Cardiac Pacing and ICDs- Ellenbogen and Wood

Chapter 1- Indications for Permanent and Temporary Pacing

I. Anatomy
   A. SA node- junction of RA and SVC; fed by SA nodal artery and proximal branch of RCA or left circumflex; lots of P cells (initiate impulses-automaticity); **normal impulse generator**
   B. AV node- fed by large AV nodal artery, RCA, and septal branches of LAD, P cells too (not as many as SA); **delays impulse (.04 seconds) to allow complete atrial emptying, subsidiary ppm, acts as filter limiting ventricular rates**
   C. His Bundle- purkinje fibers emerge from AV node to form this; fed by AV nodal artery, branches of LAD; **conduct impulses from AV node to bundle branches**
   D. Bundle branches- starts in muscular septum and branches out in ventricles, fed by LAD, RCA; **activates ventricles**

II. Indications for Permanent Pacing
   A. Class I- conditions for which there is evidence and/or general agreement that ppm placements is beneficial, useful, effective
   B. Class II- conflicting evidence and/or divergence of opinion about the usefulness/efficacy
      i. IIa weight of evidence in favor of
      ii. IIb usefulness/efficacy less well established
   C. Class III- evidence or general agreement that ppm is not useful/effective or might cause harm

III. Acquired AV Block
   A. Acquired AV block with syncope (**Stokes-Adams attacks**) was first indication for pacing
   B. In the presence of symptoms documented to be DT AV block, pacing is indicated regardless of the site of the block
   C. **AV Block- see cause of acquired high-grade AVB pg 11 Ellenbogen**
      i. Class I. Third degree and advanced second degree AV block at any anatomic level (**Neuromuscular disease- Kearnes-Sayre syndrome, Erb’s dystrophy**)
      ii. Class IIA
         1. Asymptomatic third-degree AVB with rate >= 40bpm
         2. Asymptomatic type II second degree AVB with a narrow QRS (if QRS is wide= class I)
         3. Asymptomatic type I
      iii. Class IIB pg 7-8 Ellenbogen
   IV. Chronic Bifascicular or Trifascicular Block
      Class I
         1. Intermittent 3rd degree AVB
2. Type II 2nd degree AVB
3. Alternating BBB

V. Sinus Node Dysfunction
   A. Disorder includes brady, sinus arrest, SA block, SVT (atrial or junctional), alternating periods of brady/asystole
   B. Quite common, incidence increases with age; important to correlate symptoms with bradycardia; more patients are implanted for this reason than any other indication
   C. Common meds that cause SA dysfunction or AVB- digitalis, antihypertensive agents, beta adrenergic blockers, calcium channel blockers, antiarrhythmic drugs, psychotropic meds
   D. Class I- (see pages 17-18 ellenbogen)
      i. SA node dysfunction with documented symptomatic brady or sinus pauses
      ii. Symptomatic chronotropic incompetence

VI. Neurocardiogenic Syncope/ Hypersensitive Carotid Sinus Syndrome- neurally mediated syncope- form of abnormal autonomic control of the circulation (syncope accounts for 6% of hospitalizations)
   A. Three types
      i. Cardioinhibitory- ventricular asystole of at least 3 seconds due to sinus arrest or CHB- this one will be benefited most by ppms
      ii. Pure vasodepressor response
      iii. Mixed
   B. Pacemaker therapy considered in pts only when symp severe, recurrent and cannot be controlled by more conservative measures (avoidance of stimuli, beta blockers, midodrine hydrochloride- ProAmatine and or fludrocortisone acetate- Florinef)
   C. Class I- Recurrent syncope caused by CSS see pg 22 ellenbogen

V. Causes of Torsades- see pg 26 ellenbogen

VI Permanent PPM mode selection
   A. largely based on the desire to maintain AV synchrony

VII. Disorders of AV conduction
   A. Most commonly caused by inferoposterior infarct, supplied by LCX
   B. Risk of progression from 1st degree to high grade AVB is 10-30%

Chapter 2- Basic Concepts of Pacing

I. Basic EP
   A. Excitability- the property of biologic tissue to respond to a stimulus with a response that is out of proportion to the strength of the stimulus
      i. Cardiac myocytes- there is a separation of charge across the cell membrane that results in a resting transmembrane electrical potential; the concentration of NA+ outside the cell exceeds the
concentration inside; the inside of the cell has a 35-fold greater concentration of K+ versus outside

ii. See pg 48 ellenbogen for resting action potential

II. Stimulation Threshold- the delivery of a polarizing electrical impulse from an electrode in contact with the myocardium with the generation of an electrical field of sufficient intensity to induce a propagating wave of cardiac action potentials.; definition: the minimum stimulus intensity and duration necessary to reliably initiate a propagated depolarizing wavefront from an electrode.

A. Strength Duration Relation- a small change in pulse duration is associated with a significant change in the threshold amplitude at short pulse durations; however, only a small change at longer pulse durations

i. Rheobase- the least stimulus voltage that will electrically stimulate the myocardium at any pulse duration (usually determined at 2.0ms)

ii. Chronaxie- as the threshold pulse duration at a stimulus amplitude that is twice rheobase voltage (pg50)

iii. Energy-

\[ E = \frac{V^2}{Rt} \]

E= the stimulus energy
V=stimulus voltage
R=total pacing impedance
t= pulse duration

****Since the energy of a pacing stimulus increases by the square of the voltage, doubling the stimulus voltage results in fourfold increase in the stimulus energy.

-The chronaxie pulse duration approximates the point of minimum threshold energy on the strength-duration curve; with pulse durations greater than chronaxie, there is relatively little reduction in the threshold voltage; the wider pulse duration results in the wasting of stimulation energy without providing a substantial increase in safety margin; at pulse durations less than the chronaxie there is a steep increase in threshold voltage and stimulation energy.

