Management of Cardiac Arrythmias in the ICU

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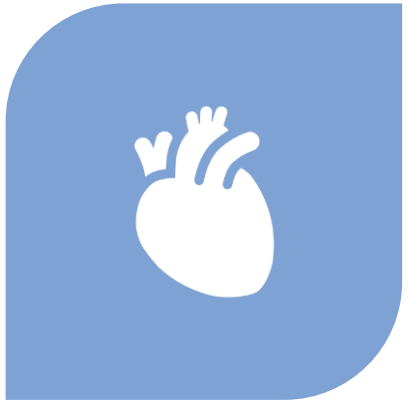
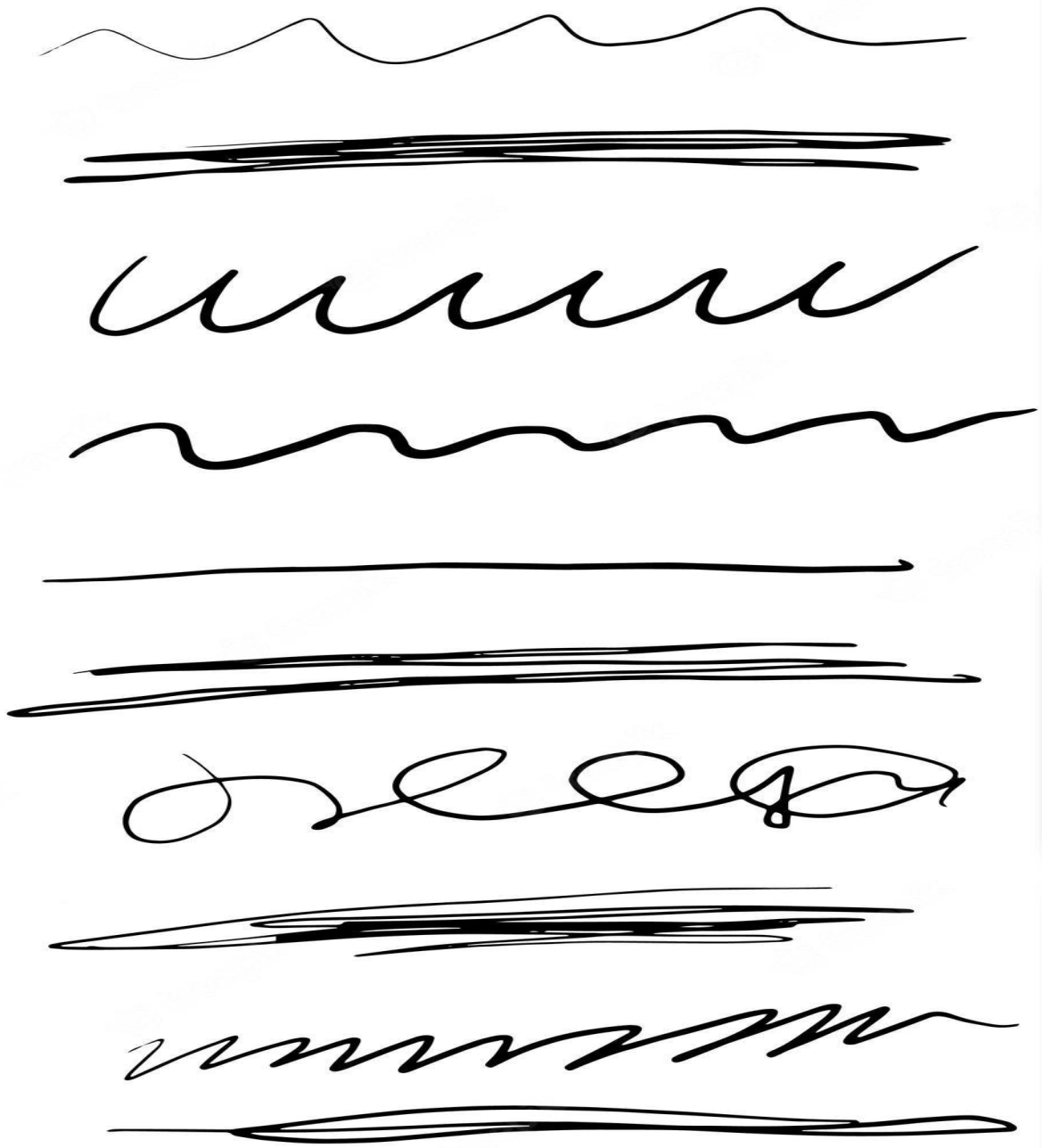
What will we cover today?

6 STEP APPROACH TO RHYTHM RECOGNITION



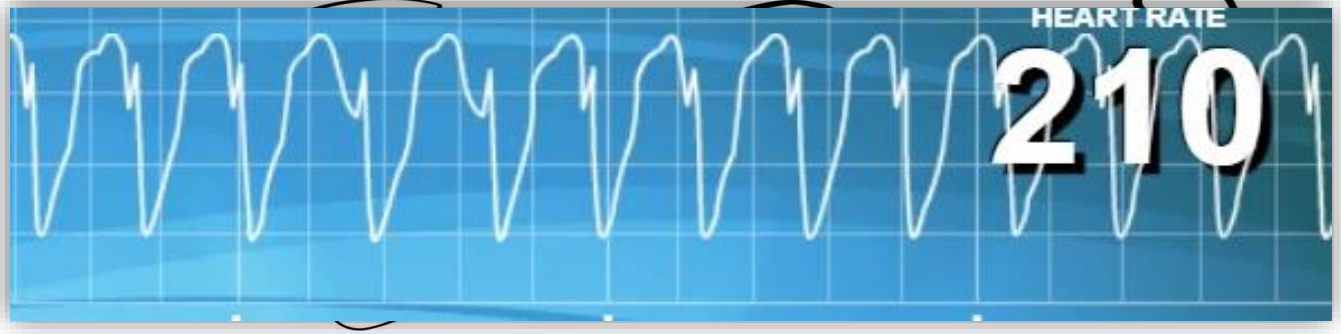
ECG HACK – RELATING THE ‘PQRST’ TO THE ANATOMY OF THE HEART AND

