Basic Science

Important Formulas

1) CO=SV+HR – Cardiac Output
2) (EDV-E SV)/EDV=SV/EDV=EF% - Ejection Fraction
3) Ohm’s law – V=IR (in Volts - V)
4) I=V/R (in Ampere - A)
5) R=V/I (in Ohms - Ω)
6) C=Q/V – Capacitance (Q – charge)
7) T=(Q/I)*114 – Battery longevity
   Example: V=5V, R=500Ω, Q=1Ah, T=?
   Conversions: 1Ah=1,000,000µA 5V=5,000mV
   I=V/R=5,000mV/500Ω=10µA
   T=(1Ah/10µA)*114=11.4 years
   Units for calculation: V in mV, I in µA, R in Ω, C in µF or Ah, Q in J, T in Year
8) E=V*I*t if I=V/R then
9) E=V*(V/R)*t then E=(V²/R)t
10) LR = AVD + VA
11) AVD+PVARP=TARP
12) WI=UTR(MTR)-TARP – Wenckebach Interval
13) TARP=2:1 Block
14) If UTR>TARP then WI – Upper Rate Behavior
15) If TARP>UTR then 2:1 Block – Upper Rate Behavior
(R) Resistance (impedance) – Ohms (Ω)
1KΩ=1,000Ω
(V) Pressure electrical – Volts (V)
1mV=1/1,000V 1V=1,000mV
(I) Current = Ampere (A)
1A=1,000mA 1mA=1,000µµA 1A=1,000,000µA
(E) Energy – Joules (J)
Charge – Coulomb (C) – stored by a capacitor
Frequency – Hertz (Hz)
(t) Time – Pulse Width (ms)
Conversions:
   Unit given   Convert to before calculations
   mA       A       to get V
   V       mV       to get mA

Closed circuit
Electrons – flow from “negative” to “positive”
Currents – flow from “positive” to “negative”

Battery – Li+I–
(-) Anode – produces Li+
(+) Cathode – produces I–
at BOL – low (normal) impedance
at EOL – very high impedance
Expected Battery Life Time=Capacity(Ah)/Drain(µA)

Lead
Polarization – electrode/tissue interface
Initial polarity:
• Lead tip – anode
• Lead ring – cathode

Leading edge – start of pacing pulse
Trailing edge – end of pacing pulse

Strength Duration Curve
- Rheobase – the voltage level at which a further increase in pulse duration does not result in a continued fall in pulse amplitude
- Chronaxie point – the pulse duration threshold at twice the Rheobase. Chronaxie point in most pacing systems is 0.4ms to 0.6ms
- Pacemaker pulse must fall to the right of the curve in order to be effective
- Doubling the pulse amplitude will quadruple the delivered energy
- Doubling the pulse duration will only double the delivered energy
- Halving the pulse amplitude reduces the delivered energy by a factor of four
- Halving the pulse duration reduces the delivered energy by a factor of two

Cardiac Cell Action Potential

Shape of the Action Potential determines conduction velocity, refractory period, and automaticity of cardiac tissue.
Phases 1,2,3 – Repolarization

**Phase 2** – Plateau – mediated by slow Ca\(^{+}\) channels: positively charged calcium ions slowly enter the cell, thus interrupting repolarization and prolonging the refractory period

**Phase 4** – Resting - no net movement of ions across the cell membrane

Automaticity – in some cardiac cells there’s a leakage of ions across the cell membrane during Phase 4 in such was as to cause a gradual positively directed increase in transmembrane potential. When it reaches threshold voltage, the appropriate channels are activated to automatically generate another action potential.

**Action Potential of SA and AV Nodes** – dependent entirely on slow Ca\(^{+}\) channel for depolarization; they lack the rapid Na\(^{+}\) channel (responsible for rapid depolarization).