-Tripling a threshold pulse duration that is greater than 0.3ms may not provide adequate safety margin

-Stimulus thresholds that are measured by decrementing stimulus voltage until LOC are usually 0.1 to 0.2V lower than when the stimulus intensity is gradually increased from subthreshold until capture is achieved.

B. Strength-Duration Curves for Constant-Voltage and Constant-Current Stimulation

i. Constant-voltage stimulation usually results in a flat curve at pulse durations greater than 1.5 milliseconds, whereas constant-current stimulation curve may be slowly downslping beyond this duration. (see. Pg 54).

ii. Small changes in pulse duration less than 0.5ms may result in significantly greater reduction in stimulation safety margin for constant-current than for constant-voltage generators.
Because of this diff. In the shape of the strength duration curves, the chronaxie pulse duration of a constant-current strength duration relation is significantly greater than that observed with constant-voltage stimulation. Therefore a constant-voltage generator can be set to deliver a narrower pulse duration than a constant-current generator and yet provide the same safety margin.

C. Time-Dependent Changes in Stimulation Threshold.
   i. See pg. 55 chart; acute rise in threshold that begins within the first 24 hours. Threshold usually continues to rise over the next several days, usually peaking at approximately one week. The typical stimulation threshold then gradually declines over the next several weeks. By 6 weeks, the myocardial stimulation threshold has usually stabilized at a value that is significantly greater than that measured at implantation, but less than acute peak.
   ii. Active fixation electrodes may produce, immed. Following implant, an increased stimulation threshold that gradually decreases over the next 20 to 30 minutes. This is likely RT acute injury at the myocardial-electrode interface and is generally not observed with atraumatic passive fixation leads (current of injury).
   iii. In general, the more stable and the less traumatic the interaction of the electrode and lead with the myocardium, the lower the rise in threshold over time.
   iv. Programming the stimulus intensity greater than 2.8v results in a marked increase in current drain from the battery.
   v. Patients subject to major shifts in potassium concentration or acid-base balance, such as those with renal failure, may have transient increases in pacing threshold.
   vi. The smaller the surface area of the electrode, the lower the pacing threshold.

D. Effects of Pacing Rate on Myocardial Stimulation
   i. At pacing rates exceeding 250 bpm, pacing stimuli might be delivered during the relative refractory period, resulting in an increase in threshold, which may have implications for ATP.

E. Pharmacologic and Metabolic Effects on Stimulation Threshold
   i. Stimulation threshold generally increases during sleep and falls during waking hours. Decreases during exercise DT autonomic tone and circulation catecholamines.
   ii. Increases following eating, hyperglycemia, hyperkalemia, hypoxemia, hypercarbia, and metabolic acidosis and alkalosis.
   iii. Increases dramatically during acute viral illnesses, esp. in children.
iv. Isoproterenol may restore exit block in some patients. Beta-blockers increase threshold. Corticosteroids can decrease stim threshold.

v. Raise stim threshold: type I antiarrhythmic drugs quinidine, procainamide, flecainide, and encainide.

vi. Amiodarone (Type III) is questionable if raises stim. thresh.

III. Impedance- the sum of all factors that oppose the flow of current in an electric circuit. Not necessarily the same as resistance.

Ohm’s Law: \[ V=IR \] current= I, resistance=R

i. the leading edge voltage of a constant-voltage ppm is fixed, and the lower the resistance, the greater the current flow. In contrast, the greater the resistance, the lower the current flow.

ii. Powered by lithium iodine batteries with a fixed amount of charge; pacing impedance is an important determinant of battery longevity.

iii. Total pacing impedance is determined by factors that are RT the lead conductor (resistance), the resistance to current flow form the electrode to the myocardium (electrode resistance), and the accumulation of charges of opposite polarity in the myocardium at the electrode-tissue interface (polarization).

iv. The resistance to current flow by the lead conductor results in a voltage drop across the lead with a portion of the pacing pulse converted into heat = inefficient use of electrical energy and does not contribute to myocardial stimulation. The ideal pacing lead would have a very low conductor resistance.

v. In contrast, the ideal pacing lead would also have a relatively high electrode resistance to minimize current flow and maximize battery life. The electrode resistance is largely a function of the electrode radius, with higher resistance provided by a smaller electrode.

vi. An electrode with a small radius minimizes current flow in an efficient manner.

vii. Electrodes with a small radius also provide increased current density and lower stimulation thresholds

viii. A lead with 500 ohms of resistance vs. a lead with 1000 ohms, would decrease current drain by 50% with each pacing pulse.

ix. But can’t get too small because risk microdislodgement.

x. Polarization impedance- is an effect of electrical stimulation and is RT the movement of charged ions in the myocardium toward the cathode. Caused by the negatively charged cathode inducing the accumulation of two layers of oppositely charged ions in the myocardium.

xi. Because polarization impedes the movement of charge in the myocardium, it is inefficient and results in an increased voltage requirement for stimulation. Thus polarization impedance reduces the effectiveness of a pacing stimulus to stimulate the myocardium and wastes current.
xii. Polarization is directly RT the duration of the pulse and can be minimized by the use of relatively short pulse durations. It is inversely RT surface area.

xiii. To maximize electrode resistance but minimize polarization, the surface area of the electrode can be made large but the geometric radium small by the use of a porous coating on the electrode.

xiv. Electrodes constructed with activated carbon, or coated with platinum black or iridium oxide are effective in minimizing the wasteful effects of polarization and in diminishing afterpotentials which can interfere with sensing.

