1. Electrical Heart

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Origin & Spread of Excitation

Anatomy
- SA node = junction SVC & R atrium
- AV node = R post portion of intraatrial septum
- 3 bundles of atrial fibres which connect SA & AV node:
  - anterior tract of Bachman
  - middle tract of Wenckebach
  - post tract of Thorel
  - conduction also through atrial myocytes
- AV node gives of bundle of His:
  - Left BB – from top of interventricular septum
    - Anterior fascicle
    - Post fascicle
  - R BB – continuation of bundle of His
- Branches and fasicles run subendocardially each side of septum
- Contact Purkinje system
- Conduction system composed of modified cardiac mm
- SA node & AV node contain small round cells with gap junctions
  - pacemaker cells
- Atrium separated from ventricle by fibrous ring ∴ only conduction between 2 is through His bundle
- Vagus & sympathetic distribution:
  - Left ⇒ AV node
  - Right ⇒ SA node
- Sympathetic fibres:
  - From stellate ganglion
  - Norad fibers = epicardial
- Vagal fibers = endocardial
- Cross connection between symp & parasymp:
  - Ach acts presynaptically ⇒ ↓Norad from symp nerves
  - Neuropeptide Y from symp system ⇒ ↓Ach release

Cardiac Muscle APs & Pacemaker APs
- Seen atrial, ventricular & purkinje fibres:

Cardiac AP
- Resting potential -90mV
- Depolarisation spreads rapidly between cells due to gap junctions
1. Electrical Heart

- Phases:
  - 0 = rapid depolarisation towards threshold
    - Na influx via fast voltage gated Na channel opening in response to AP
    - Overshoot seen briefly as Na channels self inactivating
  - 1 = rapid repolarisation –
    - Na channels close
    - Ca open – Ca start to flow in
    - K channels open – K flow out
  - 2 = plateau –
    - Ca influx in which maintains depolarisation (via L type channel)
    - Na channel closing continues which contines to repolarisation
  - 3 = repolarisation –
    - rapid K channel
    - slow K channel
    - both show K out
    - Na & Ca channels return to baseline state
    - Ion channels & electrogenic pumps return membrane to resting potential
  - 4 = return to resting membrane potential –
    - K channels return to baseline state
    - Na/K/ATPase electrogenic pump

Refractory Period

- Ventricular mm AP’s refractory period = ~250ms:
  - ~200ms = absolute (ARP)
  - ~50ms = relative (RRP)
- ARP:
  - extends into phase 3
  - Na channels are still in an inactive state
- RRP:
  - -50 to -90mV
  - possible to elec stim the cell but need a larger stim and resultant AP will be smaller
  - contraction weaker as well (less Ca influx)
- refractory plateau impot:
  - tetany:
    - mechanical response to multiple elect stim is one single twitch
    - in skeletal mm multiple stim ⇒ fused twitches ie tetany which would be bad in heart
  - strength of contraction:
    - influx of Ca during plateau phase ↑s intracellular Ca
    - adrenaline stim ⇒ longer plateau ⇒ +ve inotropy
By Adam Hollingworth

Pacemaker Potential

- seen only in SA, AV nodes
- are other latent pacemaker cells in conduction system if nodal disease
- resting potential -60mV but not stable
- pacemaker cells display auto-rhythmicity ie will always want to move to threshold
- phase 1 & 2 of cardiac AP are absent as no depolarisation plateau
- phases:
  - prepotential (4) ⇒ slow drive to threshold
    - fall in membrane K permeability
    - \( I_f \) = inward slow positive current displayed: (for “funny” current)
      - opening of transient Ca (T type) ⇒ Ca influx
        - not effecting by catecholamines
        - only found in cells which lack a T tubule system ie pacemaker cells & vasc smooth mm (not ventricular myocardium)
      - ↑activity of electrogenic 3Na-2Ca exchange system
        - driven by inward movement of Ca
  - depolarisation (0)– opening long lasting Ca channel ⇒ Ca influx
    - (L type) long lasting Ca channel
    - produce long lasting current relative to Na
    - the most predominant Ca type
    - start opening during initial upstroke
    - verapamil & nifedipine block them
    - catecholamines activate them
  - repolarisation (3) – K channel opening ⇒ K efflux
  - hyperpolarisation (4)– closing of K channel, opening of H channel
    - passes Na & K

∴ action potential in pacemaker cells has:
  - auto-rhythmicity by ↓K ⇒ ↑Ca ⇒ ↑K permeability
  - no contribution by Na

