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CARDIAC EDUCATION group

Atrial Fibrillation: Diagnosis and Clinical Management

Atrial fibrillation is one of the most common arrhythmias encountered in veterinary medicine, second in frequency only to isolated atrial or ventricular premature complexes. This arrhythmia can arise secondary to any cardiac disease that causes pathology in the cardiac atria, but it can also arise spontaneously in patients with no structural heart disease, or in patients with autonomic nervous system imbalance. This summary will review the anatomic and electrophysiologic causes of atrial fibrillation, clinical presentation of patients with this arrhythmia, and treatment recommendations focused on the associated underlying cause(s) and clinical syndromes.

Electrophysiology Review

In most patients with a structurally normal heart and cardiac conduction system, the sinoatrial (SA) node is responsible for initiation of the cardiac rhythm. Automaticity of cells within the SA node causes them to depolarize, and this electrical impulse travels through the atria via a series of specialized conduction fibers (Bachmann's bundle) to propagate electrical activation of the atrial myocytes. This is represented by the P-wave on the surface electrocardiogram (Fig. 1, black arrows). This electrical wavefront then reaches the AV node, where impulse conduction slows entering the bundle of His. On the surface ECG, this is manifest as the PR (occasionally referred to as the PQ) interval (Fig. 1, red brackets). Once the electrical impulse reaches the specialized conduction fibers below the AV node (bundle branches and Purkinje fibers), it is propagated rapidly through the ventricles and ventricular depolarization is represented by the QRS complex on the surface ECG (Fig. 1, red arrows). The final waveform on the ECG, the T-wave (Fig 1. blue arrows), represents repolarization of the ventricular myocardium. A similar repolarization wave exists for atrial repolarization (Ta wave), but it is rarely seen as it coincides temporally with ventricular depolarization and the QRS complex.

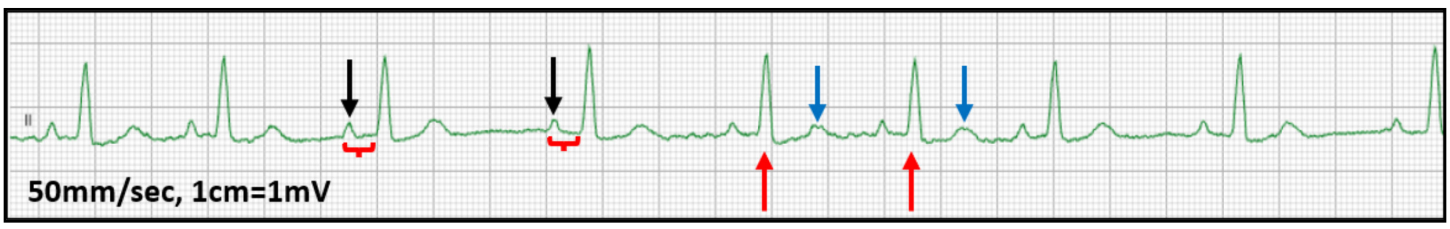
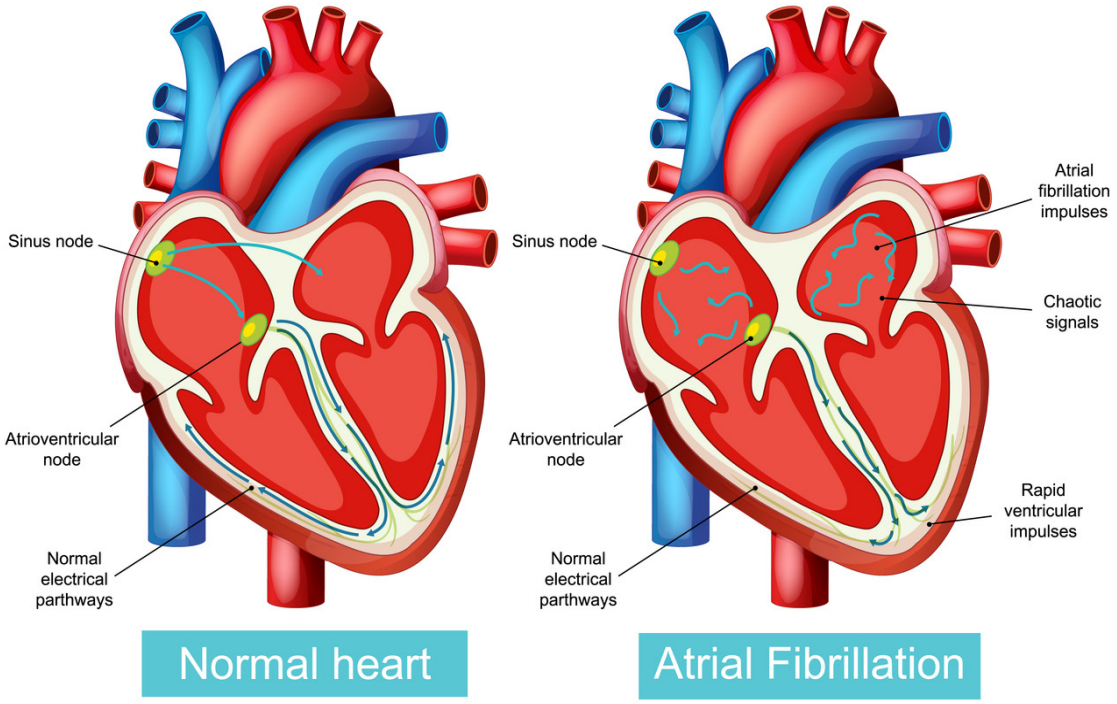


