Atrial fibrillation – a comparative review of one of the most common arrhythmias in dogs and humans

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Abstract

Atrial fibrillation is the most prevalent arrythmia in dogs and humans and is characterised by chaotic depolarisation of the atria, resulting in damage to the atrial myocardium. It can lead to heart failure or worsen an existing condition and cause sudden death. Electrocardiographically, atrial fibrillation is characterised by the absence of P waves and an irregularly irregular rhythm, with narrow QRS complexes. Treatment of atrial fibrillation requires knowledge of pathophysiology and the pharmacology of antiarrhythmic drugs. In contrast to human medicine, the treatment of dogs revolves mainly around rate control strategies, although new studies propose the consideration of several rhythm control strategies. Considering the complexity of atrial fibrillation, there is a strong need for a consensus on classification, diagnosis and treatment in dogs.

Key words: *arrythmia; atrial fibrillation; antiarrhythmics; dog; rate control; rhythm control*

Introduction

Atrial fibrillation (AFIB) is the most common arrythmia in dogs (Bonagura and Ware, 1986; Guglielmini et al., 2020; Pedro, 2020), and humans (Hindricks et al., 2020). It can be defined as supraventricular tachyarrhythmia with uncoordinated atrial electrical activation and consequently ineffective atrial contraction (Hindricks et al., 2020). Although it can occur without concomitant heart disease, atrial fibrillation is most often diagnosed in dogs suffering from heart condition in which the left atrium is severely enlarged, as is the case in dilated cardiomyopathy (DCM) (Guglielmini et al., 2020) and myxomatous mitral valve disease (MMVD) (Menaut et al., 2005; Guglielmini et al., 2023).

Factors that can induce and maintain AFIB have been described in both dogs and humans and include electric and structural remodelling of the atrial myocardium. AFIB is a consequence of atrial stretch or enlargement resulting

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in atrial tissue damage, with consequent inflammation and fibrosis. Fibrous tissue has slow conduction properties, allowing for reentry to take place. Additionally, electrical remodelling has also been described in both dogs and humans that involves calcium and sodium channels, where the impairment of these channels leads to shortening of action potential duration. Electrical and structural changes (such as loss of myofibrils, changes in mitochondrial properties and structural proteins) contribute to development as well as maintenance of chronic AFIB (Meijler et al., 1987; Li et al., 2000; Laurent et al., 2008).

Several risk factors have been described and associated with AFIB in dogs (Arcuri et al., 2024) and humans (Psaty et al., 1997; Waldo, 2003). Considering its prevalence and impact on survival in dogs (Pedro et al., 2018, 2020) and humans (Hindricks et al., 2020; Lippi et al., 2021), it is an important topic for further medical research. This review represents the latest knowledge on the epidemiology, pathophysiology, classification, diagnostic and treatment strategies of AFIB in dogs. Additionally, this review also presents human research for comparison and better understanding.

Epidemiology

In dogs, AFIB is the most common arrythmia, accounting for approximately 33% of arrythmias in the reference population (Noszczyk-Nowak et al., 2017). Large and giant breeds are overrepresented in the general canine population, likely because of the larger atrial dimensions (Pariaut, 2017; Pedro et al., 2020). In a retrospective statistical review by Westling et al. (2008), the overall prevalence of AFIB was 0.15%, with overrepresentation of giant dog breeds like Irish Wolfhounds (5.84%), Newfoundlands (1.68%), Great Danes (1.64%), Mastiffs (1.24%), Saint Bernards (0.85%) and large dog breeds such as Dobermans (0.76%) and Bull Mastiffs (0.58%).

Distribution of age at initial AFIB diagnosis by breed group revealed that giant breed dogs developed AFIB early in life while small breed dogs were diagnosed later in life. Gender analysis showed that males are overrepresented (Guglielmini et al., 2000).

