Cardiac conduction system

- Sinoatrial (SA) node:
  Located in the right atrium
  Heart’s main pacemaker
  Initiates 60-100 beats/minute

- Internodal tracts and Bachmann’s bundle
  In the R atrium, the impulse travel along three internodal tracts
  In the L atrium, the impulse travel via Bachmann’s bundle
- **Atrioventricular (AV) node**
  Positioned in the R atrium
  Doesn’t possess pacemaker cell, but the junctional tissue around it does
  Conducts atrial impulse to the ventricles with 0.04 sec delay (filling time of ventricles)

- **Bundle of His**
  Divides into R & L bundle branches
  The L bundle branch splits into two branches or fascicles
  Impulse travel faster down the left bundle branch
  Pacemaker site, firing rate 40-60 beats/minute
- **Purkinje fibers**

  Transmit impulses quicker than any other part of the conduction system

  Usually fires when SA, AV nodes fail or when normal impulse is blocked in both bundle branches

  Firing rate 15-40 beats/minute

- **Accessory pathways**

  Plays role in some arrhythmias (i.e. Bundle of Kent)
Electrocardiography

- Heart electrical activity produces currents radiates to the surroundings (skin)
- Skin leads
- Corresponds to the heart’s depolarization and repolarization
- The resulting graph is called Electrocardiogram (ECG)
ECG uses

- Identify rhythm disturbances, conduction abnormalities and electrolytes imbalances
- Contributes information about the size of the heart chambers and relative position of the heart in the chest
- Diagnosis and progression of MI, ischaemia and pericarditis
- Monitoring recovering from MI
- Monitoring drug effects
- Evaluates the function of artificial pacemakers
Types of ECGs

- **Resting**: primary conduction disorders, arrhythmias, cardiac hypertrophy, pericarditis, electrolyte imbalances, site and extent of MI, recovery from MI, evaluate pacemaker performance and the effect of cardiac glycosides and antiarrhythmics.

- **Exercise**: heart functional capacity and the origin of chest pain, screen for asymptomatic CAD, arrhythmias that develop during exercise, effect of antiarrhythmics and antianginal, changes in CV function after exercise.

- **Ambulatory**: cardiac arrhythmias, effect of antiarrhythmic drugs, evaluate chest pain and cardiac status after MI or pacemaker implantation, assessment of SOB, syncope, light-headedness and palpitations and evaluation of ST-segment changes in ischemia.
Leads & planes

- A lead provides a particular view of the heart electrical activity.
- When current flows towards the positive pole, the waveform deflects upwards - positive deflection.
- Away from the positive pole, downwards - negative deflection.
- Absent electrical activity, straight line - isoelectric deflection.
Leads & planes

Standard 12-lead ECG

- **Limb leads** (I, II, III, aVR, aVL, aVF): frontal plane (bipolar and unipolar)
- **Precordial (chest) leads** (V1 to V6): unipolar (from the centre of heart to electrode)
Leads & planes

Single lead ECG

- Rhythm strip
- Continuous information about the heart’s electrical activity
- Three bipolar leads (I, II, III) and two others (MCL1, MCL6)…modified versions of V1 and V6
Leads I, II, III

- Standard limb leads or bipolar limb leads
- R arm, L arm, L leg and R leg (stabilize ECG tracing)
- Lead I: records electrical activity between R and L arm
- Lead II: R arm and L leg
- Lead III: L arm and L leg
Einthoven’s triangle

- The sum of the electrical potentials at any specific moment as recorded in leads I and III equals the electrical potential recorded in lead II (error in electrode placement)
Leads $aV_R$, $aV_L$, $aV_F$

- Augmented unipolar leads
- Same electrode locations as (I, II, III)
- No need for R leg electrode
- Measure electrical potential between the limbs and the centre of the heart (neutral)
  - $aVR$: right shoulder
  - $aVL$: left shoulder
  - $aVF$: left foot
Leads $V_1$ to $V_6$