Figure 1

ECG Characteristics of Atrial Fibrillation

Atrial fibrillation is characterized by disorganized atrial electrical activity. The simultaneous formation and propagation of numerous small “wavelets” of electrical activity exists within either or both atria. These wavelets can travel around a physical structure (i.e. pulmonary vein) or circulate around microscopic pathology within the atria, such as myocardial fibrosis. These wavelets cause multiple, disorganized, and uncoordinated electrical impulses of varying strength to bombard the AV node in rapid succession (Fig 2). The slower electrical conduction of the AV node limits the number of impulses that are conducted to the ventricles, and the impulses reach the ventricle at varying intervals creating an irregular, chaotic rhythm. The ensuing heart rate may be highly variable and is often very rapid.

Figure 2



Age and Treatment Considerations

Due to the irregular activation of the ventricles in atrial fibrillation, the surface ECG reveals no regular pattern, but the QRS morphology is similar to sinus and other supraventricular rhythms since conduction through the Purkinje fibers of the ventricles is unchanged. The hallmark ECG features of atrial fibrillation are:

- Irregularly irregular rhythm
- Supraventricular origin (narrow, normal QRS morphology)
- No visible P-waves
- +/- Rapid rhythm, particularly with high sympathetic tone

These characteristics are noted in Fig 3. If a rapid, irregular rhythm with narrow QRS complexes is documented on a surface ECG, atrial fibrillation is the most likely differential.

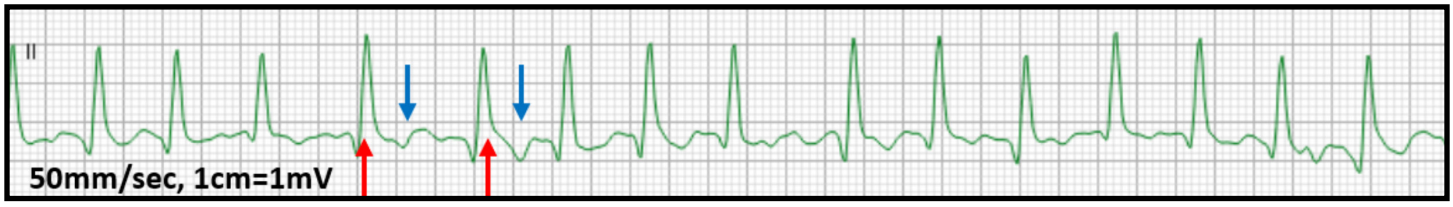


Figure 3

History/Physical Examination Findings

The development of atrial fibrillation results in a sudden loss of the atrial contribution to ventricular filling, since the atrial myocardium is not contracting in a synchronized fashion before ventricular contraction. Furthermore, the rapid heart rate that often ensues with atrial fibrillation limits diastolic ventricular filling. The net effect of these changes lead to a significant drop in forward cardiac output, up to 30% at rapid heart rates. Coupled with underlying structural heart disease, this often leads to overt clinical signs such as lethargy, weakness, or syncope. If there is severe pre-existing heart disease, patients may have a relatively rapid onset of congestive heart failure symptoms, including tachypnea, dyspnea, coughing, or abdominal distension due to ascites. In patients without structural heart disease (i.e. "lone," or primary atrial fibrillation), the arrhythmia may be detected incidentally on a routine physical examination in the absence of clinical signs.

