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**INTRODUCTION**

Atrial fibrillation (AF) is the most frequent pathological arrhythmia diagnosed in horses and the one that usually causes a decrease in performance or a bad sport performance. This severe arrhythmia is the result of a disorder in the stimulation of the natural electrical conduction of the heart, since there is a disorganized atrial activation that produces a mechanical dysfunction of both atria. Therefore, there is a loss of atrial input to ventricular filling, which may lead to a more or less severe impact depending on the hemodynamic demand of the moment.

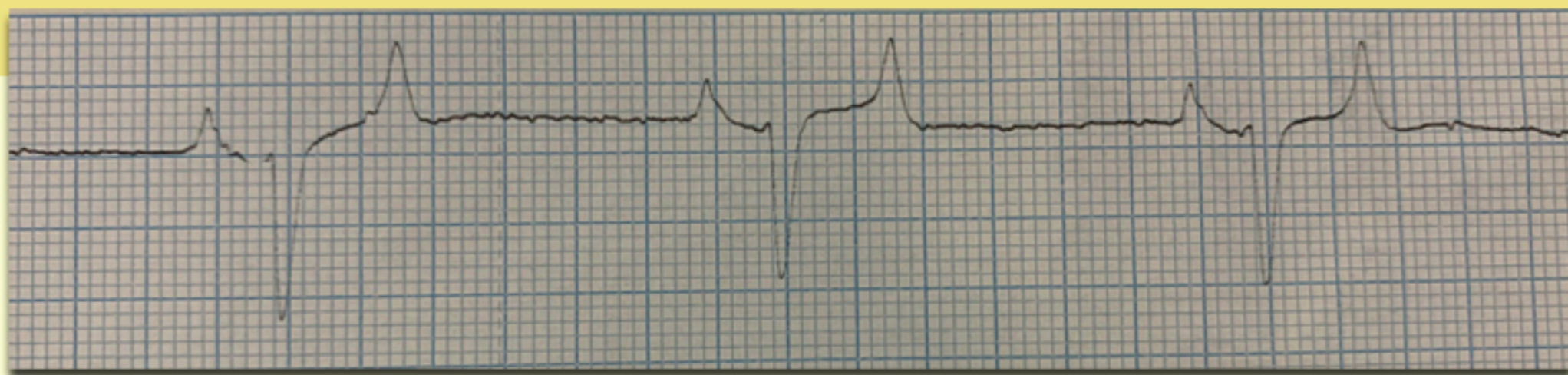


FIGURE 1. ECG register of a horse with FA. Source: Hospital Clínic Veterinari UAB. Unitat Equina

**OBJECTIVES**

- ◇ Knowing how atrial fibrillation works by relating it to cardiac physiology and pathophysiology.
- ◇ Comparing the treatment that utilises quinidine sulfate (main drug used to treat atrial fibrillation) with new treatments that have been proposed due to the need to reduce the side effects of atrial fibrillation.
- ◇ To compare the quinidine sulfate treatment with new treatments that have been proposed due to the need to reduce the side effects of atrial fibrillation.

