The Effect of Antiarrhythmic Drugs on the Ventricular Fibrillation Waveform

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COMPUTER - ASSISTED ANALYSIS OF SLEW RATE

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For optimal lead localization information regarding lead impedance (1), number of activated myocardial cells (2) and their membrane depolarisation kinetics (3) should be available. Thus usually impedance (1), R-amplitude (2) and slew rate (3) are measured. Supposed these parameters were independent on each other, no correlation should exist between them.

However, analysing a data pool of 200 successive patients with an unipolar lead (4011 Medtronic) in right ventricular position, we found that slew rate (y) was a function of R-amplitude (x) described by the equation: $y = 2.77\,x + 7.31$; $s_x = 0.083$; s_y 0.38 ; T = 10.413 ; R=0.60 ; $p < 0.001$.

In order to find an independent parameter for membrane de-/repolarisation kinetics, the undamped intracardial ECG was recorded and digitized at a converting frequency of 400 Hz. A monoexponential function was fitted to the "intrinsic deflection" and to the time course of repolarisation. The respective time constants neither depended on the R-amplitudes nor on the lead impedances. This argues for an independent parameter. Low values for the time constants indicate rapid voltage changes and vice versa. The time constants (Mean ± SEM, n = 200) were 0.27 + 0.006 ms for intrinsic deflection and 13.50 + 0.27 ms for repolarisation kinetics. As there was a distinct correlation between both time constants similar factors may be involved in the control of de- and repolarisation kinetics.

In summary our analysis shows, that lead impedance, R-amplitude and the time constants of voltage changes are independent and sensitive electrophysiological parameters. In contrast as slew rate depends on R-amplitude only orientating values regarding depolarisation kinetics should be expected from this parameter.

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

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Does pretreatment with an antiarrhythmic agent have any effect on the frequency(f) of ventricular fibrillation(VF)? VF was electrically induced after the IV administration of the test drug in anaesthetised greyhounds. The ECG was recorded on tape (lead 2 and endocardial lead). The dominant f's of consecutive periods of fibrillation were measured by fast fourier transform analysis offline (Bruel and Kjaer Spectrum Analyzer). Five dogs were given no drug (control group); the dominant f of lead 2 VF remained at over 9 Hz for 70 secs and then fell in the next 20 secs to about 5 Hz. The dominant f of VF 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 IV, n=5) prevented this fall in f in the lead 2 ECG, and maintained the dominant f above 10 Hz for several mins ($p<0.01$). Pretreatment with lignocaine (10 mg/kg IV, mean plasma concentration at VF 5.0±0.5 µg/ml, n=5) significantly lowered the dominant f's of VF in lead 2 during the initial 80 secs ($p<0.01$), but had no additional effect thereafter. Verapamil significantly increased the dominant f of endocardial VF ($p<0.05$), while lignocaine reduced the dominant f ($p<0.05$). Propranolol (0.4 mg/kg IV, mean plasma concentration 118±22 µg/ml, n=5), had no significant effect on the dominant f of VF recorded from lead 2 or the endocardium. The fall in the dominant f of lead 2 VF 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 f of the initial VF recorded from outside or inside the heart. Blockade of beta-adrenergic receptors had little effect on the f of VF. The endocardium appears resistant to the metabolic deterioration in VF.