NURSING CARE AND MANAGEMENT OF COMMONLY OCCURRING ARRYTHMIAS SEEN




HEART RATE
72

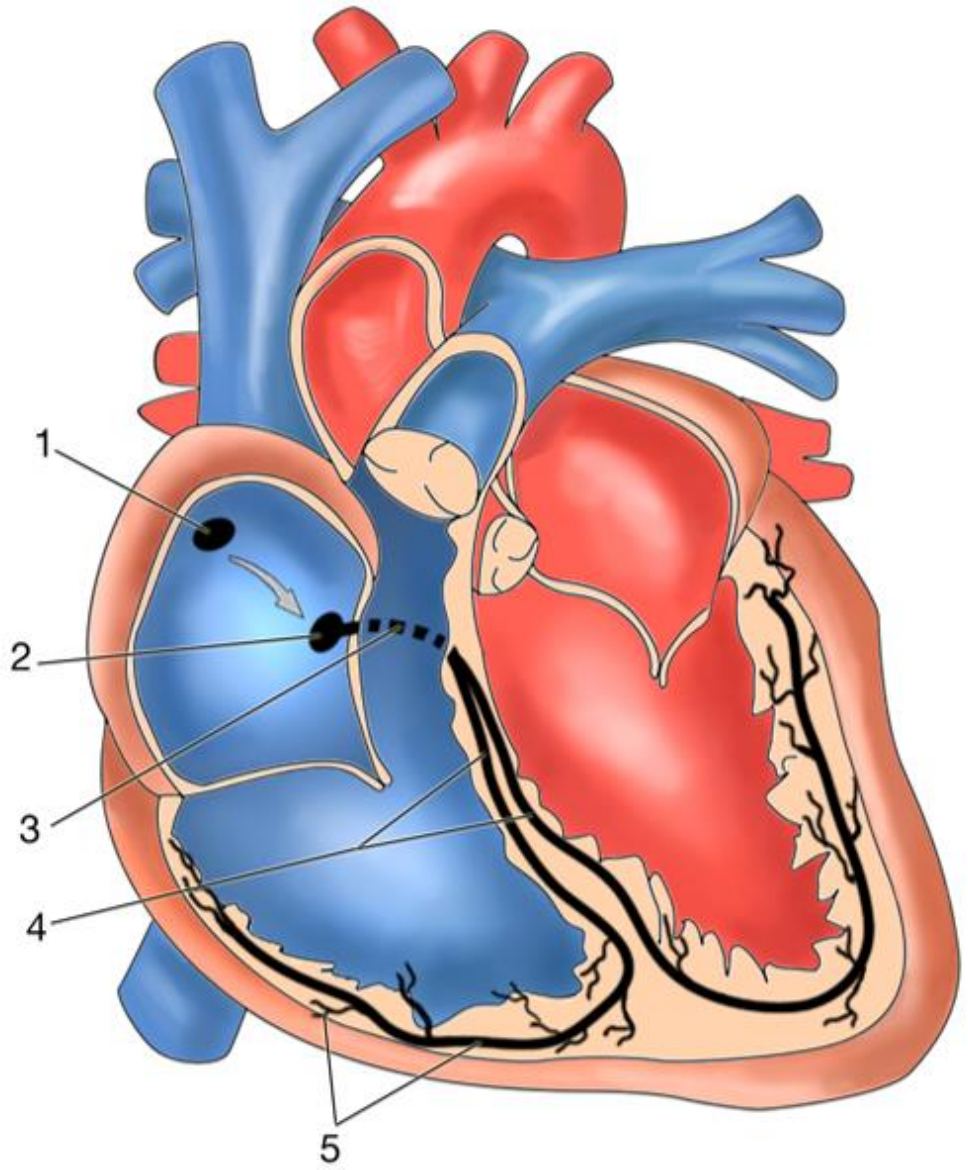

HEART RATE
90

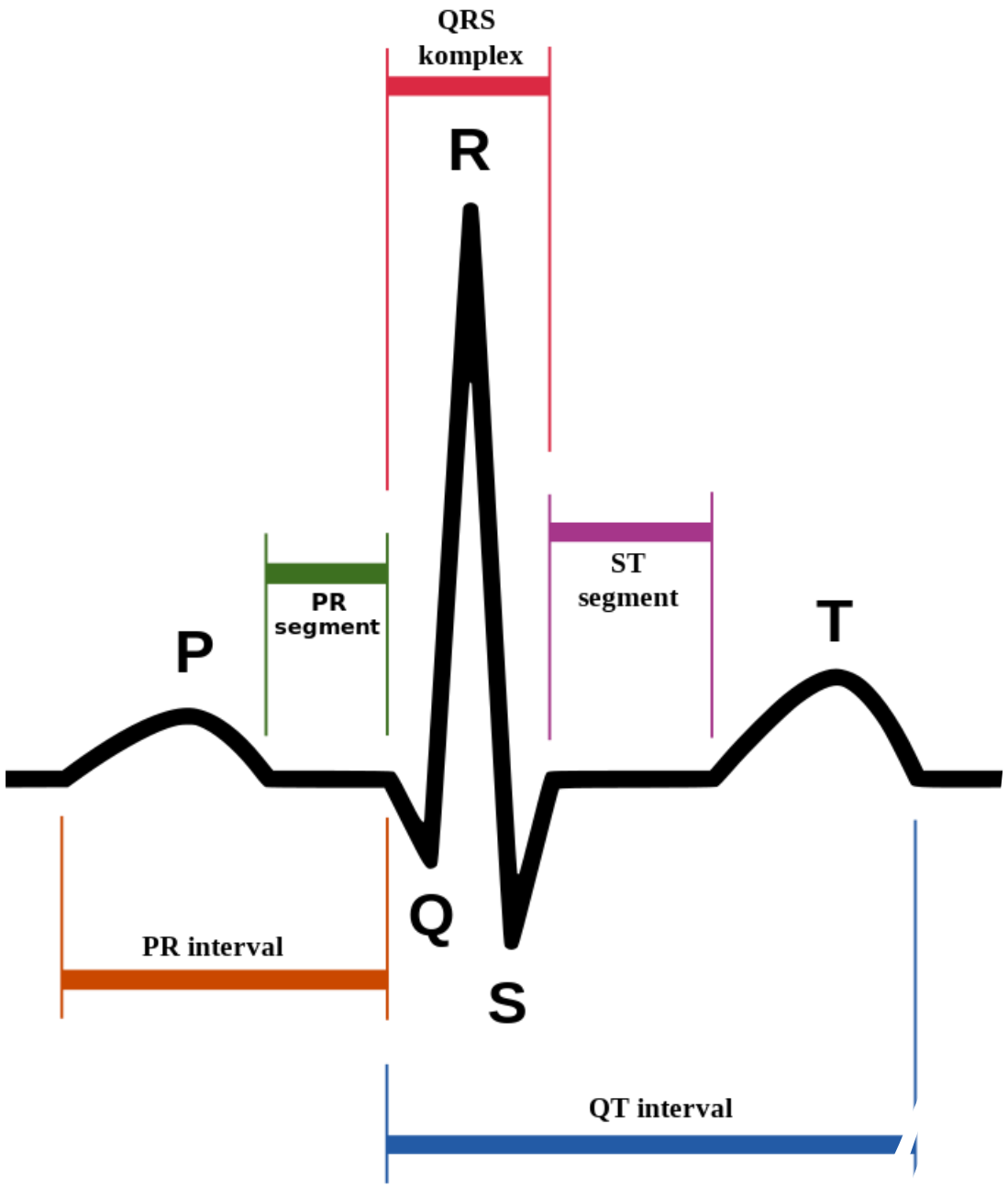

HEART RATE
210


HEART RATE
36

Cardiac anatomy & Conduction system

1. Sinoatrial (SA) node
2. Atrioventricular (AV) node
3. Bundle of HIS
4. Left and Right Bundle Branches

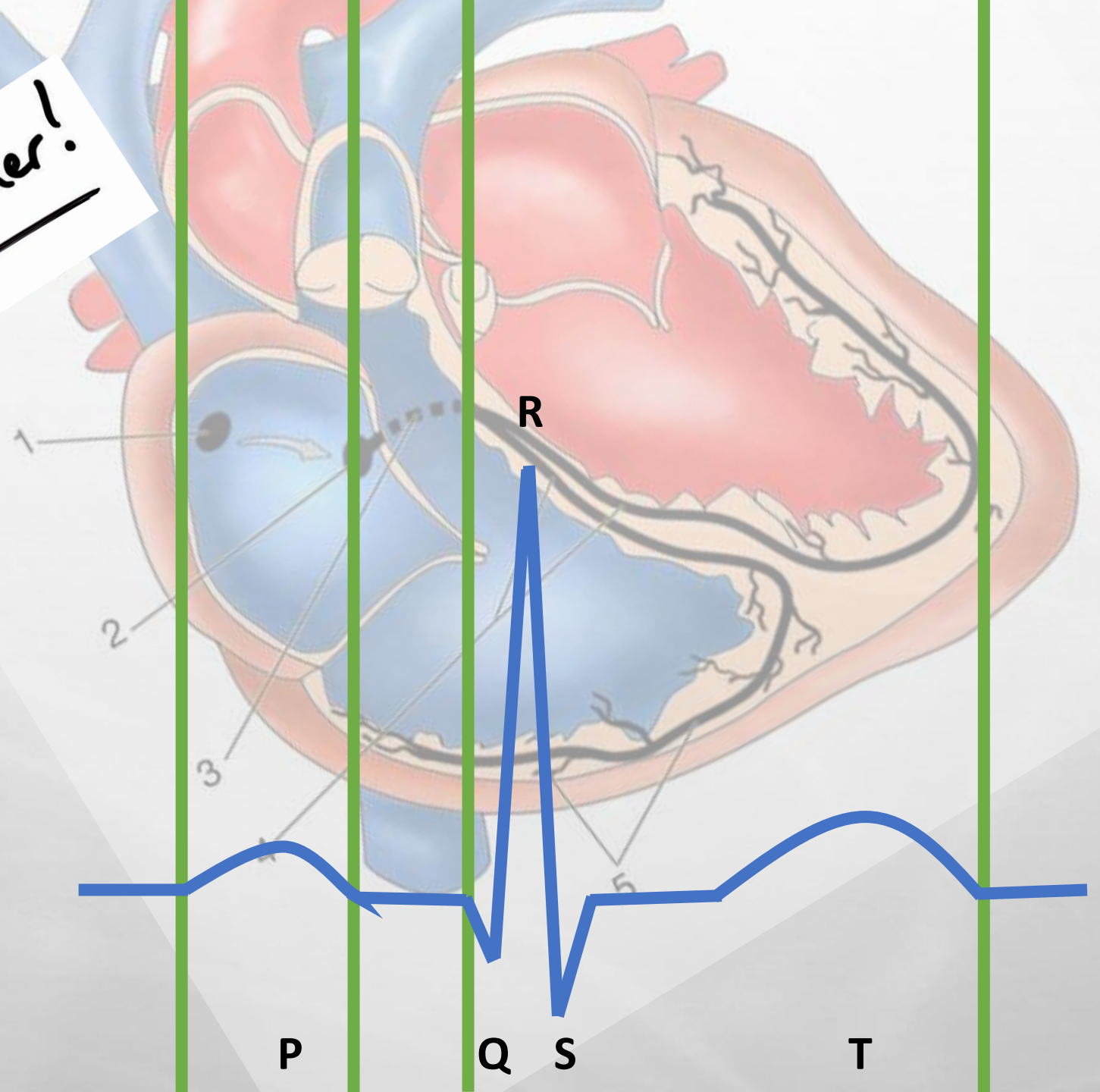




Cardiac cycle

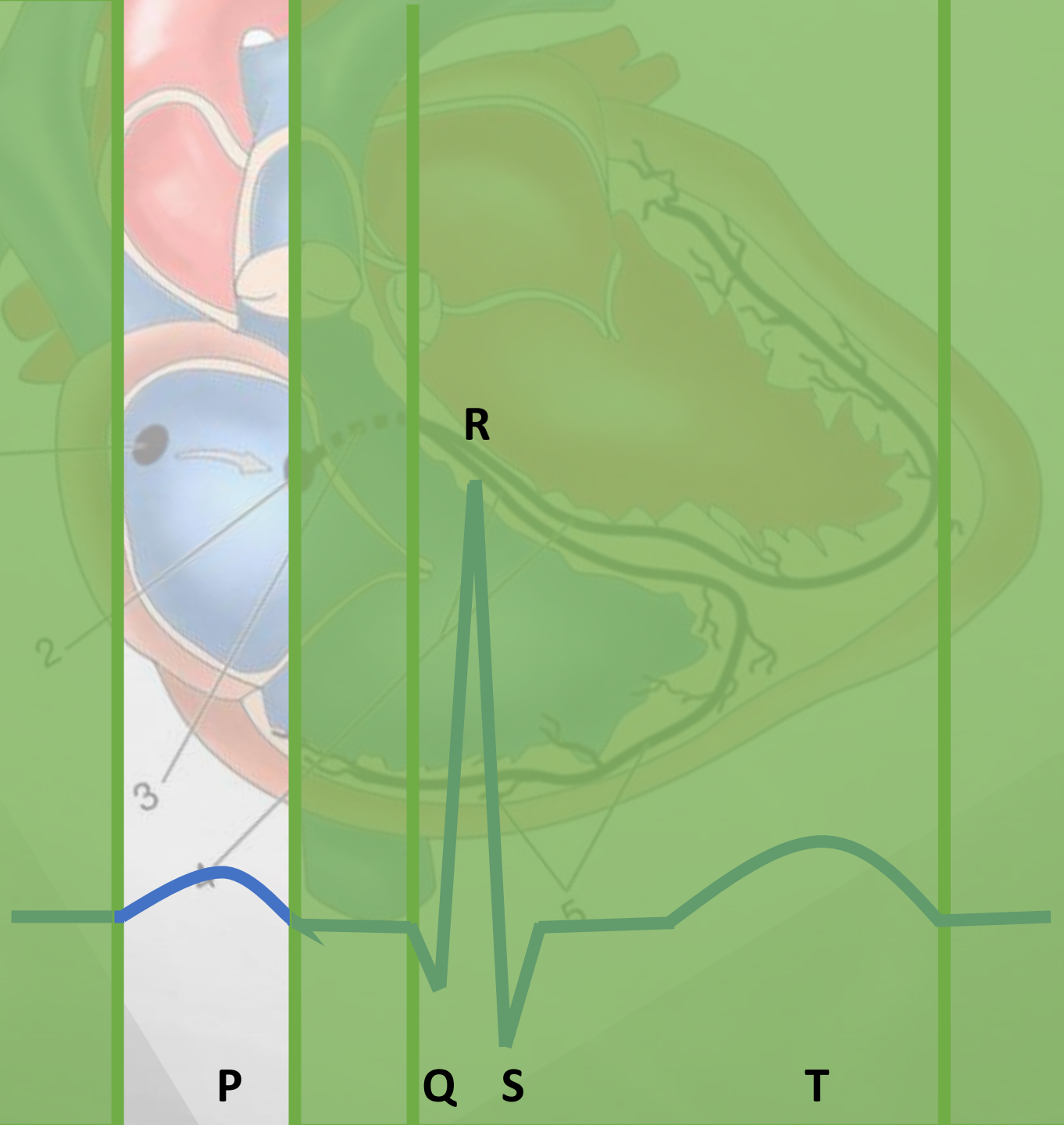
- P Wave = Atrial contraction
- QRS Complex = Ventricular contraction
- T Wave = Ventricular relaxation

Let's Pull Together!



P Wave
- Atrial contraction
- SA node firing
- Should be smooth and round

SA Node
- Natural pacemaker cell

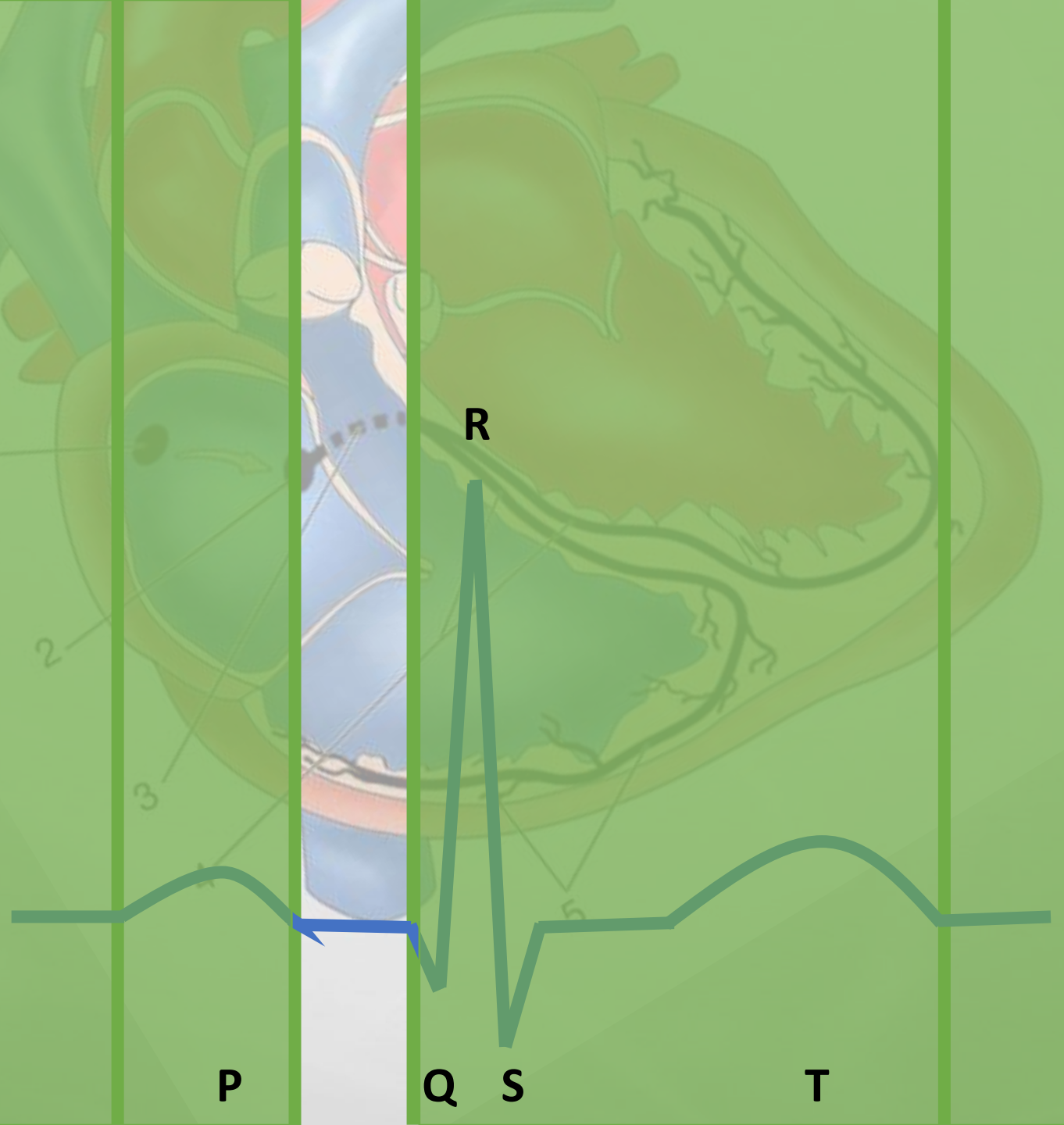


PR Segment

- Electrical impulse delay though AV node
- Holds onto electrical impulse to allow the ventricles to fill up

AV Node

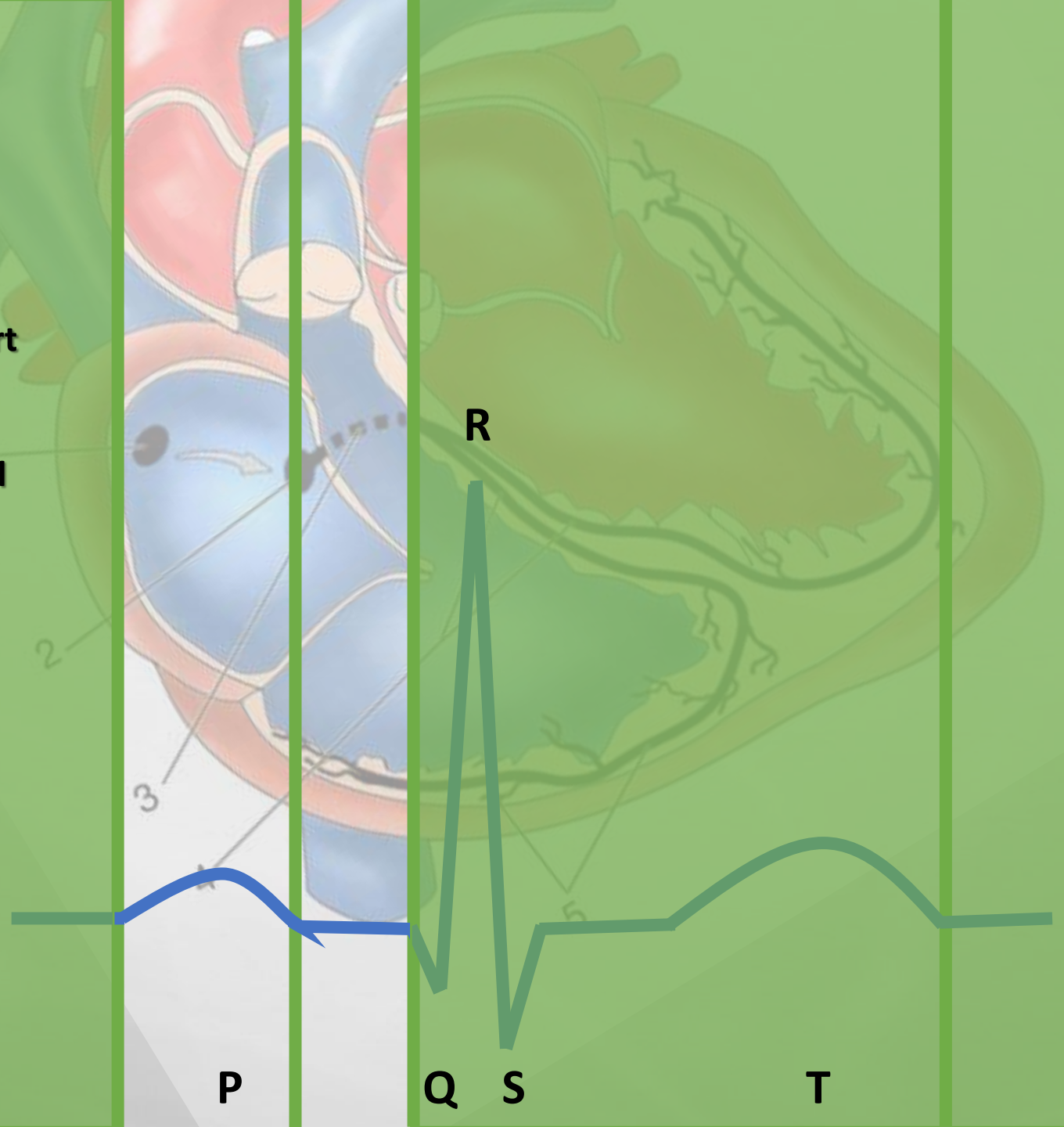
- ‘Gate keeper’ cell
- Safety rate filter
- Secondary pacemaker (if SA node fails)
- Back up rate 40-60bpm



PR Interval

- **Atrial Contraction + AV Delay**
- **Start of P wave to the start of QRS complex**

- **Normal timing is 3-5 small squares**
- **0.12-0.2 seconds or 120-200 milliseconds**



QRS Complex

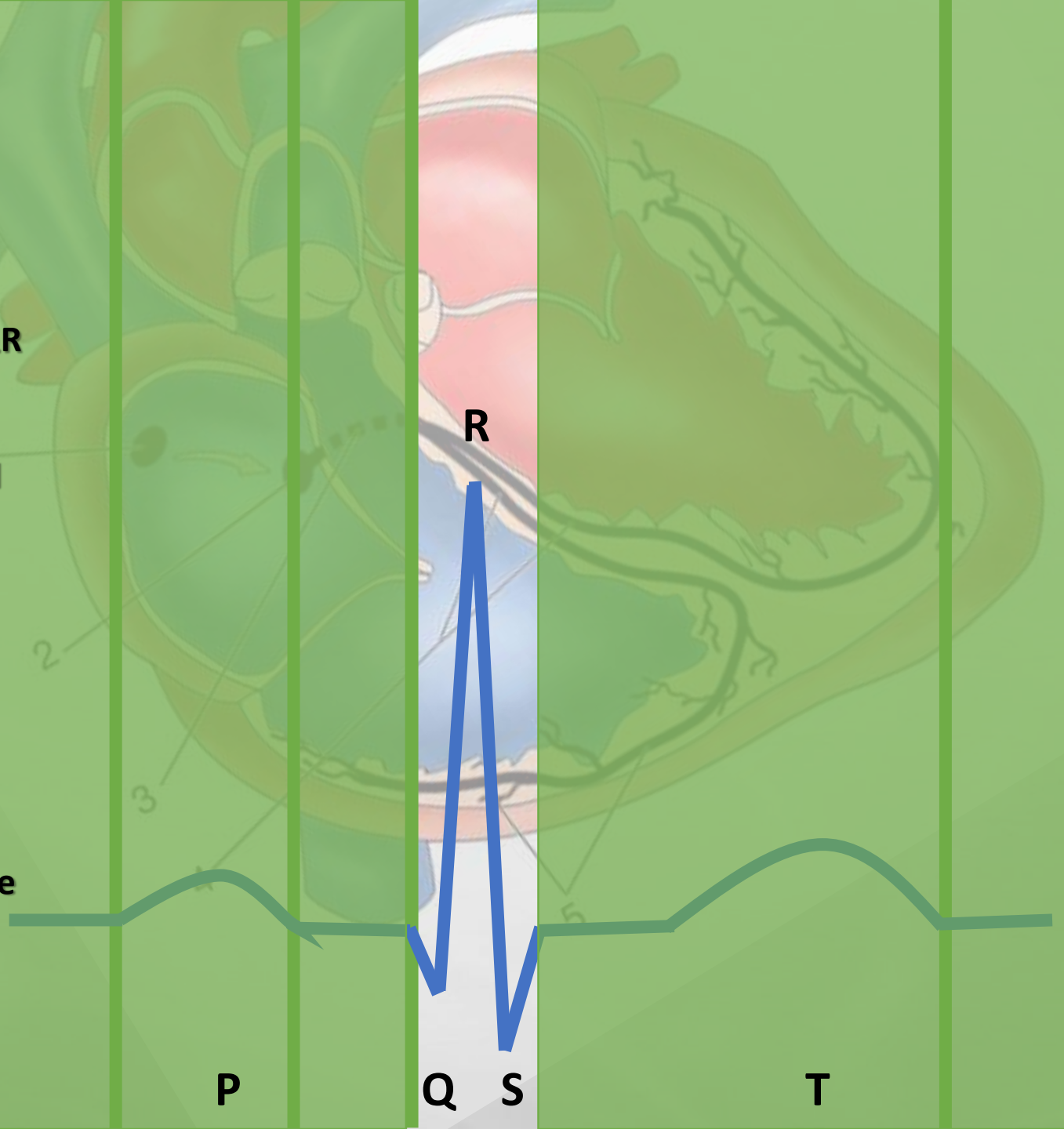
- Ventricular contraction
- Electrical Impulse travels through Bundle of HIS, L&R Bundle Branches and Purkinje Fibers
- Normal timing is < 3 small squares
- < 0.12 seconds or < 120 milliseconds