xv. Pacing impedance is characterized by a fall over the first 1 to 2 weeks following implantation. The chronic pacing impedance then rises to a stable value that is, on average 15% higher than at implant. See pg 64 for chart

IV. Biventricular Pacing - review impedance

IV. Sensing

A. Intracardiac Electrograms- produced by the movement of electrical current thru myocardium.

i. During depolarization, the outside of the cell becomes electrically neutral with respect to the inside. Therefore, as a wavefront of depolarization travels toward an endocardial electrode that records from resting myocardium, the electrode becomes positively charged relative to the depolarized region. This is manifested in the intracardiac electrogram as a positive deflection. As the wavefront of depolarization passes under the recording electrode, the outside of the cell suddenly becomes negatively charged relative to resting myocardium, and a brisk negative deflection is inscribed in the EGM. The peak negative deflection in the EGM is known as the intrinsic deflection, is considered the moment of myocardial activation underlying the recording electrode.

ii. B/c of greater mass, the normal vent EGM is usually of far greater amplitude than the normal atrial EGM. See pg 70 for deflection

iii. By using Fourier transformation one can express the frequency spectrum of an electrical signal as a series of sine waves of varying frequency and amplitude. Maximum density of frequencies for R waves is usually found between 10 and 30 Hz. Removing frequencies below 10Hz markedly attenuates the T wave amplitude without significantly influencing the R wave. The T wave is usually a slower, broader signal that is composed of lower frequencies, generally less than 5 Hz.

iv. Similarly, the FFRW in the atrial EGM is composed predominantly of low-frequency signals. Therefore by high-pass filtering of the intracardiac EGM, many of the low frequency components can be removed.

v. In contrast, the frequency spectrum of myopotentials ranges approx 10 to 200 Hz, with overlap of P and R waves. Despite the
high frequency filtering, inappropriate sensing of myopotentials remains a potential prob with unipolar config.

vi. For the EGM to be sensed by the sense amplifier of an implantable ppm, the signal must be of sufficient amplitude, measured in peak-to-peak voltage. In addition, the intrinsic deflection must have a sufficient slope. The peak slope (dV/dt) of the EGM (slew rate) is of critical importance to sensing. The sense amplifier of most ppm has a center frequency (the freq for which the amplifier is most sensitive) in the range of 30 to 40 Hz, so frequencies greater than this are attenuated and less likely to be sensed. In general, the higher the slew rate, the higher the frequency content.

vii. Thus slow, broad signals with a low slew rate may not be sensed, even if peak-to-peak amplitude is large.

B. Unipolar and Bipolar Sensing

i. Bipolar lead- electrodes are within the heart with an interelectrode distance that is usually less than 3cm. A unipolar lead has interelectrode distance of 30 to 50 cm.

ii. Because the bipolar config represents the signal at the cathode minus the signal at the anode, the net EGM may be considerably different.(see pg 72 for further explanation)

iii. Normal PVARP begins after a sensed or paced vent event

C. Time related Changes in EGM

i. The injury of current usually returns to the isoelectric line over a period ranging form several minutes to several hours.

ii. The amplitude of the EGM typically declines abruptly within several days following implantation, with a gradual increase toward the acute value by 6 to 8 weeks. Chronic R wave amplitude about 85% of implant value and slew rate about 50-60% of implant value.

iii. Active fixation leads follow a different path...immediately following lead placement, there is a markedly attenuated amplitude and slew rate. Over the next 20-30 minutes, the EGM amplitude increases- likely due to current of injury.

D. Sensing Impedance

i. The electrode resistance is inversely RT electrode surface area. Polarization impedance is also inversely RT to electrode surface area. Thus electrodes with large surface area minimize source impedance and contribute to improved sensing.

E. Automatic Capture Features

i. The Autocapture feature of STJM ppm allows the ppm to automatically adjust the amplitude of the stimulation pulse by detecting capture in the vent. These ppms require a bipolar vent pacing lead that must have low polarization properties for the distal electrode. The presence or absence of vent capture is determined by sensing ER from the ring electrode. See pg 79 for further explanation.
ii. The MDT Vent Capture Mgmt features a strength duration threshold at a programmed interval see pg 81-82 for further details.

V Lead Design- leads have 5 major components- the electrode, the conductor, insulation, connector pin, the fixation mechanism.

A. Electrodes
   a. The smaller the radius of the electrode, the greater the current density.
   b. The resistance at the electrode-myocardial interface is higher with smaller electrodes, providing efficient use of a constant-voltage pulse and improving battery longevity.
   c. Sensing impedance and polarization are decreased with electrodes of larger surface area.
   d. The ideal pacing lead would have an electrode with a small radius (to increase current density) and a large surface area (to reduce polarization). The solution to these conflicting considerations for optimal stimulation and sensing characteristics has been addressed by the development of electrodes that have a small radius but a complex surface structure that provides a large surface area.

B. Electrode Shape
   a. Electrodes with a smooth, hemispherical shape produce a uniform current density. In contrast, electrodes with more complex shapes typically produce an irregular pattern of current density, with “hot spots” at the edges and points of the electrode (good).

C. Surface Structure
   a. The use of electrodes with a textured surface has resulted in a dramatic increase in the surface area of the electrode without in increase in radius. The textured surface of modern leads minimizes polarization and improves sensing and stimulation efficiency (Elgiloy, platinum, or iridium oxide).
   b. The performance of carbon electrodes has been improved by roughening of the surface, a process known as activation; this structure can lead to the ingrowth of tissue into the electrode.
   c. The importance of low polarization has greatly increased the ability of ppms to sense evoked response.

