

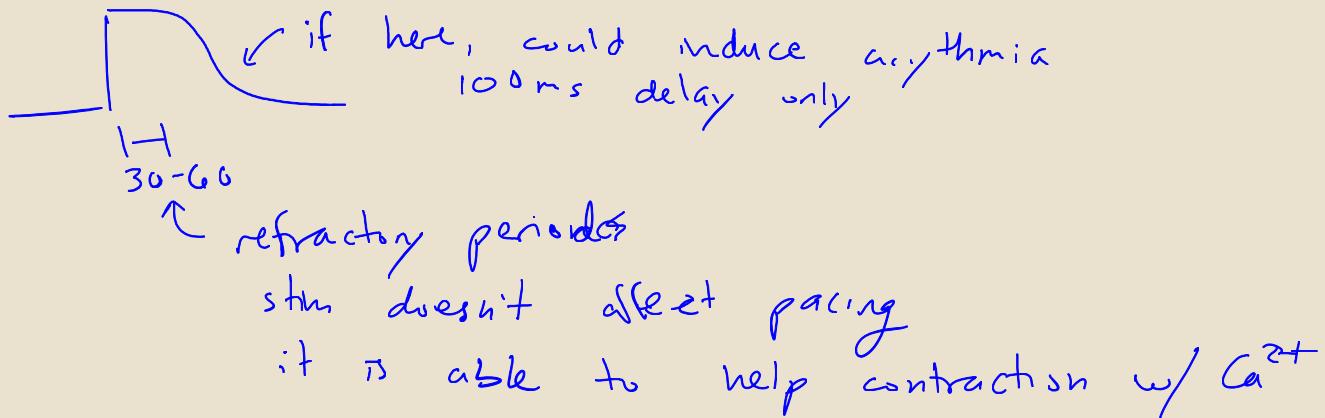
Cardiac Pacing & Defibrillation

CCM - cardiac Contract Modulation

Pacing artifacts are large vertical lines

Amplitude is rather high, just below sensation

Calcium → contractility
excitation-contraction coupling



Chronic

NL → normal hearts
upregulate important proteins

CSQ - calcium sequestering protein

SERCA - pumps Ca^{2+} back in

recovery of contractility machinery

chance of induced arrhythmia

$\uparrow \text{Ca}^{2+}$ into cell

CRT

biventricular leads

both ventricles

Pace at same time

25% - 30% of HF patients

very effective in target population
who will respond based on QRS duration
 $>150\text{ ms}$ → good responders

left ventricular fxn more important

affects a lot of different things

APD prolongation → diastolic heart failure

strains are different throughout the heart but near normal

changes activation pattern → ↑ stress on heart

→ bad for people with QRS $< 120\text{ ms}$

Activation Wavefronts

A → anodal → hyperpolarized center w/ depolarized end

Anisotropy: direction matters for conductivity

* Cathodal Malce & Anodal Malce → know these

Break → fast activation along the fiber when turned OFF

BAS

lead has tip and ring electrodes for source and return
or use block for return

Cathodal stimulation usually

reduce diaphragm stimulation with LV tip to RV ring stimulation

↑ SA of ring → ↓ current density → E field strength

Controls directionality or magnitude?

HIS Bundle Pacing

Natural activation pattern via Purkinje fibers

Hard to locate HIS bundle and place electrodes

Replaces biventricular pacing

↓ QRS complex time

Pacemaker Intervale Transient Asynchrony

Inverse of CRT

Fors population

Induces desynchronization

Stresses heart during sleep to "exercise" it

Leadless Pacemaker

Size limited by batteries and electronics

Requires surgery grab and pull out or place

Needs subcutaneous ICD for defibrillation bc power too low

Fibrillation

Tachycardia

VT → abnormal pathway

Dark region → slowly conducting

Has already recovered / not in refractory period, so a unidirectional block can occur

Premature beat needed to get reverse flow (re-entry)

Heterogeneity → fast vs slow conduction

Wavelength WL < PL Pathlength

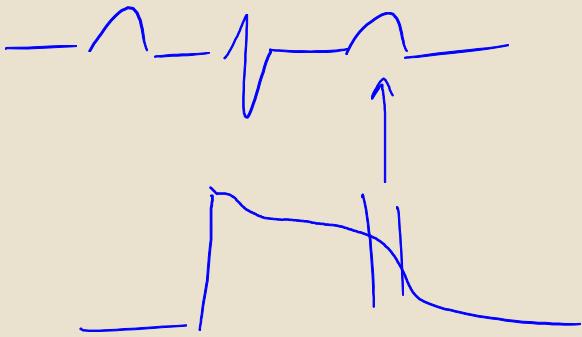
In order to have space to propagate w/o hitting refractory period

Anti-tachycardia

Pace faster than VT rate to extinguish wavefront

Avoid defibrillation pulse

λ : wavelength (m) | RP: refractory period (s) | θ : conduction velocity (m/s)



Lots of wavefronts

Tachycardia \rightarrow one wavefront

Fibrillation \rightarrow many wavefronts

Biphasic shock \ggg Monophasic shock

VEP

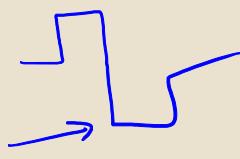
β can induce ~~asystole~~ fibrillation
 extinguish during shock, reintroduced when turned OFF
 strong post-shock gradient

Defibrillation

different effects at different areas
 pair curve with virtual anode or virtual cathode



These thresholds are no longer tested, just use high power
 at maximum possibility of device

Biphasic
balance second part  to try to get
homogeneity after the initial activation