Bundle of HIS

- Gateway to the ventricles
- Back up rate 25-40bpm

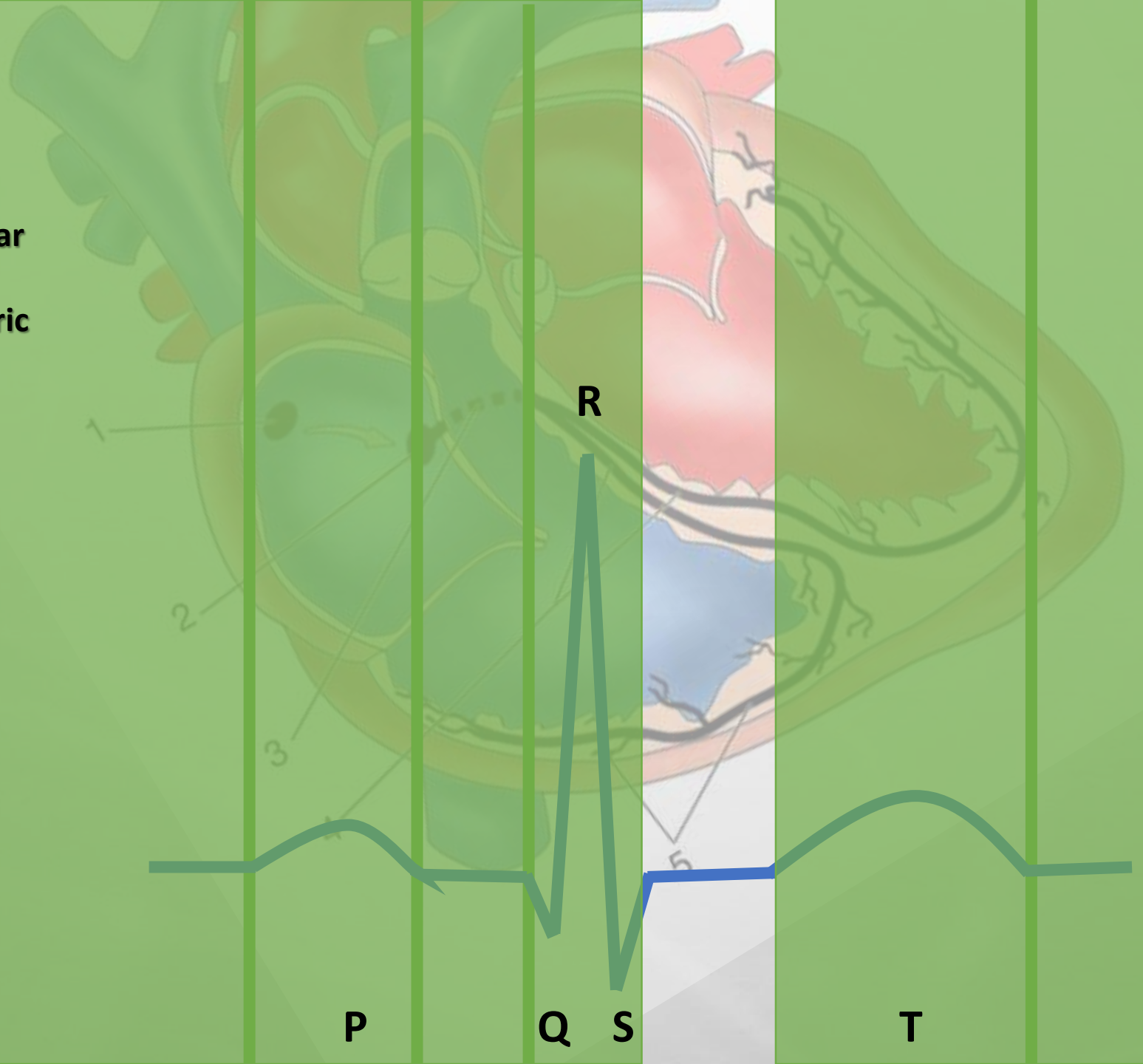
Bundle Branches and Purkinje Fibers

- Maintain Ventricular synchrony
- Back up rate 15-30bpm



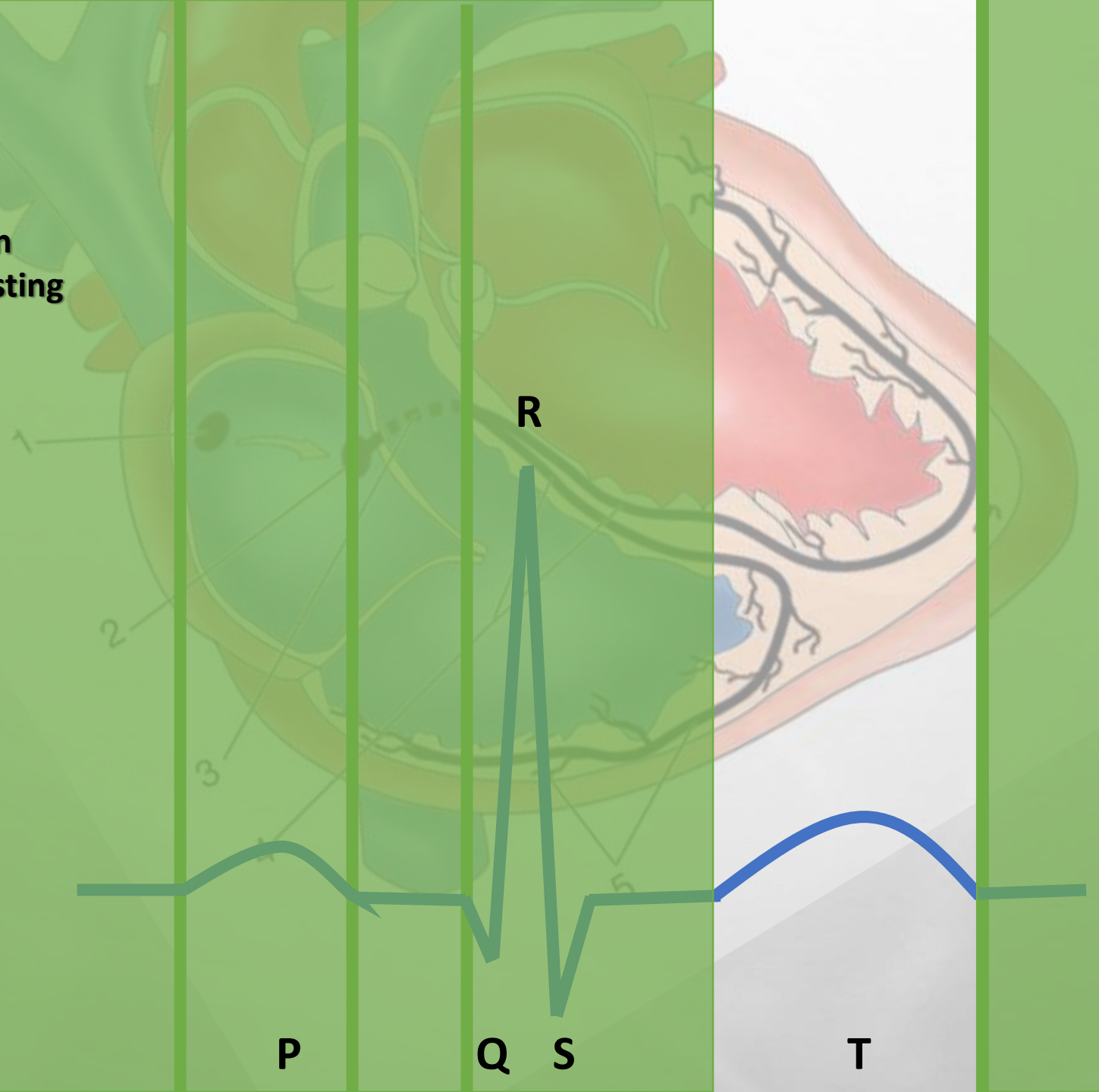
ST Segment

- **End of Ventricular contraction**
- **Normal Isoelectric**

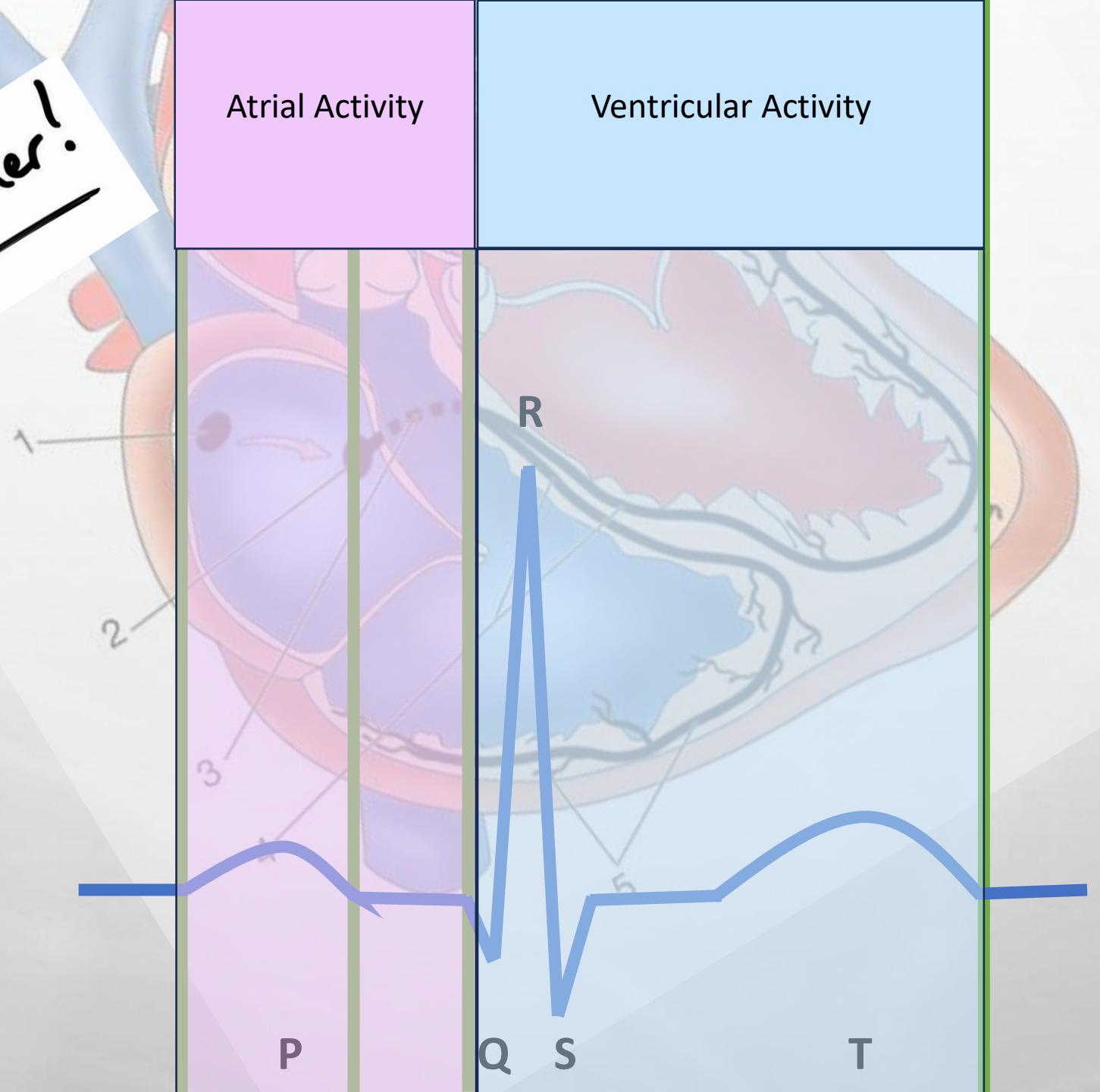


T Wave

- **Ventricular Relaxation**
- **Heart returning to resting state**

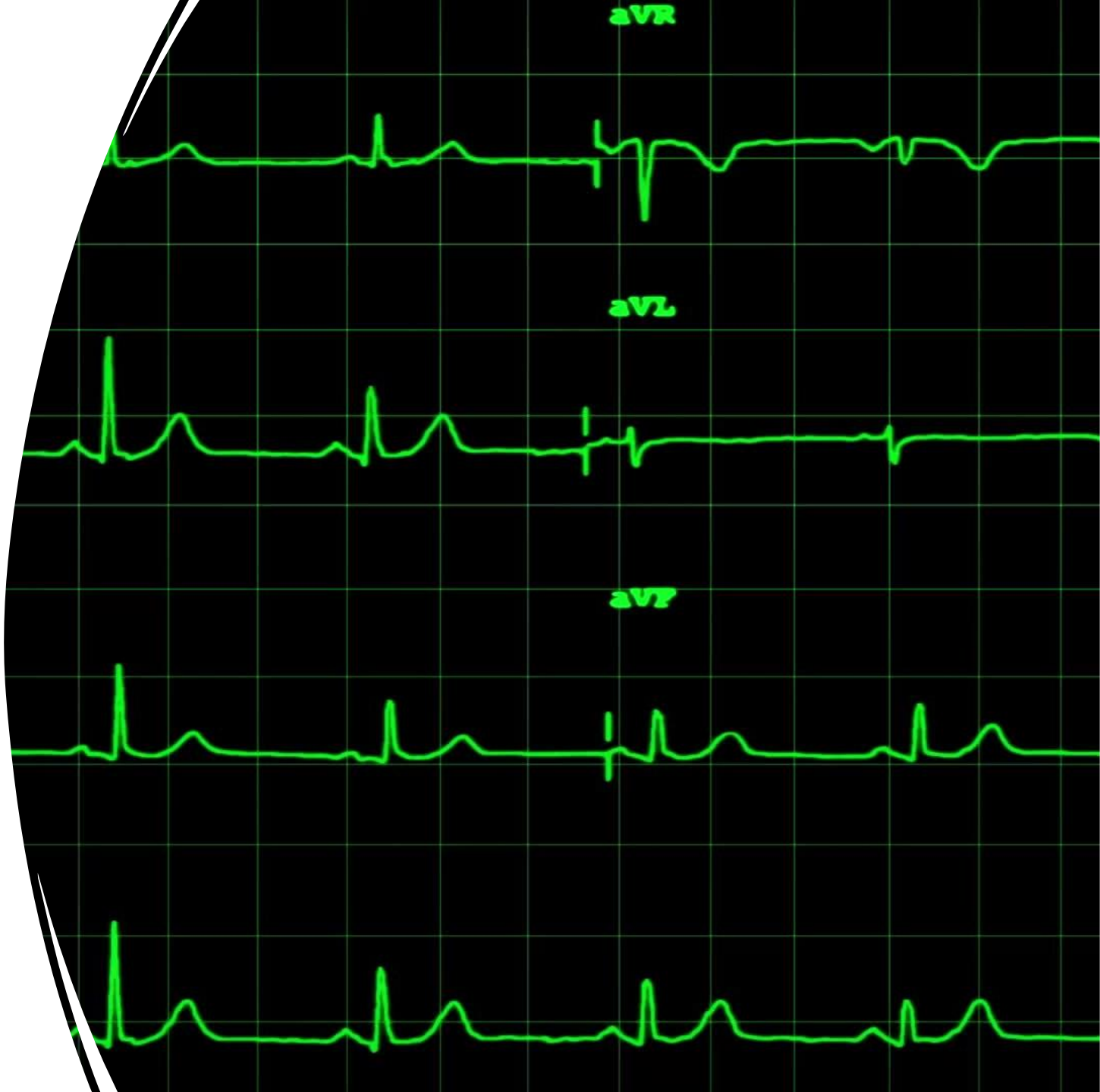


Let's Pull Together!



Why bother cardiac monitoring?

- Baseline assessment for cardiac function
- Understand the patients ‘normal’
- Recognise deterioration



Rhythm recognition

Is there electrical activity?

Is the rate fast or slow?

Is the rhythm regular or irregular?

Is the QRS complex broad or

Can you see atrial activity?

How is the atrial activity related to

<60 bpm = bradycardia 60-100 bpm = normal >100 bpm = tachycardia

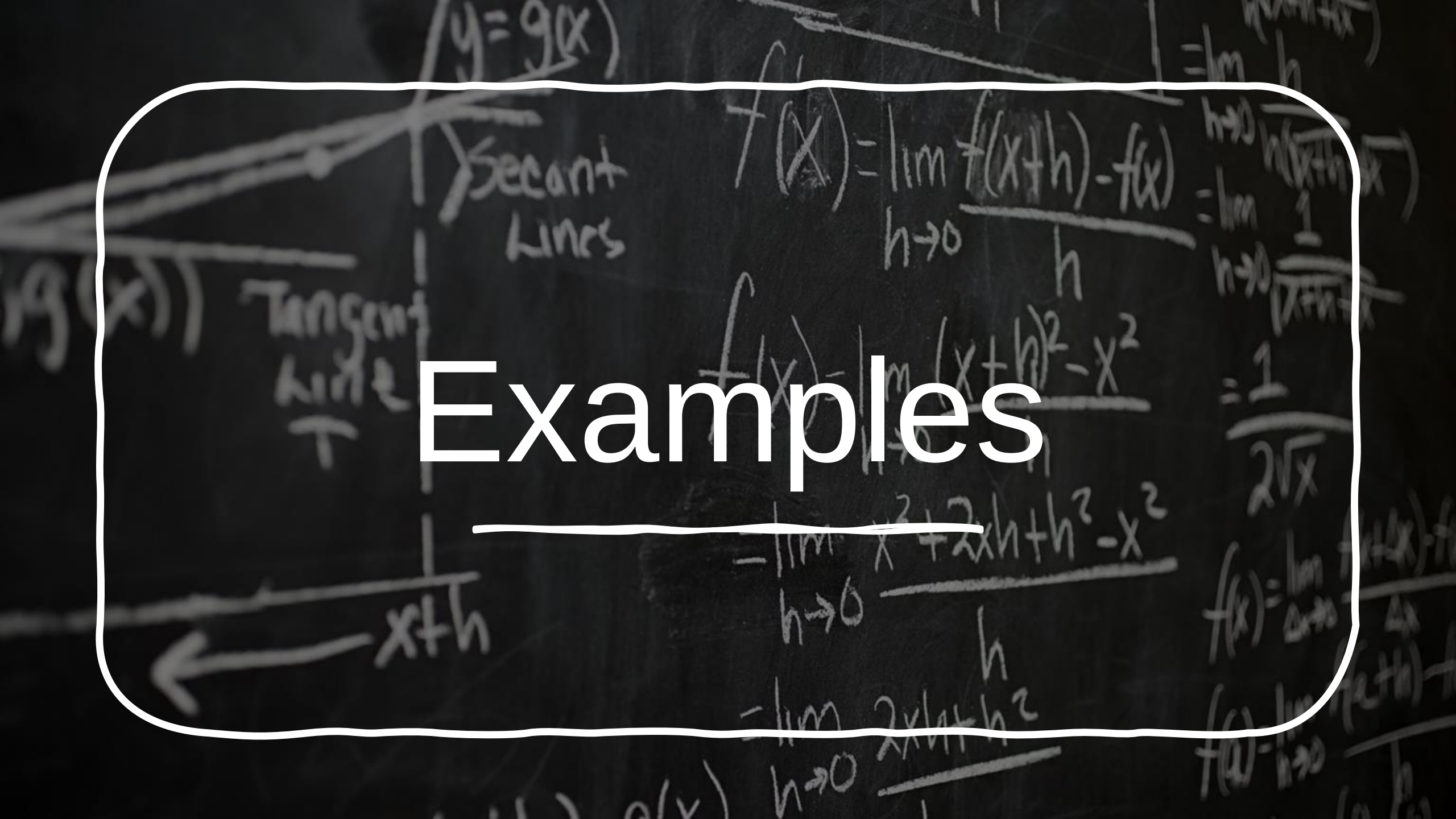
If irregular, try to establish if there is a pattern to the irregularity