- $V_1$: 4\textsuperscript{th} IS, R sternal border
- $V_2$: 4\textsuperscript{th} IS, L sternal border
- $V_3$: midway between $V_2$ and $V_4$
- $V_4$: 5\textsuperscript{th} IS in L midclavicular line
- $V_5$: 5\textsuperscript{th} IS in L anterior axillary line
- $V_6$: 5\textsuperscript{th} IS in L mid axillary line
- $V_1$, $V_2$ (R precordial leads); $V_3$, $V_4$ (mid precordial); $V_5$, $V_6$ (L precordial leads)
Electrodes and heart walls

- Leads I, II and VL: lateral surface of the heart
- Leads III and VF: inferior surface of the heart
- VR: right atrium
- V1- V2: right ventricle
- V3-V4: septum & anterior left ventricle
- V5-V6: anterior & lateral left ventricle
Graph paper

- Horizontal axis: length of particular event and duration (small block 0.04 sec, large block 0.2 sec)
- Vertical axis: electrical voltage (amplitude) in millivolts (small block 0.1 mV, large block 0.5 mV)
ECG waveform

- Wave forms: P wave, QRS complex, T wave
- Segments and intervals: PR interval, ST segment, QT interval
- U wave: may appear sometimes
- J point: marks the end of the QRS complex and the beginning of ST segment
P wave

- Atrial depolarization
- Originates in SA node, atria or AV junctional tissue
- P wave normal (originates in the SA node)

- If a P wave precedes each QRS, the impulse are being conducted from the atria to the ventricles
P wave - characteristics

- Location: precedes the QRS complex
- Amplitude: not more than 0.25mV (< 2.5 small squares)
- Duration: 0.06 to 0.11sec (< 3 small squares)
- Configuration: usually rounded and upright
- Deflection: +ve leads I, II, aVF, V2-6; -ve in lead aVR; biphasic lead V1; variable lead III, aVL
P wave - variations

- Peaked: R atrial abnormality
- Broad, notched: L atrial abnormality
- Inverted: SA node isn’t the pacemaker (junctional arrhythmias)-except in aVR
- Varying (shapes and sizes vary): pulses originating at various sites
- Missing: third degree AV block, sinus exit block, atrial flutter or AF
PR interval

- Beginning of atrial depolarization to the beginning of ventricular depolarization
- Time taken from SA node-atria-AV node to the bundle branches
- Any variation in the PR interval suggests a conduction delay (AV block)
PR interval-characteristics

- **Location**: extends from the beginning of P to the beginning of QRS
- **Amplitude**:
- **Duration**: 0.12 to 0.2 sec (< 1 big square)
- **Configuration**:
- **Deflection**:
PR interval- variations

- Short: impulse is originating in an area other than SA node (junctional arrhythmias)
- Prolonged: impulse is delayed as it passes through the AV node (1\textsuperscript{st} degree or 2\textsuperscript{nd} degree AV block)
- Depressed: pericarditis
QRS complex

- Ventricular depolarization

- If the P wave doesn’t appear before the QRS complex, then the impulse probably originated in the ventricles (ventricular arrhythmia)

- Q wave: Any initial negative deflection

- R wave: Any positive deflection

- S wave: Any negative deflection after an R wave
Depolarization is moving towards the dead
The transition point

- Normal position: leads V3/V4
- Can detect hypertrophy (RV hypertrophy, the transition point will be at leads V4/V5 or V5/V6)... “clockwise rotation of the heart”
QRS complex - characteristics

- Location: follows the PR interval
- Amplitude: differs for the 12 leads
- Duration: 0.06 to 0.10sec (< 3 small squares)
- Configuration: 3 waves (Q: -ve, R: +ve S: -ve)
- Deflection: +ve (complex above the baseline); -ve (complex below the baseline); biphasic (complex above and below the baseline)

+ve: I, II, III, aVL, aVF, V4-6
-ve: aVR, V1-2
Biphasic: V3
QRS complex - variations

