the case. Atrial arrhythmias are also detected due to electrolyte (potassium) disturbances; these are often short-lived and not caused by underlying heart disease.

Another special example of a supraventricular arrhythmia that can occur without over structural heart disease is the **reentrant supraventricular tachycardia** (SVT) using an accessory pathway. This tissue provides a second electrical bridge between the atria and ventricles, which are normally insulated by the AV valves and the fibrous cardiac skeleton. As shown in class, this can lead to a reentrant circuit that sequentially depolarizes the AV node → ventricles → accessory pathway (retrograde direction) → atria (retrograde) → AV node (antegrade direction) → ventricles. These accessory pathways are a special form of congenital heart disease (and beyond the scope of this overview). These will sometimes respond to lidocaine in hospital or to other sodium-channel blocking agents chronically if ablation cannot be performed.

A **premature atrial complex** (PAC or APC) arises outside of the SA node, timed early (prematurely) relative to the dominant sinus cycle. The arrhythmia is characterized by premature P wave (P') with a morphology that differs from P waves of sinus origin. The premature P' wave is followed by a normal duration to prolonged P-R interval (if the AV node has not fully repolarized) and then by a related QRS complex. In most cases, the QRS complex is of normal-duration and morphology, indicating the impulse

originated above the ventricles ("supraventricular"). Occasionally the conducted QRS complex will be slightly different than a sinus conducted QRS or even much wider: this indicates the ventricular conduction system had not yet repolarized fully; the phenomenon is called aberrant ventricular conduction. This situation is analogous to a conducted beat with bundle branch block (in fact right or left BBB can occur when premature atrial complexes are conducted into the ventricle). Sometimes the AV node is so refractory to premature stimulation from the atrium that the impulse is blocked. Isolated nonconducted PACs are especially common in people (and horses) but not often observed in dogs and cats. However, these can be an explanation for sudden pauses in the rhythm. In these situations, the P' is so early it appears within the T-wave of the previous supraventricular complex.

The rhythm **atrial tachycardia** is essentially a "run" of repetitive PACs. This usually arises from a single atrial focus, and the term "focal atrial tachycardia" is sometimes used for such arrhythmias. The rhythm is characterized by an abrupt increase in the heart rate with abnormal P' waves preceding each QRS complex. Finding the P' waves can be difficult because they are usually buried in the T-wave of the preceding QRS-T complex. Although the QRS complexes should be relatively normal in appearance during a PAT, sometimes the first premature QRS complex of the atrial tachycardia exhibits aberrancy (abnormal ventricular conduction), leading to confusion with premature ventricular complexes. When the atrial tachycardia is nonsustained (<30 sec) with a sudden onset and termination, the term paroxysmal atrial tachycardia ("PAT") is often used. Focal atrial tachycardias can also be sustained or persistent. Atrial tachycardia can be confused with atrial flutter if the atrial rate is very fast and large atrial repolarization waves (Ta waves) deviate the baseline. Similarly, there are multiple types of atrial atrial flutter and flutter waves can sometimes appear similar to ectopic P' waves. Regardless, both are forms of atrial-based supraventricular tachycardias and will be managed in veterinary practices in a similar manner.

Sustained atrial arrhythmias include focal atrial tachycardias, atrial flutter, and atrial fibrillation (AF). As indicated above, atrial tachycardia is a series of premature atrial complexes. **Atrial flutter** (or macro-reentry atrial tachycardia) is thought to represent a macro-reentrant circuit involving the right atrium and is characterized by saw-toothed flutter waves in the baseline and the lack of an isoelectric shelf (i.e. the atrial activation does not return to the baseline but "saws" it back and forth). It is more common in cases of right atrial dilatation, as with tricuspid valve malformations or severe heartworm disease. The atrial rate in atrial flutter is about ~300 to 400/minute in (unanesthetized) dogs. QRS complexes are caused by conduction of flutter waves into the ventricles. Inasmuch as the normal AV node, His bundle and bundle branches are available for conduction, the resultant QRS complexes are usually normal in morphology.

