ELECTRICAL ACTIVITY OF THE HEART

INTRODUCTION:

- Contraction of the atria (ATRIAL SYSTOLE) is followed by contraction of the ventricles (VENTRICULAR SYSTOLE)
- During DIASTOLE, all four chambers are RELAXED
- The heartbeat originates in a specialised CARDIAC CONDUCTION SYSTEM and spreads via this system to all areas of the myocardium
- Structures that make up the conductions system include:
    - SINOATRIAL NODE
    - INTERNODAL ATRIAL PATHWAYS
    - THE ATRIOVENTRICULAR NODE
    - THE BUNDLE OF HIS
    - THE PURKINJE SYSTME
- The various parts of the conduction systems (and, under abnormal conditions, the myocardium) are capable of SPONTANEOUS DISCHARGE
- The SA node is the normal CARDIAC PACEMAKER, its rate of discharge determining the rate at which the heart beats

ORIGIN AND SPREAD OF CARDIAC EXCITATION:

ANATOMIC CONSIDERATIONS:

- SA node is located at the junction of the SVC with the RA
- The AV node is located in the right posterior portion of the interatrial septum
- There are **three** bundles of atrial fibres that connect the SA node to the AV node
    - Conduction occurs through atrial myocytes, but it is more rapid in these bundles
- The AV node is normally the only conducting pathway between the atria and ventricles
    - It is continuous with the bundle of His
- Bundle of His gives off a left bundle at the top of the interventricular septum and continues as the right bundle branch
- The left bundle branch divides into an anterior fascicle and a posterior fascicle
    - The branches and fascicle run subendocardially and then come into contact with the Purkinje system
- The SA node develops from structures on the right side of the embryo and the AV node from structures on the left
    - **This is why in the adult the right vagus is distributed mainly to the SA node and the left vagus mainly to the AV node**
- On each side, most sympathetic fibres come from the **stellate ganglion**

PROPERTIES OF CARDIAC MUSCLE:

- Depolarisation spreads RADIALLY through cardiac myocytes as if they were a SYNCYTIUM, because of the presence of GAP JUNCTIONS
- The transmembrane action potential of single cardiac muscle cells is characterised by:
    - RAPID DEPOLARISATION
    - A PLATEAU
    - SLOW REPOLARISATION PROCESS
- The initial depolarisation is due to Na influx through rapidly opening Na channel
- Ca influx through more slowly opening Ca channels produces the plateau phase
- Repolarisation is due to net K efflux through multiple tups of K channels

PACEMAKER POTENTIALS:

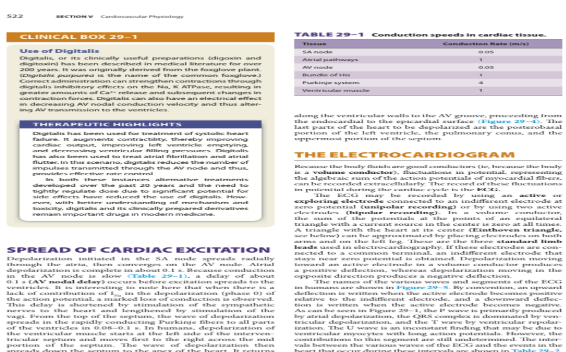
- Rhythmically discharging cells have a membrane potential that, after each impulse, declines to the firing level
    - This PREPOTENTIAL OR PACEMAKER POTENTIAL triggers the next impulse
- At the peak of each impulse, K efflux begins and brings about repolarisation
    - IK then declines and as K efflux decreases, the membrane begins to depolarise, forming the first part of the prepotential
    - **I_{funny} Na channels are responsible for the first part of the pacemaker potential**
    - Ca channels then open and these are of TWO TYPES:
        - T (TRANSIENT) – **end of the pacemaker potential**
        - L (LONG-LASTING) CHANNELS - **upstroke**
            - The calcium current due to opening of T channels completes the prepotential and the calcium current due to opening of L channels produces the impulse
            - The action potentials in the SA and AV nodes are largely due to Ca, with no contribution by Na influx
                - Consequently there is no sharp, rapid depolarising spike before the plateau
- When the cholinergic vagal fibres to nodal tissue are stimulate, the MEMBRANE BECOMES HYPERPOLARISED and the slope of the pacemaker potential is DECREASED
    - This is because acetylcholine increases the K conductance of nodal tissue
        - Mediated by M2 receptors (Gi)
        - Results is a decrease in the firing rate
- Conversely, stimulation of the sympathetic cardiac nerves makes the membrane potential fall more rapidly
    - Noradrenaline binds to β1 receptors (Gs)
        - Resulting increase in intracellular CAMP facilitates the opening of L channels, increasing the Ca current and the rapidity of depolarisation