- Widened: bundle branch block, premature ventricular contractions, ventricular tachycardia
- Configuration variation: bundle branch block, WPW syndrome
- Varying complexes: ectopic impulse
- Missing: AV block, ventricular standstill
**ST segment**

- End of ventricular depolarization and the beginning of ventricular repolarization

- The point that marks the end of the QRS complex and the beginning of the ST segment is known as the *J point*

- A change in ST segment may indicate myocardial damage
**ST segment - characteristics**

- **Location:** from the end of S wave to the beginning of T wave
- **Amplitude:**
- **Duration:**
- **Configuration:**
- **Deflection:** usually isoelectric, if elevated no more than 0.1mV
ST segment-variations

- Elevation: myocardial injury
- Depression: myocardial injury or ischemia
- Changes: pericarditis, myocarditis, L ventricular hypertrophy, pulmonary embolism, electrolyte disturbances and antiarrhythmic (amiodarone)
T wave

- Ventricular repolarization
**T wave-characteristics**

- **Location:** follows S wave
- **Amplitude:** 0.5mV or less in I, II, III; 0.1 or less in V1-6
- **Duration:**
- **Configuration:** typically rounded and smooth
- **Deflection:** +ve leads I, II, V3-6; -ve in lead aVR; variable lead III, aVL, aVF, V1-2
T wave - variations

- Inverted: in I, II, V3-6 may indicate myocardial ischemia
- Peaked: hyperkalemia, myocardial ischemia
- Heavily notched: children (normal), adults (pericarditis)
- Large or small: electrolyte imbalance
- With bumps: (P wave hidden within T wave) impulse originated above the ventricle
**QT interval**

- The time needed for the ventricular depolarization-repolarization cycle

- Abnormal duration may indicate myocardial irregularity

- Shouldn’t be greater than half the distance between consecutive R wave (R-R interval) when the rhythm is regular
QT interval - characteristics

- **Location:** extends from the beginning of the QRS complex to the end of the T wave
- **Amplitude:**
- **Duration:** varies according to age, sex, and heart rate, usually (0.36-0.44 sec)
- **Configuration:**
- **Deflection:**
QT interval - variations

- Prolonged: antiarrhythmics, myocardial ischemia, MI, life threatening ventricular arrhythmia
- Shortened: hypercalcemia, digoxin toxicity
U wave

- Theory: repolarization of the His-Purkinje fibres
U wave - characteristics

- Location: follow T wave
- Amplitude:
- Duration:
- Configuration: typically rounded and upright
- Deflection: upright
U wave - variations

- Prominent: hypokalemia, healthy people
- Inverted: heart disease
Applying your knowledge

- Rate
- Rhythm
- Cardiac axis
- Abnormalities
Heart Rate

- Locate the QRS complex that is closest to a dark vertical line
- Count either forward or backwards to the next QRS complex
- 300-150-100-75-60-50-43
- 2 lines means HR=150 beat/minute
Heart Rhythm

- **Source and its Regularity**
- Source of the rhythm is the SA node or an ectopic pacemaker
- Relationship of the P-wave to the QRS-complex
- P wave before each QRS and the P is in the same direction (sinus).
Heart Axis

- Sum of the vectors
- The same direction (down-left) for a normal heart: SA node (top right) to the purkinje fibers (bottom left)
- I and AVF = +ve = normal axis
The normal range for the cardiac axis is between -30° and 90°
**R axis deviation:** long thin individuals, RV hypertrophy, pulmonary conditions (PE), congenital heart disease, conduction defect

**L axis deviation:** short fat individuals, LV hypertrophy, conduction defect

<table>
<thead>
<tr>
<th></th>
<th>Normal axis</th>
<th>Right axis deviation</th>
<th>Left axis deviation</th>
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</thead>
<tbody>
<tr>
<td>Lead I</td>
<td>Positive</td>
<td>Negative</td>
<td>Positive</td>
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<tr>
<td>Lead II</td>
<td>Positive</td>
<td>Positive or negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Lead III</td>
<td>Positive or negative</td>
<td>Positive</td>
<td>Negative</td>
</tr>
</tbody>
</table>
The 3 pathways of the depolarization wave