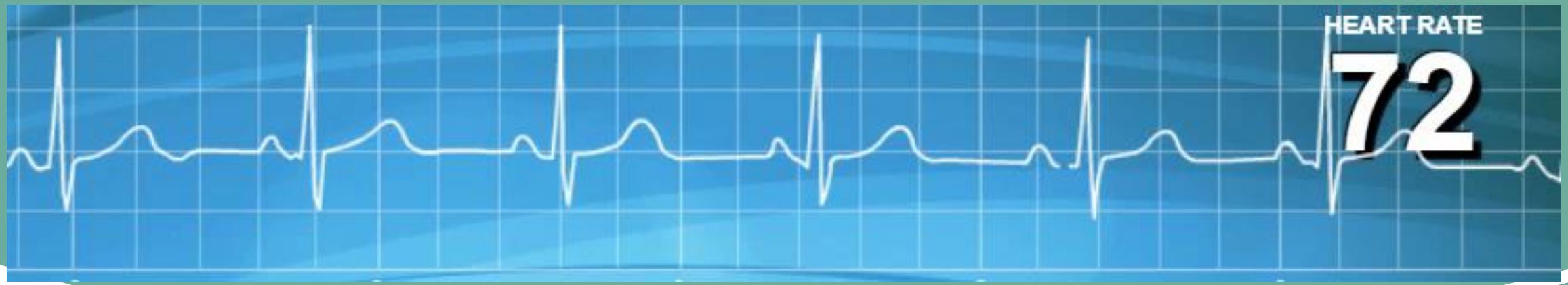
Normal is narrow (<3 small squares or 0.12 secs)

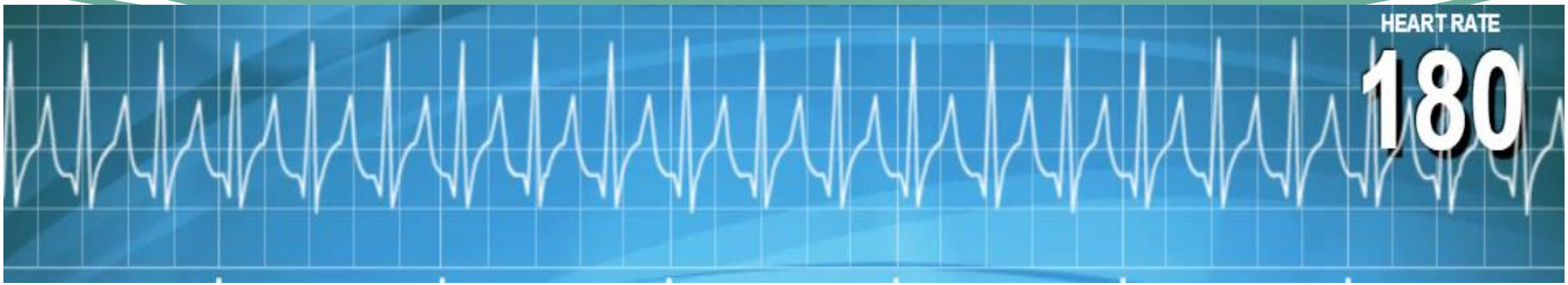
Broad QRS suggests a Ventricular issue

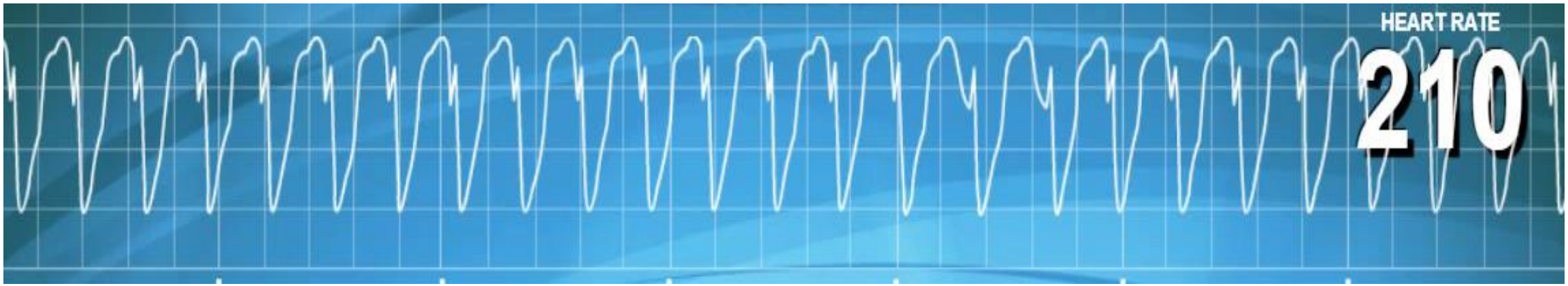
Look for P Waves or any activity before the QRS complex

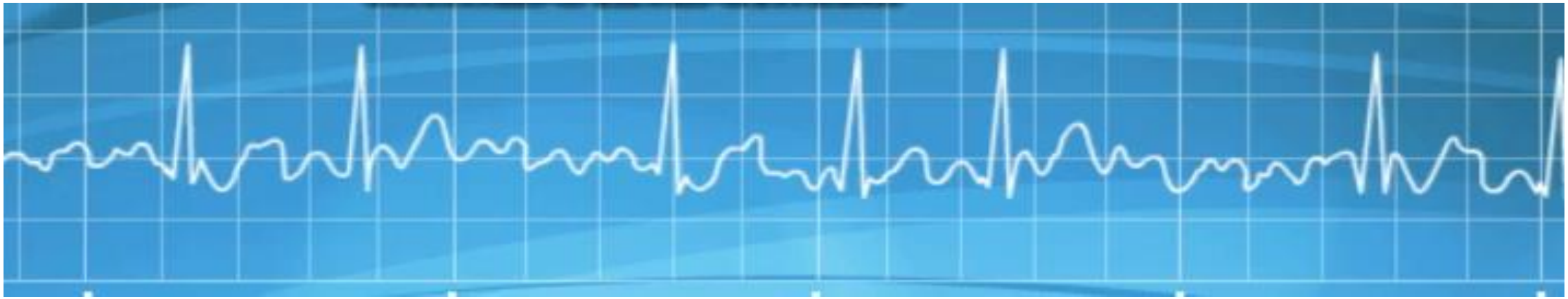
How are the P Waves (if present) related to the QRS complexes? Should be a 1:1 ratio.


Examples


HEART RATE
72


HEART RATE
180


HEART RATE
210

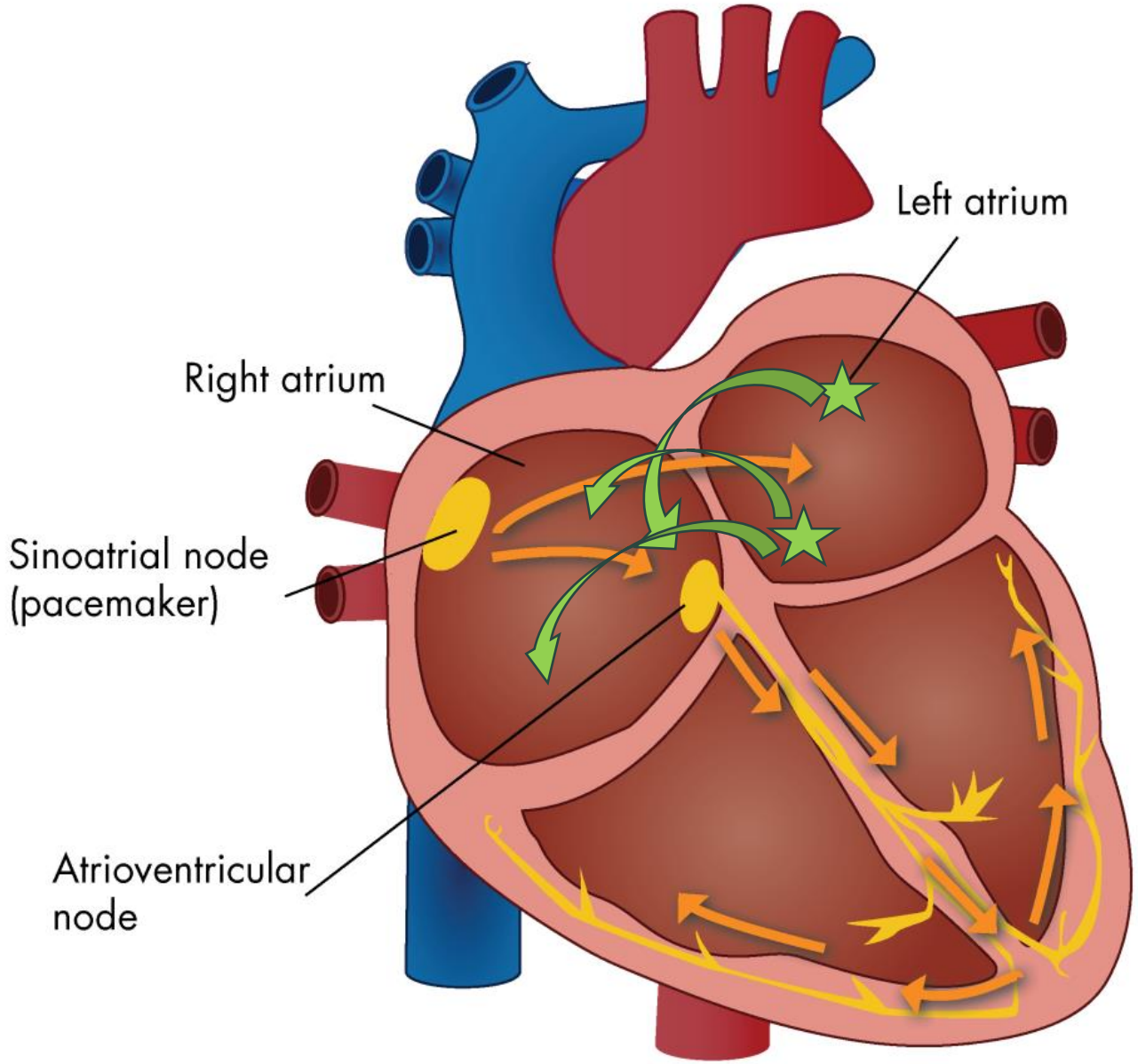


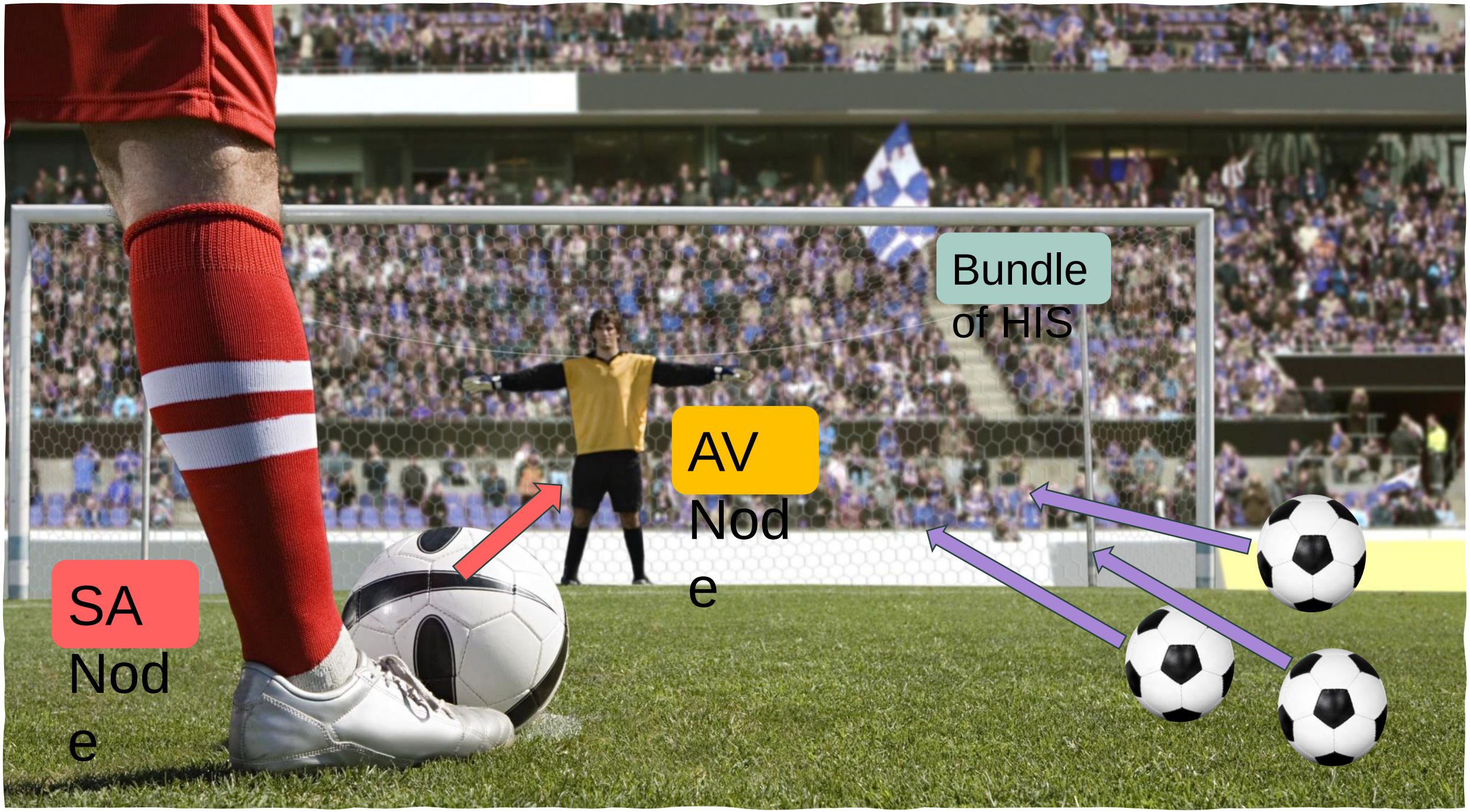
Atrial fibrillation (AF - RVR)

- New onset - Rapid Ventricular Response
- Irregularly irregular
- Narrow QRS complex
- Absence of P waves

Understanding AF

- Atrial arrythmia → disorganised electrical activity causing ineffective atrial contraction and irregular ventricular contraction
- Paroxysmal, persistent or permanent




Bundle of HIS
AV Node
SA Node

Priorities in treating fast AF

Assess using ABCDE approach

LIFE THREATENING FEATURES?

Yes?

1. Shock 2. Syncope 3. Myocardial Ischaemia 4. Severe heart

DC Cardioversion

No?

Identify and treat underlying cause

Anticoagulation

Priorities in treating fast AF

PATIENT REASSURANCE

ASK FOR HELP

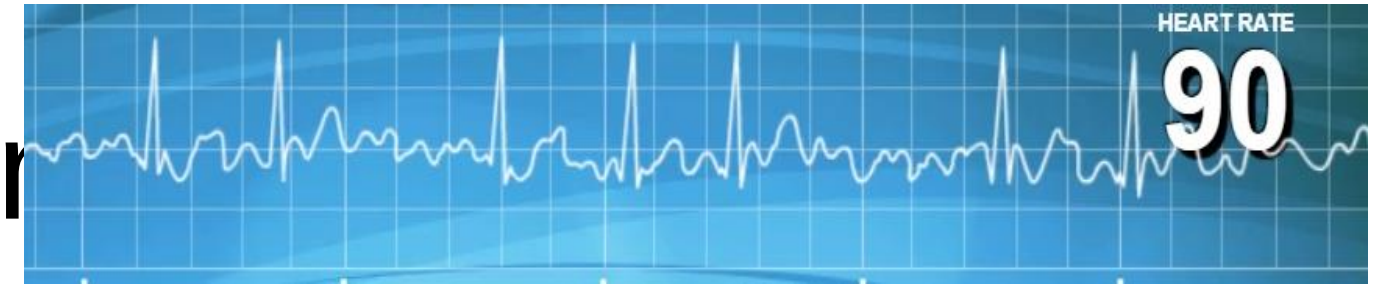
ABCDE ASSESSMENT

INVESTIGATIONS

RATE VERSUS RHYTHM CONTROL

ANTICOAG.