- Sympathetic stimulation doesn't change the baseline degree of polarisation of the SAN.

- Rate of SA node discharge is influenced by TEMPERATURE AND BY DRUGS

SPREAD OF CARDIAC EXCITATION:

- Depolarisation initiated in the SA nodes spreads radially through the atria then converges on the AC node
- Because conduction in the AV node is SLOW, a **delay of about 0.1s** (AV NODAL DELAY) occurs before excitation spreads to the ventricles
- This delay is shortened by stimulation of the sympathetic nerves to the heart and lengthened by stimulation of the VAGI
- From the top of the septum, the wave of depolarisation spreads in the rapidly conducting Purkinje fibres to all parts of the ventricles in 0.08-0.1s
- In humans, depolarisation of the ventricular muscle starts at the left side of the interventricular septum and moves FIRST TO THE RIGHT and then down the septum to the apex of the hear
- I.e. septum depolarises L → R
- It returns along the ventricular walls to the AV groove, proceeding from the endocardial to the epicardial surface



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CLINICAL BOX 29–1

Use of Digitalis

Digitalis, or its clinically useful preparations (digoxin and digitoxin) has been described in medical literature for over 200 years. It was originally derived from the foxglove plant. (*Digitalis purpurea* is the name of the common foxglove.) Correct administration can strengthen contractions through digitalis inhibitory effects on the Na, K ATPase, resulting in greater amounts of Ca^{2+} release and subsequent changes in contraction forces. Digitalis can also have an electrical effect in decreasing AV nodal conduction velocity and thus altering AV transmission to the ventricles.

THERAPEUTIC HIGHLIGHTS

Digitalis has been used for treatment of systolic heart failure. It augments contractility, thereby improving cardiac output, improving left ventricle emptying, and decreasing ventricular filling pressures. Digitalis has also been used to treat atrial fibrillation and atrial flutter. In this scenario, digitalis reduces the number of impulses transmitted through the AV node and thus, provides effective rate control.

In both these instances alternative treatments developed over the past 20 years and the need to tightly regulate dose due to significant potential for side effects have reduced the use of digitalis. However, with better understanding of mechanism and toxicity, digitalis and its clinically prepared derivatives remain important drugs in modern medicine.

SPREAD OF CARDIAC EXCITATION

Depolarization initiated in the SA node spreads radially through the atria, then converges on the AV node. Atrial depolarization is complete in about 0.1 s. Because conduction in the AV node is slow (Table 29–1), a delay of about 0.1 s (**AV nodal delay**) occurs before excitation spreads to the ventricles. It is interesting to note here that when there is a lack of contribution of I_{Na} in the depolarization (phase 0) of the action potential, a marked loss of conduction is observed. This delay is shortened by stimulation of the sympathetic nerves to the heart and lengthened by stimulation of the vagi. From the top of the septum, the wave of depolarization spreads in the rapidly conducting Purkinje fibers to all parts of the ventricles in 0.08–0.1 s. In humans, depolarization of the ventricular muscle starts at the left side of the interventricular septum and moves first to the right across the mid portion of the septum. The wave of depolarization then spreads down the septum to the apex of the heart. It returns along the ventricular walls to the AV groove, proceeding from the endocardial to the epicardial surface (Figure 29–4). The last parts of the heart to be depolarized are the posterobasal portion of the left ventricle, the pulmonary conus, and the uppermost portion of the septum.

