ELECTRICAL EVENTS IN THE CARDIAC CYCLE

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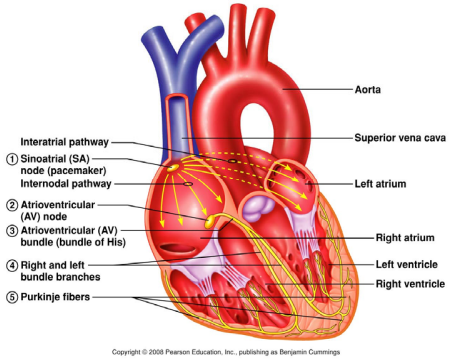
OBJECTIVES OF THIS LECTURE

- Describe the path of action potentials through the conduction system of the heart
- Understand the ionic basis of the different phases of the pacemaker potential
- Describe the effects of the autonomic nervous system on pacemaker potentials
- Understand the ionic basis of the different phases of the ventricular action potential
- Describe the different waves of the electrocardiogram and how they relate to the events of the cardiac cycle

The heart has a specialised system for

- generating rhythmical impulses ⇒ rhythmical contraction (autorhythmic)
- conducting these impulses throughout the heart

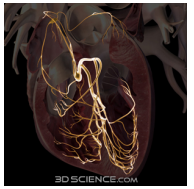


- impulses generated in the sinus node (sinoatrial node / SA node)
- flattened strip of specialised muscle in lateral wall of the upper RA
- discharges 70 times/min at rest
- SA nodal cells have fastest inherent rate of spontaneous depolarization and so set heart rate
- from SA node impulses spread through atria by interatrial pathways
- impulses also travel through internodal pathways to the atrioventricular node
- small bundle of specialised cardiac muscle cells at base of right atrium

- ±30msec from SA node to AV node, ±100msec delay in AV node itself - AV nodal delay.
- Impulses then enter Bundle of His (atrioventricular bundle) – further delay of ±40msec, then divides into
- right and left bundle branches – travel down septum, curve around tips of ventricles & travel back towards atria
- Purkinje fibres extend from bundle branches and spread into ventricular myocardium
- Purkinje fibres – large elongated cylindrical cells, numerous mitochondria, few myofibrils, specialised for fast conduction
- then impulses are transmitted through ventricular muscle fibres

RATES OF CONDUCTION

SA node	0.05m/sec
Atrial muscle	1m/sec
AV node	0.05m/sec
Bundle of His	1m/sec
Purkinje fibres	4m/sec
Ventricular muscle	1m/sec



These various autorhythmic cells have different rates of discharge

SA node (fastest)	70-80 times/min
AV node	40-60 times/min
Bundle of His/purkinje fibres	20-40 times/min

ION MOVEMENT ACROSS CELL MEMBRANES

ion	IC conc	EC conc
Na^+	15mM	150mM
K^+	150mM	5mM
Ca^{++}	low	high

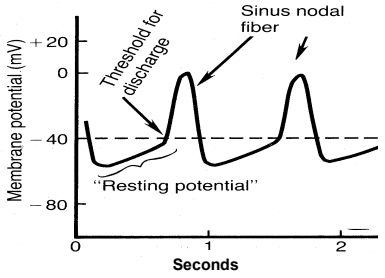
$[Na^+]$ outside cell much higher (150mM) than inside (15mM) so tendency is for Na^+ to enter cell, increasing positivity inside cell.

$[K^+]$ inside cell much higher (150mM) than outside (5mM) so tendency is for K^+ to leave cell, increasing negativity inside cell.

$[Ca^{++}]$ outside cell higher than inside so tendency is for Ca^{++} to enter cell, increasing positivity inside cell.

PACEMAKER POTENTIALS

An action potential recorded from a sinus nodal fibre



resting membrane potential about -60mV

membrane depolarises to around 0mV

A) Slow Depolarisation (unstable baseline)

membrane potential drifts up to -40mV because

i) progressive spontaneous reduction in membrane permeability of SA node cell to K^+ i.e. closing of K^+ channels - less K^+ moves out of cell → ↑ internal potential. This is coupled with a constant inward Na^+ current.

ii) small progressive ↑ in Ca^{++} permeability - Ca^{++} moves into cell → ↑ internal potential (opening of slow or T Ca^{++} channels)