Atrial fibrillation is the most common arrhythmia associated with canine DCM, however it may also develop in dogs with MMVD and other structural heart diseases in which atrial enlargement is present (Jung et al., 2016; Friederich et al., 2020; Pedro et al., 2020). Studies in humans have also shown that AFIB is the most common arrythmia, with approximately 33% of arrhythmia-related hospitalisation due to AFIB. In humans, incidence of AFIB is age and gender related, ranging from 0.1% per year before 40 years of age to 1.5% in women and 2% in men older than 80 years (Lippi et al., 2021). Predisposing factors are congestive heart failure, aortic and mitral valve disease, systemic hypertension, obesity, obstructive sleep apnoea, and alcohol abuse (Roger et al., 2011; Hindricks et al., 2020).

Pathophysiology

AFIB is characterised by chaotic atrial electric activity. It can be maintained by reentry and/or rapid focal ectopic firing. The electrophysiological mechanism for induction and maintenance of AFIB is reentry, although triggered activity (as is the case in focal ectopic firing) can also induce atrial fibrillation (Iwasaki, 2011). Such ectopic foci are most commonly found in the myocardial sleeves of pulmonary veins (Hocini et al., 2002; Hel-

lemans et al., 2023). There can be one or more reentry circuits and factors promoting reentry include dispersion of refractoriness, short atrial refractory period, fibrosis and atrial enlargement (Iwasaki, 2011). Currently there are two models that have been described in both dogs and humans: multiple wavelet model and focal model, although other mechanisms are known to be also involved (Brundel et al., 2005; Iwasaki, 2011; Schotten et al., 2011). The multiple wavelet model is characterised by minimal number of circuits, usually six or more, that provide the substrate for reentry currents that can maintain AFIB (Moore and Spear, 1987). These reentry currents are the consequence of shorter refractory period of atrial myocytes, low conduction velocity, and larger atrial mass. The term critical mass is used to describe sufficient atrial mass for the maintenance of AFIB and is defined as the minimal required area for propagation of reentry circuits (Moore and Spear, 1987; Li et al., 1999). The focal model is characterised by one or more atrial ectopic foci with fast discharging rates that create irregular wavelets that promote AFIB. These ectopic foci are most commonly found in the area of pulmonary veins, as stated earlier (Moore and Spear, 1987; Li et al., 1999).

Atrial fibrosis is a significant factor for the induction but also the maintenance of chronic AFIB both in dogs and humans. Fibrosis promotes slow pathways in atrial myocardium allowing for the creation of macro- and micro-reentry circuits that can serve as a substrate for permanent AFIB (Nattel et al., 2005; Nguyen et al., 2009).

AFIB in dogs is predominantly secondary in nature, and occurs in association with various pathologic substrates, most commonly structural remodelling and ischemic changes (Brundel et al., 2005). Primary atrial fibrillation occurs without obvious structural heart diseases, and it is most commonly triggered by focal atrial tachycardia arising within the tributary atrial veins (Hassani and Saremi, 2011; Hellemans et al., 2023).

Detailed classification of AFIB based on onset and duration has been described in human medicine (Hindricks et al., 2020). In veterinary medicine, there is no consensus on AFIB classification and classification used in humans has been also used in dogs. Again, AFIB found in dogs is most commonly secondary and associated with structural heart diseases. Primary AFIB is also called idiopathic or lone AFIB and is found in a small number of cases. It is assumed that giant dog breeds are genetically predisposed (Menaut et al., 2005; Westling et al., 2008). Primary AFIB in dogs has been best described in Irish Wolfhounds, where 48 of 500 dogs had chronic atrial fibrillation without signs of DCM (Vollmar et al., 2000). AFIB can also be induced by increased parasympathetic tone.