TABLE 29–1 Conduction speeds in cardiac tissue.

Tissue	Conduction Rate (m/s)
SA node	0.05
Atrial pathways	1
AV node	0.05
Bundle of His	1
Purkinje system	4
Ventricular muscle	1

THE ELECTROCARDIOGRAM

Because the body fluids are good conductors (ie, because the body is a **volume conductor**), fluctuations in potential, representing the algebraic sum of the action potentials of myocardial fibers, can be recorded extracellularly. The record of these fluctuations in potential during the cardiac cycle is the **ECG.**

The ECG may be recorded by using an **active** or **exploring electrode** connected to an indifferent electrode at zero potential (**unipolar recording**) or by using two active electrodes (**bipolar recording**). In a volume conductor, the sum of the potentials at the points of an equilateral triangle with a current source in the center is zero at all times. A triangle with the heart at its center (**Einthoven triangle,** see below) can be approximated by placing electrodes on both arms and on the left leg. These are the three **standard limb leads** used in electrocardiography. If these electrodes are connected to a common terminal, an indifferent electrode that stays near zero potential is obtained. Depolarization moving toward an active electrode in a volume conductor produces a positive deflection, whereas depolarization moving in the opposite direction produces a negative deflection.

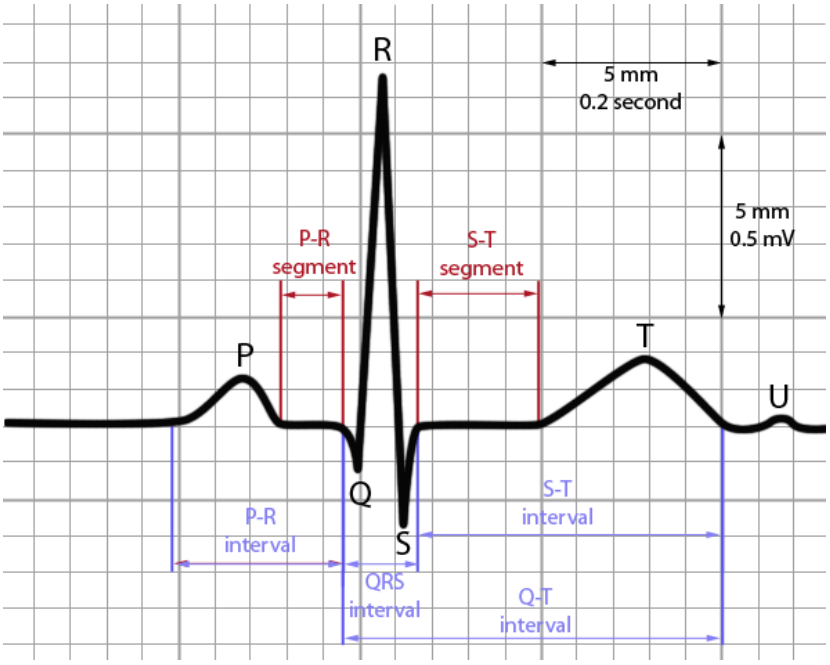
The names of the various waves and segments of the ECG in humans are shown in Figure 29–5. By convention, an upward deflection is written when the active electrode becomes positive relative to the indifferent electrode, and a downward deflection is written when the active electrode becomes negative. As can be seen in Figure 29–1, the P wave is primarily produced by atrial depolarization, the QRS complex is dominated by ventricular depolarization, and the T wave by ventricular repolarization. The U wave is an inconstant finding that may be due to ventricular myocytes with long action potentials. However, the contributions to this segment are still undetermined. The intervals between the various waves of the ECG and the events in the heart that occur during these intervals are shown in Table 29–2.