**Autonomic innervation**

**Increased sympathetic tone** – enhanced automaticity, increased conduction velocity, decreased AP duration and refractory periods

**Increased parasympathetic tone** – depressed automaticity, decreased conduction velocity, increased refractory periods

SA and AV nodes – richly innervated → changes in parasympathetic tone have greater effect

### Device Troubleshooting

**Brady**

1) **Evaluation of Pacing Mode – Device Behavior**

<table>
<thead>
<tr>
<th>DDD</th>
<th>DDI when ApVs</th>
</tr>
</thead>
<tbody>
<tr>
<td>DDI</td>
<td>DVI when LOAS – more pacing</td>
</tr>
<tr>
<td>DVI</td>
<td>DVI when LOAS – possible Atrial competition</td>
</tr>
<tr>
<td>VDD</td>
<td>VVI when AV synchrony is lost</td>
</tr>
<tr>
<td>VDD</td>
<td>DDD when AsVp</td>
</tr>
<tr>
<td>DVI</td>
<td>DDI = DDD when ApVp</td>
</tr>
</tbody>
</table>

2) **Evaluation of sensing and pacing**

Whatever is FASTER is in control (intrinsic or paced):

- LR faster than SN = A pacing (90-100bpm LR)
- SN faster than LR = P waves (40bpm LR)
- AVD shorter than PRI = V pacing (90-100ms AVD)
- PRI shorter than AVD = R waves (250-350ms AVD)

AS to AP – increase rate
AP to AS – decrease rate
VS to VP – decrease ADV
VP to VS – increase ADV

ApVs + AsVp confirms both A&V sensing&capture

To confirm A capture – increase AVD
To confirm A&V sensing – decrease rate ad increase AVD

ApVp look the same for both A and V based timing

- **Factors that lower pacing thresholds** – corticosteroids (Dexamethasone), exercise, cathecolamines, hypocapnia, symptomatic agents (Isopro, Epinephrine, Ephedrine, Atropine), hyperoxia

- **Factors that increase pacing thresholds**

- **Ventricular safety pace** – triggered when the ventricular channel senses right after coming out of blanking

- **Pseudo-Wenckebach** (Upper Rate Behavior) – when spontaneous atrial rate exceeds the programmed URL

- **Crosstalk vs. “safety pacing”** – look between the two spikes: if there’s R-wave – that’s what triggered (with frequent PVCs or LOAS) – safety pacing; if there’s no R-wave – crosstalk likely

- **Crosstalk** – with unipolar configuration, Ap event sensed on V channel, no R between spikes; 1) Ap/inhibition, 2) persistent safety pacing, 3) Ap/Vs at >AV → no safety pacing or Vp at end of AV clock and A-A interval maintained at LRL → extend Blanking period and/or reduce Atrial output

- **PVC – retrograde algorithms** – PVARP extension after the first PVC; Tracking preference – shortens PVARP after two consecutive ventricular events with atrial events in PVARP

- **Tissue vs. device refractory periods**

ERP(ARP) = PVC – in ERP extrasystole will fail to propagate

RRP = noise sampling – in RRP extrasystole results in a slowed conduction (last part of Phase 3)

ERP & RRP – longest coupling interval

FRP – smallest possible interval between two impulses that are conducted
3) **Evaluation of Lead Integrity**
- High impedance – loose set screw if a new implant, high-impedance lead (passive fixation), lead fracture, terminal lead pin not fully inserted in header
- Low impedance - lead insulation break, current leakage - header
4) **Evaluation of Device Statistics**
5) **PMT/ELT vs. the unknown**
6) **Failure to ModeSwitch – causes**
- Atrial undersensing
- P-waves in PVAB
- Too high MS rate
7) **Repetitive Non-Reentrant VA Synchronous Rhythm (AV Desynchronization Arrhythmia)** – can be initiated only in modes DDDR or DDIR; initiates by retrograde P-wave not sensed (falls in the PVARP) leading to functional Atrial noncapture and/or pacemaker syndrome; can be triggered by PAC or PVC; the following conditions are needed in order to be initiated:
- Intact retrograde conduction
- Long PVARP (retrograde conduction does not induce ELT)
- High Base Rate (short Atrial escape interval) Management
- Short Av delay at higher rates or
- Lower higher rates or
- Extend PVARP or
- If persistent at higher rates (above 90pm) program to DDD and further investigate
8) **Loss of CRT at UTR** – tracking of a fast atrial rate causes loss of CRT @ 120bpm → program high UTR (140bpm) and AVD extension to avoid ventricular pacing above UTR
9) **Bi-V capture to...** - look at QRS complex
- LV capture – lead /(-) & lead III(+)
- RV capture – lead I(+)& lead III(-)
10) **Loss of Bi-V capture to...** - look for increase in positivity of the QRS complex: a) positive to more positive; b) negative to positive; c) negative to less negative; d) negative to isoelectric; e) isoelectric to positive
- RV-only capture – the QRS morphology in Lead I becomes more positive
- LV-only capture – the QRS morphology in Lead III becomes more positive
11) **RV Anodal stimulation** – more common with dedicated bipolar leads and the inclusion of the RV-ring in the pacing configuration of the LV lead (such as LV-tip – RV-ring) is not necessary for it to occur. It has to do with the small surface area of the anode. A small study in Europe has shown a benefit of RV anodal capture. It is unclear whether RV anodal capture may cause some patients to not respond to CRT therapy. The European study showed that integrated bipolar leads are less susceptible to RV anodal capture since the distal coil acts as the anode, therefore large surface area.
12) **V-V timing optimization** – it is recommended for non-responders to CRT (about 30% of CRT implants).
- Identify intrinsic RVs-LVs interval by obtaining an EGM with approximately 10 intervals; identify a representative averaged RVs-LVs interval
- Using the averaged RVs-LVs interval, select the recommended LV offset from the table below (if the RVs precedes the RVs on the EGM, program the LV offset to Zero).
- Select the recommended offset from the choices provided. Lengthen the AV delay by the absolute value of the suggested LV offset (Example: AVD=80+|-40|=80+40=120ms)