D. Chemical Composition
   a. Metals such as zinc, copper, mercury, nickel, lead and silver are assoc with toxic reactions in the myocardium and are unsuitable in the use chronically implanted leads.
   b. These metals also result in increased chronic stimulation thresholds.
   c. Stainless steel alloys are variably assoc with potential for corrosion.
   d. Titanium and tantalum have been shown to acquire a surface coating of oxides, which may impede charge transfer.
e. However, titanium that is coated with microscopic particles of platinum or vitreous carbon has been found to have excellent long-term performance as a pacing electrode.

f. Elgiloy is acceptable when used as a cathode, but corrosive when used as an anode.

g. The materials presently in use for the electrodes of permanent pacing leads include platinum-iridium, Elgiloy, platinum coated with platinized titanium, vitreous or pyrolytic carbon coating a titanium or graphite core, platinum, or iridium oxide. The platinized-platinum and iridium-oxide have been associated with a reduced degree of polarization. Carbon corrodes the least.

E. Steroid-Eluting Electrodes
   a. Incorporates a silicone core that is impregnated with a small quantity of dexamethasone. The core is surrounded by a porous titanium electrode that is coated with platinum.
   b. It should be emphasized that the corticosteroid eluted from the lead does not affect acute stim thresholds. Rather, the steroid controls chronic evolution of pacing thresh.

D. Fixation Mechanism
   a. Passive fixation leads, in general, are more difficult to extract than active fixation.

F. Active Fixation Leads
   a. Inactive helix leads are assoc with lower acute thresholds.
   b. Active fixation leads have significantly reduced the risk of atrial lead dislodgement, but the chronic pacing thresholds are somewhat higher.
   c. Risk of myocardial perf is higher

F. Conductors
   i. The most common site for lead fracture is at the fulcrum of a freely moving conductor with a stationary point (subclavian vein and first rib).
   ii. Leads also may fail at the site of mechanical injury, such as with tight fixation sutures.
   iii. Stainless steel has the potential of corrosion and therefore was abandoned for the use of conducting coils of early multifilar leads. It was replaced by Elgiloy or MP35N, an alloy of nickel.
   iv. More recently, conductors made of DBS (drawn-brazed-strand), six nickel alloy wires that are drawn together with heated silver, has been introduced.
   v. DBS is no longer used with polyurethane because of the potential for internal oxidation of the poly by the silver chloride from the DBS conductor.

G. Insulation
   i. Platinum-cured silicone rubber, which is characterized by improved mechanical strength. The coefficient of friction has been
greatly reduced by the development of a lubricious “fast pass” coating. Improved leads are smaller external diameter and are far easier to manipulate when in contact with another lead.

ii. See chart pg 93 on pacemaker lead insulation.

iii. The 55D polymer is now the predominant form of poly used for leads (greater tensile and tear strength but stiffer than P80A).

iv. Surface cracks are likely RT environmental stresses rather than to biologic degradation. The surface cracks likely develop in the manufacturing process as the heated poly cools more rapidly than the inner core, leading to opposite stresses within insulation.

H. Epimyocardial Leads
   i. Used in clinical situations involving abnormalities of the tricuspid valve, congenital heart disease, or when ppm leads are implanted during intrathoracic surgical procedures.
   
   ii. The use of three turns has been shown to reduce exit block as compared with a two-turn screw.

I. Connectors
   i. Industry wide standard of IS-1, sealing rings on a 3.2mm lead connector see pg 96-97 for lead connectors

J. Specialized Coatings to Prevent Tissue Ingrowth
   i. The first consideration is to make the lead isodiametric so that there are no ridges or areas of increased diameter along the lead that would increase the risk of removal.
   
   ii. ePTFE- prevents the ingrowth of tissue into the coil electrodes while having no significant effect on the conduction of electrical energy.

V. Pulse Generators
A. Power Source- modern ppms use lithium as the anodal element and iodine as the cathodal element
   i. A major advantage of the LiI battery is the solid nature of the material- allowing the cell to be hermetically sealed and relatively persistent to corrosion
   
   ii. LiI cell generates approx 2.8v at BOL. A voltage multiplier may be used to allow output pulses of greater than that generated by the battery.
   
   iii. The net result is that by programming the ppm to a voltage that is double the battery voltage, the charge taken from the battery is equal to twice that delivered to the lead and the total amount of charge drained from the battery increases fourfold.
   
   iv. The longevity of the battery is determined by the chemical elements of the battery, the size, the amount of internal discharge, and the voltage decay charact.
   
   v. To maximize battery life, the ideal electrochemical cell would have no internal discharge.
   
   vi. LiI has a self discharge of about 1% a year.
vii. For a battery to be suitable for a ppm, the decay character should be predictable. The ideal battery should have a predictable fall in voltage near the end of life, yet provide sufficient service life after the initial voltage decay to allow time for the ERI to be detected.

viii. The voltage produced by a LiI cell is inversely related to the internal battery impedance. The internal impedance of the battery increases with the thickness of the LiI electrolyte layer, from less than 1k ohm at the beginning of life to over 15k ohm at the extreme EOL. The voltage generated by the cell declines almost linearly from the initial value of 2.8v to 2.4v at approx 90% of the usable battery life. Following this, the voltage declines exponentially to 1.8v at the EOL. The magnet related pacing rate of a ppm is RT to the cell voltage.

ix. In order, output pulse and impedance are major factors of current drain.

x. The cost of doubling the output voltage (2.8 to 5.6- uses two capacitors in parallel) is a fourfold increase in current drain from the battery. A threefold increase (8.4v- three capacitors in parallel) results in a nine-fold increase in current drain.

xi. The lower the impedance, the greater the current delivered and the greater the drop in voltage from leading edge to trailing edge during the pulse. Even, though the term constant voltage is used to describe the stimulus waveform of ppms, in the reality the output voltage of the pulse is not constant beginning from end.

xii. The constant current pulse is typically flat, with little or no change in current from leading edge to trailing edge. However as the polarization impedance rises during the pulse, the resulting voltage must also rise proportionally to maintain the current at a constant level. At extremely high lead impedances, the voltage required to maintain a constant-current pulse may exceed the capabilities of the battery.

xiii. The output waveform of the pulse generator is followed by a low-amplitude, long-duration wave of opposite polarity known as the afterpotential. The afterpotential is caused by the polarization at the electrode-tissue interface and is dependent on the stimulus amplitude and duration.

xiv. To reduce the afterpotential, the output circuit of some manufacturers incorporates a fast recharge pulse, during which the electrode polarity is reversed for a short period following the output pulse.