Purkinje system conducts the fastest

TABLE 29–2 ECG intervals.

Intervals	Normal Durations		Events in the Heart during Interval
	Average	Range	
PR interval[a]	0.18[b]	0.12–0.20	Atrioventricular conduction
QRS duration	0.08	to 0.10	Ventricular depolarization
QT interval	0.40[c]	to 0.43	Ventricular action potential
ST interval (QT minus QRS)	0.32	…	Plateau portion of the ventricular action potential

[a]Measured from the beginning of the P wave to the beginning of the QRS complex.



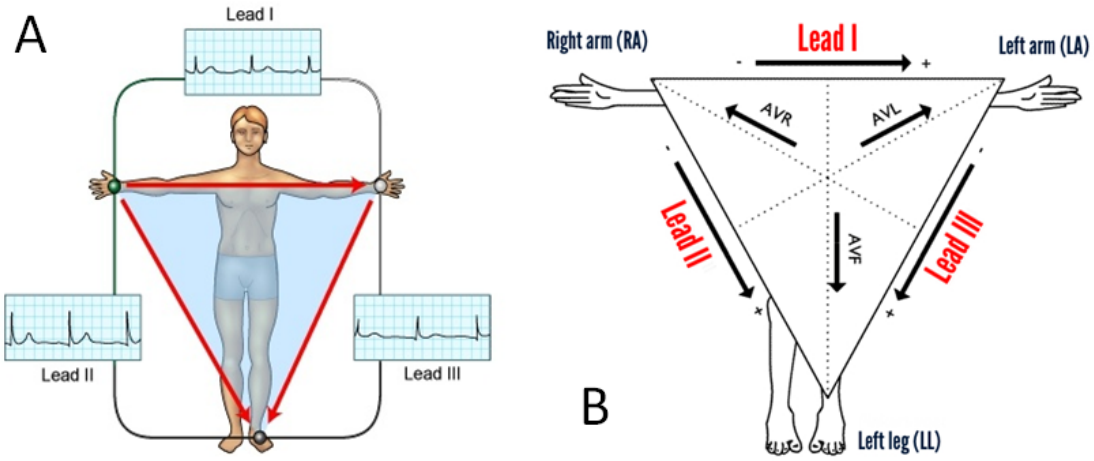
PR – beginning of P to end of QRS
QT – beginning of Q to end of T
ST – QT-QRS

<u>THE ELECTROCARDIOGRAM:</u>

- The ECG may be recorded by using an active or exploring electrode connected to an indifferent electrode at zero potential (UNIPOLAR RECORDING)
    - Can be recorded using two active recordings (BIPOLAR RECORDING)
- Depolarisation moving toward an active electrode in a volume conductor produces a POSITIVE DEFLECTION
    - Depolarisation moving in the opposite direction produces a NEGATIVE DEFLECTION

- The P-wave is produced by atrial depolarisation
- The QRS complex by VENTRICULAR DEPOLARISATION
- The ST segment and T wave by VENTRICULAR REPOLARISATION
- The manifestations of atrial repolarisation are not normally seen because they are obscured by the QRS complex
- The U-wave is an inconstant finding, believed to be due to slow repolarisation of the papillary muscles

- **BIPOLAR LEADS – limb leads**
    - The standard limb leads (I, II and III) each record the difference in potential between two limbs
    - In I, the electrodes are connected so that an upward deflection is inscribed when the left arm becomes positive relative to the right
    - In II, the electrodes are on the right arm and left leg, with left leg being positive
    - In III the electrodes are on the left arm and left leg, with the leg positive