Atrial fibrillation is a very important heart rhythm disturbance. The genesis of AF is likely an electrical scroll wave, micro-reentry circuit, or focus of abnormal automaticity within the pulmonary venous entries or the left atrium itself. Varying conduction patterns across the atrial mass leads to fragmented and chaotic myocardial activation. Left atrial dilation is commonly present as a substrate for maintaining this arrhythmia.

The ECG diagnosis is straightforward and characterized by a lack of any consistent P-waves, presence of fibrillation waves in the baseline, and an irregularly irregular (unpatterned) ventricular rhythm of supraventricular complex morphology. As with the other atrial tachyarrhythmias, the *QRS complexes are due to conduction of atrial impulses across the AV node*; consequently, the ventricular rate depends on

AV nodal conduction. The amplitude of the fibrillation waves varies: these are obvious in some cases but often less clear as the animals decreases in size. In cats the diagnosis is secured by recognizing a rapid rhythm, irregular R-R intervals, narrow (supraventricular) QRS complexes, and absence of consistent P-waves.

In dogs and cats, AF is usually characterized by a rapid, irregular supraventricular tachycardia. The admonition: "if it's rapid and irregular it is atrial fibrillation until proven otherwise", is a useful guideline. However, the *ventricular response rate depends on AV conduction*. In **lone AF**, cardiac output is usually normal at rest so arterial BP is normal, sympathetic tone low and resting heart rate normal. In contrast, during exercise – when sympathetic activity is high and vagal traffic low – the AV nodal cells conduct supraventricular impulses rapidly so ventricular rate response is higher than would be normal for the degree of activity. This is why animals with lone AF are often fine at rest, but at peak activity develop exercise intolerance. Recall: most ventricular filling occurs in early to middle diastole, but when the ventricular rate response is rapid and irregular, the diastolic filling periods are abbreviated and preload is often reduced. Furthermore, the normal response to faster heart rates is a greater atrial contribution to filling in the form of a vigorous atrial contraction; however, this contribution is absent in AF. Carrying this explanation to a patient with heart failure, where sympathetic tone is generally high, it should be clear that AF will lead to a sudden increase in resting heart rate, chaotic cardiac cycles, reduced ventricular filling, and decreased cardiac output. In fact, AF often precipitates CHF in previously compensated animals with heart disease.

Physiological AV block is often observed with atrial tachycardia and atrial flutter and is always present in AF. This block helps to prevent excessive ventricular rate response and can be understood by appreciating that AV nodal cells might not conduct current if there has been insufficient time for repolarization. Whereas the ventricular rate response to AF is "always" irregular, with atrial tachycardia or atrial flutter the rhythm can be regular or irregular as the atrial waves are better organized and regularly spaced. It can be challenging if there is a regular AV conduction sequence, for in these cases, ectopic P' or F-waves are buried in the QRS or ST-T and it might require multiple lead traces or sudden depression of AV conduction for identification (as with a vagal surge or after administration of diltiazem).

Thus, the **ventricular rate response** in any supraventricular tachycardia is determined by the type of atrial arrhythmia and the AV conduction pattern: the ventricular response can be slow or fast; regular or irregular. In high-sympathetic states, AV conduction of supraventricular arrhythmias can be very rapid, as with *AF in the setting of congestive heart failure* (CHF). Even in the absence of heart failure, a SVT due to focal atrial tachycardia, atrial flutter or re-entrant SVT can induce a ventricular response of nearly 400/minute (in small animals). Unlike AF, other supraventricular tachyarrhythmias form regular and organized impulses within the atria. With atrial tachycardia or atrial flutter, the ventricular rate can suddenly double or half as the atrioventricular conduction ratio suddenly changes. Supraventricular tachyarrhythmias also can be conducted with subtle electrical alternans (a diagnostic clue!) or even with a bundle branch block pattern so that the resultant QRS complexes are so wide as to be confused with ventricular tachycardia.

Conceptually it can be helpful to consider that the most common atrial arrhythmias (PACs, FAT, atrial flutter and AF) are inter-related. Many patients exhibit more than one of these rhythm disturbances at one point or another.