Normal axis: effect of normal conduction
L anterior fascicular block

L posterior fascicular block
RBBB

RBBB+L ant hemi block
Normal axis or Slight L axis deviation
Conduction and its problems
SA node block

- Seen as a complete pause of 1 beat - "skipped beat"
- Can occur occasionally in normal patients
AV nodal blocks (heart block)

- **AV Node Block** is a block which delays the electrical impulse as it travels between the atria and the ventricles in the AV node
- **1º AV node block:**
  - One P wave per QRS complex
  - PR interval > 1 large box
2ºAV Block is when sometimes excitation fails to pass through the AV node or the bundle of His

Mobitz type II:
Constant PR interval
One P wave is not followed by QRS complex

Wenckebach type:
Progressive lengthening of the PR interval
One non-conductive P wave
Next conductive P wave has a shorter PR interval
- Second degree heart block (2:1 type)
  Two P waves per QRS complex
  Normal and constant PR interval
- Second degree heart block (3:1 type)
- **3° Block** is a **complete** block of signals from the atria to the ventricles

P waves 90/min, QRS complex 36/min

No relationship between P & QRS (lack of synchronization)

Abnormal shape of QRS (abnormal spread of depolarization)

- **Morphology (QRS complex):** narrow complex (origin is likely to be nodal), wide complex (ventricular)
Bundle branch block

- The key to recognizing a bundle block is to find a $\textbf{R-S-R'}$ pattern.
- The criteria consist of a QRS wider than 0.12 seconds and the 2 R waves.
- In a left bundle block, the left ventricular firing is delayed, while in right bundle block, the right ventricular firing is late.
RBBB

- Remember to consider what precordial lead the block is presenting in
- V1-V2: RBBB, V5-V6: LBBB
- The axis will be hard to accurately determine
Diagnostic criteria RBBB:

- QRS duration >0.12 s
- RSR in V1-V2
- Wide slurred S wave in leads I, V5, and V6
LBBB

- Note the R-S-R' pattern repeating as well as the QRS complex being wider than (0.12sec).
- In the LBBB, the R wave is the right ventricle and the R' is the left ventricle
- “W” pattern in V1 & “M” pattern in V6
Diagnostic criteria LBBB:

- QRS duration of $>0.12$ s
- “W” pattern in V1 & “M” pattern in V6
- Absence of Q waves in leads V5 and V6
Fascicular (hemi) blocks

- L ant fascicular block (L ant hemiblock): L axis deviation
- L post fascicular block (L post hemiblock): R axis deviation
- RBBB: normal cardiac axis
- Bifascicular block (RBBB+L ant hemiblock): L axis deviation
- Bifascicular block (RBBB+L post hemiblock): R axis deviation
Fascicular blocks

- RBBB with L ant hemiblock is the commonest type of bifascicular block.
- The left posterior fascicle is fairly stout and more resistant to damage.
- Trifascicular block: bifascicular block + first degree heart block
- RBBB+ L ant hemiblock + L post hemiblock = complete heart block (as if the His bundle is damaged)
Trifascicular block:
right bundle branch block
left anterior hemiblock
first degree heart block
Useful information

- First degree block: normal people, acute MI, rheumatic fever
- Second degree block: acute MI; Mobitz type II & Wenckebach type require no treatment, but 2:1 type block may need temporary or permanent pacemaker
- Third degree block: fibrosis more than ischemia; temporary or permanent pacemaker
Useful information

- **RBBB**: ASD
- **LBBB**: aortic stenosis, ischemia, acute MI
- L axis deviation: LV hypertrophy
- L axis deviation + RBBB: severe conducting tissue disease; may need pacemaker
The rhythm of the heart
Sinus bradycardia

- Unusually slow heartbeat
Sinus Tachycardia

- Increased in demand for cardiac output, which is successfully met by the heart and whose rhythm originates in the SA node.
- Remember that if the rate is high enough, the P-wave can be obscured in the ST-segment, but is still present.
Sinus arrhythmia