- Vagal effect on pacemaker potential:
  - Cholinergic fibres
  - Membrane hyperpolarised by ↑ing K membrane permeability
  - Prepotential is slower to depolarise
    - Ach act on M2 receptor⇒
      - βy subunit of G protein ⇒ opening special K channel ⇒ K efflux ⇒ slows depolarising effect H channels
      - ↓cAMP in cell ⇒ slows opening of Ca channel
  - all ⇒ ↓speed of firing
Sympathetic input:
- Norad acts on $\beta_1$ receptor $\Rightarrow$
  - $\uparrow$cAMP $\Rightarrow$ quicker opening of Ca channels $\Rightarrow$ quicker depolarisation of prepotential
- other effects on pacemaker potential:
  - $\uparrow$temp $\Rightarrow$ $\uparrow$speed of firing
  - digoxin $\Rightarrow$ $\downarrow$speed of firing esp AV node

SAN vs AVN
- ionic basis same for AVN & SAN
- rate of depolarisation of the pacemaker cells is slower in AVN
- $\therefore$ SAN has the highest depolarisation rate & thus drives all pacemakers cells downstream

Spread of Cardiac Excitation

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Conduction Rate (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA node</td>
<td>0.05</td>
</tr>
<tr>
<td>Atrial pathways</td>
<td>1</td>
</tr>
<tr>
<td>AV node</td>
<td>0.05</td>
</tr>
<tr>
<td>Bundle of His</td>
<td>1</td>
</tr>
<tr>
<td>Purkinje system</td>
<td>4</td>
</tr>
<tr>
<td>Ventricular muscle</td>
<td>1</td>
</tr>
</tbody>
</table>

Atria
- AP generated by SAN spreads from cell to cell directly via gap junctions
- $\therefore$ simultaneous contraction both atria
- atria separated from ventricle by fibrous tissue – must go via AVN & bundle of His
- Atrial depolarisation complete in 0.1s
- SAN through atrial mm at 1m/sec
- SAN $\Rightarrow$ Left atrium via Bachmann’s bundle
- SAN $\Rightarrow$ AVN via internodal pathways:
  - Anterior
  - Middle
  - Post

SAN
- $=$ normal pacemaker of heart
- lies in RA close to entry of SVC
- 2mm thick & 8mm long
- perfused with blood via sinus node artery
- 2 cell types:
  - small round cells (probably PMs)
  - longer elongated cells
- inherent d/c rate $\sim$ 100/min
- influenced by autonomic & humeral activity
- depression of activity $\Rightarrow$ next fastest pacemaker to take over $=$ escape rhythms

AVN
- at base of RA on R side of inter-atrial septum near opening of coronary sinus
- same types of cells of SAN but fever round cells
- conduction speed = 0.05m/sec
- has 3 zones:
  - AN zone = transitional zone between atria & node
  - N zone –cells here have long RRP so AV conduction slows with $\uparrow$atrial firing
  - NH zone = origin of His bundle
- Vagal & symp influence on the node apparent as explained previously
- AV nodal delay of 0.1s
allowing atria to finish filling ventricles before they contract
delay can be altered by autonomic system

Ventricles
• Impulses pass down R side of IV septum via Bundle of His
• After ~1cm His bundle splits:
  o R
  o L – splits into ant & post divisions
• His bundle has intrinsic rate & can take over if total AVN block
  ~30-40/min
• Bundle branches supply dense network of Purkinje fibres which innervate ventricles
• Purkinje cells:
  o largest cells in heart
  o fastest conduction velocity 1-4m/s
  o have long ARP – help block premature atrial impulses esp at slow HRs
• Depolarisation of ventricles
  o starts L side of septum and moves to R
  o then down to apex heart – so apex activated before bases
  o return along vent walls to AV groove
  o depolarises from inside to outside of vent walls:
    ▪ endocardial surfaces
    ▪ interventricular septum & papillary mms – prevents valve regurg & base for contraction
    ▪ epicardial surfaces – outside of RV activated 1st because of thinner walls
    ▪ last part to to depolarise = postero-basal LV
• conduction speed:
  o fastest purkinje system 4m/s
  o slowest SA & AV node 0.05m/s

Cardiac Arrhythmias

Classification
• According to site of origin:
  o Supraventricular vs ventricular
  o Narrow vs broad complex
• According to heart rate:
  o Tachy
  o brady

Cardiac Rate
• Normal sinus rhythm – originated in SA node
• Bradycardia –
  o Sleep
  o expiration
• Tachycardia:
  o Inspiration-
    ▪ Stretch receptors in lung ⇒ ↑parasymp inhibition of cardio-inhibitory area in medulla oblongata ⇒ ↑HR
  o Emotion
  o Exercise
  o Fever