<table>
<thead>
<tr>
<th>RVs-LVs</th>
<th>LV Offset</th>
<th>RVs-LVs</th>
<th>LV Offset</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-15</td>
<td>-20</td>
<td>106-135</td>
<td>-60</td>
</tr>
<tr>
<td>16-45</td>
<td>-30</td>
<td>136-165</td>
<td>-70</td>
</tr>
<tr>
<td>46-75</td>
<td>-40</td>
<td>&gt;166</td>
<td>-80</td>
</tr>
<tr>
<td>76-105</td>
<td>-50</td>
<td>LVs first</td>
<td>0</td>
</tr>
</tbody>
</table>

13) **False positive IEGM storage of Atrial arrhythmias – causes**
- ST above the detection rate
- Far-field R-wave sensing
- Premature Atrial events
- Myocardial oversensing
- External interference

**Tachy**
1) **ATP schemes**
- Scan – increment/decrement from burst to burst
- Ramp – increment/decrement from pulse to pulse within a burst
- Ramp/Scan – BCL increment/decrement both within bursts and between bursts – very aggressive therapy
2) **False positive IEGM storage for VT – causes**
• Sinus or Atrial tachycardia with or without Atrial undersensing
• P-waves in refractory
• Double counting QRS (oversensing on the Ventricular channel such as T-wave oversensing)
• Myopotential oversensing
• External interference

Device System Troubleshooting
Lead Perforation
• TEE – evaluate for epicardial effusion
• Pericardial friction rub
• Chest pain
• RBBB on ECG
• Dyspnea
• Hypotension
• Predictors of lead perforations – concomitant temporary pacemaker or steroid therapy within the last seven days

Infections
• Acute – Staphylococcus Aureus – may cause endocarditis – system extraction
• Chronic – Staphylococcus Epidermidis
Subclavian Puncture – watch for subcutaneous emphysema and pneumothorax

Management of high DFT
• More distal RV position
• Change polarity
• Remove SVC coil
• Sub-Q array
• High energy device
• Azygos vein single coil DF lead

Less Known Indications
Pacemaker
1) Linègre or Lev’s disease – idiopathic, age-related, progressive, leading to AV block

ICD
1) Graves Disease – Hypokalemia & Hypomagnezia – thyroid disorder leading to LQT & Torsades-De-Pointes
2) Timothy Syndrome – LQT8 – gene mutation
3) Brugada Syndrome – mutation of SCN5A gene; leading to SCD in young Asian men, night screams; ECG characteristics – ST elevation in V1 to V3, RBBB

4) Naxos Disease – Arrhythmogenic RV Cardiomyopathy, mutation in plakoglobin, at risk for SCD
5) Arrhythmogenic RV Dysplasia (ARVD) – pathophysiologic process in RV where myocardial cells are replaced by fibrotic tissue, leading to VTs
6) Inherited LQT Syndromes – gene mutations
   • Romano-Ward Syndrome – mutation of SCN5A gene
   • Jervlle-Lange-Nielson Syndrome – mutation of SCN5A gene
   • Anderson Syndrome – QT>440ms for males and QT>450ms for females

7) Chaga’s Disease – the “kissing bug” Trypanosoma Cruzi insect – infections by bites and blood transmissions in Latin America. ECG characteristics – RBB, SN Dysfunction, Ventricular Arrhythmias, AV Block, abnormal Q-waves, S-segment and T-wave abnormalities, autonomic dysfunction; leading to SCD from VTs, deterioration in ANS and LV function, swelling of one eye, enlarged Lymph Nodes.