- The normal increase in heart rate that occurs during inspiration
- Sinus arrhythmia is generally a good thing
- Absence of any sinus arrhythmia suggests an autonomic neuropathy
Tachycardia

- The source of a tachycardia, if ventricular, is always pathologic, while non-ventricular tachycardias can be thought of as "sinus" and supraventricular (SVT) rhythms, and may or may not be pathologic.
- The most common cause of pathologic tachycardias and arrhythmias results from reentry.
Tachycardia-reentry

This is due to a signal splitting around a defect and one side of that split being conducted significantly slower than the other. If the slow signal meets the fast side ready to be depolarized it can cause both normal and retrograde depolarization.
Paroxysmal Atrial Tachycardia

- This arrhythmia is seen with reentry in the atria
- Causing reentry to give a rapid tachycardia
- There is a single ectopic pacemaker, which can even be the AV node itself
- Inverted P-waves (source being lower down in the atria)
- Can be differentiated from sinus tachycardia by vagal maneuvers (if the rhythm slows and then resumes after cessation of the maneuvers, sinus rhythm is present. If the rhythm terminates abruptly or there's no change, then it's PAT).
SVT- Multifocal Atrial Tachycardia

- Ectopic pacemaker somewhere in the atria
- This causes there to be 2 or more asynchronous pacemakers for the heart
- The hallmark of this form of SVT is the 2 or more P-wave morphologies you see (one P-wave from each pacemaker)
- Each of the pacemakers is not tachycardia, it is the sum of their rates that produces the tachycardia
Atrial extrasystoles

- These arise from ectopic atrial foci
  Commonly, the ectopic beat always arises at about the same time after the sinus beat

- Distinguish between an atrial extrasystole, and an atrial *escape* beat, where the SA node falters, and a subsidiary pacemaker takes over
Regular SVT - Atrial flutter

- Undulating saw-toothed baseline F (flutter) waves
- Atrial rate 250-350 beats/min
- Regular ventricular rhythm
- Ventricular rate typically 150 beats/min (with 2:1 atrioventricular block)
- 4:1 is also common (3:1 and 1:1 uncommon)
Irregular SVT-Atrial fibrillation

- P waves absent; oscillating baseline f (fibrillation) waves
- Atrial rate 350-600 beats/min
- Irregular ventricular rhythm
- Ventricular rate 100-180 beats/min
Ventricular extrasystoles

- The QRS complex is wide, bizarre, and unrelated to a preceding P wave.
- There is usually a constant relationship (timing) between the preceding sinus beat and a subsequent ventricular beat, because the preceding beat influences the ectopic focus.
Ventricular extrasystoles

- Because the intrinsic rate of an ectopic focus often tends to be slow-ish, extrasystoles will tend to arise more commonly with slower rates.

- In addition, if the rate is varying, extrasystoles will tend to 'squeeze in' during long RR intervals. Some have called this the "rule of bigeminy".
Ventricular Tachycardia

- When there is ischemic, infarcted or necrotic conductive tissue around the bundles, there can be a reentry of the downward propagating depolarization, that causes the signal to repeat itself.
- This causes the **Ventricular Tachycardia**; this can be a life threatening condition.
- The hallmark of ventricular rhythms is the **wide QRS complex**.
Ventricular flutter

- Ventricular 'flutter' is a bizarre sine-wave like rhythm, and usually degenerates into ventricular fibrillation
Ventricular Fibrillation

- complete breakdown in the synchronization of the myocardial conduction system; ranging from course (large amplitude) to fine (close to asystole) in amplitude. The only "cure" for V-fib is electrical cardioversion (defibrillation).
Abnormalities of:
P waves
QRS complexes
T waves
Atrial hypertrophy

- V1, which is mostly over the right atrium,
- tall P wave (3 blocks or more) signifies right atrial enlargement; widened bifid one, left atrial enlargement
- P-wave can become biphasic in bilateral atrial hypertrophy
Large P waves in leads II, III, and aVF (P pulmonale)
Biphasic P wave in V1. The large negative deflection indicates left atrial abnormality.