Rate VS Rhythm



Rate Control

- Beta-Blockers (Bisoprolol/Metoprolol)
- Digoxin (slow acting in pts with increased sympathetic tone)
- Verapamil – cardiac depressant effect

Rhythm Control

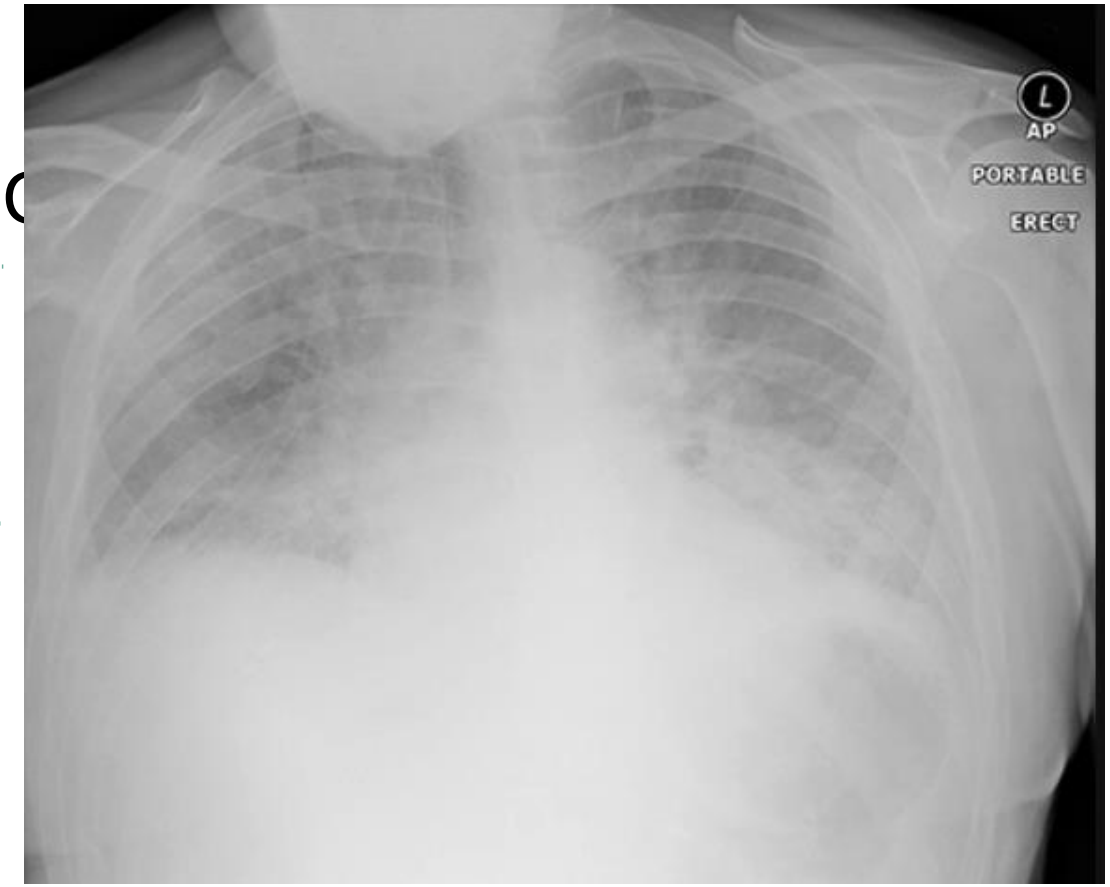
- Electrical (Synchronised D.C Cardioversion)
- Used in episodes of haemodynamic comprise
- Chemical (Amiodarone)

Clinical Sce

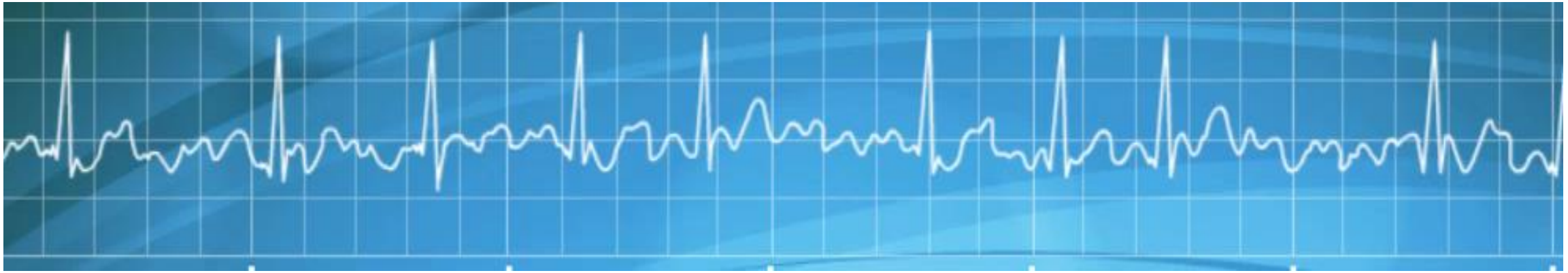
- Donald Sim (68); ♂
- CICU → C.A.B.G x3 [2 POD]
- Increased O2 demand escalating NC

0.75

- CXR; acute pulmonary oedema
- Oliguria – UO 20/20/10
- Imp: Pulmonary Oedema
- Plan: 40mg IV Furosemide
- Assessment → UO: 200/220/160



HFNC: 50L / FiO2 0.3



Cardiac Rhythm: Atrial Fibrillation (Fast AF)

Priorities?
Thoughts?

R.R 24/min SpO2 86% HFNC 50L / FiO2 0.3

HR 160bpm 86/44 (58)
Noradrenaline 2ml/hr

nediate Nursing managem

R.R 24/min SpO2 86% HFNC 50L / FiO2 0.3
Hypoxia due to increased myocardial oxygen demand
Increase oxygenation

HR 160bpm 86/44 (58) Noradrenaline 2ml/hr

Reduction to Cardiac Output due to loss of atrial 'kick' → decreased ventricular filling
BP = CO x SVR Fluid challenge or increase vasopressors

ABG → Assess for Metabolic abnormalities & electrolyte replacement Replace K+

12lead ECG → Confirm the diagnosis of AF + assess for evidence of ischaemia

Impression: Hypokalemia secondary to Furosemide

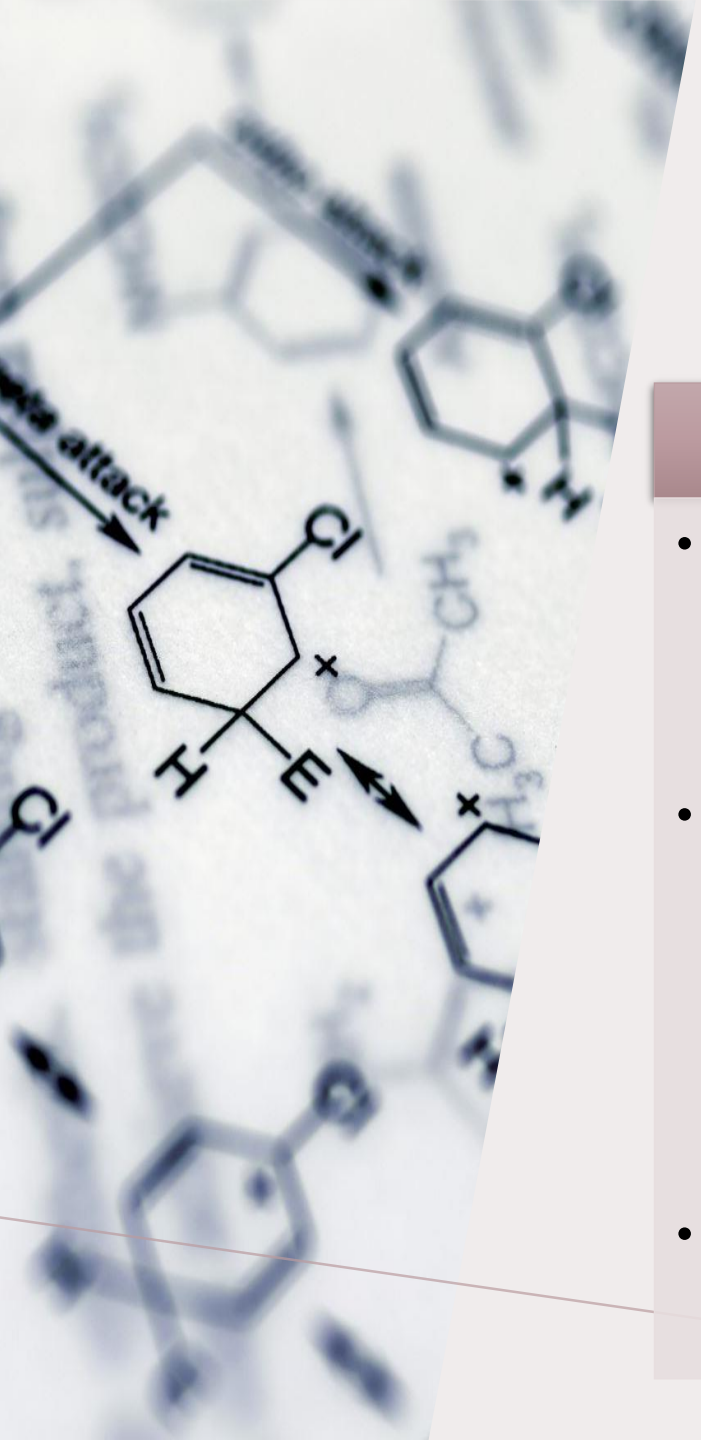
Plan: Titrate FiO2, Titrate Noradrenaline, replace electrolytes

RADIOMETER ABL90 SERIES

ABL90 WARD 201		09:15	06/10/2023
PATIENT REPORT	Syringe – S 65uL	Sample #	18779

50L / 30% HFNC

Identification			
Patient ID	0123456		
Patient last name	Mouse		
Patient first name	Mickey		
Sample Type	Arterial		
Operator	Neil Maddison		
Blood gas values			
cH+	28 nmol/L		
pH	7.49	[7.350 – 7.450]	
pCO2	3.9 kPa	[4.3 – 6.4]	
pO2	8.4 kpa	[11.1 – 14.4]	
Oximetry values			
Hct	30.6 %		
tHb	81.0 g/L	[115 – 178]	
sO2	86.0 %	[94.0 – 98.0]	
FO2Hb	92.6 %	[94.0 – 98.0]	
FCO2Hb	1.2 %	[0.0 – 3.0]	
FHHb	2.8 %	[0.0 – 2.9]	
FMetHb	0.3 %	[0.0 – 1.5]	
Electrolyte values			
K+	3.1 mmol/L	[3.5 – 5.1]	
Na+	138 mmol/L	[136 – 145]	
Ca2+	0.991 mmol/L	[1.15 – 1.33]	
Cl-	101 mmol/L	[98 – 107]	
Metabolite values			
Glu	7.1 mmol/L	[4.1 – 5.6]	
Lac	2.4 mmol/L	[0.2 – 1.8]	
Acid-base status			
Base	-3.2 mmol/L		
HCO3- (P, st)	22 mmol/L		
HCO3- (P)	18.4 mmol/L		



ELECTROLYTE AND METABOLIC DERANGEMENT

POTASSIUM

- **Hypo**kalaemia can lead to result in AF, PVCs and ventricular arrythmias
- **Hyper**kalaemia can lead to asystole

- Target a potassium between 3.5 – 5.0 mmol/L

MAGNESIUM

- Helps to regulate regular cardiac conduction and the movement of potassium and calcium over the cardiac muscle
- Magnesium deficiency is known to lead to excessive Potassium wastage!!!

- Target a Magnesium >/= 1.0 mmol/L

CALCIUM

- Hypocalcaemia is associated with QT prolongation and ventricular arrythmias

- Target a Calcium >/= 1.0 mmol/L

Brady-arrythmias

Sinus bradycardia

- HR slow <60 bpm
- Regular
- Narrow QRS
- P wave : QRS comples → 1 : 1 ratio
- **If symptom free** – no treatment is required but ongoing monitoring for further conduction changes

Understanding brady-arrhythmias

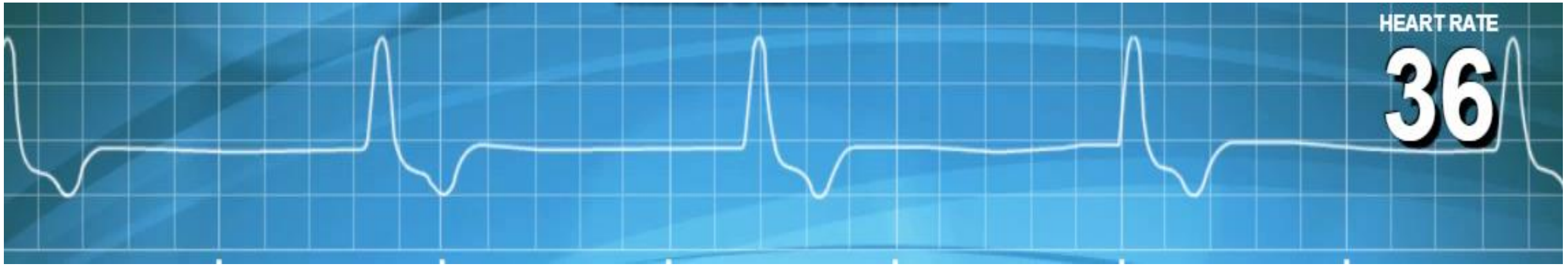
Defined as a resting Heart Rate <60bpm

Conduction issues can be delayed or blocked at any point in the conduction system

Potential causes:

- Physiological (athletes or sleeping)
- Cardiac origin (AV blocks or Sinus-node disease)
- Non-cardiac origin (Vasovagal, hypothermia, hyperkalaemia)
- Drug-induced (Beta-blockers, Digoxin, Amiodarone)

Often can be asymptomatic, but may be cardiovascularly unstable if there is a drop in Cardiac Output → reduced end-organ perfusion

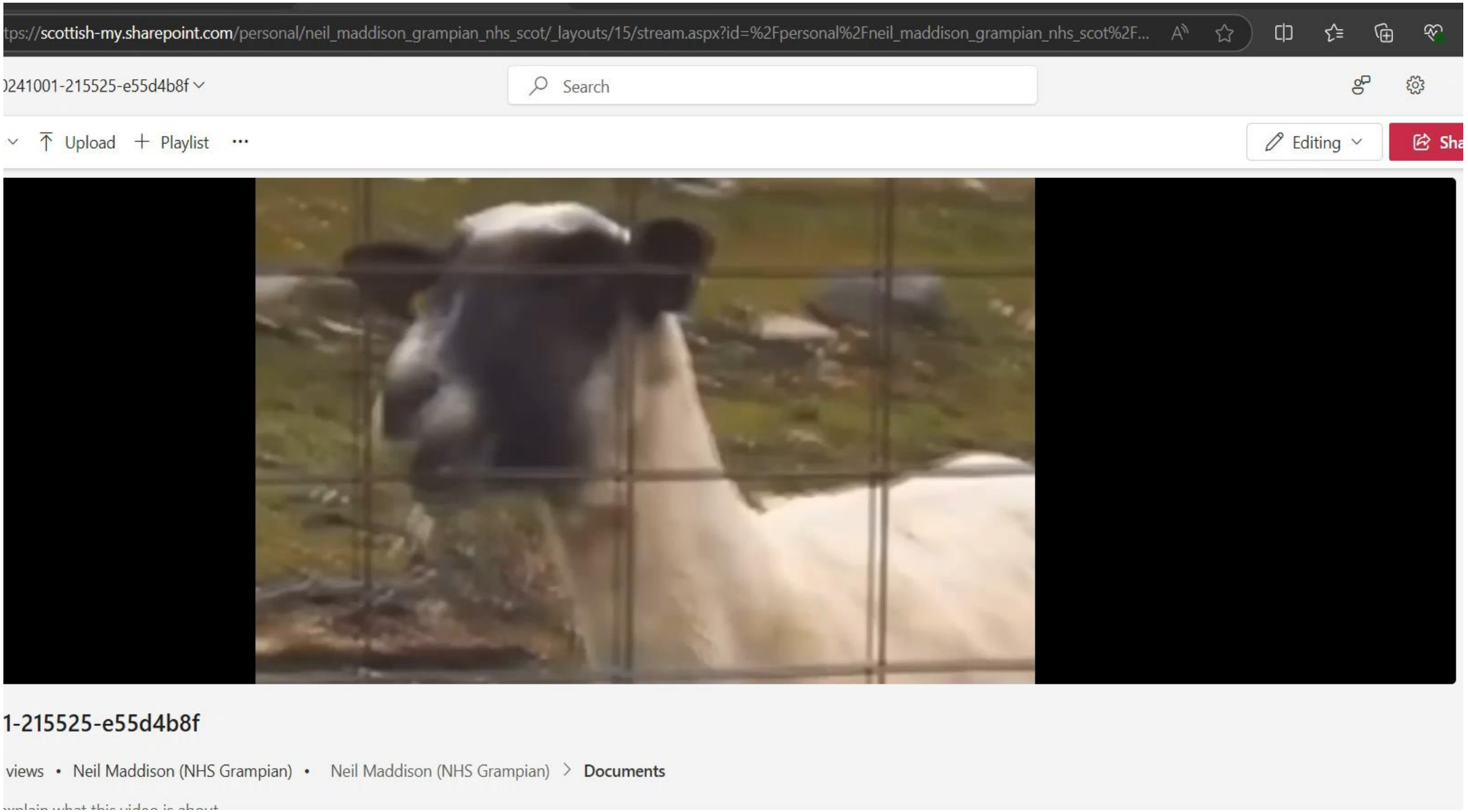


Idioventricular rhythm "Rhythm of the last resort"

- Broad QRS
- No P-waves : QRS complex ratio → 0 : 1 ratio
- Hearts safety mechanism to prevent ventricular standstill (asystole) when no impulse is conducted above the His-Purkinje system

Prioritie

Though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Nursing Care and Medical Management

What effect would a slow ventricular HR have on the patients Cardiac Output?