« Bear Dropping » Theory
CAST Trial – Antiarrhythmogenic suppression of frequent PVCs (>10/min) – increased mortality due to proarrhythmic effect

Commotio Cordis
Mechanical stimulation of the heart – rhythm disturbances, 15% survival only, R-on-T phenomena

Bezold-Jarisch Reflex
A normal reflex caused by activation of myocardial mechanoreceptors that transmit vagal afferent impulses via the C-fibers. This causes withdrawal of efferent sympathetic activity to peripheral blood vessels, which leads to hypotension, and an increase in efferent vagal output, which leads to bradycardia. It’s thought to be a major cause of Vasodepressor Syncope (VVS).
   • Malignant Vasovagal Syncope – β-blocker to stop the stimulation of C-fibers

 Syndromes that can cause AV Block
• Myotomic Muscular Dystrophy
• Kearns-Sayre Syndrome – genetic, young <20y.o., external opthalmoplegia & retin degeneration
• Limp-Girdle Dystrophy of Erb
• Peroneal muscular atrophy

Sites of AV Block
• In the AV node - 1° and 2° - benign and non progressive
• Distal to the AV node - 1° and especially 2° - tends to progress to a higher degree of block; prophylactically pacing often indicated

SCD in the young – causes
• HCM – most common
• ARVD
• Coronary Artery abnormalities
• Myocarditis
• Unexplained

SCD – Sudden Cardiac Death
450,000 cases a year due to VT/VF
80% SCD due to CAD
25% SCD due to structural heart disease within 1hr of onset of symptoms

HCM – Risk factors
• Prior Cardiac Arrest
• Family history of SCD
• Ventricular Arrhythmias
• LV wall thickness >3mm
• Hypotension or blunt BP with exercise
• Unexplained Syncope
• Genetic

Pulseless VT/VF
CPR and defibrillation 120-200J biphasic, 360J monophasic
Epinephrine 1mg IV/IO, repeat every 3-5min or
Vasopressin 40U IV/IO instead of first and second doses of Epi

Heart Failure
Progressive and complex mechanisms on cellular, metabolic, and neurohormonal level
Affects 5 million Americans
450,000 new cases a year
1 year mortality is 45%