Secondary AFIB in dogs is called chronic or permanent and is by far the most common type (Menaut et al., 2005). This is in contrast to humans where the most common form is paroxysmal AFIB (Hindricks et al., 2020). Paroxysmal AFIB has been described in dogs, but its prevalence is unknown. It is most commonly associated with changes in autonomic nervous system tone and certain drugs (Pariaut et al., 2008; Porteiro et al., 2016). Because muscarinic receptors are several times more present in the atria than in ventricles, high parasympathetic tone can induce depression of the sinoatrial (SA) and atrioventricular (AV) nodes and consequently induce paroxysmal atrial flutter or AFIB in humans (Waldo et al., 2003). Paroxysmal, vagally induced AFIB has been described in German Shepherd

dogs and successfully converted to sinus rhythm with lidocaine (Pariaut, 2008). In humans paroxysmal AFIB is more common than in dogs, accounting for 25% of AFIB cases (Zoni-Berisso et al., 2014). One of the main reasons for this is that Holter-ECG monitoring is not as often used in dogs as in humans. More research is needed in dogs to better understand the prevalence and pathophysiology of paroxysmal AFIB. Pedro et al. (2020) stated that, similar to humans, paroxysmal AFIB in dogs may precede chronic AFIB.

There are three main mechanisms by which AFIB promotes heart failure: (1) loss of atrial contraction, (2) rhythm irregularity coupled with high ventricular response rate, and (3) atrial fibrosis (Menaut et al., 2016; Friederich et al., 2020; Pedro et al., 2020). Loss of atrial contraction results in decreased atrial emptying, lower ventricular filling and consequently lower cardiac output. The effects of tachycardia on myocardial function have been shown to occur, both by rapid atrial and by ventricular pacing, and are further compounded by dyssynchrony and irregularity (Pedro et al., 2020). The atrial rate in AFIB is between 400 to 700 beats/min. The ventricular rate is dependent on the number of atrial depolarisations that pass through the AV node that acts as a filter between the atria and ventricles. In the AV nodes, slow pathways block excessive conduction from the atria, by an underlying mechanism called ventricular penetrance. The usual ventricular response in dogs is 120 to 260 beats/min, but the autonomic nervous system (mainly the sympathetic nervous system) and certain drugs can enhance conduction frequency through the AV node (Langendorf and Pick 1965; Kirsh et al., 1988; Menaut et al., 2005).

AFIB is a functional reentry supraventricular tachyarrhythmia in which irregular FF and RR intervals are found, and the nature of the distribution of the irregular RR interval is mainly the consequence of irregular FF intervals (Kirsh et al., 1988). This mechanism is very important considering that dogs mainly have chronic AFIB, in which the goal of treatment is rate control or lowering of the ventricular rate.

Diagnosis

Considering its complexity, high prevalence and health impact, it is important to ensure proper diagnosis to initiate appropriate treatment. Although in a thorough physical examination one might suspect AFIB based on a fast and irregular heartbeat, the gold standard for AFIB diagnosis is electrocardiography (ECG) and in some cases 24-hour ECG (or Holter-ECG) monitoring. AFIB is characterised by the absence of P waves and an irregular RR interval or irregularly irregular rhythm. The QRS morphology and duration are normal (duration less than 70 ms), unless anatomical or functional intraventricular conduction disturbances are present (Pedro et al., 2020). To differentiate AFIB from other fast supraventricular rhythms, 12-lead ECG should be performed, with lead V1 placed in the right first intercostal space as proposed by Santilli et al. (2019). This novel precordial system allows for easier differentiation of complex and fast supraventricular rhythms where atrial deflection is not visible on the frontal plane ECG (Santilli et al., 2008; Battaia et al., 2023). The F waves are hallmarks of AFIB and are best visualised in lead II, which is parallel to the interatrial septum, lead aVL, which is closest to the left atrium, and lead V1 which is closest to the right atrium (Park et al., 2019).