**PHYSIOPATOLOGY**

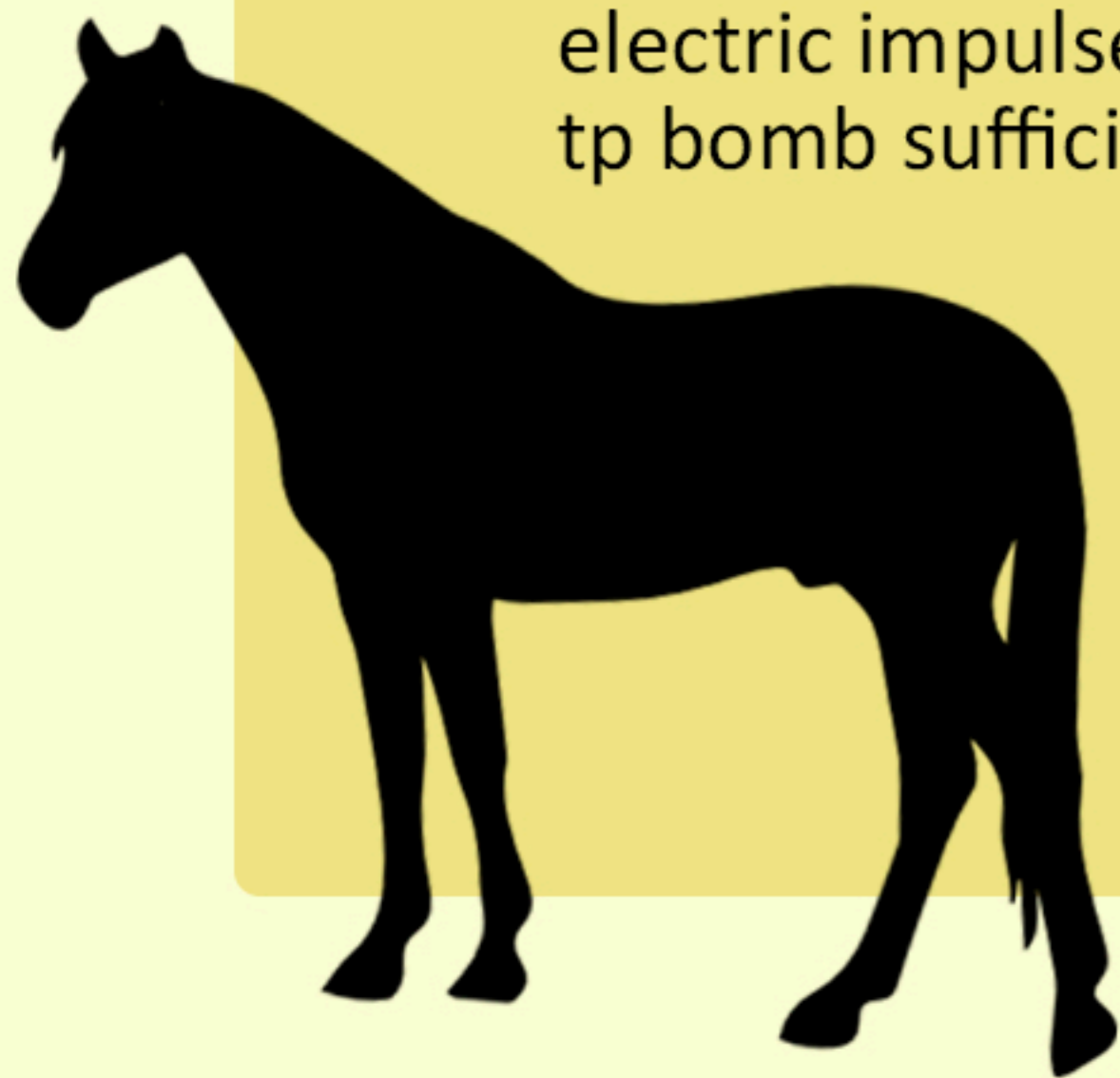
Uncoordinated contraction of the atria caused by electrical disruption

Altered ventricular filling

**A) AT REST**

Increased vagal tone + Ectopic atrial electric impulses blocked by A-V node = Heart able to pump sufficient flow blood