<table>
<thead>
<tr>
<th>Ischemic</th>
<th>Non-Ischemic</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/3 of the patients</td>
<td>1/3 of the patients</td>
</tr>
<tr>
<td>Underlying CAD</td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Valvular</td>
</tr>
<tr>
<td></td>
<td>Viral</td>
</tr>
<tr>
<td></td>
<td>Alcohol/Drug induced</td>
</tr>
<tr>
<td></td>
<td>Familial</td>
</tr>
<tr>
<td></td>
<td>Idiopathic</td>
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<table>
<thead>
<tr>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/3 of patients</td>
<td>1/3 of patients</td>
</tr>
<tr>
<td>Decreased SV</td>
<td>Impaired early diastolic relaxation due to ischemia</td>
</tr>
<tr>
<td>Impaired inotropic state</td>
<td>Increased stiffness of the ventricular wall – LV</td>
</tr>
<tr>
<td>due to impaired contractility or hypertension</td>
<td>hypertrophy</td>
</tr>
<tr>
<td><strong>Contractility/Ejection</strong></td>
<td><strong>Filling/Relaxation</strong></td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Left-sided</th>
<th>Right-sided</th>
</tr>
</thead>
<tbody>
<tr>
<td>Damaged pump fails to discharge load</td>
<td>Difficult time with increased afterload from the lungs</td>
</tr>
<tr>
<td>Pressure rises in atrium and “backwards” to lungs</td>
<td>Caused by left-sided HF</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Left-sided - Symptoms</th>
<th>Right-sided - Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea on exertion</td>
<td>Abdominal pain</td>
</tr>
<tr>
<td>Paroxysmal nocturnal dyspnea</td>
<td>Anorexia</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Nausea</td>
</tr>
<tr>
<td>Cough</td>
<td>Bloating</td>
</tr>
<tr>
<td>Hemoptysis – blood in spit</td>
<td>Swelling</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Left-sided – Signs</th>
<th>Right-sided – Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basilar rales</td>
<td>Peripheral edema</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>Jugular venous distension</td>
</tr>
<tr>
<td>Ss Gallop</td>
<td>Abdominal-jugular reflux</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>Hepatomegaly</td>
</tr>
<tr>
<td>Cheyenne-Stokes respiration</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>ACC/AHA Stages</th>
<th>NYHA Class</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage A</strong></td>
<td>None</td>
</tr>
<tr>
<td>High risk for HF</td>
<td></td>
</tr>
<tr>
<td>No structural disease or symptoms (CAD, Hypertension)</td>
<td></td>
</tr>
<tr>
<td><strong>Stage B</strong></td>
<td>Class I – 35% of patients</td>
</tr>
<tr>
<td>Structural heart disease without symptoms</td>
<td>Asymptomatic with exertion</td>
</tr>
<tr>
<td><strong>Stage C</strong></td>
<td>Class II – 35% of patients</td>
</tr>
<tr>
<td>Structural heart disease with prior or current symptoms</td>
<td>Symptomatic with moderate exertion</td>
</tr>
<tr>
<td><strong>Stage D</strong></td>
<td>Class III – 25% of patients</td>
</tr>
<tr>
<td>Refractory HF requiring specialized interventions (such as heart transplant)</td>
<td>Symptomatic with minimal exertion</td>
</tr>
<tr>
<td></td>
<td>Class IV – 5% of patients</td>
</tr>
<tr>
<td></td>
<td>Symptomatic at rest</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>CRT-P Indications</th>
<th>CRT-D Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>EFS≥35%</td>
<td>Standard ICD Indication</td>
</tr>
<tr>
<td>QRS≥120ms</td>
<td>COMPANION NYHA Class IV population</td>
</tr>
<tr>
<td>NYHA Class III</td>
<td></td>
</tr>
<tr>
<td>Ambulatory Class IV HF</td>
<td></td>
</tr>
<tr>
<td>Symptomatic despite OPT</td>
<td></td>
</tr>
</tbody>
</table>
Causes of HF
- Ischemic heart disease (CAD, MI)
- Hypertension
- Idiopathic Cardiomyopathy
- Infections (Viral Myocarditis, Chaga’s Disease)
- Toxins (alcohol or cytotoxic drugs)
- Valvular Disease
- Prolonged Arrhythmia

Deaths related to HF
NYHA Class II & III – SCD
NYHA Class IV – pump failure

Notes on HF
- **Autonomic Nervous System (ANS)**
  - Sympathetic – increase HR & contractility
  - Parasympathetic – decrease HR
- **Preload** – the degree of stretch in the heart before it contracts
- **Contractility** – the strength of contraction at any given preload
- **Afterload** – the pressure that must be overcome before the semilunar valves can open
- **Preload, Contractility, and Afterload are all a function of the Stroke Volume (SV)**
- A decreased CO results in compensatory mechanisms produced by the nervous and humoral systems
- The purpose of the compensatory mechanisms is to restore CO to meet the need of vital organs
- The compensatory mechanisms increase SV by impacting Preload, Contractility, and Afterload
- The ANS and Renal Systems control the release of the compensatory mechanisms
- ANP – released in the atrium
- BNP – released by the ventricular myocardium
- CNP (C-natriuretic peptide) – released by the endothelium
- TNF – increases during HF and stimulates ventricular remodeling
- Systolic Dysfunction – pressure overload, impaired contractility
- Diastolic Dysfunction – decreased ventricular compliance

β-Receptors
- Stimulation increases HR, force of contraction, CO
- May lead to ischemia and arrhythmias, decreased contractility
- As HF progresses, downregulation of β-receptors take place which is a protective response
- Reduced HR variability is linked to increased mortality in HF

Factors that WORSEN the prognosis of HF
- CAD, advanced NYHA Class, decreased functional capacity, S3 Sound, Cardiomegaly
- Increased norepinephrine, rennin, ANP, BNP, vasopressin (ADH), LVEDD pressure, systemic vascular resistance
- Decreased (low) serum sodium, potassium, magnesium, VO2max, 6-min walk, cardiac index