B. Power Source

i. To minimize attenuation of the signal the sensing amplifier must have an input impedance greatly in excess of the sensing impedance. The greater the input the impedance, the less the EGM is attenuated by the amplifier. The input impedances of the sense amplifiers used in ppm systems are greater than 25,000 ohms.
ii. The bandpass filters of different manufacturers vary significantly with regard to center freq (from approx 20 to 40Hz), so intracardiac egms measured with a PSA of one manufacturer may provide different values compared to the next.

iii. Signals with amplitude greater than the sensitivity threshold level are sensed as intracardiac events, whereas signals of lower amplitude are discarded as noise.

iv. Ppms also contain noise reversion circuits that change the pulse generator to an asynch pacing mode when the sensing threshold is exceeded at a rate faster than the noise reversion rate.

v. The Zener diode protects the integrated circuit from high external voltages such as may occur during defib shocks or electrocautery. When the input voltage carried by the pacing leads exceeds the Zener voltage, the excess energy is shunted back to the myocardium thru the leads.

vi. During the ventricular blanking period, the ventricular sensing amplifier is turned off immediately following the atrial pacing pulse.

C. Timing Circuits
   i. The pulse generator contains a rate-limiting circuit that prevents the pacing rate from exceeding an upper limit in the case of a random component failure. This runaway protection rate is typically in the range of 180 to 220ppm.

D. Microprocessors
   i. ROM (typically 1 to 2 kilobytes of 8 to 32 bits) is used to guide sensing and output circuits. RAM is used to store diagnostic information regarding pacing rate, intrinsic heart rates, and sensor output.

E. Rate-Adaptive Sensors
   i. The ideal sensor demonstrates sensitivity and specificity.

F. Activity Sensors and Accelerometers
   i. A piezoelectric ceramic crystal functioning as a strain gauge is bonded to the inside of the pulse generator case or to the circuit board. As the crystal flexes and deforms in response to mechanical vibration or pressure, an electric current is generated. The magnitude of the electric current for the crystal is RT the freq and amplitude of vibrations. The output of the sensor is processed electronically and used to modulate changes in pacing rate.

   ii. The piezoelectric accelerometer is mounted to the hybrid circuitry-standard method for detection of body vibrations.

   iii. In contrast, piezoresistive accelerometers measure changes in electrical resistance that occur with mechanical deformation of the sensor and require a somewhat greater current to drain to power the sensor.

   iv. Exercise workload is more proportional in the anterior-posterior axis than in the vertical axis.
v. Accelerometers have been shown to produce a RR that is closer to the expected behavior of the SA node during exercise.

vi. Because the frequency range of the accelerometer signal associated with exercise is known to have a maximal amplitude less than 4 Hz, the signal can be filtered to remove external noise, which typically is between 10 and 50 Hz.

vii. B/c accelerometers have a constant coupling mass of the sensor that is independent of the body, these devices offer a more predictable range of responses during exercise that do activity sensors.

G. MV Sensors
i. Resp rate, tidal volume, and minute ventilation (the product of these 2 parameters) increase in proportion to changes in carbon dioxide production (VCO2). At exercise workloads less than anaerobic threshold, the MV is closely assoc with oxygen consumption (VO2).

ii. MV is estimated by freq measurements of transthoracic impedance between an intracardiac lead and the ppm using the tripolar system. A low energy pulse of 1mA @15ms is delivered from the ring electrode. The resultant voltage between the tip and the pulse generator is measured and an impedance is calculated. The impedance pulses are subthreshold and are delivered every 50ms.

iii. The impedance is mostly RT the volume and resistivity of blood in the right heart chambers and systemic venous system.

iv. The impedance signal fluctuates in response to both respiration and cardiac motion (right ventricular ejec). The signal may also change in response to arm movement.

v. To minimize the cardiac-related component of the impedance signal, low-pass filtering of frequencies greater than 48 to 60 hz is performed.

H. QT Interval
i. QT interval has been demonstrated to shorten with exercise or sympathetic tone and to lengthen at rest.

ii. Measured from the onset of a pacing stimulus.

iii. Although it varies widely among individuals, it is quite consistent in an individual at rest.

iv. Can be markedly influenced by meds or electrolyte concentrations.

v. Disadvantages of the QT sensor involve the requirement for a low-polarization electrode.

vi. Advantages are its responsiveness to emotional factors and the lack of a specialized lead.