Underlying Etiologies

As mentioned previously, any disease that causes significant pathology or enlargement of the atria can result in atrial fibrillation. This is often referred to as "secondary" atrial fibrillation. Common causes include, but are not limited to:

- Myxomatous valvular degeneration with substantial AV valve regurgitation
- Dilated cardiomyopathy (canine)
  - Idiopathic/Inherited
  - Diet-associated
- Congenital heart disease (tricuspid valve dysplasia, patent ductus arteriosus, subaortic stenosis, pulmonic stenosis, etc.)
- Hypertrophic and restrictive cardiomyopathies (feline)
- Myocarditis
- Cardiac neoplasia
- Pericarditis

One exception is primary, or "Lone," atrial fibrillation, which is seen almost exclusively in large or giant breed dogs. In these patients, it is felt that the relatively large atrial mass, in conjunction with a preponderance of either vagal or sympathetic stimulation, can result in the development of spontaneous atrial fibrillation without underlying structural heart disease or atrial enlargement.

Given the large number of potential causes of atrial fibrillation, as well as the different prognosis associated with primary vs. secondary atrial fibrillation, the discovery of this arrhythmia always warrants further investigation with an electrocardiogram, echocardiogram, thoracic radiographs (particularly in symptomatic individuals), routine laboratory evaluation (including a thyroid level), and possibly a cardiac troponin I level to screen for myocarditis. Ideally, referral to a cardiologist should be considered.

Treatment

The treatment for atrial fibrillation depends heavily on any underlying cause of the arrhythmia as well as the patient's clinical signs.

Heart rate control (negative chronotropic therapy): In most patients with atrial fibrillation, clinical presentation is affected by a rapid ventricular rate. Rate control is achieved by increasing the refractory period of the AV node, thereby decreasing the number and speed of atrial impulses that reach the ventricles. The resulting reduction in heart rate helps to improve ventricular filling and forward cardiac output, even though the rhythm remains irregular. Negative chronotropic therapy is ideally monitored with serial ECGs (target heart rate in hospital <160bpm average) or 24hr Holter monitoring (target heart rate average <120bpm over 24hrs). Some commonly used negative chronotropic medications are listed here:

- **Diltiazem:** A commonly used calcium channel blocker that is readily available, cost effective, and usually well tolerated. Side effects may include nausea, vomiting, or lethargy/weakness associated with a drop in heart rate or blood pressure. The drug comes in a regular release preparation that typically requires dosing three times a day in dogs. However, an extended-release preparation is also available that can be effective when dosed twice daily. ***CEG Canine Formularies***
- **Digoxin:** This is a glycoside compound found naturally in the foxglove family of plants that are known to have cardiotoxic effects. Cardiac glycosides have been used for more than a century, but are less readily available due to the infrequency of use in human medicine and their side effect profile, which can include nausea, vomiting, diarrhea, and other cardiac arrhythmias. The use of digoxin also requires regular therapeutic monitoring, with a narrow

therapeutic window. However, when used/monitored appropriately, it can be effective and is the only negative chronotrope that has a small positive effect on contractility (positive inotropy). ***CEG Canine Formularies***

- **Beta-blockers:** This class of drugs includes several readily available medications that are clinically and cost effective. However, the relatively potent negative inotropic effects of many of these medications may make them more difficult to use in patients with significant myocardial disease or dysfunction (dilated cardiomyopathies). They should not be used in patients with active heart failure symptoms unless other options have been exhausted
  - **Sotalol:** While not a "pure" beta blocker, this drug appears to have less potent negative inotropic effect than other "pure" beta blockers.
  - **Atenolol**
  - **Metoprolol**
  - ***CEG Canine Formularies***
- **Potassium channel blockers:** This class of drugs includes both sotalol and amiodarone, in addition to a few less commonly used agents in veterinary medicine. While sotalol is relatively common and has a limited side-effect profile, amiodarone is used infrequently and has a higher number of side effects that limit its use to patients that have failed other therapies. Consequently, amiodarone is typically not considered until a patient is under the care of a veterinary cardiologist. However, both drugs may be effective at slowing the ventricular response rate, either alone or in combination with other drug classes. ***CEG Canine Formularies***
- **Combination therapy:** Several publications have documented a benefit to the use of combination therapy to reduce ventricular response rate in patients with symptomatic atrial fibrillation, with heart rate control that may be superior to single agent therapy. However, these therapeutic protocols require more intensive monitoring, typically under the guidance of a veterinary cardiologist:
  - **Diltiazem + digoxin**
  - **Diltiazem + beta blocker**
  - **Diltiazem + sodium channel blocker**