Delayed After Polarisations
• Caused by inward current assoc with abnormally raised intracellular Ca
• Cause oscillations which ⇒ ectopic beats
Abnormal pacemakers
- Other parts of conduction system can become pacemakers
- SA node most rapid discharger of conduction system ⊂ normal pacemaker
- Complete heart block ⇒ idioventricular rhythm indep of atria
  - due to either:
    - AV nodal block – other AV node takes over ~45/min
    - infranodal block in His bundles – new pacemaker 15-35/min
  - Stokes Adams syndrome fainting & cerebral ischaemia
- 1st degree block
- 2nd degree block:
  - 2:1 mobitz
  - 2:2 Wenkebach
- RBBB
- LBBB - also see hemiblock in a fascicle:
  - Ant hemiblock ⇒ L axis deviation
  - Post hemiblock ⇒ R axis deviation
  - combinations – bifascicular or trifascicular block

Ectopic Foci of Excitation
- Can be encouraged by:
  - ↑ sympathetic activity
  - abnormal electrolytes
- Abnormal condition ± ⇒ spont d/c of His/Purkinje system or myocardium
  - ↑ ed automaticity of heart
- Extrasystole = Ectopic focus discharges ⇒ beat before next expected beat
- Paroxysmal tachycardia = repetitive d/c of ectopic focus at rate over SA node

ReEntry
- Common cause of paroxysmal tachyarrhythmia’s = defect in conduction permits circus movement
  - transient block on one side of conduction system ⇒ impulse down good side then back up diseased side ⇒ then repeat
  - in AV node re-entry:
    - retrograde flow back up diseased side ⇒ atrial depolarisation
    - next beat = echo beat
    - depolarisation then circus back down good side and continue
  - non-AV node re-entry:
    - abnormal extra bundle of connecting system connecting atria & vent
    - wave down AV node, retrograde through bundle of Kent
    - circus movements seen in atrium & vent

Atrial Arrhythmias
- atrial extrasystole:
  - ECG changes:
    - P wave abnormal
    - QRST normal
  - Extrasystole may depolarisation SA node ⇒ must repolarise before next normal beat
  - see pause which allows natural reset of rhythm
- Atrial tachycardia
  - Rate up to 220/min
By Adam Hollingworth

1. Electrical Heart

- when:
  - Regular atrial focus d/c
  - Re-entry circus tachy

- Atrial flutter:
  - Atrial rate 200-350
  - Anticlockwise circus in R atrium
  - Assoc 2:1 or greater Av block
    \[\text{as AV cannot conduct } >230\text{ beats/min}\]

- AF:
  - Atrial rate 300-500 irreg disorganised fashion
  - Vent rate 80-160
  - Cause unknown but include:
    - Multiple re-entrant circus excitation
    - Ectopic foci – often seen in pulmon veins ~4cm from heart

**Consequences Atrial Arrhythmias**
- If ⇒ high vent rate ⇒ ↓diastolic filling ⇒ heart failure
- Vagal stimulation effects:
  - Ach ⇒ ↓conduction in AV node & atrial myocardium
  - ↑AV block
- digoxin also blocks AV node

**Ventricular Arrhythmias**
- ectopic vent focus ⇒ extrasystole:
  - ECG:
    - abnormal QRS
    - P may be buried in QRS
  - unable to depolarise bundle of His \(\therefore\) no retrograde flow
  - no resetting of rhythm as in atrial ectopics
    \[\text{longer compensatory pause after ectopic}\]
    \[\text{SA node continues to fire regularly irrespective of vent activity}\]
  - common & usually benign
  - if early in diastole: may not be strong enough to create pulse at wrist
- Vent tachycardia (VT) =
  - Due to circus movement in ventricles
  - Torsade de pointes:
    - Form of VT with varying QRS morphology
  - Serious as ⇒ ↓CO
  - VF occasional complication of VT
- SVT with conduction block can be diff to differentiate with VT
  \[\text{need HBE – VT will not have a H spike}\]
- VF:
  - Muscle fibres contract in irreg and ineffective way
  - Due to multiple ectopic focus or circus movemt
  - Can be induced by defib during vulnerable period
    - Vulnerable period = midportion of T wave
      \[\text{when ventricular muscle at diff stages of de \\& repolarisation}\]
      \[\text{great time to start circus movmt}\]
  - most common cause of death in ACS is VF