Chapter 3- Hemodynamics of Cardiac Pacing
I. AV Synchrony
A. Advantages of AV Synchrony
a. AV synchrony generally provides similar or slightly greater systolic and mean blood pressure than vent pacing

B. Cardiac Output
   a. Properly timed atrial contraction provides a significant increase in vent end-diastolic volume and is responsible for the so-called atrial kick
   b. There has been a general perception that patients with abnormal cardiac function benefit most from maintenance of AV synchrony—not freq DT cardiac output

C. Effects of AV Interval
   a. An excessively long AV delay may inadequately fill the ventricle by causing early mitral valve closure and truncating diastolic filling time
   b. Diastolic AV valvular regurgitation may occur with re-opening of the valve before ventricular systole.
   c. An excessively short AV delay may limit active filling of the ventricle and promote systolic AV valvular regurgitation as ventricular contraction begins while the AV valves are still open.
   d. At rest, 125-200ms is generally the optimal range if the right atrium and ventricle are paced.
   e. Doppler echo is commonly used to optimize the AV interval by assessing the pattern of early and late diastolic mitral valve flow and the aortic flow velocity integral. While AV interval can be optimized at rest, assessing the adequacy of this parameter upright and during exercise presents greater technical difficulty.
   f. With exercise there is a relatively linear decrease in PR interval. The total reduction in spontaneous PR interval in normal individuals appears to be about 20-50ms or approx 4ms for each 10-beat increment in HR.
   g. Because some atrial activation has already occurred at the time a sensed event marks the initiation of the ppm AV interval, the SAV interval should be about 20-50ms less than a PAV interval.

D. Pacemaker Syndrome
   a. Most often results from VVI pacing, but can result in any mode that cause AV dyssynchrony, even AAI pacing with long PR interval.
   b. Syncope, a very uncommon result, is RT profound hypotension—and in some, a decrease in cardiac output.
   c. Additional symptoms of decreased CO include, malaise, easy fatigability, a sense of weakness, lightheadedness, and dizziness.
   d. Symptoms RT higher atrial and venous pressures include dyspnea, orthopnea, paroxysmal nocturnal dyspnea, fullness and/or pulsations in the neck and chest, as well as palpitations, chest pain, nausea, and/or peripheral edema.
   e. You sometimes can visualize continuous or fluctuating pulsations in the neck, neck vein distension, with prominent “cannon” A waves, pulmonary rales and rarely peripheral edema.
   f. PPM syndrome is most severe when intact retrograde VA is present.
g. This symptoms result from reduced stroke volume and cardiac output from the loss of atrial kick, and elevated venous pressures resulting from atrial contraction against closed AV valves.

h. For patients with PPM syndrome, to help can upgrade to DDD system, reduce LR to encourage intrinsic conduction, use of hysteresis, or withdrawal of meds that impair SA node function, ensure atrial capture, avoidance of atrial non-pacing modes (VDD) or atrial non-tracking modes (DDIor DVI).

II. Hemodynamics of Pacing in HOCM

Hypertrophic obstructive cardiomyopathy represents a special situation for which pacing may have a role in some patients. These patients have obstruction to LV outflow caused by hypertrophy of the interventricular septum typically in the subaortic valve area, combined with systolic anterior motion of the mitral valve. Studies show that by producing dyssynchrony of LV contraction, dual-chamber pacing reduces the degree of outflow obstruction and symptoms in many patients.

Chapter 4 Techniques of Pacemaker Implantation and Removal

I. Implant Procedure

A. Site

a. Persistent Left Superior Vena Cava (0.3-0.5%) - drainage into the CS, which complicates lead positioning. Suspicion of this anomaly may be raised by finding greater distension and a double A wave in the left jugular vein compared with that of the right vein, a left paramediastinal venous crescent on the chest radiograph, and an enlarged CS on the echo.

b. With PLVC, right sided implants are easier

B. Ventricular Lead Position

a. Under fluro, the RV apical lead is one in which the lead’s tip is well to the left of the spine and in pointing anteriorly and slightly caudal.

b. In the AP projection it may not be possible to distinguish whether a lead is in a posterior coronary vein, the LV, or the RV apex.

c. In the right anterior oblique position, you can observe the position of the lead with respect to the tricuspid valve which allows you to visualize how far the lead is in the RV.

d. Failure to record a large current of injury after fixing active-fixation leads suggests an unstable position.

e. All lead positions should be confirmed by both left anterior oblique and right anterior oblique views in the lab.

f. It is common for capture thresholds to decrease significantly 15 to 30 mins after active fixation.

C. Atrial Lead Implantation

a. Right atrial appendage preferred site

b. Studies suggest that dislodgement is not more common with atrial leads, but reliance on the appendage location may mandate the acceptance of less than ideal pacing characteristics that become unacceptable over time.
c. There is no evidence that the atrial stimulation site influences hemodynamics per se although atrial septal pacing near Bachman’s bundle may be of some importance when atrial tachycardia algorithms are applied.
d. The j shaped active fixation can be positioned almost anywhere in the atrium but may increase dislodgement risk because of the undue tension at the site of attachment to the endocardium.

D. Single lead VDD Pacing
a. Proximal atrial sensing electrode as well as a tip electrode to sense and pace the ventricle
b. Good for AVB patients who have normal sinus mechanism.
c. Functionally VVI(R)

E. Complications of Implantation
a. PASE study, 6.1% of 407 patients had a complication- lead dislodgement, pneumothorax, perforations.
b. Harcombe and coworkers found that the rate of late complications (later than 6 weeks after the procedure) is higher for elective replacement than initial system implantation.
c. Air embolus can occur when a central vein is accessed by a sheath. May be signaled by a hiss as air is sucked into the sheath by negative intrathoracic pressure and may occur suddenly when a heavily sedated pt, deeply inspires and control over the sheath’s orifice is uncontrolled.
d. Symptoms of air embolus include respiratory distress, chest pain, hypotension, and arterial oxygen desaturation (is significant blockage of pulmonary artery), however is air amount is small, usually well tolerated.
e. Perforation may occur internally (into another cardiac chamber) or externally (into the pericardial space) by the pacing lead.
f. Old age, female gender, steroid therapy, recent RV infarct, stiff leads or stylets may be considered risk factors.
g. Silent venous thrombosis is not uncommon, 11-12% of patients suffer asymptomatically with this.