Treatment of congestive heart failure: Since congestive heart failure often develops with the onset of atrial fibrillation, management of congestive signs is often needed concurrently with negative chronotropic therapy. Typical heart failure therapy involves the use of diuretics, medications to inhibit the renin-angiotensin-aldosterone system (RAAS), and positive inotropic therapy. In patients that develop ascites after the onset of atrial fibrillation, adequate rate control alone may control their congestive symptoms without the use of aggressive diuretic therapy. Therapeutic strategies for managing congestive heart failure are beyond the scope of this article but are highlighted in numerous other references:

- ***ABCDs of Myxomatous Mitral Valve Disease***
- ***ABCDs of Canine Cardiomyopathy***
- ***ABCDs of Feline Cardiomyopathy***

Addressing non-cardiac factors: In patients with pericarditis, myocarditis, neoplasia, or other systemic diseases that contribute to the development of atrial fibrillation, the underlying condition should be addressed, if possible (i.e. pericardiectomy, removal of intrathoracic masses, etc.)

Cardioversion: This approach to managing atrial fibrillation aims to convert a patient back to a normal sinus rhythm. This re-establishes atrio-ventricular synchrony, thereby improving ventricular filling by restoration of the atrial "kick" and establishes normal heart rate control by the sinoatrial node. Cardioversion can occur medically with some drugs mentioned above (amiodarone, sotalol, etc.), but is more frequently performed with synchronized electrical cardioversion using a cardiac defibrillator. While this is theoretically the ideal approach to managing atrial fibrillation, it is often unsuccessful or only temporarily successful in patients with significant structural heart disease or atrial enlargement; recurrence back to atrial fibrillation is common. Consequently, cardioversion is usually reserved for patients that have primary, or "lone," atrial fibrillation. When successful, it can reduce or eliminate dependence on chronic oral medications. This therapy is typically performed under the guidance of a veterinary cardiologist.

Ablation: Although one of the mainstays of therapy in human patients, ablation has not yet been utilized for successful elimination of atrial fibrillation in clinical veterinary patients. This therapy is aimed at eliminating the micro (or macro) re-entrant loops that serve to propagate fibrillation waves. Since this is a permanent treatment, successful ablation would eliminate the need for long-term medical therapy. This may be an emerging therapy in the future as tertiary referral centers develop electrophysiology laboratories.

Anticoagulation: Use of anticoagulation strategies is an important consideration in human patients with atrial fibrillation. Fortunately, anticoagulation is rarely needed in dogs with atrial fibrillation, restricted only to patients with pre-existing diseases that promote hypercoagulability (protein-losing nephropathy, etc.) or in feline patients, which are more prone to cardiogenic thromboembolism.

Prognosis

As with many arrhythmias, the ultimate prognosis for patients with atrial fibrillation depends on the underlying etiology. In patients with substantial structural heart disease, the onset of atrial fibrillation is typically a negative prognostic factor, and it may occur late in the course of disease when other heart failure therapies are already in use. Consequently, atrial fibrillation in this population carries a poor prognosis. However, adequate rate control can still be rewarding and improve clinical signs in patients with active congestion, thereby improving their quality of life.

In patients with primary, or "lone," atrial fibrillation, the prognosis is typically good. With adequate control of the heart rate, most of these patients can live a normal length and quality of life with minimal restrictions. The lack of atrial myocardial contraction that occurs with atrial fibrillation results in atrophy of the atrial myocardium.This can cause gradual atrial enlargement, secondary valvular regurgitation, and ultimately signs of congestive heart failure. In patients with lone atrial fibrillation, atrial enlargement may take years to develop after arrhythmia diagnosis, with many patients dying from other, non-cardiac causes. Yearly recheck echocardiography, electrocardiography, and therapeutic monitoring (regular digoxin levels) is recommended for these patients.

Summary

Atrial fibrillation is a relatively common arrhythmia encountered in veterinary medicine. Recognition of the arrhythmia is important as it often causes significant clinical signs, particularly in patients with underlying structural heart disease. While it is uncommon to be able to convert patients with this arrhythmia back to a normal sinus rhythm, pharmaceutical control of the ventricular response rate can significantly improve clinical signs and maintain quality of life in affected individuals. In patients with primary or lone atrial fibrillation, therapy may be curative (electrical or medical cardioversion), but patients often do very well with negative chronotropic medications alone.

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