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THE EFFECT OF ANTI-ARRHYTHMIC DRUGS ON THE VENTRICULAR FIBRILLATION WAVEFORM

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Does pretreatment with an anti-arrhythmic agent have any effect on the frequency of ventricular fibrillation (VF)?

Ventricular fibrillation was electrically induced after the i.v. administration of the test drug in anaesthetised greyhounds; the ECG was recorded on tape (lead 2 and endocardial lead). The dominant frequencies of consecutive periods of fibrillation were measured by Fast Fourier Transform Analysis performed offline (Bruel and Kjaer Spectrum Analyzer).

Five dogs were given no drug (control group); the dominant frequency of lead 2 ventricular fibrillation remained at over 9 Hz for 70 secs and then fell in the next 20 secs to about 5 Hz. The dominant frequency of ventricular fibrillation recorded from the endocardium of the right ventricle did not fall as rapidly as in lead 2, but remained above 8 Hz for several minutes.

Pretreatment with verapamil (1 mg/kg i.v.; $n = 5$) prevented this fall in frequency in the lead 2 ECG, and maintained the dominant frequency above 10 Hz for several minutes ($p < 0.01$).

Pretreatment with lignocaine (10 mg/kg i.v.; mean plasma concentration at time of ventricular fibrillation 5.0 ± 0.5 mcg/ml; $n = 5$) significantly lowered the dominant frequencies of ventricular fibrillation in lead 2 during the initial 80 secs ($p < 0.01$), but had no additional effect thereafter. Verapamil significantly increased the dominant frequency of endocardial ventricular fibrillation ($p < 0.05$), while lignocaine reduced the dominant frequency ($p < 0.05$).

Propranolol (0.4 mg/kg i.v.; plasmaconcentration 118 ± 22 mcg/ml; $n = 5$) had no significant effect on the dominant frequency of ventricular fibrillation recorded from lead 2 or the endocardium.

The fall in the dominant frequency of lead 2 ventricular fibrillation with time may be due to the intracellular accumulation of calcium, as it can be prevented by verapamil. Lignocaine, which blocks fast sodium channels, reduced the frequency of the initial ventricular fibrillation recorded from outside or inside the heart. Blockade of beta adrenergic receptors had little effect on the frequency of ventricular fibrillation. The endocardium appears to be resistant to the metabolic deterioration which occurs in the myocardium during ventricular fibrillation.