In humans, F waves are described as highly organised in paroxysmal AFIB. As the AFIB progresses into long standing,

long standing persistent and eventually into permanent, it becomes more disorganised, characterised by a coarse sawtooth pattern on the surface ECG (Hoppe et al., 1999; Schotten et al., 2011). Sih et al. (2000) suggested that differences in organisation between acute and chronic atrial fibrillation in dogs have a pattern similar as in humans. Park et al. (2019) proposed a model that can be used to recognise longstanding AFIB, which is based on the amplitude, irregularity and dominant rate of the F waves. However, no such (or similar) model with cut-off values has been developed in dogs. Holter-ECG monitoring is a useful tool to diagnose and monitor AFIB in both dogs and humans. Holter-ECG monitoring is more often used in humans in cases of suspected paroxysmal and familial AFIB (Volders et al., 2008). Considering that paroxysmal AFIB is rare and there is currently no evidence of familial AFIB in dogs, Holter-ECG is used mostly for monitoring of therapy effects (rate control) in cases of chronic AFIB in dogs (Pedro et al., 2023).

Treatment

The main goal of therapy is to resolve clinical signs of heart failure, improve cardiac output and slow the progression of AF (Saunders et al., 2009). AFIB treatment consists of two main strategies: rate control and rhythm control. Rythm control involves either chemical or electrical cardioversion, although both are often used in humans (Hindricks et al., 2020). In dogs, the usual method of long-term treatment remains rate control (Pedro et al., 2023).

Cardioversion is a procedure in which pathological rhythm is converted into sinus rhythm. In humans, chemical cardioversion is performed for unstable patients with diltiazem or esmolol, and for stable patients with ibutilide, procainamide or amiodarone. In dogs, chemical cardioversion can be performed with amiodarone (Saunders et al., 2006) or lidocaine (Pariaut et al., 2008) but few cases of conversion are documented. In humans, electrical cardioversion is performed most commonly, by transthoracic cardioversion with biphasic waveform with a success rate of 95% in cases of acute onset AFIB. The principle is that depolarisation of a large area of the atrial myocardium will disrupt the multiple reentry circuits and produce a more electrically homogeneous environment and restore sinus rhythm (van Gelder, 1999). In dogs, Bright et al. (2005) performed transthoracic electrical cardioversion with a 92.3% success rate and the median time of the cardioverted sinus rhythm lasted for 120 days. They concluded that electrical cardioversion is beneficial in dogs although more studies are required given the variable survival outcome. Rate control in humans is indicated when cardioversion is not possible or in cases of asymptomatic patients older than 65 years of age. The goal of long-term treatment is to achieve ventricular rate while resting from 60 to 80 beats/min. Treatment options available are digitalis glycosides, beta blockers, calcium channel antagonists and amiodarone (Hindricks et al., 2020).

In dogs, rate control is the main approach to long-term management. There is no consensus as to an optimal ventricular rate for dogs, though it is widely accepted that it should be less than 150 beats/min (Pedro et al., 2023). Considering the ambulatory stress in dogs, it is recommended that optimal rate control monitoring should be performed by 24 hour-ECG. Rate control is most commonly achieved with digitalis glycosides and calcium channel antagonists, sometimes

with beta blockers and potassium channel blockers (Table 1) (Pariaut et al., 2017; Pedro et al., 2023). Although digitalis glycosides and calcium channel antagonists can be used as a monotherapy, they are often not sufficient. Calcium channel antagonists reduce AV nodal conduction rate and in combination with digitalis glycosides, which decrease sympathetic tone and increase parasympathetic tone, giving better results in dogs with overriding sympathetic tone. Beta blockers can also be used in dogs, but care must be taken to evaluate ventricular systolic function, since they have a negative inotropic effect (Jost et al., 2021; Pedro et al., 2023).

In the case of focal AFIB arising from pulmonic veins, medical treatment is not recommended. Ablation techniques are well described in humans and have been proven very effective in rhythm control strategy. Ablation is performed either by radiofrequency or cryothermal method (Ang and Earley, 2016). The anatomy of pulmonic veins in dogs is well documented (Brewer et al., 2012). As in humans, pulmonic veins can be the main site of origin of focal AFIB in dogs (Hellemans et al., 2023). Radiofrequency ablation is well known as the method of choice for ablation of the accessory pathways (Wright et al., 2018). In dogs, it is considered that radiofrequency ablation of ectopic foci and pulmonic vein isolation can be curative, but more research is needed to better understand the full therapeutic potential of this treatment method (Hellemans et al., 2023).