CO = HR X

Nursing Care and Medical Management

DRUGS

Atropine vs Glycopyrrolate

Isoprenaline

Adrenaline

PACING

Percussion

Transcutaneous

Temporary

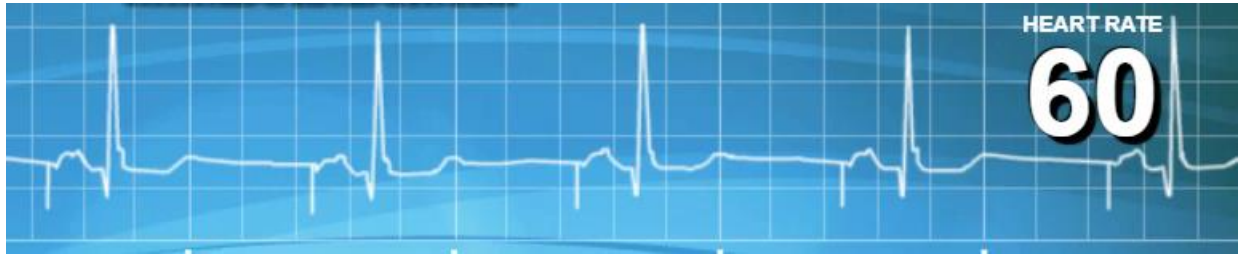
Permanent Pacing

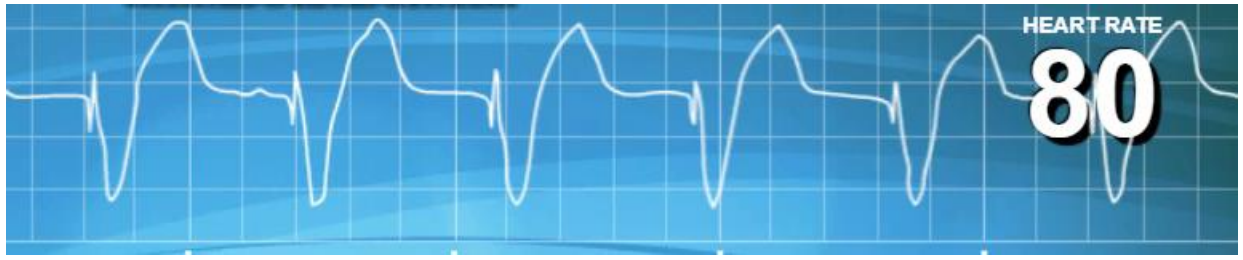
Reassuring the patient + Bedrest

ABCDE

12Lead ECG, bloods, access

Review Prescribed drugs





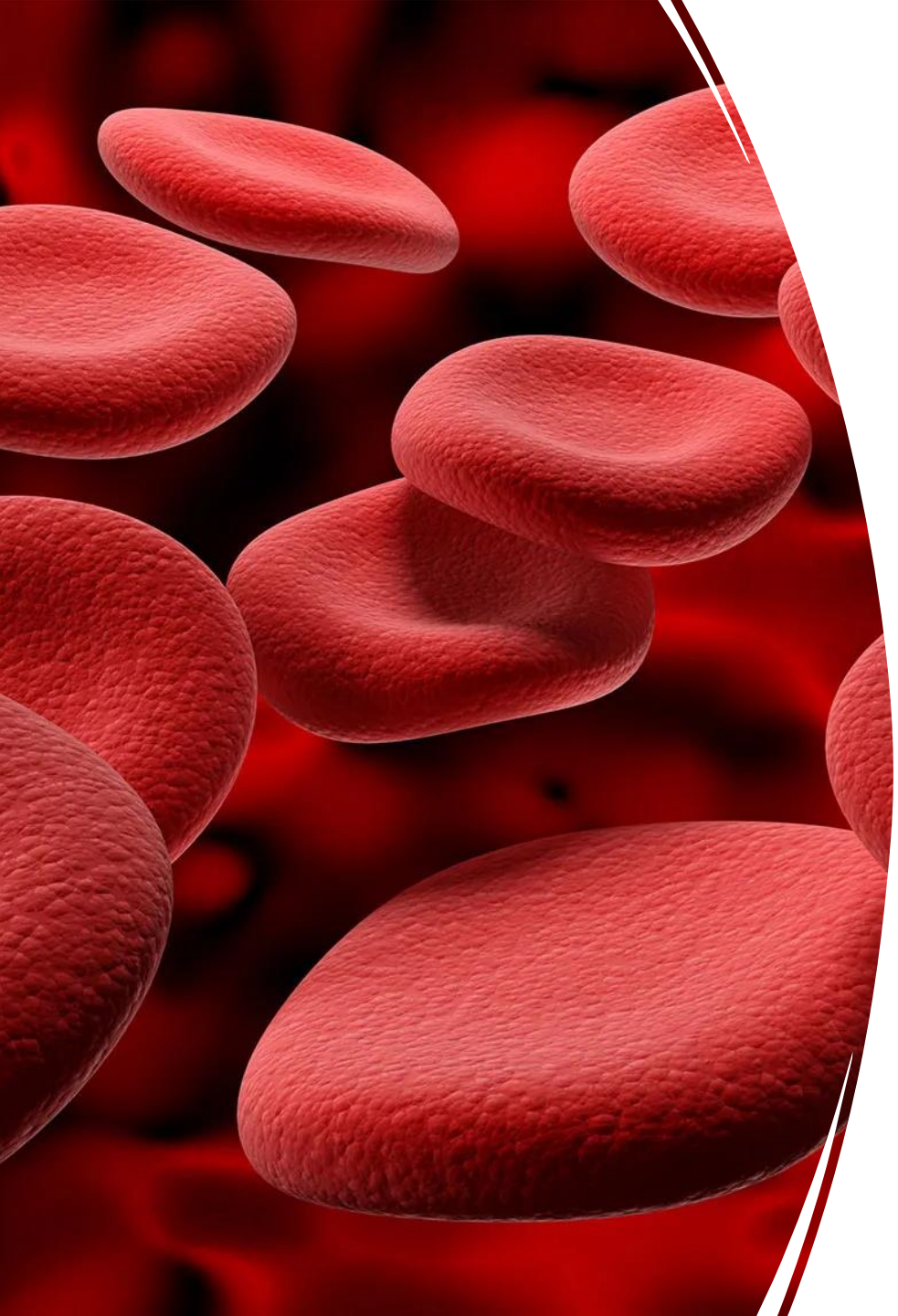
HyperKalaemia

HYPER-K-al-AEMIA

RAISED

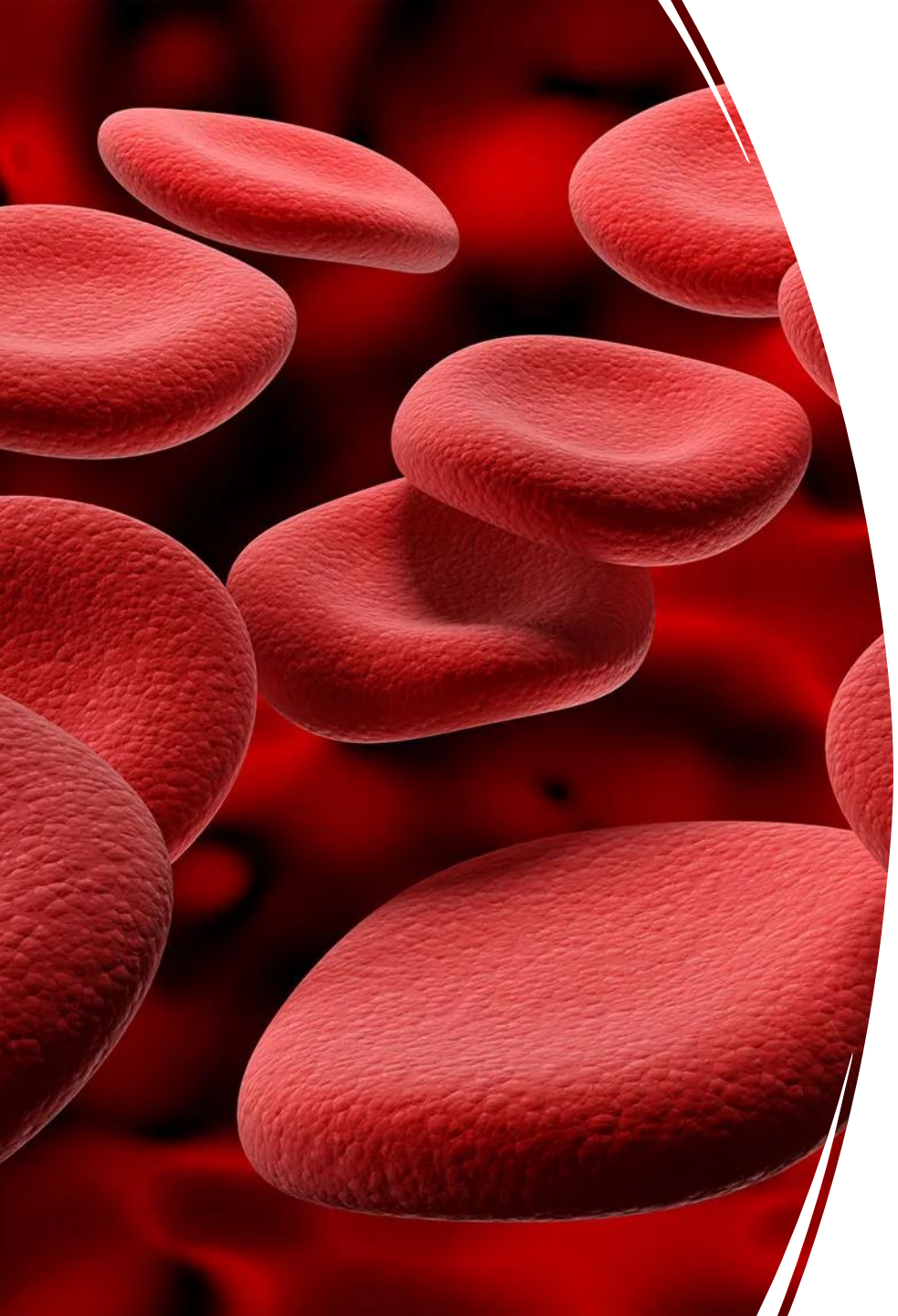
POTASSIUM

BLOOD



Understanding Hyperkalaemia

- Blood is made up of plasma, RBC, WBC, platelets
- Potassium moved in & out of cells in the Na+/K+ pump
- Normal serum K+ → 3.5 - 5.0 mmol/L
- Within the red blood cell potassium levels → K+ = 150 mmol/L
- Rely on homeostasis mechanism !!



Causes of hyperkalaemia

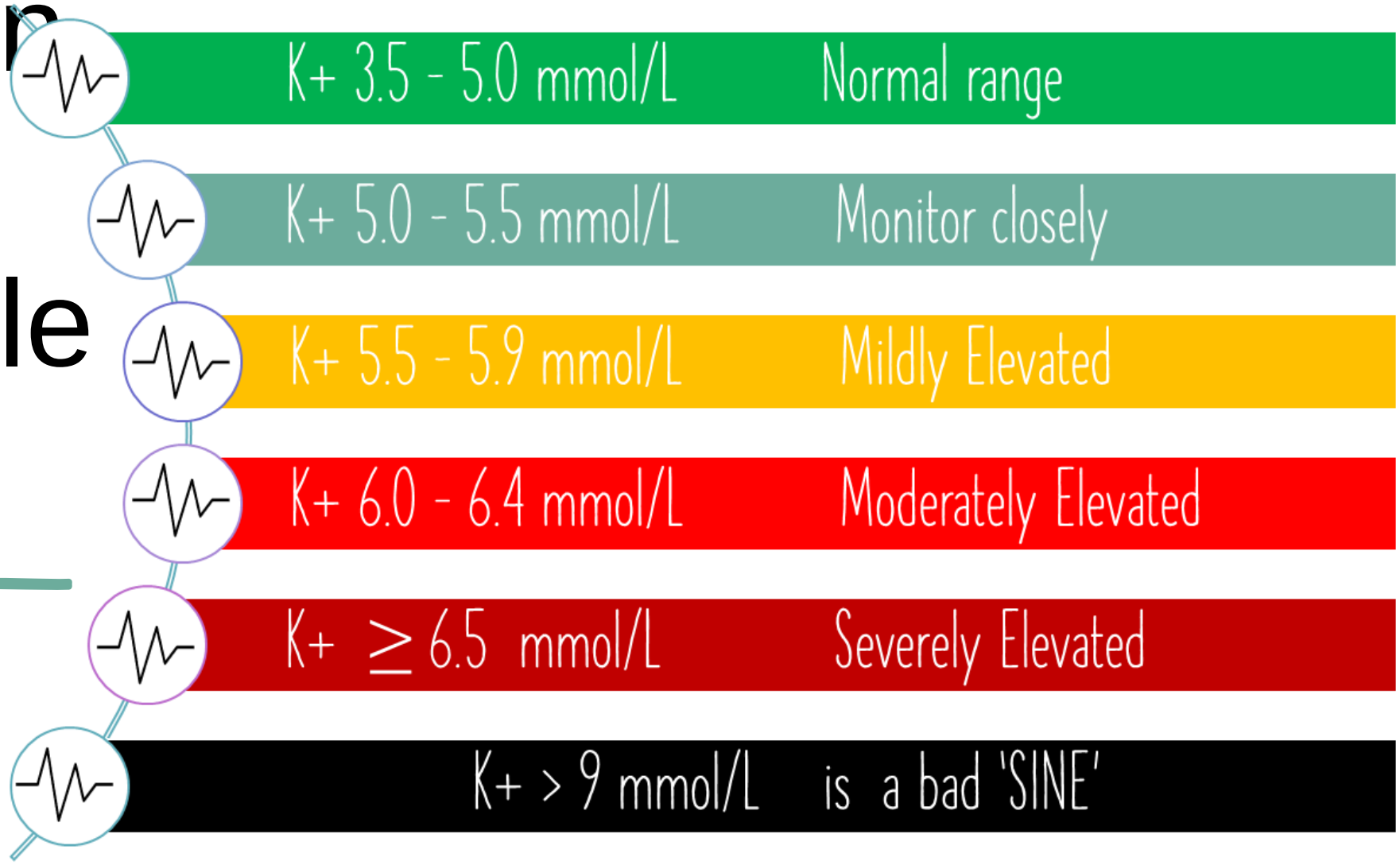
- Excreted by kidneys (90%) and GI tract (10%)
- Medications
  - Ace-inhibitors, NSAIDs, Potassium-sparing diuretics
- Trauma + Burns + Long lies
- DKA (lack of insulin)
- IV Fluids

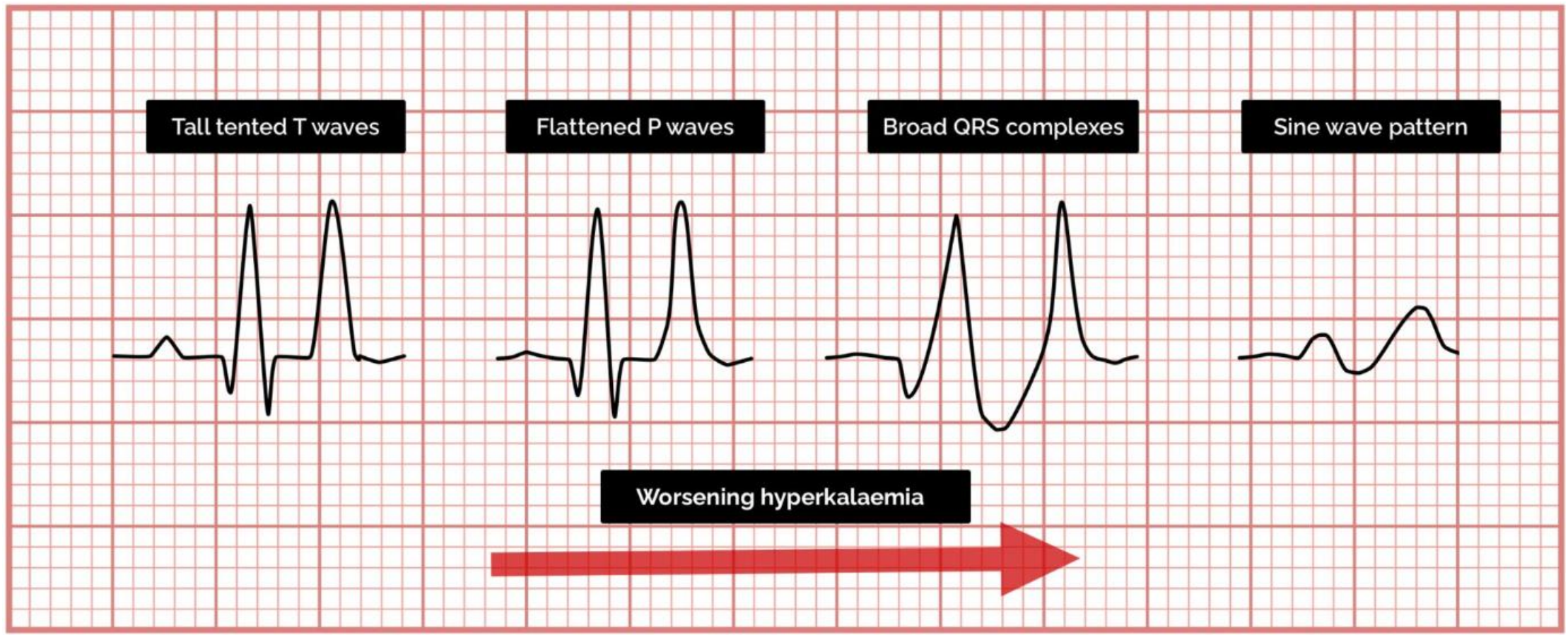
Worsening Hyperkalaemia

- Severe Hyperkalaemia is a medical emergency due to the risk of life-threatening arrythmias
- Reversable causes of cardiac arrest
- Prompt recognition + treatment is vital