F. Infection
a. Diabetes and postoperative hematoma are predisposing factors.
b. Usually staph aureus or staph epidermidis, more than 1/3 to ½ occur with new systems.
c. Most will warrant an explant with antibiotic therapy.

G. Complications of BiV Pacing
a. Unable to place LV lead, 9%
b. Unique complications such as extensive coronary sinus dissection, 4-7% and coronary venous dissection 2%

H. Lead Extraction
a. A lead that has been in place for 3 months or less should be easily removed whereas one in place longer than a year may well present difficulties to fibrosis.
b. Non-isodiametric leads and those with anchoring appendages (tines or fins) present additional problems

c. Lead extraction/explantation can be done percutaneously or transthoracically

d. The Accufix J- there was a small metal wired placed within the lead only to maintain a J shape is subject to fracture under the stress and strain of clinical use

e. Lead extraction using modern tools has an associated risk of death of 0.6% and a 2.5% risk of potentially life-threatening complications.

f. With modern tools, there is about a 90% success rate of lead extraction- laser

**Chapter 6 Pacemaker Timing Cycles**

A three-letter code describing the basic function of the various pacing system was first proposed in 1974 by a combined task force from the American Heart Assoc and the American College of Cardiology

- Elimination of crosstalk may be accomplished by extending the ventricular blanking period, decreasing atrial output, or reducing the ventricular sensitivity

- in an atrial-based timing system, the AA interval is fixed whereas in a ventricular-based system, the AEI is fixed

- in an atrial-based system, the alternation of the longer AVI with the shorter AR interval results in ventricular rates that are both faster and slower than the programmed base rate.

- in ventricular based timing, the LR is never violated

- NCAP is used to minimize competition between the sensor and sinus rhythm. If atrial depol occurs within the alert period, it inhibits atrial output and triggers ventricular output.

**Chapter 7 The Implantable Cardioverter Defibrillator**

I. Indications - initially devices were only implanted in patients who had survived a cardiac arrest or an episode of sustained VT  see page 381 for indications of ICD

A. AVID study- compared ICD implantation with antiarrhythmic drug use (primarily amiodarone) in patients with aborted cardiac arrest or poorly tolerated VT. There was a significant reduction in mortality in the grps randomized to ICD implantation. Benefit showed most in pts with a reduced EF of <35%.

B. MADIT and MUSTT trials evaluated pts with CAD, left ventricular systolic dysfunction, nonsustained VT, and inducible sustained monomorphic VT

C. In MADIT, patients were randomized to either receive an ICD or “conventional” medical therapy, most commonly amiodarone.

D. In MUSTT pts were randomized to either no antiarrhythmic therapy or EP guided drug therapy
E. Both trials showed a 50% decreased mortality in those with ischemic cardiomyopathy.

F. MADIT II was a prospective randomized study of 1232 pts with previous MI and EF of 30% or less, spontaneous VT or EP testing was not required for enrollment

G. SCD-HeFT study evaluated more than 2500 subjects with CHF (II and III) and EF <=35%, ischemic or nonischemic. ICD implantation, but not amiodarone, was shown to reduce all-cause mortality.

H. One cohort that is particularly difficult to manage is those pts with structural heart disease and a history of syncope.

I. Lead insulation can have an important impact on long-term stability and function. Silicone is inert, biostable, and biocompatible, but has a high coefficient friction. It is soft which can make it prone to damage and swell over time. Poly is biocompatible, has a high tensile strength making small lead diameters possible, and a low coefficient of friction but it is prone to environmental stress and metal ion oxidation. Fluoropolymers (PTFE and ETFE) are the most biocompatible, have high tensile strength allowing small lead size, but are stiff, susceptible to damage from traction when the lead migrates, and they are prone to insulation micro defects and have a difficult manufacturing process.

J. Today’s systems have an insulating body of silicone, and in some instances supplemented by an outside poly layer (not used for insulation) to reduce friction, abrasion, and scar formation. Metal ion oxidation is avoided as poly is not in direct contact with the conductors.

K. The first large study to evaluate directly the impact of dual chamber pacing among ICD patients was DAVID.

L. Defibrillation testing
   a. A single termination of fibrillation with a given energy level identifies an energy that may terminate fibrillation in as few as 25% of repeated attempts DT the probabilistic nature of defibrillation
   b. By demonstrating that this same shock energy terminates VF three times without failure, it may be concluded that the energy level is high on the probability curve for success and in fact has at least a 75% chance of terminating VF on further attempts.
   c. Two methods predominated DFT- the single energy success and the step-down protocol
   d. Upper limit of vulnerability (ULV)- by delivering shocks of decreasing energy synchronized to the T wave, a minimal energy level is found that does not induce VF while lower energies induce VF. The lowest energy value that does not induce VF is the ULV.

M. Undersensing was most commonly observed with the redetection of VF following failed shocks. The most likely mechanism of this phenomenon is stunning of local myocardium following shocks, resulting in diminution of the EGM near the tip of the lead. With positioning of the RV shock coil farther from the rate sensing electrode tip, post-shock undersensing is minimized (be careful bc DFTs can rise with this) see page 395 for auto adjusting sensitivity.
M. Implantable Cardioverter-Defibrillator Therapies
   a. Approx 70% of new ICD systems are dual chamber devices.
   b. BiVs are indicated for severe CHF (NYHA III-IV), left ventricular systolic dysfunction and QRS prolongation, typically left bundle branch block.
   c. The success rates for spontaneous VT are higher than induced episodes, at about 90%, whereas arrhythmia acceleration rates are low (1% to 3%).

