STEMI mimics; A mnemonic

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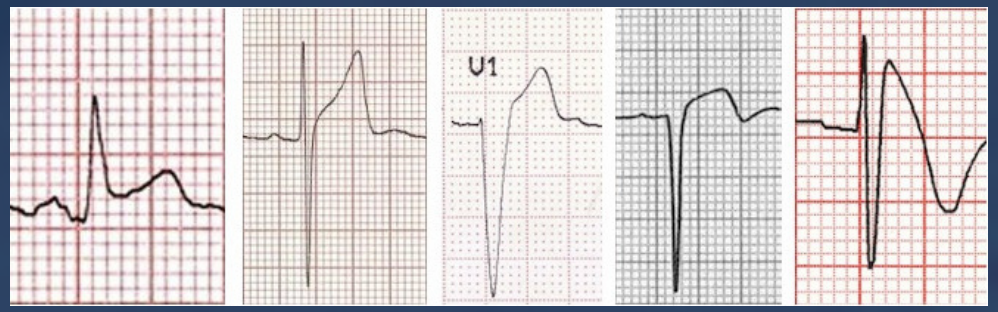


Raised intracranial pressure (Such as SAH or haemorrhagic stroke)

Abberrant conduction (Left Bundle Branch Block)

Inflammation (Pericarditis)

Spontaneous coronary artery dissection (SCAD)

Electrolytes (Hyperkalaemia)

Device (Ventricular paced rhythm)

Sodium channelopathy (Brugada Syndrome)

Thoracic aortic dissection

Spasm of the coronary arteries (Prinzmetal's/variant angina)

Embolism (Pulmonary)

Grief (Takotsubo cardiomyopathy)

Myocardial infarction recently (leading to ventricular aneurysm)

Enlarged ventricle (Left ventricular hypertrophy)

Normal for them (Early repolarisation)

Temperature (Hypothermia)

Enlarged ventricle (Left ventricular hypertrophy)

Accounts for up to 25% of ED presentations with chest pain.

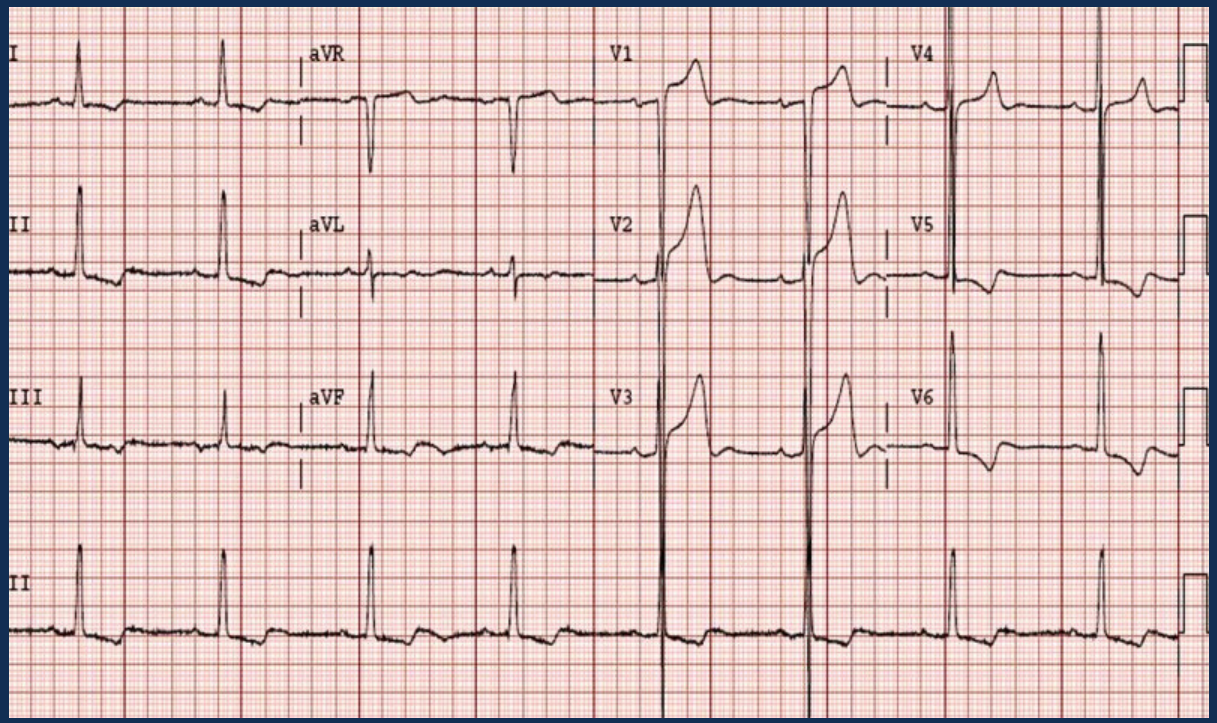
The left ventricle hypertrophies in response to pressure secondary to conditions such as hypertension and aortic stenosis, as well as aortic regurgitation, mitral regurgitation, coarctation of the aorta, and hypertrophic cardiomyopathy.