Ventricular remodeling
Components of remodeling
- Ventricular dilatation
- Myocyte hypertrophy
- Interstitial fibrosis
- Apoptosis – programmed (premature) cell death

Triggers of remodeling
- Increased stretch on myocytes (Frank-Starling law)
- Neurohormones – norepinephrine, angiotensin II, aldosterone
- Cytokines – TNF (tumor necrosis factor) – leads to muscle wasting and cardiac cahexia

HF Management Goals & Treatment
Stage A
- Control risk factors
- Ace-inhibitors (for asymptomatic patients)
- Effective treatment of Hypertension
- Prevent ventricular remodeling

Stages B,C, and D with or without symptoms
- Improve survival
- Slow progression of HF
- Alleviate symptoms
- Minimize risk factors

Resynchronization Therapy - pacing for HF
- Stable & optimal medical regimen
- NYHA Class III or IV
- QRS > 120 to 130ms, LBBB
- LVEF ≤ 35%
- *Meluzin method for AV optimization* – lengthen AVD until first signs of fusion, then
subtraction 25-30ms (negative AV Hysteresis - 30ms)

Revascularization - CABG

Drugs for HF

Class III AADs - only Dofetilide and Amiodarone are proven safe with HF

Class II AADs (β-blockers) – Atenolol, Carvedilol, Metoprolol
  - Counteract sympathetic nervous system
  - Help reverse ventricular remodeling
  - Help improve EF
  - Hypertension, angina, arrhythmias, prophylaxis of MI

Vasodilators - Ace Inhibitors

- Reduce conversion of angiotensin to angiotensin II
- Reduce degradation of bradykinin – promotes vasodilatation and causes natriuresis in the kidneys
- Reverse ventricular remodeling
- First-line chronic therapy for LV systolic dysfunction

Inotropics – Digoxin

Diuretics

Aldosterone Antagonists – only for stages C or D - Spironolactone

Antiarrhythmic Drugs (AADs)

Class I (Na⁺/fast channel blockers) – bind to Na⁺ channel, decrease speed of depolarization
  - IA – slow conduction velocity and lengthen refractory periods (Quinidine, Procainamide, Disopyramide) – convert arrhythmia and maintain NSR
  - IB – decrease AP duration and refractory periods (Lidocaine, Phenytoin, Tocainide, Mexiletine)
  - IC – pronounced depressant effect on conduction velocity, little effect on refractory periods (Flecainide, Encainide, Propafenone, Moricizine*) – potentially convert arrhythmia and maintain NSR, watch for decreased LV function

Class II (β-blockers) – decrease sympathetic tone, little effect on AP, affect mainly SA and AV nodes indirectly by blocking β-receptors (Atenolol, Labetolol, Metoprolol, Nadolol, Propranolol, Timolol, Esmolol)

Class III (K⁺ channel blockers) – increase AP duration and refractory periods, little effect on conduction velocity (Amiodarone, Bretylium, N-acetylprocainamide, Ibutilide, Dofetilide, Sotalol) - convert arrhythmia and maintain NSR, control HR

Class IV (Ca⁺/slow channel blockers) – effect mainly on SA and AV nodes – direct membrane effect (Diltiazem, Verapamil) – decrease sinus activity and AV conduction, lengthens ERP, control HR

Class V (Digitalis agents) – affect mainly SA and AV nodes indirectly by increasing vagal tone (Digitoxin, Dogoxin) – Na⁺ and K⁺ balance, control HR

Amiodarone

- Properties - Class I, II, III, IV – anti-ischemic, not proarrhythmic, reduces Digoxin clearance
- Interactions – Increases potentiation of Coumadin and Digoxin; increases levels of Quinidine, Procainamide, Phenytoin, Flecainide; if combined with Class I AADs → decrease dose of Class I AADs; may lead to bradycardia – negative inotropic effect of Class II and IV AADs
- Side effects – photosensitivity, pulmonary toxicity, polyneuropathy, GI upset, bradycardia, hepatic toxicity, thyroid toxicity, rarely Torsades-De-Pointes