**Long QT**
- long QT \(\approx\) ventricular repolarisation is irreg ⇒ ↑incidence of arrhythmia
- caused by:
  - ischaemia
  - drugs
Electrolyte abnormalities
- congenital – genes encoding for Na & K channels mutated

**Accelerated AV Conduction**

**WPW**
- additional aberrant connection between atria & vent:
  - types:
    - muscular or
    - nodal tissue ie bundle of Kent
  - conducts more rapidly than AV node ⇒ one vent excited before other
- ECG change:
  - short PR interval
  - upstroke slur
  - normal PJ interval (start P to end QRS)
- arrhythmias start:
  - following atrial extrasystole
  - beat goes 2 ways:
    - through AV node, retrograde through aberrant pathway back to atria
    - through aberrant pathway, retrograde through AV node (less common)
- congenital element:
  - mutation in AMP activated protein kinase
  - ?involved in suppressing development of abnormal pathways in utero

**Lown-Ganong-Levine Syndrome**
- short PR & normal QRS
- beat bypasses AV node by aberrant conduction pathway but then joins intraventricular conduction system before depolarising vents

**Myocardial Excitability vs Irritability**
- excitable =
  - ease with which myocardial cell can respond to stimulus by depolarization
  - index of excitability:
    - AP is initiated at different stages of the RRP
    - = the slope of phase 0 at this point
    - steeper slope = more excitable cell & faster velocity of conduction
  - hypokalaemia = hyperpolarised RMP (more –ve) but actually see ↑ ed excitability
  - hyperkalaemia = RMP moves towards zero. ∴ may see initially ↑ ed excitability. BUT in long term see ↓ excitability due to inactivation of Na channels. Significant ↑ K will eventually cause heart to stop in diastole.
- irritable =
  - used in context of a resting myocardial cell during phase 4
  - = size of stimulus needed to depolarize cell
  - index – difference between current potential & threshold potential
  - ↑ ed irritability = difference smaller ∴ depolarisation easier
    - but there is ↓ gradient & ↓ conduction velocity of phase 0
  - ↓ Ca ⇒ ↑ irritability ie = cause of tetany
ECG

- standard ECG =
  - 25mm/sec ⇒ 0.04sec/small sq, and 0.2 sec/big square
  - 1mV = 1cm

Einthoven’s triangle =
- heart at centre with 3 limb leads around
- triangle between shoulders & pubic symphysis
- electrodes record cardiac electrical activity in vertical plane
- 3 standard limb leads records electrical activity from 2 corners of triangle:
  - lead I = RA ⇒ LA
  - lead II = RA ⇒ LF (left foot)
  - lead III = LA ⇒ LF
    (4th electrode acts as an earth)

Depolarisation towards = upward deflection
Depolarisation away = downward
Repolarisation toward = downward
Repolarisation away = upward

P = atrial depolarisation
QRS = vent depolarisation & atrial repolarisation
T = vent repolarisation
QT = total duration of vent depolarisation & repolarisation
U = not always seen – repolarisation of papillary mms
J point = junction between QRS & ST segment

Lead 2 Example
- Lead 2 lies in axis of heart
- P wave: atrial depolarisation SAN ⇒ AVN which is down & Left (ie dep toward = upward)
- Small initial Q wave: depolarization starting in IV septum spreading down & right (ie dep away = downward)
- Large R wave = depolarisation spreads endo ⇒ epicardial & larger bulk of LV means net effect is down & left (ie dep toward = upward)
- Small S wave = activation of remaining ventricle, wave spreading upwards ie (dep away = downwards)
• T wave = ventricular repolarisation moving from epicardial to endocardial (ie repol away = upward)

ECG Intervals
• PR = 0.12-0.2 secs
  ↩ AV delay (0.1sec) accounts from most of delay
• QRS = <0.12
• QT = 0.3 0.43
  ↩ varies inversely with HR

ECG Pattern
• aVL & aVF look at ventricles ∴ mostly positive
• V1-V2:
  o No Q wave
  o Small initial R wave 2nd to L to R septal depolarisation
  o Large S wave – depolarisation down septum & into ventricle away from electrode
• V4-V6:
  o Small q = initial depolarisation across septum L to R
  o Large R – septal and vent depolarisation
  o Mod S – late depolarisation of vent moving back to atria

Cardiac Axis
• Mean QRS vector = -30 to +110
• L axis deviation if axis to left (up or <) of -30
• R axis deviation if axis to R (down or >) of +110