This leads to the following ECG features;

- Increased R wave amplitude in leads I, aVL and v4-v6.
- Increased S wave depth in leads III, aVR,v1-v3.
- The thickened LV wall leads to prolonged depolarisation increased R wave peak time and delayed repolarisation (ST and T wave abnormalities) in the lateral leads.

Diagnostic criteria (Sokolov-Lyon);

S wave depth in v1 + tallest R wave height in v5-v6 >35mm.



(Courtesy of LITFL)

Abberant conduction (Left bundle branch block)

Accounts for up to 15% of ED presentations with chest pain.

Caused by aortic stenosis, ischaemic heart disease, hypertension, anterior MI, primary degenerative disease of the conducting system (Lenegre disease), hyperkalaemia, digoxin toxicity.

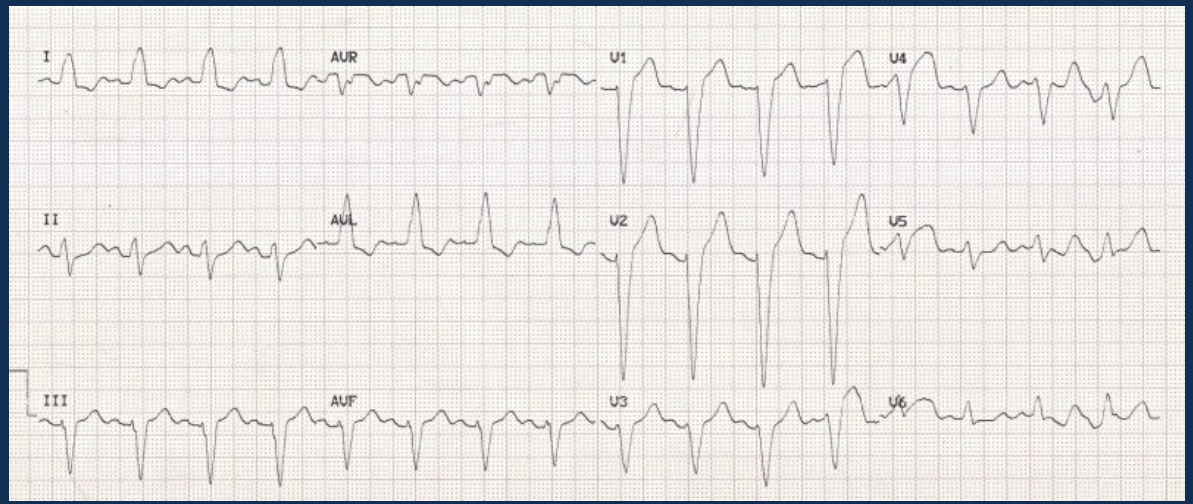
Diagnostic criteria;

- QRS duration >120ms.
- Dominant S wave in v1.
- Broad monophasic R wave in lateral leads (I, aVL, v5-v6.
- Absence of Q waves in lateral leads (I, v5-v6 - Q waves are still allowed in aVL.
- Prolonged R wave peak time >60ms in left precordial leads (v5-v6).

Associated features;

- Appropriate discordance.
- Poor R wave progression in the chest leads.
- Left axis deviation.

Can be assessed using Sgarbossa criteria to exclude presence of STEMI.