The basic management of supraventricular arrhythmias involves either heart rhythm control or heart rate control. **Rhythm control** means suppressing ectopic atrial activity. **Heart rate control** focuses on maintaining a more normal ventricular rate by depressing AV nodal conduction. For example, if a focal atrial tachycardia of 320/minute is conducted 1:1, then the ventricular rate is 320/minute. If every other impulse is blocked in the AV node, the ventricular response rate falls to 160/minute, a rate that is better tolerated hemodynamically.

When managing atrial tachyarrhythmias, the clinician should decide if the arrhythmia is more likely of recent onset or chronic in nature. Additionally, therapy will differ if the rhythm is "lone" (i.e., without structural heart disease) or associated with cardiac enlargement or heart failure. If the latter situations are evident, heart rate control is more commonly pursued.

Heart rhythm control (also called "conversion") can be obtained in some patients with drugs that suppress ectopic rhythms. These drugs are typically in the Vaughan-Williams Classes I and III, although this is a generalization. Lidocaine (Class 1B) can be effective in some peracute conditions, but typically does not suppress atrial arrhythmias. More often, the class IA drugs and class III drugs are used for rhythm control. **Procainamide** (IV) although now very expensive can also can be used for hospital therapy of atrial arrhythmias. The class III drugs (**sotalol, amiodarone**) are used for both hospital and chronic suppression of atrial arrhythmias in dogs and (sotalol) occasionally in cats. Sometimes class II drugs (beta-blockers) and class IV drugs (calcium channel blockers) will suppress an ectopic atrial rhythm, but drugs in these two classes are more often used for heart rate control (see below).

Many atrial tachycardias are resistant to drug suppression and require heart rate control. Sometimes rate control is also needed prior to cardioversion. Not every contingency can be discussed in an introductory presentation; but these are the principles of rhythm control.

Synchronized DC cardioversion is another therapeutic approach to converting a sustained supraventricular tachyarrhythmia back to NSR. This approach is particularly relevant to dogs with lone AF. **Electrocardioversion** requires general anesthesia and involves a timed, DC shock delivered on the R-wave to depolarize all of the heart muscle cells simultaneously and disrupt reentry electrical circuits. This should not be confused with **ventricular defibrillation**, which is a stronger, untimed DC shocking of a heart (without any QRS complexes) in an attempt to depolarize all the cells and allow the sinus node or another pacemaker to assume control. Amiodarone (dogs) or sotalol (dogs) is often prescribed empirically after cardioversion to maintain sinus rhythm. These drugs are usually continued for some months to prevent reversion to atrial fibrillation; however, the efficacy and the benefit to risk of such therapy have not been published.

Frequently, **heart rate control** of supraventricular tachycardia is the more logical treatment goal. In the setting of significant cardiomegaly or heart failure**,** ventricular *rate control* – not rhythm control – is generally preferred. Rate control involves administering a drug that depresses AV nodal conduction, reducing the transmission of atrial impulses into the ventricle. The three drugs used for this purpose are *digoxin, diltiazem, and beta blockers*. Recall, stimulation of parasympathetic muscarinic receptors depresses AV conduction. **Digoxin** increases the vagal input to these receptors, albeit indirectly, by *sensitizing the baroreceptor reflex* to prevailing BP. **Diltiazem** works by blocking the *L-type calcium channel*, and thereby the calcium current essential for phase 0 depolarization in AV nodal cells. **Beta-blockers** (__lol) depress calcium entry indirectly, because the beta-receptor exerts *ligand-operated control of calcium entry* across the L-type channel.

When atrial tachyarrhythmias are *associated with CHF* and if there is no contraindication for digoxin (such as renal failure or ventricular ectopy), this cardiac glycoside is administered. Digoxin is mainly used in dogs (rarely if ever in cats today due to prolonged elimination half-life). However, the CCB diltiazem (Class IV) is usually more effective for depressing AV nodal conduction than digoxin, and this drug is often used in dogs and cats for this purpose. (Infrequently diltiazem will actually convert the arrhythmia back to NSR.) There is some debate about which drug should be started first in the heart failure patient, but practically speaking, most clinicians use *combined* therapy with digoxin and diltiazem to control heart rate in atrial tachyarrhythmias of dogs. Remember that while digoxin is a positive inotropic drug, diltiazem actually depresses myocardial contractility and reduces systemic vascular resistance. Thus diltiazem should not be given to CHF patients without concurrent therapy to manage edema and reduced cardiac output (such as diuretics and pimobendan in dogs). In most cases, the dose of diltiazem is gradually increased to gain ventricular rate control while minimizing the negative inotropic impact of the drug.

