The ECG is a recording of the heart's electrical activity.

- **Analysing ECG allows us to:**
  - trace the conduction pathway through the heart
  - determine the size and orientation of the heart
  - locate injured or necrotic regions of the heart
There is some physiology you need to understand for this session:

The heart is capable of myogenicity / autonomic rhythmicity - normally a function of pacemaker tissues but if nec. any muscle cell or cell of the conducting pathways can generate an AP de novo

All muscle cells can conduct APs – although usually high speed specialised tissues are used

All cardiomyocytes are interconnected within the atria and again within the ventricles so when atrial mm cells are depolarised they will contract in unison & ditto for the ventricles.

Under normal circumstances the PM cells & conduction system orchestrate co-ordinated & effective contractions
Components of the intrinsic System:

- 1. Sinoatrial node
- 2. internodal pathway
- 3. atrioventricular node
- 4. AV tract or Bundle of His
- 5. Left bundle branch
- 5. Right Bundle branch
- Plus Purkinje fibres
The Conduction Pathway:

**Inherent Rates of Rhythmicity:**

- Normally established by the SAN under the influence of ............??

- However all cardiac muscle possesses automaticity
- This is important because ..................??

- If the SA node fails to depolarise the next fastest cells will take over & dictate HR
  - SAN 100+ /min
  - Atrial cells 60-100/min
  - The AV node 40-60 /min
  - Ventricular cells 20-40 /min.
What is happening to cardiac output as lower and lower order of ICS takes over the function of lead AP generator?

- AP generation & propagation at PM tissues, within the ICS & across muscle cell membranes
- Is due to:
  - the orderly and sequentially influx and efflux of ions
  - Namely Na+, K+, & Ca2+
- It is the sum of these ionic fluxes which are detected in the ECG at the skin surface

Electrophysiology:
## The 12-lead ECG:

- The next few slides on the 12 lead ECG are to aid your understanding of what you are looking at on an ECG print-out.
- You are **not** expected to remember all leads & all views.
- Some understanding will help you appreciate:
  - how we can look at different anatomical areas of the heart &
  - how an ECG trace can still be perfectly normal even if it looks very different from 2 different views

## The 12-lead ECG:

- Provides spatial information about the heart's electrical activity in 3 approximately orthogonal directions.

- The 12-leads are subdivided into:
  - 3 limb leads (I, II, III) (bipolar)
  - 3 augmented leads (aVR, aVL, aVF) (unipolar)
  - 6 chest leads (V1-V6)

**What is a lead????**
Electrode Sites

CHEST LEADS

Specific anatomical points
V1-V6
Precordial leads 1-6: horizontal plane

Frontal plane and horizontal plane leads
**LIMB LEADS**

- **Lead I** = RA to LA. Positive deflection
- **Lead II** = RA to LL. Very positive deflection
- **Lead III** = LA to LL. Tends to be bi-phasic

**AUGMENTED LEADS**

- Unipolar therefore ‘one fixed point’
- aVR = negative deflection
- aVL = positive or biphasic
- aVF = strongly positive
12-LEAD ECG
**The Normal ECG:**
- P-Wave – represents the impulse travelling across the atria to the AV Node (AVN)
- The QRS complex represents the impulse as it travels across the ventricles;
- T-Wave, representing the repolarization of the ventricles.

Ambulatory options are also available.
ECG Paper:
- 1 large square is 0.2 seconds
- Each small square is 0.04 seconds
- 5 large squares = 1 second
- 30 large squares = 6 secs
- 300 large squares = 1 minute

Refresh… What is? (Make sure you can answer this at all times)
- The P wave
- The PR interval
- QRS complex
- T wave

What does it all mean?
- The P wave – excitation across atria to AVN = 0.12 sec = 3 small squares max

- The PR interval – AP delayed at AVN = 0.12 – 0.2 secs = (5 small squares max)

- QRS complex – excitation across ventricles = 0.1 secs = 2.5 small squares

- T wave – repolarisation of the ventricles


Common Normal and Abnormal Rhythms:
Sinus Rhythms:

- Normal rhythms i.e. the AP originates at the SA node - travels through the entire conduction system without problem therefore **PQRST components are all present**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-100 bpm</td>
<td>Regular</td>
<td>Before each QRS, identical</td>
<td>.12 to .20</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>

When the sinus rhythm becomes *irregular* but all are components present - it is a **Sinus Arrhythmia**

**Sinus Arrhythmia**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually 60-100 bpm</td>
<td>Irregular</td>
<td>Before each QRS, identical</td>
<td>.12 to .20</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>
- **Sinus bradycardia (SB)**
  - Sinus rhythm slower than 60 BPM (adults)
  - Vagal stimulation e.g. trained athletes, the fit,
  - carotid sinus syndrome
  - Cerebral oedema (e.g. TBI)
  - Hypothyroidism
  - Damage to PM tissue 2nd to AMI or surgery
  - sympathetic blockade e.g. beta blockers.

- **Sinus tachycardia (ST)**
  - Sinus rhythm greater than 100 BPM (adults)
  - Pyrexia, ( \( \rightarrow \) ↑ excitation of the SA node).
  - Sympathetic stimulation (from a variety of causes)
  - Some drugs
Ventricular arrhythmias:

- Premature Ventricular Contractions (PVC’s).
- Occasionally a ventricular mm cell may initiate an AP → a contraction.
- A single occurrence probably not serious
- Causes inc. metabolic acidosis, hypoxaemia, infarction, electrolyte disturbances, caffeine & nicotine
- PVC's can be classified by their frequency
  - If each normal contraction is followed by single PVC = **bigeminy**.
  - If two normal contractions are followed by single PVC = **trigeminy**.

