~~7-17-98 Lt, MH~~ hc

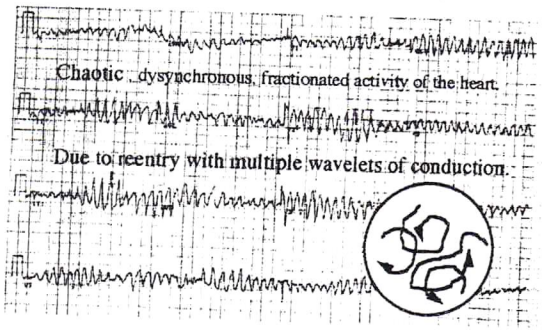
June 1998 Meeting 190

Thomas Svinarich & Mike Piper - Speakers

Ventricular Fibrillation

Mechanisms and relationship to lightning strikes

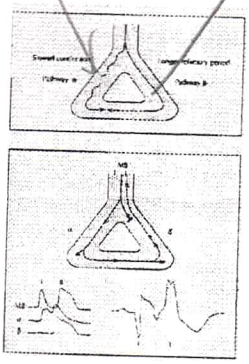
Ventricular Fibrillatioin



Slower conducting pathway

Faster conducting pathway

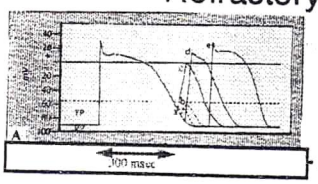
Reentry



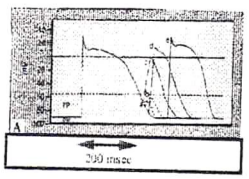
Unidirectional Block occurs when tissues have differences in refractory periods.

Dispersion of refractoriness promotes reentry.

Refractory Period



Action potential duration is the primary determinant of refractory period.



APD altered by:

ischemia

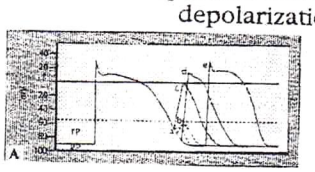
infarction

drugs, electrolytes

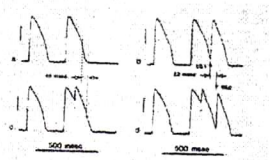
abn of ion channel expression

graded depolarizations – occurs when defibbrilated or struck by ltg

Graded depolarization versus normal depolarization

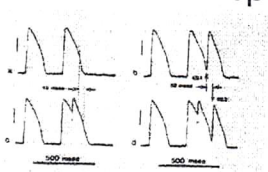


Partially depolarized cells have partially inactivated Na channels and fully depolarized cells have completely inactivated Na channels and are refractory to physiologic stimuli



Strong shock (10-20 V/cm) during absolute refractory period can reult in rapid reactivation of Na current

Graded depolarization



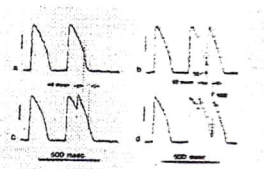
Can initiate arrythmia.

Does not probagate actively but can depolarize tissue at a distance initiating a wavefront

Prolongs refractoriness locally allowing unidirectional block

When dispersion of refractoriness is sufficient multiple wavelets may be created and if reentry is sustained result in VF

Graded Depolarization

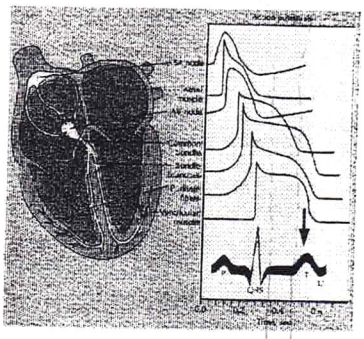


Can terminate VF

Syncronize myocardial activation by depolarizing excitable and refractory tissue

Prevent probagation by homgeneously increasing refractoriness

Vulnerable Period



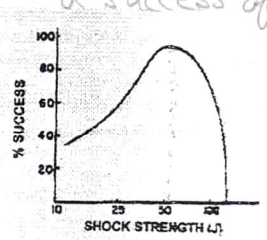
stimulation of heart just before peak of T wave gives highest probability of inducing VF

some myocarial cells are recovering excitability and some are still refractory, maximum dispersion of refractoriness

↳ vulnerable period.

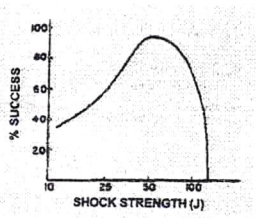
Defibrillatioin threshold is a probability function – likelihood of success of defib.

Pig model



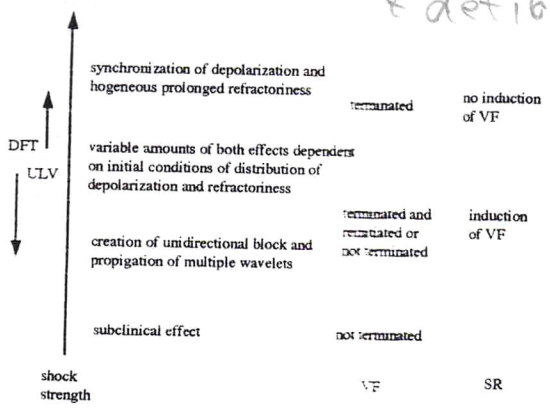
Joules

Fibrillation induction with shock during vunlerable period demonstrates probability function and upper limit of vulnerability ULV



ULV and DFT are related implying physiologic relationship between fibrillation induction and termination

Implies there's a range of shock strengths that will both fibrillate & defib the heart.



Other determinants of DFT/ULV

- electrode positioning
- anatomic variables
- cardiac disease
- drug therapy
- Shock Waveform monophasic/ biphasic

Described Cardiovascular effects of lightning strikes

- myocardial heat injury resulting in ECG changes, arrythmia or congestive heart failure
- adrenal stimulation and catecholamine release resulting in hypertension, tachycardia, arrhythmia in suseptable induviduals

How can VF occur in the absence of direct hit, side splash, or ground strike leading to direct myocardial injury?

Predictions of myocardial voltage gradients induced by internal shocks, external shocks, and AC EM sources have been attempted

Fields of 10-20 V/cm have been modeled with transvenous and transthoracic DC defibrillation in pigs taking into account electrode position, impedance of various tissues, and thaoracic shape and anaomy on CT scan

Fields of 5 V/cm shown to prolong refractoriness of depolarized tissue and may induce VF during vulnerable period?

EM sources of 550-1400 kV/m at 60 Hz suspected to be sufficient to cause VF in a ~~sperical~~ Spherical chest model

Near lightning strike and VF

If a near lightning strike is able to induce transmyocardial voltage gradients in a range that is sufficient to depolarize fully refractory myocardium, but is below the ULV, and occurs during the vulnerable period, VF can be produced.

A near lightning strike that induces voltage gradients that are lower or higher (above ULV), or which does not occur during the vulnerable period will not induce VF.

A model describing the myocardial voltage gradient induced by a near lightening strike should be created as a first test of this hypothesis.