(Courtesy of LITFL)

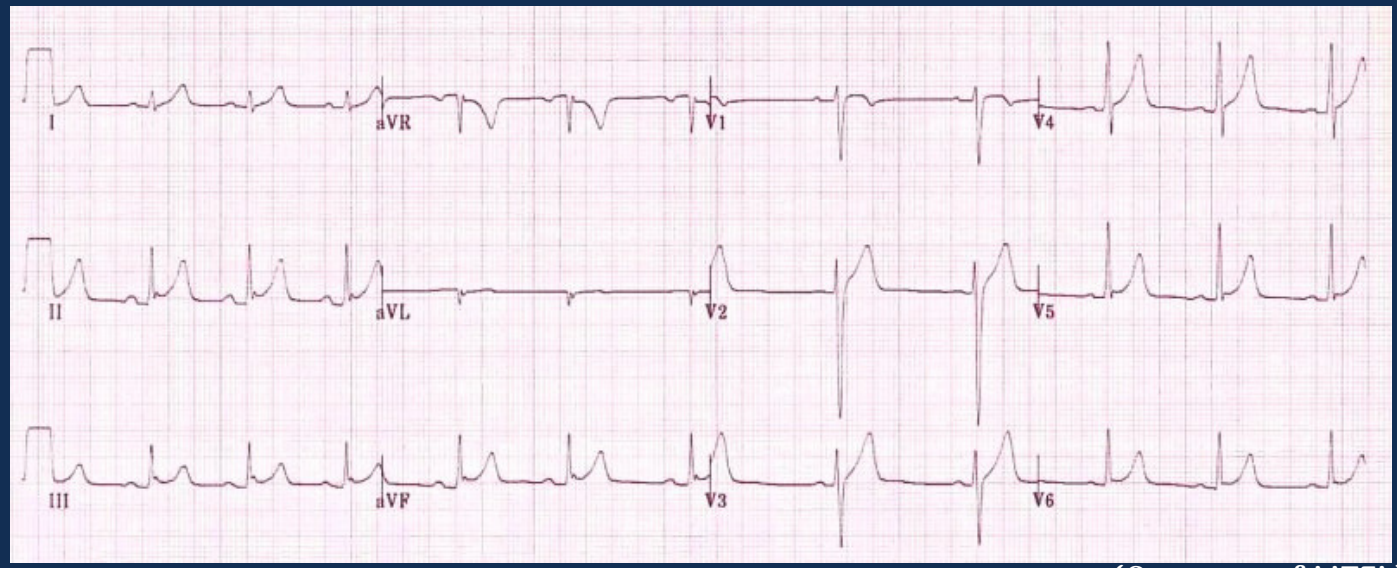
Normal for them (Early repolarisation)

Accounts for up to 12% of ED presentations with chest pain.

Most commonly seen in young, healthy patients <50. It produces widespread ST elevation that may mimic pericarditis or AMI. Generally considered to be a normal variant not indicative of cardiac disease. ER is less common in >50s, in whom STE is more likely to represent myocardial ischaemia. It is rare in the >70s. Clinicians should avoid diagnosing ER in patients >50, especially those with risk factors.

How to recognise ER;

- Widespread concave STE, most prominent v2-v5.
- Notching or slurring at the J-point.
- Prominent, slightly asymmetrical T waves that are concordant with the QRS complexes.
- The degree of STE is modest comparison to the T wave amplitude (less than 25% of the T wave height in v6).
- ST elevation is usually <2mm in the precordial leads and <0.5mm in the limb leads, although precordial STE may be up to 5mm.
- No reciprocal ST depression to suggest STEMI.
- ST segment changes are relatively stable over time (no progressive on serial ECG tracings).



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Normal for them (Early repolarisation)

The degree of ST elevation may fluctuate in response to changes in autonomic tone: diminishing with increased sympathetic tone / exercise / tachycardia or increasing when the HR slows.
The ST elevation may gradually disappear over time as the patient ages: up to 30% of patients with ER will have resolution of ST elevation on ECGs taken several years later.

- Early repolarisation no longer a "benign" finding devoid of clinical significance.
- Studies have demonstrated a 2 to 3-fold increased risk of death vs those without.
- Absolute risk remains exceedingly low in otherwise healthy individuals.
- Incidental finding of ER should not be interpreted as a high-risk marker for arrhythmic death due to the relatively low odds of SCD based on ER alone.

A consensus paper in 2015 concluded that in the absence of syncope, or strong family history of SCD, the finding of ER does not merit further investigation.