P mitrale in lead II
It is commonly seen in association with mitral valve disease, particularly mitral stenosis.
Ventricular hypertrophy

- Large S wave in V1 and a large R wave in V5
- The actual criteria are to add the height of S in V1 and the height of R in V5 (in mm) and if the sum is greater than 35mm, then LVH is probable.
Left ventricular hypertrophy in patient who had presented with chest pain and was given thrombolytic therapy inappropriately because of the ST segment changes in V1 and V2.
Ventricular hypertrophy

- V1 (and less so in V2 and V3) and notice that there is a large R-wave (the normal V1 has a small R with a large S)
Right ventricular hypertrophy secondary to pulmonary stenosis (note the dominant R wave in lead V1, presence of right atrial hypertrophy, right axis deviation, and T wave inversion in leads V1 to V3)
Ischemia, Infarct and Injury

- the QRS is not the most affected part of the EKG waveform in ventricular ischemia
- ST segment is most often affected in ischemic conditions
- **Transmural Ischemia:** elevation of the ST segment (*unstable angina, acute MI*)
- As the ischemia becomes more extensive the ST segment elevation becomes more pronounced
Ischemia, Infarct and Injury

- The lead in which the ST elevation appears, allows you to accurately locate the ischemic or infarcted area of ventricular myocardium. If the elevation appears in inferior leads, this indicates an inferior ischemic/infarcted myocardium; the lateral leads, likewise indicate lateral wall ischemia/infarction
Ischemia, Infarct and Injury

- It is important to differentiate pathologic ST segment elevation from non-pathologic J-point elevation. J-point elevation is identified by an elevation of the terminal portion of the QRS which then dips back down towards the baseline before rising back up to the ST segment. This is opposed to the pathological ST-elevation which is visualized as the terminal portion of the QRS going directly up to the T-wave.
Ischemia, Infarct and Injury

- After the ischemia has progressed to an infarct, and the tissue has scarred, the ECG will show an inverted T wave.
- A pronounced Q-wave (not normally present) and loss of all or part of the R wave may also be present.
Ischemia, Infarct and Injury

- **Sub-Endocardial Ischemia:** ST-segment depression (stable-angina)

- Unlike elevation, the ST-segment depression is not localizable to a specific lead, but is seen in the leads with the tallest R waves, which are the inferior leads (II, III and AVF and leads V4-V6). Typically, stable angina will self-resolve, however, like elevation, the depression is increased as the myocardial demand increases.
Prinzmetal's angina

- The simple (and possibly even correct) explanation of why you see ST segment elevation with this variant form of angina is that the predominant area of ischaemia is epicardial. This disorder is thought to be related to vascular spasm, and angiography shows coronaries without a significant burden of atheroma. Many other morphological abnormalities have been described with this disorder.
Hyperkalemia

- The most prominent feature of an ECG of a hyperkalemic patient is the peaked-T wave.
- The other feature of the hyperkalemic EKG is a stretching of entire waveform.
Serial changes in hyperkalaemia

- 5.5-6.5 Tall peaked T waves
- 6.5-7.5 Loss of P waves
- 7.0-8.0 Widening of QRS complexes
- 8.0-10 Sine wave, ventricular arrhythmias, asystole

ECG in hyperkalaemia

Broad complex tachycardia with a potassium concentration of 8.4 mmol/l (A); after treatment, narrower complexes with peaked T waves (B)
Hypokalaemia

- The T waves flatten, U waves become prominent (this may be falsely interpreted as QT prolongation), and there may even be first or second degree AV block.
**ECG in hypokalaemia**

- Broad, flat T waves
- ST depression
- QT interval prolongation
- Ventricular arrhythmias (premature ventricular contractions, torsades de pointes, ventricular tachycardia, ventricular fibrillation)

Electrocardiogram showing prominent U wave, potassium concentration 2.5 mmol/l (A) and massive U waves with ST depression and flat T waves, potassium concentration 1.6 mmol/l (B)
Overdoses of digitalis (Digoxin or Digitoxin) can have effects ranging from mild 1:1 AV block to junctional rhythms through fatal arrhythmias.