When atrial tachyarrhythmias in dogs or cats are not associated with heart failure, most clinicians select diltiazem or a beta-blocker such as atenolol to control ventricular rate response. Diltiazem and a beta-blocker also can be used together, but care must be taken to avoid **excessive AV nodal conduction block** that might lead to ventricular bradycardia.

Reentrant SVTs using an accessory pathway employ circuits that develop at the micro and macro reentrant levels. The best characterized in dogs use a circuit involving the atria, AV node, and an accessory AV pathway that bypasses (or longitudinally separates) the AV conduction system. The tachycardia is often triggered by a sudden change in sinus cycle length, by premature atrial or ventricular complexes, or by electrophysiologic changes in the electrical pathways constituting the circuit. In most cases the circuit is “orthodromic”; down the AV node with an associated normal (narrow) QRS. Retrograde P’-waves may be identified in the ST segment (an R–P’). In some dogs, periods of sinus rhythm are associated with ventricular pre-excitation, a helpful clue to the presence of an accessory pathway. Pre-excitation is characterized by a short PR interval and early ventricular activation (the delta wave) with narrow to wide QRS and secondary T-wave changes. Management of orthodromic reentrant SVT is done with drugs initially (diltiazem and procainamide can be tried to block the AV node and accessory pathway, respectively), but referral to a specialist for catheter ablation of the accessory path is the best treatment.

Atrial standstill is another type of atrial arrhythmia, but does not result in tachycardia. This diagnosis indicates that the atrial muscle is unexcitable. It should not be confused with sinus arrest wherein the failure resides in the SA node discharge, not the atrial muscle depolarization. Atrial standstill is caused transiently by **high serum potassium** or persistently by atrial muscle disease (dogs, cats) or severe atrial dilation (in cats). The main ECG findings of atrial standstill are due to hyperkalemia include complete absence of P-waves, widening of the QRS complex, and increased amplitude of the T-waves (with abbreviated ST-T relative to heart rate). The P-wave and QRS changes are caused by partial depolarization of the cardiomyocytes, which inactivates fast sodium channels and slows or depresses depolarization. The ST-T changes are due to opening of potassium channels allowing for more rapid repolarization (despite the higher extracellular K^+, intracellular K^+ is still >>> extracellular K^+). SA impulses can still conduct to the AV node via functional (microscopic) internodal pathways, and the rhythm is sometimes termed “sinoventricular”, as no P waves are seen. The sinus discharge rate is

usually depressed in hyperkalemia, but in cats, this is less common, so that wide QRS complexes and T waves associated with severe hyperkalemia can be confused with an ectopic ventricular tachycardia. When atrial standstill is due to primary muscle disease (replacement of atrial muscle with fibrous tissue), either no P-waves or tiny, non-conducted P waves are evident. Generally, the AV conduction system is also involved in the fibrotic process so the patient depends on a junctional (AV nodal) or ventricular escape rhythm to initiate heartbeats. Persistent standstill caused by atrial myocardial disease and fibrosis is most common in English Springer spaniels, but can also occur in larger retriever breeds. In cats, apparent atrial standstill can be observed with severe forms of cardiomyopathy.

Ventricular Arrhythmias

Arrhythmias that arise ectopically in the ventricle parallel those of the atria in terms of nomenclature. However, there are some very important differences: 1) the AV node need not be activated to generate a QRS complex; therefore, rate control is not an effective strategy; and 2) there is greater potential for sudden death if the rhythm degenerates to ventricular fibrillation or asystole. Unfortunately, currently it is difficult to predict which patients will die suddenly or will benefit from therapy. The other obvious difference between rhythms of supraventricular and ventricular origin is the **morphology of the QRS-T**. Most ventricular arrhythmias cannot enter the His-Bundle Branch-Purkinje system normally. Therefore, depolarization spreads along abnormal conduction pathways, and propagates in some regions using slower cell-to-cell conduction. This results in a wider than normal QRS complex, often in the opposite direction, and frequently a "bizarre" morphology when compared to the normal activation process typical of sinus-rhythm impulses. The T-wave is also very large and oriented in the opposite direction, representing a secondary repolarization change (due to abnormal depolarization).