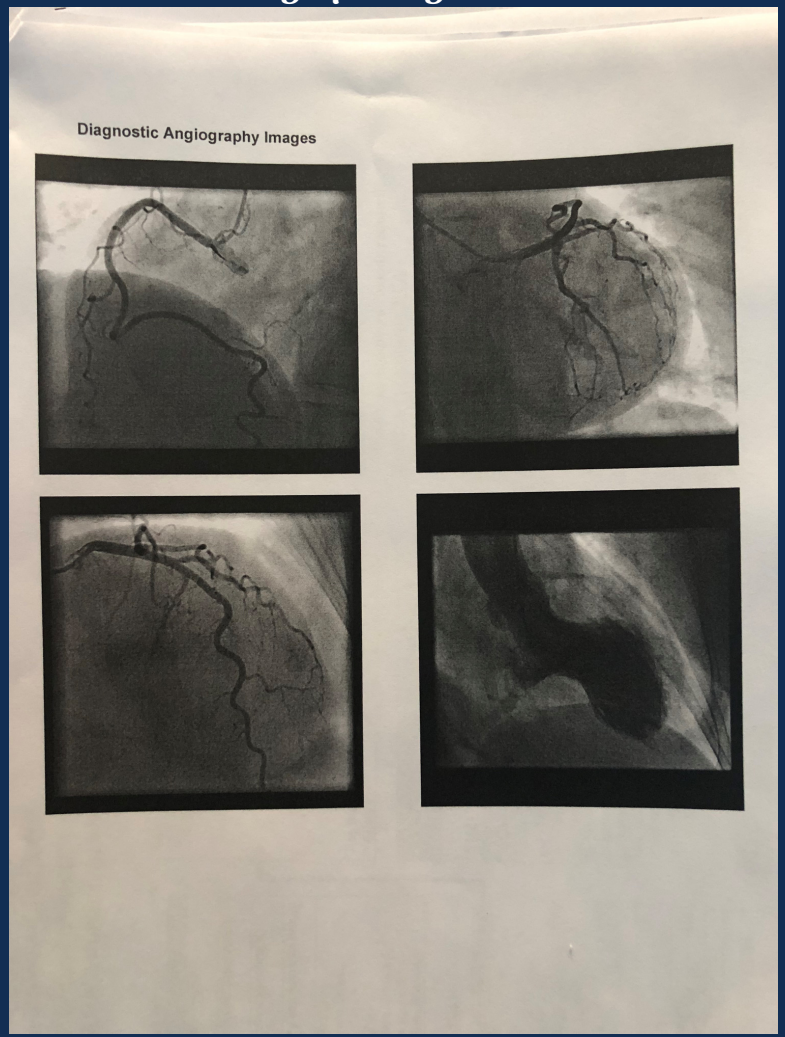
Grief (Takotsubo cardiomyopathy)

Only described within the last 20 years and increasingly recognised due to the increased use and availability of coronary angiography. Also known as stress cardiomyopathy, or broken heart syndrome, Takotsubo is typically triggered by emotional stress, or the death of a loved one.

Mayo clinic criteria (widely but not universally accepted);

- New ECG changes and/or moderate troponin rise.
- Transient akinesis/dyskinesis of LV (apical and mid-ventricular segments) with regional wall abnormalities extending beyond a single vascular territory.
- Absence of coronary artery stenosis >50% or culprit lesion.

Clinically indistinct from STEMI in the ED and should be treated as such until proven otherwise - although Takotsubo may form an early differential.



Inflammation (Pericarditis)

Inflammation of the pericardium produces characteristic chest pain (retrosternal, pleuritic, worse laying flat, relieved sitting forward), tachycardia and dyspnoea. There may be an associated pericardial rub or evidence of a pericardial effusion. Widespread ST segment changes occur due to involvement of the underlying epicardium (ie myopericarditis).

Recognising pericarditis;

- Widespread concave ST elevation and PR depression throughout most of the limb leads (I, II, III, aVL, aVF, and precordial leads (v2-v6)
- Reciprocal ST depression and PR elevation in lead aVR (+v1)

Causes;

- Infectious - mainly viral, occasionally bacterial, fungal, TB
- Immunological
- Uraemia
- Post-MI / Dresslers
- Trauma
- Post cardiac surgery
- Paraneoplastic syndromes
- Drug induced
- Post radiotherapy

Features suggesting ER;

- ST elevation limited to the precordial leads
- Absence of PR depression
- Prominent T waves
- ST segment / T wave ratio <0.25
- Characteristic "fish-hook" appearance in v4
- ECG changes usually stable over time (non-progressive)

Features suggesting pericarditis;

- Generalised ST elevation
- Presence of PR depression
- Normal T wave amplitude
- ST segment / T wave ratio >0.25
- Absence of "fish-hook" appearance in v4
- ECG changes evolve slowly over time

Sodium channelopathy (Brugada syndrome)

An ECG pattern with a high incidence of sudden death in patients with structurally normal hearts. Diagnosis requires characteristic ECG finds AND clinical criteria. Further risk stratification is controversial. Definitive treatment is ICD implantation. Brugada sign alone is of questionable signficance.