- The more frequent the PVC the more susceptible the person is to more serious ventricular arrhythmias such as Ventricular Tachycardia & Ventricular fibrillation

Which rhythm worries you most?
Has the worst prognosis?
PVCs are also classified by their origin

<table>
<thead>
<tr>
<th>Unifocal PVCs: identical shapes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Note: A single PVC is labeled isolated</td>
</tr>
</tbody>
</table>

| Multifocal PVCs: more than one shape |

Which trace worries you most / has the worst prognosis? Why

**Ventricular tachycardia:**

- 3 or more consecutive PVCs occur → ventricular tachycardia
- Ventricular rate > 100 bpm
- Extremely unstable rhythm
- Can occur in short burst < 30 secs & then rectify
- Or
- Be more sustained in which case likely to deteriorate to VF
Sinus tachycardia

Ventricular tachycardia

Ventricular Fibrillation:
- VF – erratic pattern of electrical activity in the ventricles – APs arise from multiple loci.
- No effective contraction = no cardiac out-put
- Unconscious, pulseless, BP-less i.e cardiac arrest within minutes
- Tx = immediate defibrillation, CPR & drugs e.g adrenaline into cardiac mm
Atrial Arrhythmias:

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>300-600</td>
<td>Extremely irregular</td>
<td>Absent</td>
<td>N/A</td>
<td>Fibrillatory baseline</td>
</tr>
</tbody>
</table>
Atrial Flutter

- Recognised by the distinct "saw tooth" pattern of p-waves.

AP arises **single atrial locus** (not SAN) & causes a wave of depolarisation which recurrently travels around the walls of an atria (often RA). Depolarising waves are periodically "thrown-off" to excite the LA

Re-entrant loop → flutter

Flutter is an unstable rhythm → supraventricular tachycardia & which can degenerate into AF

Flutter & AF can → intra-mural thrombus formation.
Atrial Fibrillation (AF or Afib)

• Erratic spontaneous depolarisation of multiple atrial myocardial hotspots.

• 1. lack of definitive p-waves
• 2. Irregularly occurring QRS

• Appear coarse when fewer than ~ 10 or so loci are simultaneously depolarising (hot-spots)

• Appear fine when > 10 & ? up to hundreds of fibres are simultaneously depolarising

Clinical significance of fine or coarse = not established
In AF/A-Flutter - ECG abnormality is seen in the P-wave
- Both AF & flutter result in tachycardia
- Cardiac output may be reduced – because ???
- Turbulent blood flow can → mural thrombi → necessitating anti-thrombolytic therapy
- Long-standing AF may be seen in pts with mitral valve disease and is common post C/T surgery & in any hypoxic conditions of heart or lungs

- Why is AF generally not life threatening unlike VF?

Why are ventricular depolarisation rates < atrial depolarisation rates

AVN has a conduction rate “ceiling” – safe-guards cardiac filling & therefore CO.

2. AVN capacity to conduct APs ↓ with ↑ age
3. AVN capacity may be impaired by AVN disease

4. refractory times of the individual cells of the AVN are variable & so occasionally extra APs can “sneak” through the AVN → ventricular depolarisation – hence irregularly occurring / spaced QRSs
Heart blocks:
or AV Blocks

A failure to conduct APs between atria & ventricles
May occur at the AVN, the bundle of His (AV tract) or bundle branches
AV blocks are defined by severity not location i.e 1st, 2nd or 3rd degree blocks

1st degree heart block:

Irregularity at the PR-Interval i.e - too long or irregular
All P-waves are followed by QRS complexes.
**Second Degree AV Block • Mobitz 1 (Wenckebach)**

<table>
<thead>
<tr>
<th>P Wave</th>
<th>PR Interval (in seconds)</th>
<th>QRS (in seconds)</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conducted intermittant</td>
<td>Increasingly Prolonged</td>
<td>&lt;.12</td>
<td>QRS dropped in a repeating pattern</td>
</tr>
</tbody>
</table>

**Third Degree (complete) AV Block**

<table>
<thead>
<tr>
<th>P Wave</th>
<th>PR Interval (in seconds)</th>
<th>QRS (in seconds)</th>
<th>Characteristic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal but not related to QRS</td>
<td>None</td>
<td>N/A</td>
<td>No relationship between P&amp;QRS</td>
</tr>
</tbody>
</table>
Junctional / Nodal Rhythms

Pacing

- Pacing spikes can be seen on ECGs e.g. 3rd degree heart block
- Pre P-wave is atrial pacing.
- Pre QRS-complex is ventricular pacing and
- Both is Dual Chamber Pacing.
Dual-lead pacemakers - both the atrium and the ventricle are paced. Here the atrium is paced (even though the p wave never fully develops) and then the ventricle is paced. Best visualized in lead II.

**Single-Lead Pacemaker:** Only the ventricle is paced – most commonly used in AF where an additional electrical impulse to the atrium would be ineffective. In the above e.g the pt has sinus bradycardia rather than AF. Best visualised in II
ST segment depression: suggests myocardial ischaemia
We have looked at the basic principles underpinning ECGs.

We have looked at the commonest dysrhythmias – one’s which you will regularly come across.

Next session you will use a systematic approach to analyse some common ECG traces.

You may/ will be required to identify an ECG abnormality in your exam – it will be one that we’ve looked at in T2 / 3.
ECG Interpretation made Incredibly Easy!
Includes 56 self –test strips with answers.
Lippincott, Williams & Wilkins – in library.

There are also a number of ECG Made easy texts in library.