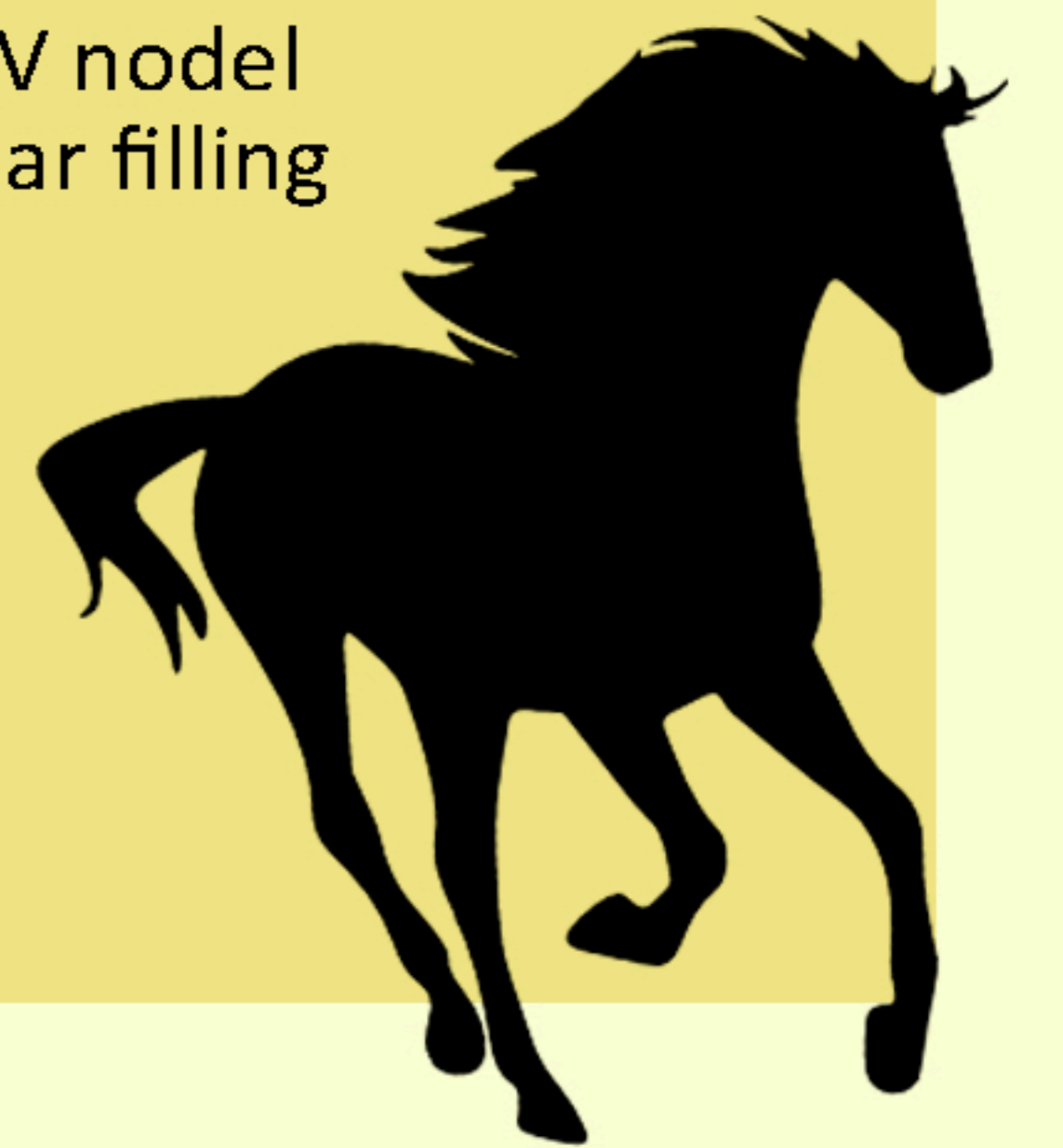
**NO CLINICAL SIGNS**



**B) DURING EXERCISE**

Decreased vagal tone + absence of A-V node block = lack of atrial input to ventricular filling

**CLINICAL SIGNS**



**TREATMENTS**

TABLE 1

Types of antiarrhythmic drugs based on Vaughan-Williams classification			
Class	Drugs	Mechanism of action	Characteristics
IA	Ajmaline Disopyramide Procainamide <b>Quinidine*</b>	Fast sodium channel blockers	Prolong action potential (AP) and effective refractory period (ERP). Prolong PR interval.
IB	Lidocaine Fenitoina		Shorten AP duration. Slow conduction velocity.
IC	<b>Flecainide*</b> Propafenone		No to minimal effect on AP duration. Slow conduction velocity.
II	Atenolol Carvedilol Esmolol Metoprolol Propranolol	Inhibit the sympathetic activation of B-adrenergic receptors	Inhibit b-adrenergic activation of adenylate cyclase and AV node activity. Slow conduction velocity. Prolong AV node repolarization and PR interval.
III	<b>Amiodarone*</b> Sotalol Dronedarone	Potassium channel blockers	Prolong QT interval and AP duration.
IV	Diltiazem Verapamil	Calcium channel blockers	Prolong AV node repolarization and PR interval.
V**	Atropine <b>Digoxin*</b> Ivabradina Ranolazina	Variable mechanisms Digitalics (digoxin): Increase vagal tone	Digitalics: increase conduction velocity.

\*Drugs used in horses for FA treatment \*\*Antiarrhythmic drugs not included in Vaughan-Williams classification.

**Side effects**

Nasal edema, hypotension, decreased heart contractility, congestive heart failure, laminitis, sudden death (Torsade de pointes)

**Side effects**

Tachycardia and severe diarrhea

**Side effects**

Neurological and hepatic signs

**CONCLUSIONS**

- ◇ Knowledge of cardiac anatomy and physiology enables us to understand the cardiac dysfunction in horses caused by atrial fibrillation.
- ◇ The treatment of this pathology is based on the use of different anti-arrhythmic drugs, since their mechanisms of action help to restore the correct cardiac functioning.
- ◇ Amiodarone has proven to be a good new candidate for the treatment due to its moderate success. Moreover, the undesirable effects that it can cause disappear within a few hours of completing the treatment.
- ◇ Whenever resources permit it, transvenous electrical cardioversion should be considered, since it has a good efficacy and the undesirable effects are non-existing in some cases.
- ◇ It is necessary that each patient suffering from atrial fibrillation undergoes an exhaustive study so that the treatment can be adapted as best as possible.