SUMMATION AND INHIBITION IN THE HUMAN HEART ARE DEPENDENT ON POLARITY OF CONDITIONING STIMULI

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Limited degrees of summation and inhibition (by up to 15%) in the human heart have been described previously, using bipolar conditioning stimuli of conventional (2ms) pulse width delivered during the refractory period.

We have used long duration constant current subthreshold conditioning stimuli (Sc) in 32 pts undergoing clinical electrophysiological studies. Continuous atrial or ventricular pacing (S1S1 = 500 or 600ms) was performed throughout the study to stabilise refractory periods. Effective refractory period (ERP) was determined with a 2ms pulse (S2) at an amplitude of twice the late diastolic threshold. A long duration conditioning pulse (duration = S1S2) was then introduced.

Bipolar stimuli were used in 8 pts, and inhibition was produced in all, the effective refractory period (ERP) being lengthened by 97%, from 238±44 ms to 469±103 ms. Sc polarity was reversed in 6 pts; the ERP was shortened in 4 pts (from 219±25 to 183±8 ms), lengthened in one, and unchanged in one.

Unipolar stimuli were used in a further 24 pts. **Cathodal** Sc produced inhibition in all 24 pts, prolonging ERP by 97%, from 241±37 ms to 474±91 ms. **Anodal** Sc shortened the ERP (from 230±37 ms to 165±34 ms) in 12 pts and lengthened the ERP (from 233±33 ms to 385±107 ms) in 11 pts.

Conclusions: Inhibition and summation may be produced in the same patient by reversal of Sc polarity. Cathodal current always produces inhibition; summation, when it occurs, is produced by anodal current.

Thursday, March 7, 1991
Poster Displayed: 9:00AM–12:00NOON
Author Present: 11:00AM–12:00NOON
Hall F, West Concourse
Ventricular Arrhythmias: Late Potentials

INCREASED MORTALITY FROM QUINIDINE THERAPY IN THE TREATMENT OF VENTRICULAR ARRHYTHMIAS

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To evaluate the potential for quinidine (QUIN) therapy to increase mortality and early pro-arrhythmia (PROA) in patients (PTS) with benign or potentially lethal ventricular arrhythmias (VEA), we performed a meta-analysis on 3 double blind randomized placebo controlled parallel trials in 904 PTS in which QUIN was compared to flecainide (FLEC), mexiletine (MEX) and tocainide (TOC). All 3 trials had similar protocols and methodology (e.g. Holter monitoring) but varying lengths of drug exposure. The analysis was performed using the Mantel-Haenszel method on the following data:

	QUIN-MEX		QUIN-FLEC		QUIN-TOC	
# PTS	245	246	139	141	66	67
Deaths	7	2	3	1	1	1
PROA	18	0	2	0	0	0

The combined relative risk of dying on QUIN versus the other 3 drugs was 2.77 (C.I.=0.93, 8.24). There were no deaths on placebo (2 wks exposure for 624 and 1 wk for 280 PTS); on the 4 active drugs there were 7 deaths within 2 wks, 6 on QUIN and 1 on MEX. PROA occurred in 20 PTS on QUIN vs 10 PTS on the 3 other drugs for a combined relative risk of PROA on QUIN of 1.88 (C.I.=1.03, 3.43). These data show that QUIN may have at least as much an effect on PROA and mortality as Class IC (FLEC) and possibly IB (MEX, TOC) drugs in the treatment of VEA.

DETECTION OF PATIENTS WITH BUNDLE BRANCH BLOCK AT RISK FOR NONISCHEMIC VENTRICULAR TACHYCARDIA

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Although it was reported that fast-Fourier transform analysis(FFTA) of signal-averaged ECGs was useful for detection of pts with ventricular tachycardia (VT) despite the presence of bundle branch block (B) in pts with ischemic heart disease, clinical significance of FFTA of signal-averaged ECGs remain unclear in pts with nonischemic heart disease. To determine whether the pts with nonischemic VT who had B could be detected during sinus rhythm, FFTA of signal-averaged ECGs was performed in 3 pts with both B and nonischemic VT and in 7 nonischemic pts with B without VT. FFTA was done on the segment from 40 ms before to 60 ms after the end of QRS complex in signal-averaged Y lead and the power spectrum curve was obtained. Area ratio was calculated by dividing the area under the curve between 20 and 50 Hz by the area under the curve between 0 and 20 Hz. Magnitude ratio were calculated by dividing the magnitude at 30 Hz by the maximum of the entire signal. The results obtained were shown below.

	VT	Control	p value
Area ratio	: 0.535±0.220	0.298±0.010	p<0.05
Magnitude ratio	: 0.383±0.101	0.180±0.083	p<0.05

We concluded that the segment from 40 ms before to 60 ms after the end of QRS complex in signal-averaged Y lead electrogram in pts with both B and VT might contain a greater component of 20 to 50 Hz range than in pts with B without VT. These results suggest that FFTA of signal-averaged ECGs could be useful for detection of pts with nonischemic, undocumented VT despite the presence of B.

HIGH FREQUENCY OF LATE POTENTIALS IN MITRAL VALVE PROLAPSE

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This study included 35 pts (22 females, 13 males, age 55±19 years) with mitral valve prolapse (MVP), absent or mild valve regurgitation, no bundle branch block, normal LV function and wall motion. They all underwent 24-hour ambulatory ECG and signal averaged ECG (SAECG, high-pass filter 25 or 40 Hz). A stress test was performed in 24 cases. In 28 pts with age>30 years, significant coronary disease was ruled out by angiography in 16, by thallium stress scintigraphy in 5, and by both tests in 7. During ambulatory ECG, 13 pts (37%) had frequent ventricular premature complexes (>10/hour), 5 (14%) had non-sustained ventricular tachycardia (VT), 5 (14%) had both. VT was documented during stress test only in 6 pts and was sustained in 2. The SAECG was abnormal in 12 pts (34%)at 25 Hz: 9 had late potentials [LPs, low amplitude signal duration (LAS)>32 ms and RMS voltage of last 40 ms of QRS (RMS40)<25 μv], 3 had both LPs and prolonged QRS duration (>115 ms). At 40 Hz, the SAECG was abnormal in 9 pts (26%): 7 had LPs (LAS>39 ms and RMS40<16 μv), 2 had both LPs and prolonged QRS duration (>111 ms). The frequency of an abnormal SAECG at 25 or 40 Hz was not significantly different in cases with or without spontaneous VT. Programmed electrical stimulation was performed in 9 pts who had syncope or sustained VT: ventricular fibrillation was induced in 2 cases (1 with LPs), no sustained VT/fibrillation in 7 (3 with LPs). Thus, in MVP: 1) spontaneous VT is common, while induced VT is rare; 2) there is no correlation between an abnormal SAECG and spontaneous or induced VT; 3) the high frequency of LPs may represent another feature of the "MVP syndrome"; its prognostic significance is uncertain but probably benign.