

Holter method is negative.

SIGNAL AVERAGING AND

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LOW FREQUENCY SPECTRAL ANALYSIS OF VENTRICULAR FIBRILLATION.

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Is the frequency (f) of ventricular fibrillation (VF) in the patient (pt) the same as in the dog, and does the dominant f of VF depend on its aetiology? In 4 groups of 4 dogs the dominant f of the first 40 secs of VF was measured using a Bruel and Kjaer analyzer (0-100Hz). For spontaneous VF following coronary artery occlusion, the mean dominant f was $12.3 \pm \text{SEM } 0.5 \text{ Hz}$. In reperfusion VF after coronary artery occlusion for 1 hr, the dominant f was $11.9 \pm 0.5 \text{ Hz}$. The f of VF induced electrically after coronary artery occlusion for 1 hr was $11.4 \pm 0.4 \text{ Hz}$. The f of glycoside-induced VF was $6.8 \pm 1.4 \text{ Hz}$ which was significantly lower than that for the first 3 groups ($p < 0.02$). In all 4 groups the f of VF fell significantly over 3 mins ($p < 0.02$). In 8 pts with VF induced by hypothermia during cardiac surgery (mean myocardial temp. 27.4°C), the mean dominant f of the first 40 secs was $4.6 \pm 0.3 \text{ Hz}$. The mean dominant f of the first 8 secs of VF did not differ significantly in 13 pts with inferior infarction ($6.50 \pm 0.2 \text{ Hz}$) from that in 7 pts with anterior infarction ($6.5 \pm 0.3 \text{ Hz}$), nor did it vary significantly with time from the onset of acute ischaemia to VF. In 1 pt, the f of VF fell from 6.0 Hz to 3.8 Hz after 90 secs. In 2 pts where VF followed ventricular tachycardia, low fs of VF were recorded (3.75, 4.25 Hz). The electro-physiological mechanisms of a dominant f in VF are not known. Spectral analysis of VF in the dog does not differentiate myocardial ischaemia from reperfusion or electrically induced VF, but may in cardiac glycoside intoxication. The higher dominant f of VF in the dog than in the pt points to possible limitations of this animal model in the study of VF.