N. Defibrillation
   a. is achieved when a critical mass of myocardium is depolarized by establishing a critical voltage gradient throughout the ventricular tissue.
   b. The resulting waveform is an exponentially declining voltage, which is prematurely terminated or truncated before full capacitor discharge. Thus the maximal delivered energy for an ICD is always less than the stored energy necessary to fully charge the capacitor.
   c. In early ICD systems, monophasic waveforms were used that were truncated at about 35% of the leading edge voltage; this is referred to as a 65% tilt monophasic shock.
   d. Adjusting pulse width is unlikely to have significant effects of defibrillation efficacy.
   e. For biphasic shocks, the polarity of the voltage pulse is reversed after the termination of the initial positive phase and a second negative phase is delivered.
   f. Initially the polarity of shocks used the RV coil as the cathode, however reverse polarity or anodal shocks result in significantly lower DFTs.
   g. Besides biphasic waveforms, the most important advance for lowering DFTs was the development of active pulse generators.
   h. With a dual coil shocking vector and an active can, mean DFTs decreased by about 36%; this benefit was due more to the lowering of impedance with the active can than to an improvement in shocking vector.
   i. In a right-sided implant, there is no significant change in thresholds with the use of an active can compared with a dual-coil transvenous lead. This is be an active can increases defibrillation current requirements due to a worsened shock vector, which is directed away from the RV and toward the right shoulder. The reduction in impedance with the active can offsets the increased current resulting in no net effect on threshold.
   j. Results from the LESS (low energy safety study) suggests that a safety margin of approx 5J may be adequate in modern ICD systems that employ biphasic waveforms, transvenous leads and active pectoral pulse generators.

O. Future Directions
a. Currently there are over 2 million people in the US with HF, including about 400,000 new cases annually.
b. With first generation devices in the 1980s, the annual mortality of patients with CHF was 20-40%, so devices with a 3 yr longevity were often sufficient.
c. The annual mortality today is less than 7% in pts receiving angiotensin-converting enzyme inhibitors, aldosterone antagonists and beta blockers, even for those with NYHA III.

Chapter 9 Cardiac Resynchronization Therapy
I. Heart Failure Epidemic
   a. There are 4-5 million people living with chronic heart failure and an additional 400,000 newly diagnosed yearly.
   b. The incidence of HF is 10 per 1000 for individuals who are older than 65 years of age.
   c. The increasing incidence of HF is due primarily to the advancing age of the population with CAD, which is the principal cause of HF, associated with reduced vent func.(dilated cardiomyopathy)
   d. In the Framingham study, total mortality was 24% and 55% within 4 years of developing symptomatic HF for women and men.
   e. Recognizing the benefits of ACE inhibitors, diuretics, digoxin, and beta-blockers has yielded substantial reductions in mortality DT progressive pump failure. However these improvements in medical therapy, symptomatic HF still confers a 20-25% risk of premature death in the first 2.5 yrs after diagnosis.
   f. Almost all HF pts will have at least one acute episode with symptoms requiring hospitalization and treatment with IV meds. Hospital discharges for HF totaled approx 1million in 2001 and have increased more than 150% form 1979. Hospitalization for management of HF imposes the highest cost by DRG
   g. There are four levels of electromechanical abnormalities associated with DCM: prolonged AV delay, interventricular delay, intraventricular delay, and intramural delay.
      i. Prolonged AV delay- the normal AV interval results in atrial contraction just before the pre-ejection (isovolumic) period of ventricular contraction that maximizes vent filling (LV end diastolic pressure, or pre-load) and CO by the Starling mechanism. The optimal timing relationship also results in diastolic filling throughout the entire diastolic filling period, prevents diastolic MR and maintains left atrial pressure at low levels.
      ii. Interventricular delay- more important that AV coupling for maximum ventricular pumping function. This refers to coordinated contraction of the RV and LV. Typically LBBB occurs where RV begins contraction before LV. Chronic DCM is often accompanied by delayed ventricular electrical activation manifested by prolonged QRS duration, most
commonly in the form of LBBB. Prevalence in HF is in the range of 25-50%. Potent predictor of mortality in HF assoc. with DCM. This delay causes abnormal septal deflections that alter EF. Regions of late activation are subject to greater wall stress and develop local myocyte hypertrophy and aggravated MR.

iii. Intraventricular delay-exists within ventricle. With LBBB, the septum begins contraction substantially earlier than the lateral wall causing the lateral wall to stretch. This causes diminished ejection at increased metabolic cost and acute decline in systolic function.

iv. Intramural delay- delay difference between endocardial and transmural activation

II. CRT

a. therapeutic strategy is that left ventricular preexcitation may correct inter- and intraventricular conduction delays and permit optimization of left-sided AV delay, thereby improving vent pumping function.

A. Mechanisms of CRT

a. When the programmed AV interval is too short, LV preexcitation occurs too early relative to atrial systole. Note that diastolic filling occurs throughout all of diastole. Atrial contraction now occurs simultaneously with LV contraction resulting in increased left atrial pressure and loss of atrial contribution to vent systole, reducing CO. Abolishes premature mitral valve closure, eliminating diastolic MR.

b. Reverse LV remodeling- reduction in left vent volume, redistribution of cardiac mass, reduced mitral orifice size and reduced mitral regurgitation.

B. Implementation of CRT

a. Inability to cannulate the CS- 1-5%

b. As many as 20% of patients don’t have a vein that reaches optimal LV free wall.

c. Epicardial LV leads are usually placed using the OM branches of the circumflex artery, approx 1 cm apical to the mitral annulus.

d. See page 444-447 for BiV pacing and differences in 12 lead EKG

e. See rest of chapter 9 to read about nonresponders

Chapter 10 ICD Followup and Troubleshooting

See pg 480 for drugs that elevate the DFT