Table 1. Drugs used to treat atrial fibrillation. The most common combination used is digoxin and diltiazem (combination or dual therapy)

Vaughan Williams class	Drug	Dose	Side effects
Class II – Beta blockers	Atenolol	Oral dose: 0.2–1 mg/kg, q12h (start low, aim high)	negative inotropy and chronotropy
Class III – Potassium channel blockers	Sotalol	Oral dose: 1–2.5 mg/kg q12h (start low, aim high)	negative inotropy and chronotropy, proarrhythmia
	Amiodarone	Oral dose: 10–15 mg/kg q12h for 7 days, followed by 5–7.5 mg/kg q12h for 14 days and then 5–7.5 mg/kg q24h	thyroid dysfunction, hepatotoxicity, blood dyscrasias (neutropenia), and proarrhythmia
Class IV – Calcium channel blockers (antagonists)	Diltiazem	Oral dose: 1–4 mg/kg q8h or up to 3–5 mg/kg q12h for sustained or extended – release preparations	systemic hypotension, negative inotropy and chronotropy
Class V – other mechanisms	Digoxin	Oral dose: 2.5–3 µg/kg q12h or 0.11 - 0.22 mg/m^2 q12h in dogs >20 kg	anorexia, vomiting, diarrhoea, depression, proarrhythmia

Although thromboembolic disease is a common comorbidity in humans (Hindricks et al., 2020; Jost et al., 2021) it is rarely seen in dogs as a consequence of AFIB (Nishida et al., 2012). Several new drugs have been introduced in veterinary medicine for the treatment of AFIB, such as ranolazine, a drug that prolongs the action potential duration, blocks sodium currents and prevents calcium overload caused by the hyperactive sodium channels, thus stabilising the membrane and reducing excitability (Burashnikov et al., 2014). Additionally, a new method of vagal nerve stimulation via an implantable device to decrease atrioventricular node conduction and slow ventricular response rate during atrial fibrillation has been successfully used in dogs and could offer an alternative to rate control medications (Ohad et al., 2008).

Conclusion

Although much new insights have been published about AFIB in veterinary medicine over the past 20 years, there is still no consensus on diagnosis and treatment in dogs. Studies in dogs lack the necessary information for categorisation of AFIB based on the time of onset and duration in contrast to human studies. There is strong need for prospective studies for long-term rate control and more studies for determining when electrophysiology mapping is needed and the importance of ablation techniques. In veterinary medicine, there is also a lack of knowledge for identifying dogs at a higher risk of developing AFIB, and a lack of a consensus on screening methods for early diagnosis of AFIB in dogs.

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Fibrilacija atrija – komparativni pregled najčešće aritmije u pasa i ljudi

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Fibrilacija atrija najčešća je aritmija u pasa i ljudi, a karakterizirana je kaotičnom i nesvrishodnom depolarizacijom atrija s posljedičnim oštećenjem miokarda atrija. Navedena aritmija može dovesti do razvoja popuštanja srca ili pogoršati već njegovo postojeće popuštanje, ali biti uzrokom iznenadne srčane smrti. Elektrokardiografski fibrilaciju atrija karakterizira odsutnost P valova, nepravilno nepravilan ritam i uski QRS kompleksi. Liječenje fibrilacije atrija zahtijeva poznavanje patofizioloških mehanizama, ali i farmakologije antiaritmika. Za razliku od humane medicine, liječenje pasa je usmjereno na kontrolu frekvencije, iako se u novijim istraživanjima raspravlja i o terapijskom pristupu usmjerenom na kontrolu ritma. S obzirom na složenost fibrilacije atrija, postoji velika potreba za konsenzusom o klasifikaciji, dijagnozi i liječenju u pasa.

Ključne riječi: *aritmija, fibrilacija atrija, antiaritmici, pas, kontrola frekvencije, kontrola ritma*