- **UNIPOLAR LEADS – 6 chest + 3 augmented limb leads**
    - An additional nine unipolar leads are present
    - There are six unipolar chest leads (praecordial leads, designated V1-V6)
    - Three unipolar limb leads:
        - VR (right arm)
        - VL (left arm)
        - VF (left foot)
    - AUGMENTED LIMB LEADS:
        - Designated by the letter a
        - Recordings between one limb and the two other limbs

        - Increases the size of the potentials by 50% without any change in configuration from the nonaugmented record
- NORMAL ECG:
    - The atria are located posteriorly in the chest
    - The ventricles form the base and anterior surface of the heart
    - The right ventricle is anterolateral to the left
    - AVR thus "looks at" the cavities of the ventricles
        - Atrial depolarisation, ventricular depolarisation and ventricular repolarisation move away from the exploring electrode
        - Thus the P wave, QRS complex and T wave are therefore all negative (downward deflections)
    - AVL and AVF "look at" the ventricles" and the deflections are therefore predominantly positive or biphasic
    - There is NO Q WAVE in V1 or V2
        - A small upward deflection because of ventricular depolarisation first moves across the midportion of the septum
            - The wave of excitation then moves down the septum, producing a LARGE S WAVE
    - In the LV leads (V4-V6), there may be an initial small Q wave (left to right septal depolarisation and there is a large R wave
        - Followed in V4 and V5 by a moderate S wave
- BIPOLAR LIMB LEADS AND THE CARDIAC VECTOR:
    - The deflection in each limb lead at any moment indicates the magnitude and direction in the axis of the lead of the electromotive force generated in the heart
    - **The normal direction of the mean QRS vector is generally said to be -30 to +110 degrees on the coordinate system**
    - LEFT OR RIGHT AXIS DEVIATION:
        - Said to be present if the calculated axis falls to the left of -30 or to the right of +100 degrees
        - Right axis deviation suggests RV hypertrophy and left axis deviation may be due to LV hypertrophy, but there are better criteria for hypertrophy
- HIS BUNDLE ELECTROGRAM:
    - In patients with heart block, the electrical events in the AV node, bundle of His and Purkinje system can be obtained with the His Bundle electrogram (HBE)
    - An A deflection occurs when the AV node is activated
    - An H spike occurs during transmission through the His bundle
    - A V deflection occurs during ventricular depolarisation
    - It is possible to accurately time THREE INTERVALS:
        - PA interval, which is the time from the first appearance of atrial depolarisation to the A wave in the HBE

        - Represents conduction time from the SA node to the AV node
    - AH interval, from the A wave to the start of the H spike
        - Represents the AV nodal conduction time
    - HV interval
        - Represents conduction time in the bundle of His and the bundle branches

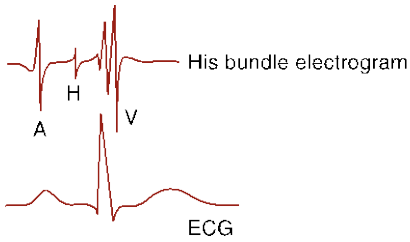


FIGURE 29–9 Normal His bundle electrogram (HBE) with simultaneously recorded ECG. An HBE recorded with an invasive electrode is superimposed on a standard ECG reading. Timing of depolarizations of the HBE are described in the text.

His Bundle Electrogram

- A-H = AVN conduction time
- HV = consuction time in bundle of His + bundle branches

CARDIAC ARRHYTHMIAS:

NORMAL CARDIAC RATE:

- In the normal human heart, each beat originates in the SA node (NORMAL SINUS RHYTHM)
- Heart normally beats at ~70bpm
    - If it is slow = BRADYCARDIA
    - If it is fast = TACHYCARDIA
- In healthy young individuals breathing at a normal rate, the heart VARIES WITH THE PHASES OF RESPIRATION
    - It accelerates during inspiration and decelerates during expiration
        - SINUS ARRHYTHMIA
        - Due primarily to fluctuations in parasympathetic output to the heart
        - During inspiration, impulses in the vagi from the stretch receptors in the lungs inhibit the cardioinhibitory area in the medulla oblongata
            - The tonic vagal discharge keeps the heart rate slow decreases and thus the heart rate INCREASES