Drugs for sedation during device implants

Pre-Op sedation

- 5-10mg Diazepam (Valium)
- 20-50mg Benadryl (Diphenhydramine)

IV sedatives/analgesics – during the procedure

- 0.5-1mg Midazolam (PRN)
- 25-50ug Fentanyl (PRN)

Reverse sedation (IV)

- Flumazenil – 0.2mg increments to reverse Midazolam
- Naloxane – 0.2mg increments to reverse Fentanyl
ECG – Axis Deviation

QRS complex orientation – positive (+) or negative (-) – look at leads I & aVF

- Normal axis – I(+) & aVF(+)
- LAD (left axis deviation) – I(+) & aVF(-) but if lead II(+) – then normal axis
- if lead III(-) – then LAD
- RAD (right axis deviation) – I(-) & aVF(+)
- Extreme RAD/NW (no man’s land) – I(-) & aVF(-)

Causes of LAD – left anterior hemiblock, Q-waves of inferior MI, artificial cardiac pacing, emphysema, hyperkalaemia, WPW syndrome – right sided accessory pathway, tricuspid atresia, ostium primum ASD, injection of contrast into left coronary artery

Causes of RAD – normal finding in children and tall thin adults, RV hypertrophy, chronic lung disease even without pulmonary hypertension, anterolateral MI, left posterior hemiblock, pulmonary embolus, WPW syndrome – left sided accessory pathway, atrial septal defect, ventricular septal defect

Causes of Extreme RAD - emphysema, hyperkalaemia, lead transposition, artificial cardiac pacing, ventricular tachycardia

Notes on Clinical Trials

AFFIRM – chronic anticoagulation recommended with an INR>2 even when the patient is in Sinus Rhythm – rate control vs. rhythm control

CARE-HF – CRT improves symptom and QoL and reduces complications and risk of death

COMPANION – (in HF) CRT+ICD+OPT – 36% reduction in mortality vs. OPT alone (OPT – optimal pharmaceutical therapy)

CONTAC CD – CRT(D) vs. no therapy - reduced morbidity and mortality, QoL and NYHA Class

DEFINITE – defibrillation in non-ischemic dilated cardiomyopathy patients with good HR variability – treatment evaluation – excellent prognosis, may not benefit from ICD; 62% absolute and 35% relative mortality reduction, arrhythmia mortality significantly reduced

DINAMIT – ICD vs. conventional therapy – ICD → decreased arrhythmic death by ≥ 50% but offset by significant increase in non-arrhythmic death

Intrinsic RV – (AV Search Hysteresis) DDDR ICD vs. VVI ICD – AVSH can dramatically reduce the percentage of RV pacing among ICD recipients (RV pacing is detrimental – DAVID and MOST trials)

MADIT II – ICD vs. no ICD – ICD reduces mortality by 31%, no EPS needed, patients with prior MI and EF ≤ 30%

MIRACLE – CRT vs. no therapy – significant improvements for NYHA Class III and IV in QoL, reduction in worsening of HF, cardiac structure and function (MUSTIC trial – same findings)

MUSTT – ICD&non-suppressible vs. AADs&suppressible vs. no therapy – ICD reduced the risk of arrhythmic death and cardiac arrest

DAVID – demonstrated an association percentage of RV pacing and HF in patients with SSS

MOST – cumulative duration of RV apical stimulation resulting in non-physiologic contraction

CHF-STAT – Amiodarone is beneficial for AF with CHF (CHF-STAT and GESICA – CHF patients with ventricular arrhythmias → supports the use of Amiodarone)

SCD-HeFT – ICD vs. Amiodarone – Amiodarone alone is not effective in preventing SCD; ICD – 21% reduction in ischemic cardiomyopathy, 27% reduction in non-ischemic cardiomyopathy; patients with NYHA Class II and III and EF ≤ 35%

PainFree Rx II – ICD, fast VT >200bpm – ATP for FVT is highly safe and effective and improves QoL

EMPIRIC – standardized set of VT/VF programming utilizing extensive SVT discrimination and ATP is at least as effective as customized physician programming for minimizing shocks after ICD implant

PAVE – Bi-V vs. RV pacing post AVN ablation (for AF) evaluation – Bi-V is superior to RV pacing among pacemaker-dependent patients particularly with LV systolic dysfunction, EF stable with CRT; Frontier II Bi-V, FDA approved after ablation for AF