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Atrial fibrillation (AF) is the most common pathological dysrhythmia in horses and pharmacological conversion to normal sinus rhythm is most frequently employed such that horses can return to their previous level of performance. With careful case selection, a successful outcome can be achieved in approximately 85% of cases of uncomplicated AF. Duration of AF and the presence of underlying pathology are the most important predictors of successful conversion. Pharmacological treatment of horses with AF of long duration or with significant cardiomegaly is unlikely to be successful. Because of the potentially life threatening complications of quinidine therapy, treatment of horses with a poor prognosis for conversion is rarely attempted. Horses with AF of less than 3 months’ duration have a good success rate for conversion to normal sinus rhythm, with a low rate of recurrence of AF (15%).

TREATMENT OF UNCOMPLICATED ATRIAL FIBRILLATION
Horses with chronic uncomplicated AF are routinely treated by administration of quinidine sulphate via nasogastric tube. The pharmacokinetics of quinidine are highly variable and therefore therapeutic drug monitoring is advisable; however, this is rarely available; thus recommendations are based upon average pharmacokinetic data in the horse. Electrocardiographic monitoring by telemetric ECG is recommended throughout the period of treatment since drug-induced cardiac dysrhythmias can occur, which if left untreated may be fatal. Additionally, a paper trace, base-apex ECG should be obtained prior to each monitoring by telemetric ECG is recommended throughout the period of treatment since drug-induced cardiac dysrhythmias can occur, which if left untreated may be fatal. Additionally, a paper trace, base-apex ECG should be obtained prior to each

The following protocol is widely employed.

- Quinidine is administered every 2 h at a dose of 22 mg/kg bwt until either: 1) normal sinus rhythm is achieved; 2) a total of 5 doses have been given; 3) there is significant QRS prolongation to greater than 25% of the pretreatment duration; or 4) other life threatening complications occur. In the event of significant QRS prolongation, the dosing interval is extended, so that quinidine is re-administered only after the QRS duration has returned to acceptable limits.
- If conversion has not occurred after the fifth dose, the dosing interval is adjusted based upon serum concentrations of quinidine sulphate (2-5 µg/ml) or to a 6 hourly interval.
- If successful conversion has not been achieved by the second day of treatment, digoxin at a dose of 11 g/kg bwt per os is combined with the 6 h quinidine doses. Diltiazam has recently been evaluated in as an adjunct to quinidine in a similar manner to digoxin and is also effective.

COMPLICATIONS OF THE USE OF QUINIDINE SULPHATE IN THE HORSE
Quinidine sulphate can induce both cardiovascular and noncardiovascular side effects. The most common complications are noncardiac and include depression, parahormis and colitis, and are usually self limiting, stopping after cessation of treatment. The development of colitis is not related to the dose of quinidine administered but, in some cases, the severity of colitis may necessitate discontinuation of therapy. Quinidine-induced colitis can be treated symptomatically with i.v. fluid therapy and gastrointestinal protectants. Nasal mucosal oedema and ataxia can occur at higher doses and are less common than gastrointestinal side effects. Cardiovascular complications can be explained by 3 of the actions of quinidine sulphate; the vagolytic effects can cause supraventricular tachycardia, prolongation of the action potential can cause ventricular tachycardia and alpha-adrenoceptor antagonism can cause peripheral vasodilatation and hypotension. Due to the potentially life threatening dysrhythmias and the fact that these effects are not dose dependent, horses undergoing quinidine therapy should ideally have continuous electrocardiographic monitoring, so that prompt intervention is possible. Horses that develop supraventricular tachycardia with a rate over 100 beats/min should be treated with i.v. sodium bicarbonate, in order to increase plasma protein binding and hence reduce the amount of free quinidine in circulation. Intravenous digoxin will slow conduction through the atrioventricular node and if unsuccessful in controlling heart rate, propanolol can also be administered to further reduce the ventricular response rate. Horses that develop ventricular tachycardia during treatment should be treated with bicarbonate, propanolol and magnesium sulphate, and if the ventricular tachycardia appears unstable, i.v. lidocaine and/or procaainamide can be administered. Hypotension can be managed by administration of i.v. crystalloids and, if refractory, i.v. phenylephrine can be administered to improve pressures.
OTHER TREATMENT REGIMES FOR ATRIAL FIBRILLATION

Amiodarone and flecainide have both been reported to be successful in small numbers of case reports and case series, although their use is beyond the scope of this presentation. Their potential benefits are in reducing the potentially life threatening complications of quinidine. Both have complications and until they can be proven to be superior in terms of clinical effectiveness they cannot be recommended over quinidine.

Other quinidine salts are more readily available in the UK compared to quinidine sulphate, these are designed to have prolonged duration of action due to slower gastrointestinal absorption. Their kinetics have not been evaluated in the horse and due to the potentially life threatening dose dependent side effects cannot be recommended.

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