- **Inspiration → increased vagal input from lung stretch receptors → inhibition of cardio-inhibitory area of medulla → ⇓tonic vagal drive to heart**

- **Inspiration → lung stretch receptors → ⇓vagal tone to heart → ⇑HR**

ABNORMAL PACEMAKERS:

- The AV node and other portions of the conduction system can in abnormal situations become the cardiac pacemaker
- When conduction from the atria to the ventricles is COMPLETELY INTERRUPTED, COMPLETE HEART BLOCK RESULTS
    - The ventricles beat at a low rate (IDIOVENTRICULAR RHYTHM) independently of the atria
- The block may be due to disease in the AV node (AV NODAL BLOCK) or in the conducting system below the node (INFRANODAL BLOCK)
- In patients with infranodal block due to disease in the bundle of His, the ventricular pacemaker is located more peripherally in the conduction system and the rate is LOWER (35 BPM)
    - There may also be periods of asystole lasting a minute or more
    - The resulting cerebral ischaemia causes dizziness and fainting (STOKES-ADAMS SYNDROME)
    - Causes of third degree heart block include:
        - Septal AMI
        - Damage to the bundle of His during surgery on congenital VSD
- When conduction between atria and ventricles is slowed but not completely interrupted, incomplete heart block is present
    - In FIRST DEGREE HEART BLOCK:
        - All the atrial impulses reach the ventricles, but the PR interval is abnormally long
    - IN SECOND DEGREE HEART BLOCK:
        - Not all atrial impulses are conducted to the ventricles
        - E.g. a ventricular beat may only follow every second or third atrial beat (2:1 or 3:1 block)
        - In another form, there are repeated sequences of beats in which the PR interval lengthens progressively until a ventricular beat is dropped (WENCKEBACH PHENOMENON)

- **Mobitz 1 = Wenkebach = not likely to progress to complete HB**
- **Mobitz 2 = 2:1 / 3:1 block = can progress to complete HB and should be paced**

- Sometimes one branch of the bundle of His is interrupted, causing RIGHT OR LEFT BUNDLE BRANCH BLOCK

- In bundle branch block, excitation passes normally down the bundle on the intact side and tehn sweeps back through the muscle to activate the ventricle on the blocked side
    - The ventricular rate is thus NORMAL, but the QRS complexes are prolonged and deformed
- LEFT ANTERIOR HEMIBLOCK produces abnormal LEFT AXIS DEVIATION
- LEFT POSTERIOR HEMIBLOCK produces abnormal RIGHT AXIS DEVIATION

Trifascicular:

- **Prolonged PR**
- **RBBB**
- **LAD (L anterior hemiblock)**

ECTOPIC FOCI OF EXCITATION:

- In abnormal conditions, the His-Purkinje or myocardial fibres may discharge spontaneously
- In these conditions, INCREASED AUTOMATICITY of the heart is said to be present
    - If an irritable ECTOPIC FOCUS discharges once, the result is a beat that occurs before the expected next normal beat
    - Also known as EXTRASYSTOLE OR PREMATURE BEAT

REENTRY:

- A more common cause of paroxysmal arrhythmias is a defect in conduction that PERMITS **A WAVE OF EXCITATION TO PROPAGATE CONTINUOUSLY WITHIN A CLOSED CIRCUIT**
- In individuals with an abnormal extra bundle of conducting tissue connecting the atria to the ventricles (BUNDLE OF KENT), the circus activity can pass in one direction through the AV node and the other through the accessory bundle

ATRIAL ARRHYTHMIAS:

- Excitation spreading from an independently discharging focus in the atria stimulates the AV node prematurely and is conducted to the ventricles
    - The P-waves of atrial extrasystoles are ABNORMAL, but the QRST configurations are usually normal
- In **ATRIAL FLUTTER**:
    - Atrial rate is 200-350/minute
    - In the most common form of this arrhythmia, there is large COUNTERCLOCKWISE CIRCUS MOVEMENT in the right atrium
    - This produces a characteristic SAW-TOOTH PATTERN OF FLUTTER WAVES due to atrial contractions
    - Nearly always associated with 2:1 or greater AV block

- ATRIAL FIBRILLATION:
    - The atria beat very rapidly in a completely irregular and disorganised fashion
    - Because the AV node discharges at irregular intervals, the ventricles beat at a completely irregular rate
- CONSEQUENCES OF ATRIAL ARRHYTHMIAS:
    - Occasional atrial extrasystoles occur from time to time in most normal humans and have no pathologic significance
    - In paroxysmal atrial tachycardia and flutter, the ventricular rate may be so high that diastole is TOO SHORT for adequate filling of the ventricles
        - Consequently, cardiac output is reduced and symptoms of heart failure appear
        - Same problem may occur in AF
    - Acetylcholine liberated at vagal endings depresses conduction in the atrial musculature and AV node
        - This is why stimulating reflex vagal discharge
            - Pressing on the eyeball (OCULOCARDIAC REFLEX)
            - Massaging the CAROTID SINUS
            - These measures often convert tachycardia and even flutter to normal sinus rhythm

VENTRICULAR ARRHYTHMIAS:

- Premature beats that originate in an ectopic ventricular focus usually have bizarrely shaped prolonged QRS complexes because of the slow spread of the impulse from the focus through the ventricular muscle
- Ventricular premature beats are followed by a COMPENSATORY PAUSE that is often longer than the pause after an atrial exytrasystole
- PAROXYSMAL VENTRICULAR TACHYCARDIA:
    - A series of rapid, regular ventricular depolarisations usually due to a circus movement involving the ventricles
    - TORSADES DE POINTES is a form of VT in which the QRS morphology VARIES
    - VT is serious because cardiac output is decreased and VF is an occasional complication of VT
- VENTRICULAR FIBRILLATION:
    - The ventricular muscle fibres contract in a totally irregular and ineffective way because of the very rapid discharge of multiple ventricular ectopic foci or a circus movement
    - Can be produced (OR REVERSED) by electric shock or an extrasystole during a period of vulnerability
        - The VULNERABLE PERIOD coincides in time with the midportion of the T-wave

        - I.e. it occurs at a time when of the ventricular myocardium is depolarised, some is incompletely repolarised and some is completely repolarised
    - The fibrillating ventricles cannot pump blood effectively and circulation of the blood stops => FATAL IF NOT REVERSED

ACCELERATED AV CONDUCTION:

- Aka WOLFF-PARKINSON WHITE SYNDROME
- Prone to attacks of paroxysmal atrial arrhythmias
- Normally the only conducting pathway between the atria and the ventricles is the AV node
- Individuals with WPW have an additional aberrant muscular or nodal tissue connection (BUNDLE OF KENT) between the atria and ventricles
    - This conducts more rapidly than the slowly conducting AV node and on ventricle is excited EARLY
    - Produces:
        - SHORT PR INTERVAL
        - PROLONGED QRS DEFLECTION SLURRED ON THE UPSTROKE
- The beat conducts normally down the AV node but spreads to the ventricular end of the aberrant bundle and the impulse is transmitted retrograde to the atrium
    - A CIRCUS MOVEMENT IS THUS ESTABLISHED

ECG FINDINGS IN CARDIAC AND OTHER SYSTEMIC DISEASES:

- MYOCARDIAL INFARCTION:
    - When the blood supply to part of the myocardium is interrupted, profound changes take place in the myocardium that lead to irreversible changes and death of muscle cells
    - The first change is **ABNORMALLY RAPID REPOLARISATION AFTER DISCHARGE** of the infracted muscle fibres as a result of accelerated opening of K channels
        - This develops SECONDS after occlusion
        - **Resting membrane potential of the infracted fibres then DECLINES because of the loss of intracellular K**
        - Infracted fibres also begin to depolarise more slowly than the surrounding normal fibres