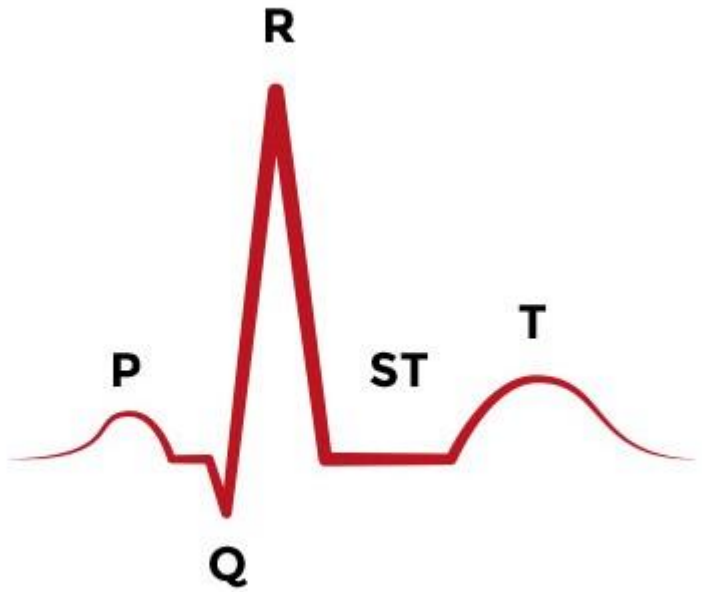
- Complications and changes to ECG worsen as Potassium increases

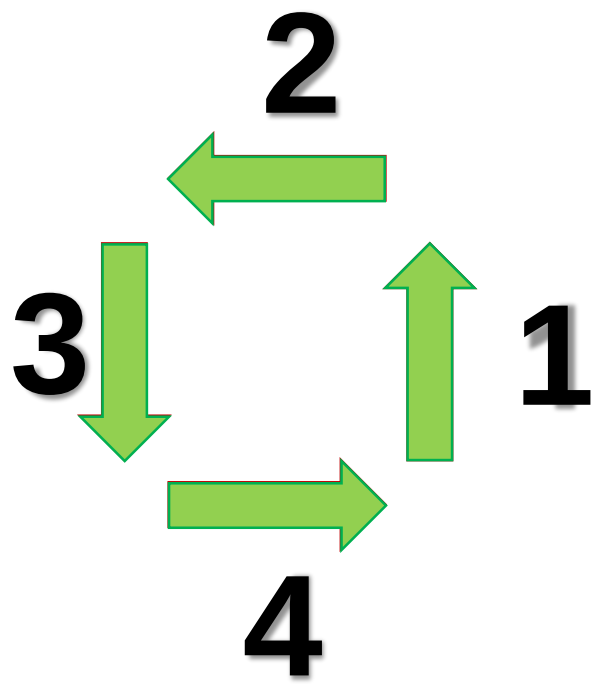
Classifying Hyperkalemia




Tall tented T waves
Flattened P waves
Broad QRS complexes
Sine wave pattern
Worsening hyperkalaemia

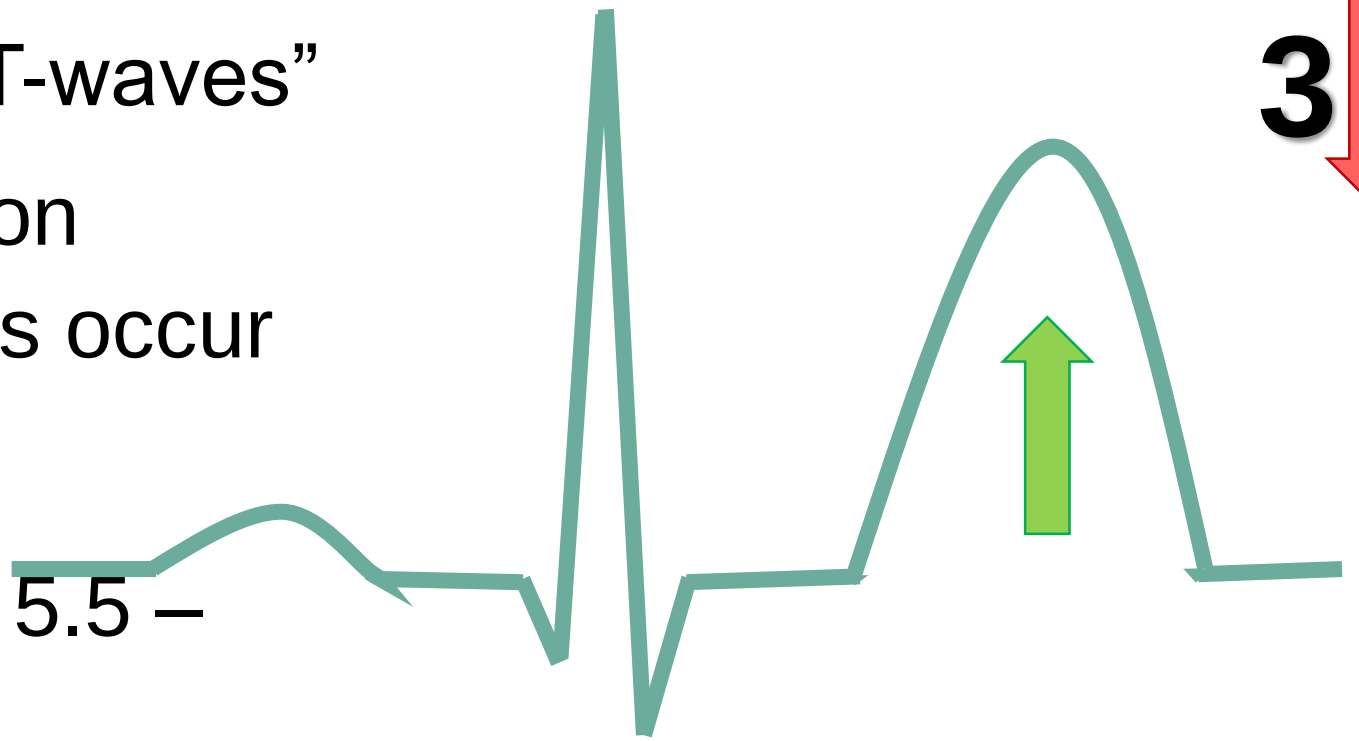
Hyperkalaemia ECG hack

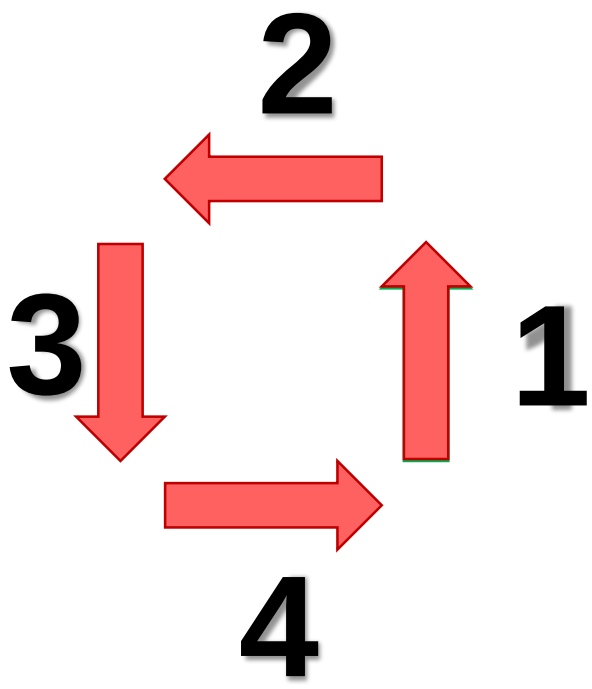




1. Peaked T-waves

- T-wave increases in amplitude
- “Tall tented T-waves”
- Repolarisation abnormalities occur

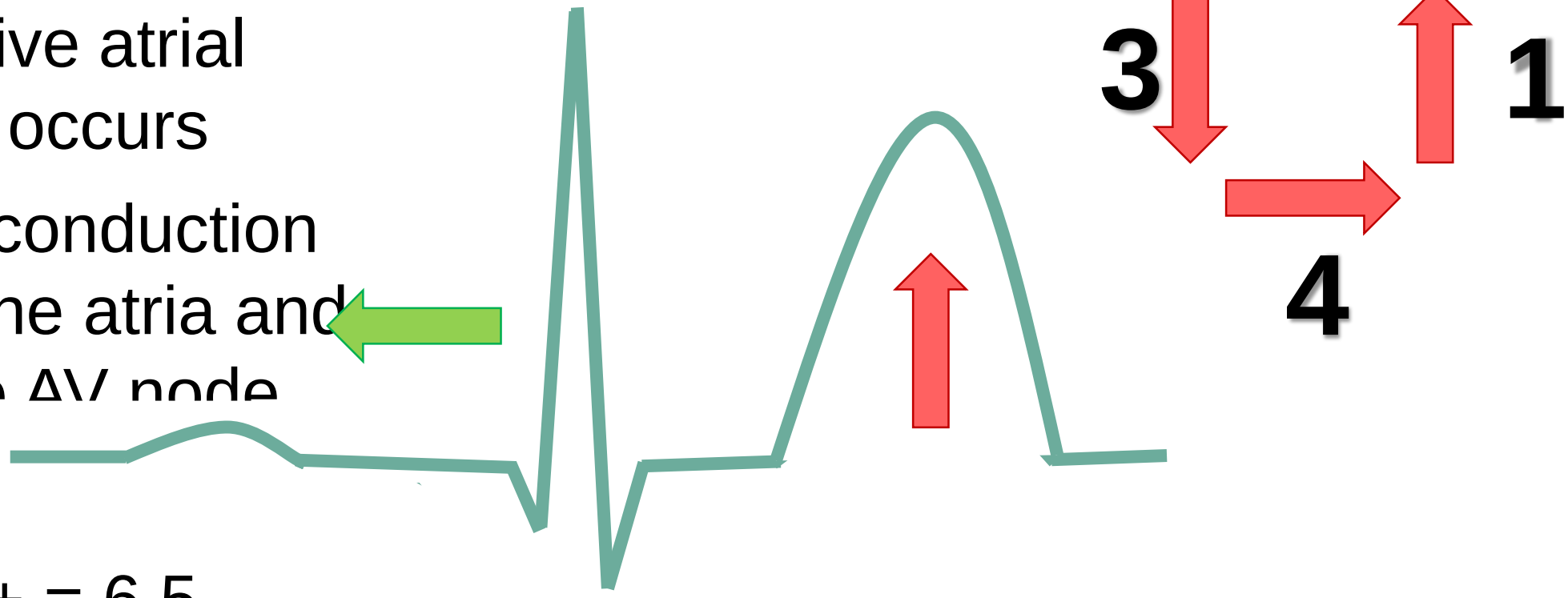




- Serum K+ = 5.5 – 6.5mmol/L

2. Prolonged PR interval

- Widening of P-waves and PR prolongation
- Progressive atrial paralysis occurs
- Delayed conduction through the atria and within the AV node

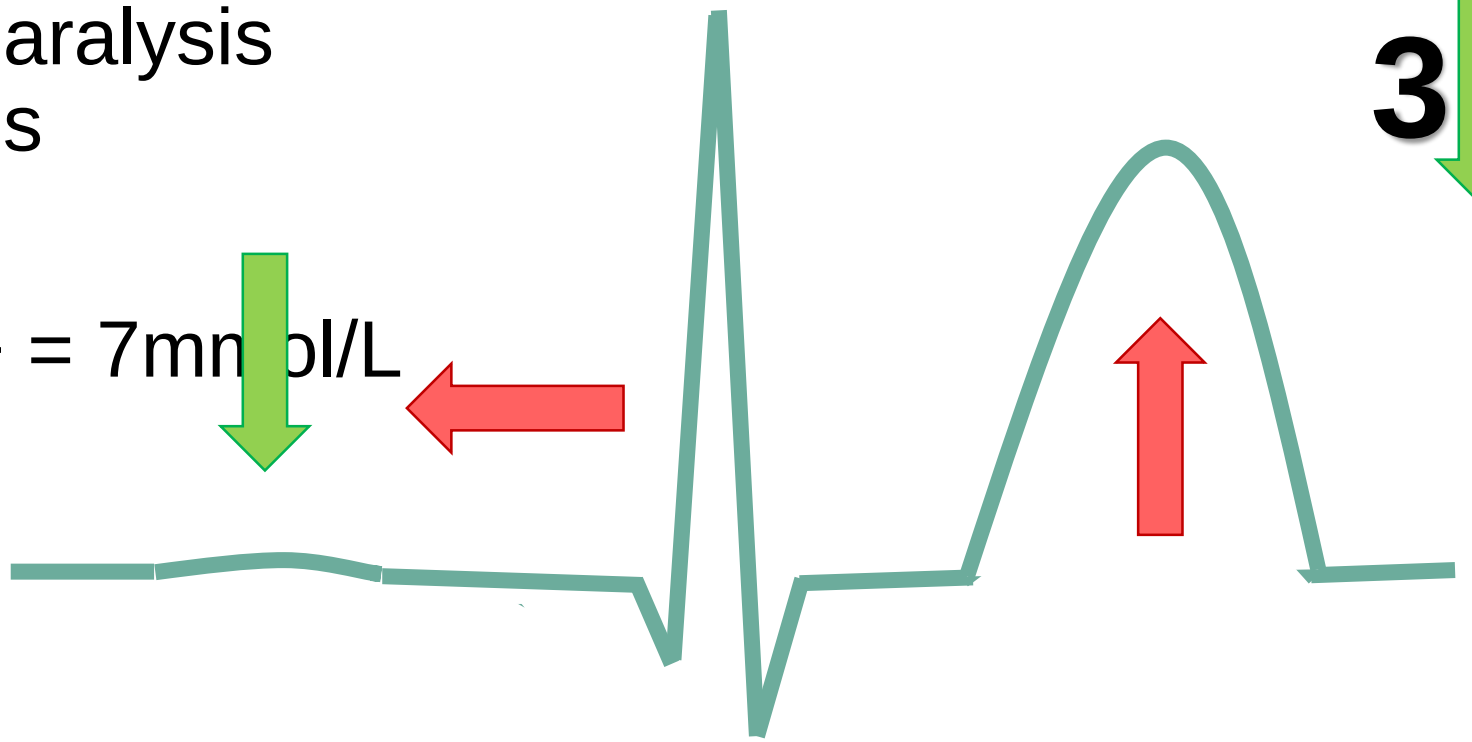


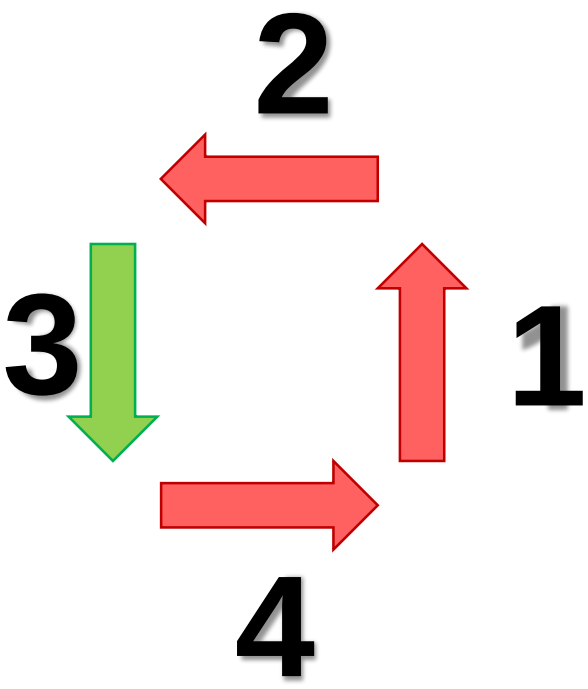
- Serum K+ = 6.5 –

3. Dropped P waves

- P-waves become ‘dropped’
- SA node suppression
- As atrial paralysis progresses

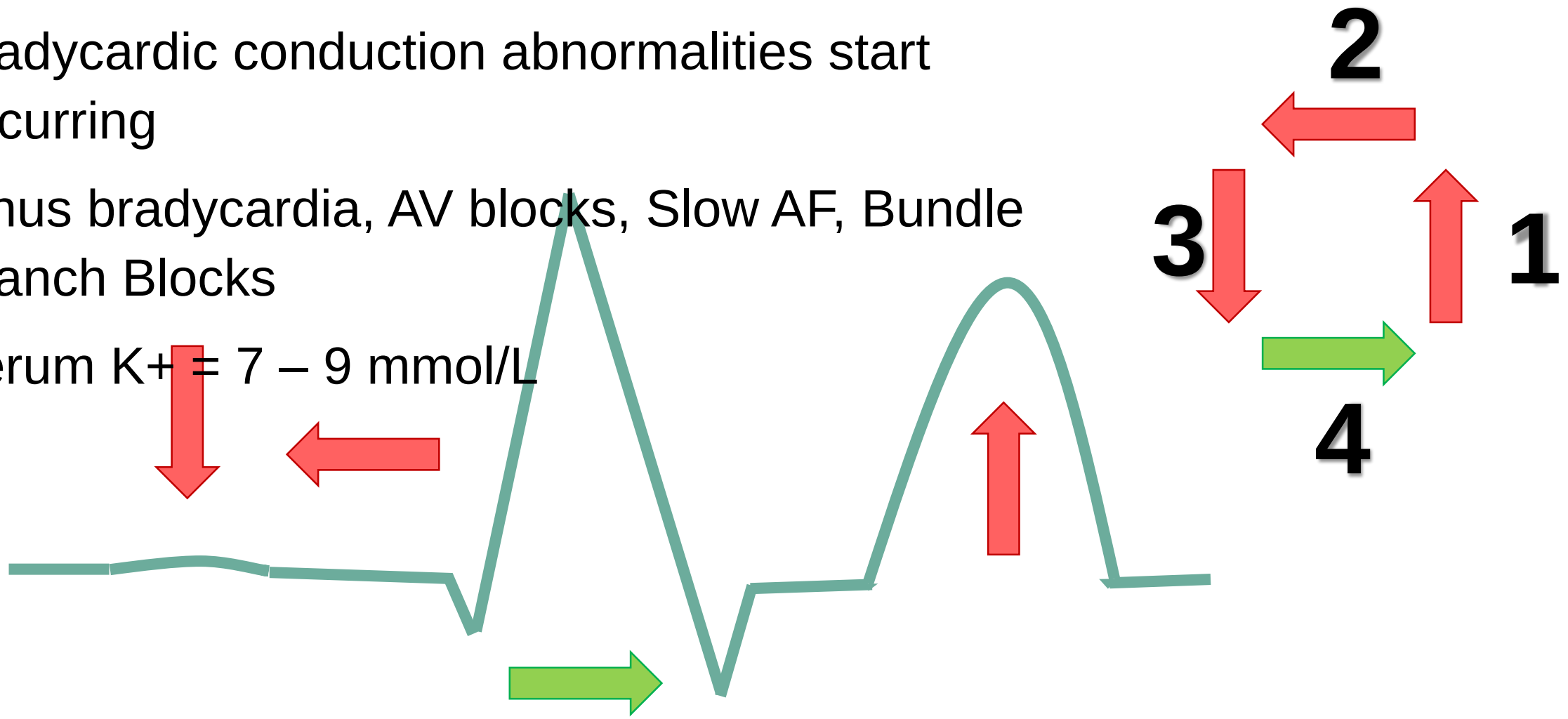
- Serum K+ = 7mmol/L





4. Widening qrs complex

- Bradycardic conduction abnormalities start occurring
- Sinus bradycardia, AV blocks, Slow AF, Bundle Branch Blocks
- Serum K+ = 7 – 9 mmol/L