Unipolar Leads
• 3 electrodes of standard limbs leads are connected to each other
• use resistances of 5000 Ohms
• can create a central terminal with zero potential
  ↩ = common electrode
• exploring electrode can be combined with common electrode to create central terminal
• here potential difference between them = actual potential
• central = reference = zero

• 12 lead ECG 3 unipolar leads are recorded ie aVR, aVL, aVF
• a = augmented
• Goldberger modification = resistances removed & exploring electrode disconnected from central terminal ⇒ larger deflections
**Precordial Leads**

- Place electrodes closer to heart around thorax
- Neutral electrode formed by standard leads
- Exploring electrode placed at 6 different sites on chest wall (V1-6)

**His Bundle Electrogram**

- Catheter placed next to tricuspid valve:

  ![His bundle electrogram](image)

- A = AV node activated
- H = transmission through His bundle
- V = ventricular depolarisation
- With HBE & ECG can measure 3 intervals:
  - PA interval =
    - start of P to A wave
    - = conduction time from SA node to AV node
  - AH interval:
    - Start of A wave to start of H spike
    - =AV nodal conduction time
  - HV interval
    - Start H to start of QRS
    - = conduction in bundle of His & bundle branches
- AH time >double others ie AV node very slow to conduct
ECG Monitoring Systems in Anaesthetics

3 Electrode Systems
- ECG observed alone 1 bipolar lead between 2 electrodes
- 3rd electrode = ground
- selector switch allows you to change between electrodes
- simple system to monitor rate & rhythm
- limited info on myocardial ischaemia

5 Electrode System
- allows for recording of all standard 6 limb leads as well as one precordial lead

Modified 3 Lead System
- 3 lead system modified to give better results:
  - maximise P wave height for diagnosing arrhythmias
  - incr sensitivity to detect ant ischaemia
- several modifications exist
- one example = CS₅ lead:
  - bipolar lead best & easiest alt to true V5 lead for monitoring ischaemia
  - RA electrode placed under R clavicle
  - LA electrode placed in V5 spot
  - LL in usual spot for grounding
- Leads:
  - Lead 1 = detection ant ischaemia
  - Lead 2 = inferior ischaemia/arrhythmias

Surface Recordings Compared to Actual APs
Diseases Effecting ECGs

Myocardial infarction

Early Pattern
- 3 major changes - all cause ST elevation

1. Rapid Repolarisation – current out of infarct
- rapid repolarisation of infarcted muscle fibres
- due to faster opening K channels
- develops seconds after infarct
- lasts few minutes

2. Decreased Resting Membrane Potential – current in
- more K channels open ⇒ ↑ed K efflux ⇒ resting membrane potential lower
- causes TQ segment depression
- ↓on ECG looks like ST elevation

3. Delayed Depolarisation – current out
- 30min after infarct

Later Pattern
- hours – few days
- dead mm & scar tissue electrically silent
- infarcted area ↓: negative compared to norm myocardium during systole ⇒ path Q waves
- anterior L vent infarction ⇒ failure of progression of R wave
- septal infarction ⇒ BBB or other heart block

Late Established Pattern
- days to weeks
- ECG:
  - Q waves persist
  - ST segments isolectric
  - T inversion – where previous ST elevation; Tall Ts where prev ST depression
- May persist for rest of life

Very Late Pattern
- Months to years
- Path Q waves persist
- T waves gradually return to norm

Post MI Vent Arrhythmias
- timing:
  - 30mins post – re-entry arrhythmias common
  - 12hrs post – due to ↑ed automaticity
  - 3days post – due to reentry
- damage to epicardial regions interrupt sympathetic nerve fibres⇒
  - denervation super-sensitivity to catecholamines in area beyond infarct
- damage to endocardial regions interrupt vagal fibres ⇒ unopposed sympathetic action

Electrolyte Effects on ECG

Na
- ↓Na: low voltage ECG

Hyperkalaemia
- changes in sequence:
  - tall peaked T waves
  - paralysis of atria – loss of P waves
  - prolonged QRS
• resting membrane potential of myocardium ↓’s ⇒ ↓excitability of myocardium ⇒ heart stops in diastole

**Hypokalaemia**

• in sequence:
  o ST segment depression
  o U wave
  o PR prolongation
  o ±T inversion

• not as fatal as hyperkalaemia

**Hypercalcaemia**

• ↑ed myocardial contractility & ↓ed ability to relax
  ↓in vivo difficult to get plasma Ca levels high enough to effect heart

**Hypocalcaemia**

• prolongation of ST segment ∴ prolongation of QT
  ↓mimicked by drugs incl TCAs, phenothiazines