The most noticeable change to the ECG is the "swooping" ST-segment depression, extended PR intervals or 1:1 block, although this alone is indistinguishable from primary 1:1 AV Block.
Others
Wolff-Parkinson-White syndrome

- PR interval under 0.12s
- A *delta* wave
- QRS duration of 0.12s (or more)
- A normal P-wave axis
Acute pulmonary embolism

- Patients who present with a small pulmonary embolus are likely to have a normal electrocardiogram or a trace showing only sinus tachycardia.

- If the embolus is large and associated with pulmonary artery obstruction, acute right ventricular dilatation may occur. This may produce an S wave in lead I and a Q wave in lead III. T wave inversion in lead III may also be present.

   S1, Q3, T3
Sinus tachycardia and S1, Q3, T3 pattern in patient with pulmonary embolus
COPD ECG

P pulmonale
low amplitude QRS complexes
poor R wave progression
Left ventricular hypertrophy
Left atrial enlargement
Abnormal inferior and anterior and/or lateral Q waves
Bizarre QRS complexes masquerading, for example, as pre-excitation and bundle branch block

Hypertrophic cardiomyopathy
ECG
Dilated cardiomyopathy ECG

Left bundle branch block
Left atrial enlargement
Abnormal Q waves in leads V1 to V4
Left ventricular hypertrophy
Arrhythmias—ventricular premature beats, ventricular tachycardia, atrial fibrillation
Restrictive cardiomyopathy

ECG

Low voltage QRS complexes
Conduction disturbance
Arrhythmias—supraventricular, ventricular
Tremor artefact from shivering
Atrial fibrillation with slow ventricular rate
J waves (Osborn waves)
Bradycardias, especially junctional
Prolongation of PR, QRS, and QT intervals
Premature ventricular beats, ventricular tachycardia, or ventricular fibrillation
Asystole

Hypothermia ECG
**Most common**
Sinus tachycardia
Increased QRS voltages
Atrial fibrillation

**Less common**
Supraventricular arrhythmias
(premature atrial beats, paroxysmal supraventricular tachycardia, multifocal atrial tachycardia, atrial flutter)
Non-specific ST and T wave changes
Ventricular extrasystoles

Thyrotoxicosis (ECG)
Most common
Sinus bradycardia
Prolonged QT interval
Flat or inverted T waves

Less common
Heart block
Low QRS voltages
Intraventricular conduction defects
Ventricular extrasystoles

Hypothyroidism (ECG)
Simple test
2:1 AV block
Acute anterior myocardial infarction
Acute inferior myocardial infarction
Acute myocardial infarction in the presence of left bundle branch block
Acute posterior myocardial infarction
Acute pulmonary embolus
Atrial fibrillation and complete heart block
Atrial fibrillation with pre-existing left bundle branch block
Atrial fibrillation with rapid ventricular response
Atrial flutter
Atrial flutter with 2:1 AV conduction
Atrial Premature Beat (APB)
Complete Heart Block
Hyperkalaemia
Implantable cardioverter defibrillator
Left anterior hemiblock & L atrial & ventricular hypertrophy
Left atrial & ventricular hypertrophy
Mitral Stenosis
Normal
Old inferior myocardial infarction
Pericardial effusion with electrical alternans
Right atrial hypertrophy
Right Bundle Branch Block
Sinus bradycardia
Sinus tachycardia
'Trifasicular' block
Ventricular bigeminy
Ventricular fibrillation
Ventricular pacemaker
Ventricular tachycardia
Ventricular tachycardia
Wolf-Parkinson-White syndrome with atrial fibrillation
Hypokalaemia
Digitalis effect
I hope you can read ECG now

Thank you