B) Depolarisation / Upstroke

sudden increase in internal potential

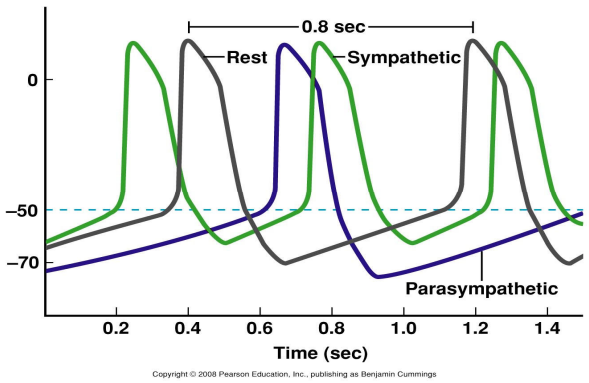
- opening of L (long lasting) Ca^{++} channels → ↑↑ in Ca^{++} permeability & large inward flow of Ca^{++} → cell depolarisation

C) Repolarisation

internal potential drops because

- reversal of Ca^{++} permeability - both Ca^{++} channels close so no further Ca^{++} moves into cell
- K^+ channels activated, K^+ permeability increases, K^+ leaves cell

EFFECTS OF ANS ON PACEMAKER POTENTIALS



EFFECTS OF ANS ON PACEMAKER POTENTIALS

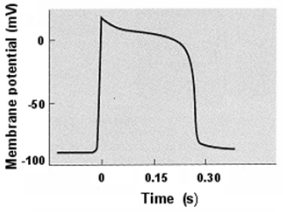
- SA node receives direct input from ANS
- ↑ SNS activity → ↑ frequency of action potentials generated by
  - ↑ slope of the spontaneous depolarisation
  - ↓ level of repolarisation

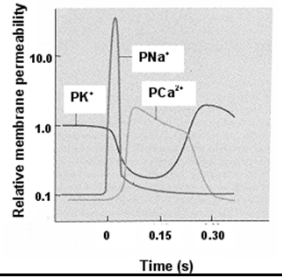
  so threshold is reached more quickly.
  ↑ frequency of APs → ↑ in heart rate

- ↑ PSNS activity → ↓ frequency of action potentials generated by
  - ↓ slope of spontaneous depolarisation
  - causing a hyperpolarisation of the membrane potential

so ↑ time taken for threshold to be reached.
↓ frequency of APs → ↓ in heart rate

ACTION POTENTIAL OF NON PACEMAKER CELL





resting potential -80 to -90mV

depolarises to +30mV

duration 300msec

long plateau phase - unique for cardiac muscle – relatively long absolute refractory period

Resting State

- cardiac muscle cell much more permeable to K^+ than Na^+ so K^+ leaks out keeping resting potential at -90mV.

Depolarisation / Fast Upstroke

- fast Na^+ channels open → rapid ↑ in Na^+ permeability, Na^+ flows rapidly into cell increasing membrane potential
- ↓ permeability to K^+ stopping K^+ moving out of cell - contributes to membrane depolarisation

Plateau Phase

↑ in Na^+ permeability is very transient and Na^+ channels quickly close again

At start of plateau, K^+ permeability is high so K^+ leaves through open K^+ channels

Both these cause membrane potential to fall a little but membrane remains depolarised at a plateau of about 0mV for up to 300msec because

i) activation of slow Ca^{++} channels → slow inward infusion of Ca^{++}

ii) concomitant ↓ in K^+ permeability reducing outflow of positively charged K^+

Both these prolong positivity inside cell

Repolarisation

Internal potential drops because

i) inactivation of Ca^{++} channels so less Ca^{++} moves into cell

ii) activation of K^+ channels causing rapid outward movement of K^+

Relative permeabilities of different ions

<u>In resting phase</u>: membrane more permeable to K^+ than Na^+ so more K^+ is moving out than Na^+ moving in so potential is negative

<u>Fast upstroke</u>: permeability to Na^+ ↑ dramatically but permeability to K^+ drops - potential ↑

<u>Plateau phase</u>: Na^+ permeability ↓, K^+ permeability continues to ↓ (reducing outflow of positive charges) and Ca^{++} permeability ↑ (increasing inflow of Ca^{++})

<u>Repolarisation</u>: K^+ permeability ↑ (K^+ flows out) and Ca^{++} permeability ↓ (Ca^{++} stops flowing in) - potential ↓

THE ELECTROCARDIOGRAM (ECG)

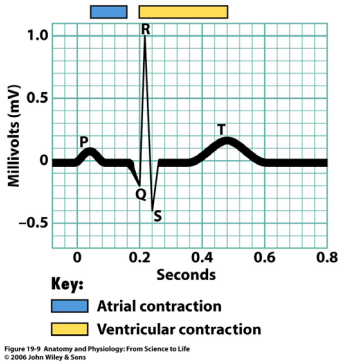


Figure 19-9 Anatomy and Physiology: From Science to Life
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P wave - atrial depolarisation

QRS complex - ventricular depolarisation

T wave - ventricular repolarisation

(atrial repolarisation not evident on ECG because it occurs at the same time as ventricular depolarisation)

Between waves – horizontal isoelectric line

Certain intervals and segments can provide information about heart function