Idioventricular "escape" complexes are rescue mechanisms for sinus arrest, atrial standstill, or AV blocks and should not be suppressed by drugs. These complexes or rhythms originate from subsidiary pacemakers, usually residing in the AV junction or Purkinje system of the ventricle. The idioventricular (escape) rhythm is initiated when there is a failure of a normal impulse to depolarize the ventricles. If the escape originates near the bundle of His, the QRS is relatively normal, but will not be preceded by a related P wave. If the impulse starts deeper in the ventricle, the resultant QRS complex is wide with a more bizarre morphology when compared to normal sinus QRS complexes. These escape foci discharge at 20 to 40/minute in dogs, but in the cat, the rate is much faster, approaching 130/minute in many cats with complete AV block.

In contrast to a ventricular escape, the **premature ventricular complex (PVC, VPC)** arises early compared to the dominant R-R cycle to suddenly assumes pacemaker control of the ventricles. These ectopic complexes can be uniform or multiform in morphology suggesting a single focus or multiple foci (or different conduction patterns). A **fusion complex** might also be seen in some cases. This complex is a "sandwich" of a PVC and a sinus impulse that occurs when both impulses arrive simultaneously and collide in the ventricles. These are usually recognized by a preceding P wave, but shorter than normal PR interval, along with intermediate QRS morphologies between sinus and PVC. The finding of three or more linked PVCs constitutes a "run" of **ventricular tachycardia (VT)**. These ectopic ventricular rhythms can be "slow" (near the sinus node rate) or "fast"; paroxysmal or sustained; monomorphic or polymorphic; or rapidly varying in orientation (torsade de pointes = turning on point). The ventricles also

can **flutter** (creating sine waves), or **fibrillate** (disorganized reentry currents without mechanical contraction = a lethal activation). In very sick animals or in those with CHF, death can occur from **asystole**, which is essentially ventricular standstill.

PVCs are among the most common rhythm disturbances. **Causes** include primary electrical or structural heart diseases, including arrhythmogenic cardiomyopathies and dilated cardiomyopathy. Other causes include electrolyte and metabolic disturbances, autonomic imbalance, drugs, toxins, and the "usual suspects", such as splenic masses and gastric dilatation in dogs systemic inflammation. The electrophysiologic mechanism for the arrhythmia is rarely known.

It can be difficult to decide if PVCs are "clinically significant" or not, but the issue is important. For example, most cats with chronic ventricular ectopy have structural heart disease (cardiomyopathy) or at least an elevated serum troponin suggestive of active myocardial injury or myocarditis. A Doberman pinscher with PVCs on a routine ECG is likely to progress towards overt dilated cardiomyopathy. When an ECG demonstrates even a few PVCs in a dog of this breed that has collapsed or fainted, the risk of sudden cardiac death is very high. Arrhythmogenic right ventricular cardiomyopathy is common in Boxers and English bulldogs. These predispositions can prompt antiarrhythmic therapy in a Doberman or Boxer dog, recognizing there is no proof treatment will prolong life. Conversely, some asymptomatic boxers have PVCs for years without signs and these patients are best assessed by reviewing history and an ambulatory (Holter) ECG recording before starting any treatment.