1. Rapid repol (current flows out of infarct)
2. ⇓ Resting membrane potential (current flows into infarct)
3. Delayed depol (current flows out of infarct)

- All three of the above changes cause current flow that produces elevation of the ST segments in the ECG:
  - Because of the rapid repolarisation, the membrane potential of the area is greater than it is in the normal area during the latter part of repolarisation, making the normal area NEGATIVE RELATIVE TO THE INFARCT
    - Extracellularly, current therefore flows out of the infarct into the normal area
    - This current flows toward electrodes over the injured area, causing increased POSITIVITY between the S and T waves of the ECG
  - Delayed depolarisation of the infracted cells causes the infracted area to be POSITIVE relative to the healthy tissue during the early part of repolarisation =>ST elevation
  - Decline in the resting membrane during diastole, causes a current flow into the infarct during ventricular diastole
    - The result of this current flow is a DEPRESSION OF THE TQ SEGMENT
      - Recorded as an ST segment elevation
- After some days or weeks, the ST segment abnormalities subside
  - The dead muscle and scar tissue become electrically silent
  - The infracted area is therefore negative relative to the normal myocardium and fails to contribute its share of positivity
    - Appearance of Q-waves
    - Poor R-wave progression
    - If the septum is infracted, the conduction system may be damaged, causing bundle branch block or other forms of heart block
- Myocardial infractions are often complicated by serious ventricular arrhythmias, which occur during three periods:
  - First 30 minutes of an infarction (due to re-entry)
  - 12 hours after an AMI (as a result of increased automaticity)
  - Occurring days to weeks down the track (usually due to re-entry)

- **AMI damage to <u>epicardium</u>:**
  - **Interrupt <u>sympathetic</u> nerve fibres, producing denervation supersensitivity to the circulating catecholamines**

- **AMI damage to <u>endocardium</u>:**
  - **Interrupt <u>vagal</u> fibres, leaving the actions of sympathetic fibres unopposed**

EFFECTS OF CHANGES IN THE IONIC COMPOSITION OF THE BLOOD:

- A fall in the plasma level of Na may be associated with low-voltage ECG complexes, but changes in the plasma K level produce SEVERE CARDIAC ABNORMALITIES
    - HYPERKALEMIA:
        - Very dangerous and potentially lethal condition
        - As the level rises, the first change in the ECG is TALL PEAKED T WAVES
            - This is a manifestation of altered repolarisation
        - At higher K levels, **PARALYSIS OF THE ATRIA and prolongation of the QRS complexes occur**
            - **Ventricular arrhythmias** may develop
            - The resting membrane potential of the muscle fibres decreases as the extracellular K concentration increases
                - The fibres EVENTUALLY **BECOME UNEXCITABLE AND THE HEART STOPS IN DIASTOLE**

⇑K abolishes the resting membrane potential → myocardium becomes unexcitable

1. **Repolarisation abnormalities** (peaked T)
2. **Atrial paralysis** (prolonged PR)
3. **Conduction abnormalities + bradycardia** (brady)
4. **Asystole**

    - HYPOKALEMIA:
        - Prolongation of the PR interval
        - Prominent U waves
        - Late T wave inversion in the praecordial leads
            - If the T and U waves merge, the apparent QT interval is often prolonged
        - Hypokalemia is a serious condition but not as rapidly fatal as hyperkalaemia

    - **HYPERCALCAEMIA:**
        - **Increases in extracellular Ca ENHANCE MYOCARDIAL CONTRACTILITY**
        - In most clinical conditions associated with hypercalcaemia, the plasma calcium level is rarely if ever high enough to affect the heart