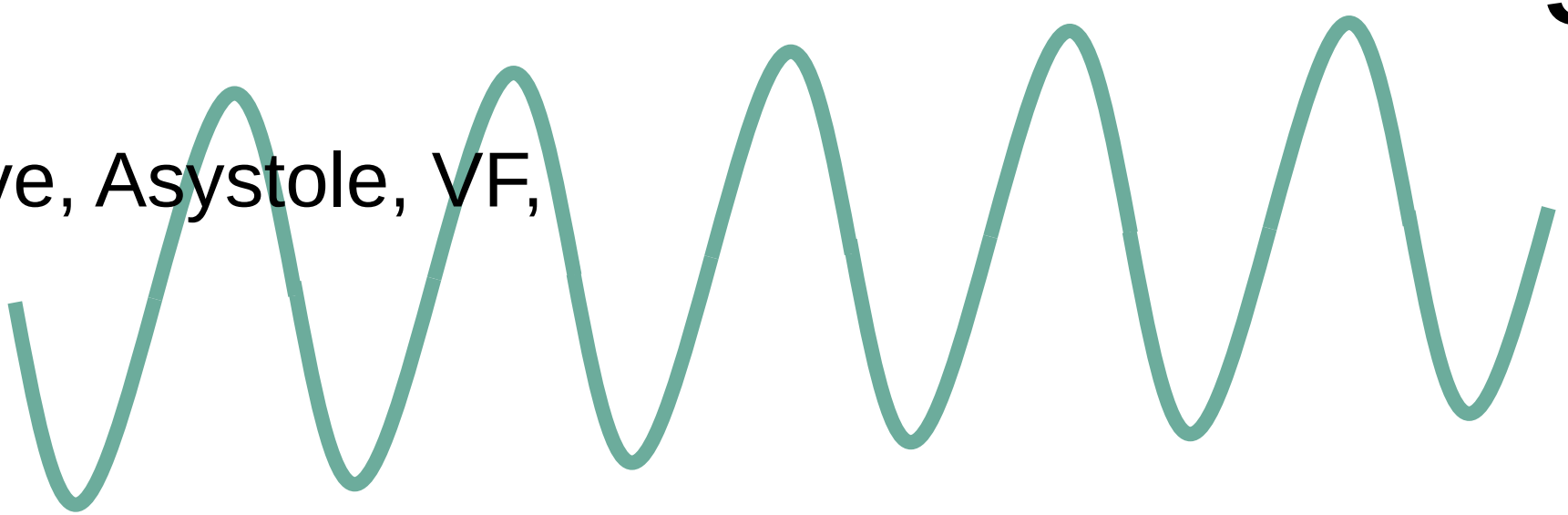


Potassium Greater than 9 is a bad ‘Sine’

- As levels increase above 9mmol/L life threatening arrythmias can begin to occur
- Sine Wave, Asystole, VF, PEA

Priorities

Thoughts



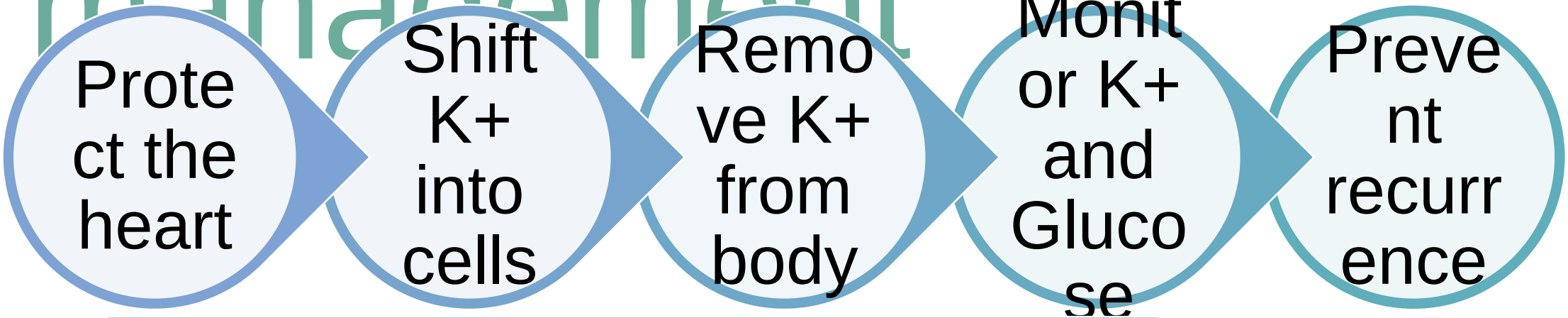
Atrial Paralysis

Potassium Greater than 9 is a

Treatment and Management

- Prompt treatment and ongoing management is essential to prevent hyperkalaemia cardiac arrest
- Goals: stabilise the myocardial from cardiotoxicity, shift & remove K+, and manage the precipitating factors
- Patients often present asymptomatic / non-specific / generalised weakness
- Assessment of effectiveness is monitored using 12lead ECG + continuous cardiac monitoring

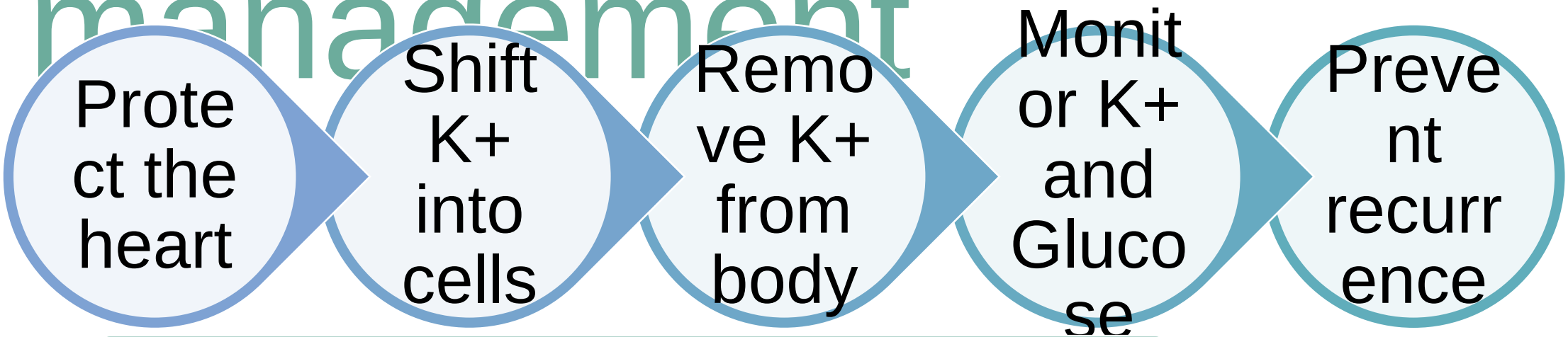
Treatment and management



Protect the heart

- Calcium is known to stabilise the cardiac membrane excitability provoked by excess potassium
- Preventing life threatening arrythmias
- Rapid onset for improving adverse ECG appearances; 3 mins
- Effective for 30-60 mins

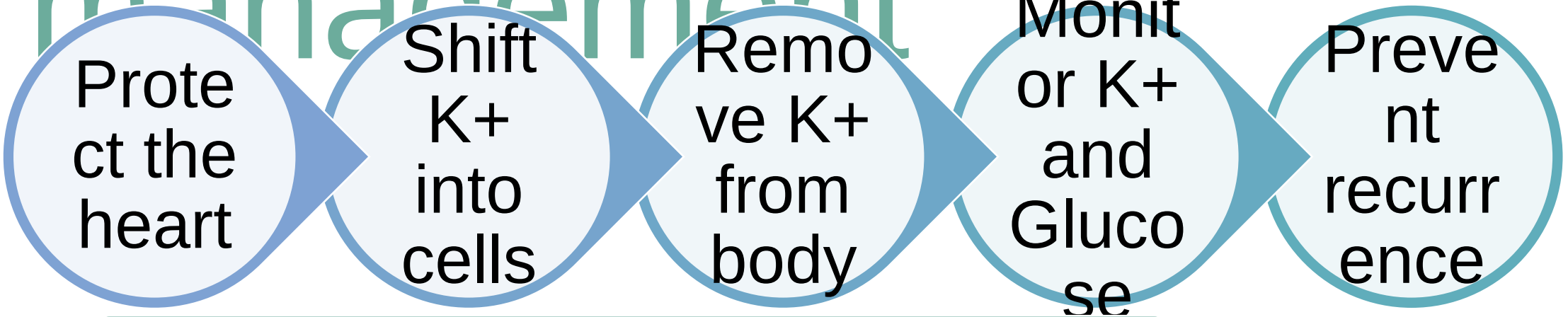
Treatment and management



Shift K+ into cells

- Administer Insulin which activates Na+/K+ pump to move potassium intracellularly
- Onset of effectiveness – 15mins; peaking around 30-60 mins
- Gradual rebound K+ usually after 2 hours
- Effectiveness if increased when given alongside

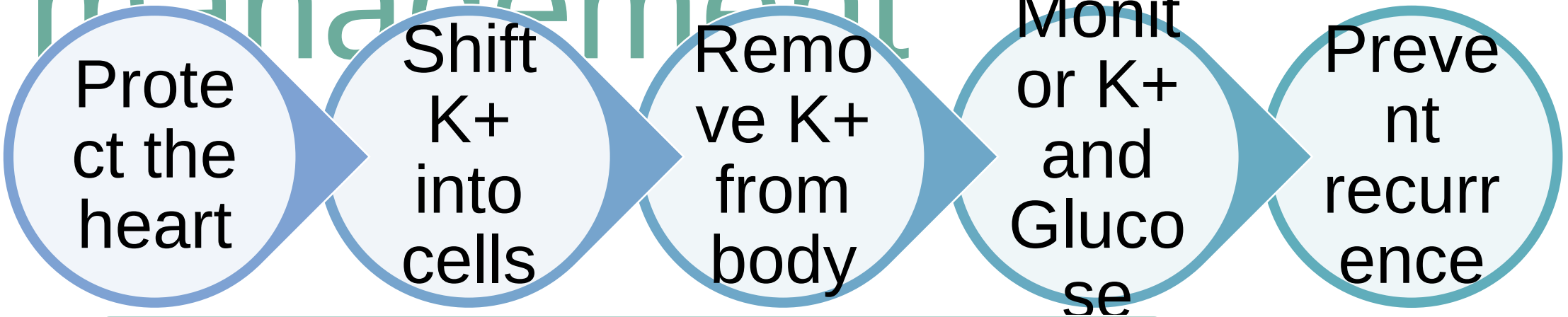
Treatment and management



Remove K+ from body

- Lower whole body Potassium renally with Diuresis or CRRT / IHD
- Growing body of evidence of K+ binders – potential role in delays to dialyse
- Sodium Zirconium Cyclosilicate oral binder that works

Treatment and management



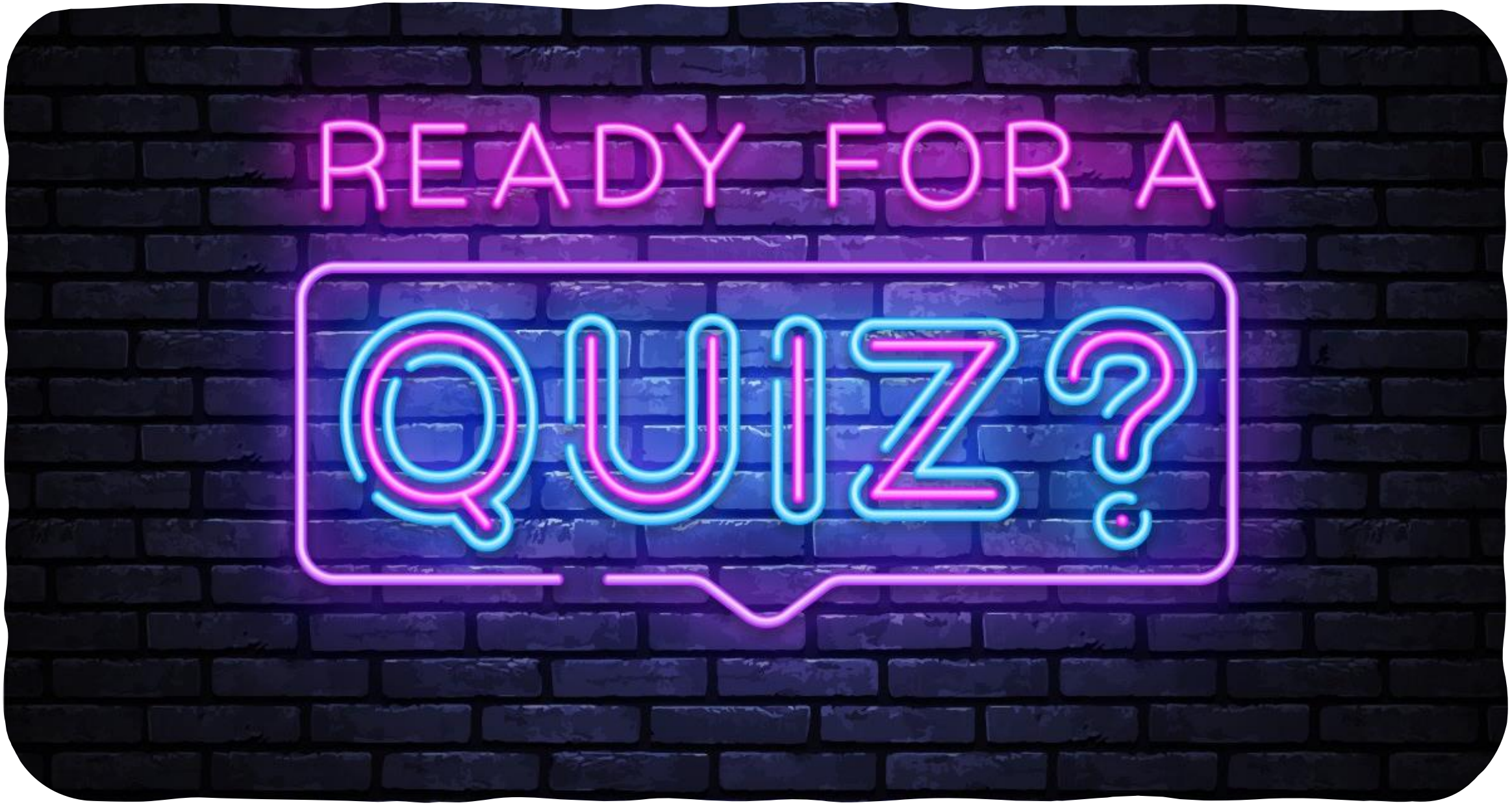
Monitor K+ and Glucose

- Regular monitor of blood glucose is essential following insulin-dextrose infusion to prevent Iatrogenic hypoglycaemia until 6hrs after treatment
- Glucose maintenance infusions should be commenced if Glucose is <7mmol/L pre-treatment

Treatment and management

Prevent Recurrence

- Review prescribed medications that are worsening hyperkalaemia
- Liase with dietitian for nutritional support and avoid constipation
- Regular blood electrolyte monitoring
- Attend regular dialysis in chronic renal failure patients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What is the most common type of cardiac arrhythmia in the ICU?

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Which cardiac rhythm is characterized by a sawtooth pattern on an ECG?

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When does the PR interval on an ECG represent a first-degree heart block?

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Why is bradycardia a concern?

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Which medication is commonly used to treat ventricular arrhythmias in the ICU?

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What is the most common complication of atrial fibrillation?

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When should electrical cardioversion be considered for atrial fibrillation in the ICU?

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Which medications are used to shift potassium intracellularly in the management of hyperkalaemia?

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When should dialysis be considered in the management of hyperkalaemia in the ICU?

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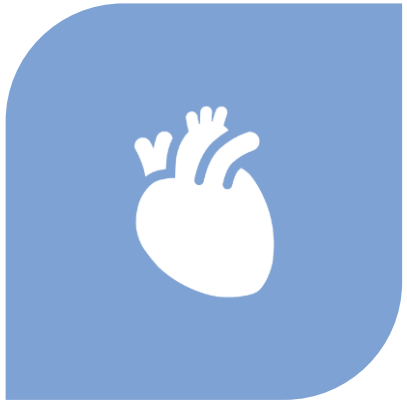
What we covered today!

6 STEP APPROACH TO RHYTHM RECOGNITION



ECG HACK – RELATING THE ‘PQRST’ TO THE ANATOMY OF THE HEART AND

NURSING CARE AND MANAGEMENT OF COMMONLY OCCURRING ARRYTHMIAS SEEN


That's all Folks!