ECG diagnosis of PVCs or of VT is generally straightforward, although it can be confused by supraventricular tachycardias conducted with aberrancy (bundle branch block) or by hyperkalemia. A full medical **workup** includes drug and medical history, consideration of clinical signs (weakness, collapse or syncope), Echo findings, laboratory tests (CBC, chemistries, cardiac troponin-I), and abdominal ultrasound in older dogs at risk for splenic disease. These are obtained to determine the most likely cause and overall clinical significance of the arrhythmia. A Holter (24h ambulatory) ECG can contribute to assessing the severity and complexity of the PVCs as well as response to therapy. Based on some Holter ECG studies, >10/day in cats and >50/day in dogs would be considered abnormal (others use *lower* limits). The absolute number of PVCs per day needing treatment is controversial. Spontaneous daily variation is common (up to ~85%); this should be taken into account. In general, **clinical signs** (collapse, syncope); **clinical situation (anesthesia; hypotension)**; and modified "**Lown criteria**" are used to assess severity and need for therapy. The latter include (a) "uniform" vs. "multiform" PVCs; (b) "monomorphic" vs. "polymorphic" VT; (c) "late" versus "R on T" timing of premature complexes; and (d) "slow" vs. "fast" ventricular rhythms – with the second of each pair considered more hemodynamically or electrically destabilizing.

Management of ventricular ectopic rhythms involves determining the most likely cause, advancing an educated guess about the clinical significance, considering the need for therapy, and possibly choosing one or more drugs. All antiarrhythmic drugs carry the potential for side effects and worsening of the arrhythmia (proarrhythmia).

Lidocaine remains the drug of choice for acute management of serious PVCs or VT in animals. Doses are important with cats more sensitive to neurotoxicity of the drug. Any underlying electrolyte disturbances, especially involving potassium or magnesium, should be corrected. As sympathetic input can trigger or worsen VT, any pain should be controlled. Fluids or colloids might be needed to support blood pressure. Second-line treatments include intravenous procainamide (when available),

amiodarone, esmolol, and magnesium salts (which stabilize membranes). These are often trial-and-error treatments, and experience has brought these drugs to the fore.

For **chronic oral therapy**, **sotalol** (class III drug with beta-blocking properties) is generally the best tolerated, and it often reduces the frequency and complexity of ventricular ectopy (beware: negative inotropic effects in CHF). Sotalol can be administered to dogs and cats. Alternatives for dogs are **mexiletine** (class IB, similar to lidocaine, but oral) or sotalol plus mexiletine. **Amiodarone** (class III) can be effective in dogs, but deserves respect, especially in terms impairing liver function; it can be co-administered with atenolol. Flecainide is a potentially useful drug but experience is low and the drug can lead to serious "pro-arrhythmia", especially in settings of a failing heart.

Conduction Disturbances

In addition to sick sinus syndrome, persistent atrial standstill, and ventricular pre-excitation (each mentioned above), conduction disturbances include the AV blocks; bundle branch blocks, and intraventricular conduction disturbances. Bundle branch blocks and fascicular blocks do not cause clinical signs.

The AV blocks are classified as first, second (Mobitz I, Mobitz II), and complete (third-degree block). **First-degree AVB** is characterized by a P wave preceding each QRS complex but with a longer than normal PR interval for the species. **Second-degree AVB** is "incomplete", meaning some impulses conduct to the ventricles and others do not. Mobitz type I, second-degree AVB (also called *Wenckebach* type) is typical of high vagal tone and drug effects and is characterized by a progressively longer PR interval until a P wave is blocked. Mobitz type II second-degree AVB is often due to conduction disease, as occurs with senile degeneration in the bundle of His or bilateral bundle branches. In this rhythm, the PR interval is relatively fixed, and often prolonged, and one or more P-waves are blocked at a time. "High-grade" second-degree AVB usually refers to blocking of three or more P waves before one is conducted. **Third-degree AVB** is "complete", meaning no atrial impulses conduct into the ventricles. The block occurs in the AV node, bundle of His, or both bundle branches. It is usually due to degeneration of the conducting pathways, although other causes (myocarditis, tumors, concentric hypertrophy in the area) are other potential causes. Since atrial impulses are blocked, the heart can only discharge from an escape pacemaker (idioventricular rhythm), typically from the His-Purkinje system and below the level of the block (see previous discussion under Ventricular Arrhythmias).

Treatment of symptomatic AV blocks generally involves referral for permanent pacing. Single or dual chamber pacing systems can be used, depending on the variety of patient and technical factors. When AVB is due to vagal input or drugs, atropine can often abolish the block.

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