BrS is a mutation of the cardiac sodium channel gene (often referred to as a sodium channelopathy). ECG changes are often transient and can be unmasked or augmented by;

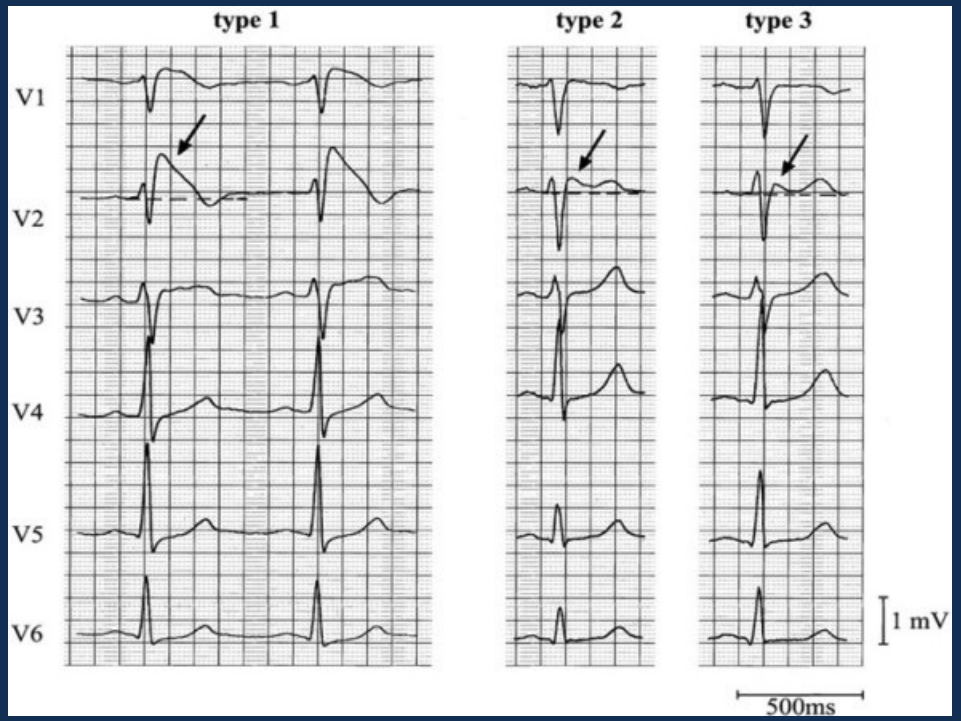
- Fever
- Ischaemia
- Drugs
- Hypokalaemia
- Hypothermia
- Post DCCV

- Diagnostic criteria;

Only type 1 is potentially diagnostic.

But must be accompanied by one of the following;

- Documented VF or polymorphic VT
- FH of SCD <45yrs
- Brugada sign in family members ECG
- Inducibility of VT with programmed electrical stimulation
- Syncope
- Nocturnal agonal respiration



- Type 2 and 3 may warrant further investigation.

Embolism (Pulmonary)

Ranges from asymptomatic to life-threatening catastrophe. PE occurs when a DVT migrates to the pulmonary arterial tree.

- Massive PE - Acute PE with obstructive shock or SBP <90mmHg
- Sub-massive PE - Acute PE without symptomatic hypotension (SBP>90mmHg but with either RV dysfunction or myocardial necrosis
- Those without the above severe features are non-massive or low risk PEs

ECG features;

- Non-specific ST changes or T wave changes(seen in up to 50% of cases)
- Sinus tachycardia (44%)
- s1 q3 t3 (less common than perceived, only around 20%)
- Complete or incomplete RBBB (18%)
- Right axis deviation (16%)
- P pulmonale (right atrial enlargement) - Peaked P wave in lead II >2.5mm (9%)
- Atrial arrhythmias (8%)
- Dominant R wave in v1 - a manifestation of acute RV dilatation
- Clockwise rotation - shift of the R/S transition point towards v6 ("pulmonary disease pattern"), implying rotation of the heart due to RV dilatation

Thoracic aortic dissection

The most common catastrophe of the aorta (3:100,000): 3 times more common than abdominal aneurysm rupture. Aortic dissection is a type of acute aortic syndrome (AAS) characterised by blood entering the medial layer of the wall with the creation of a false lumen.

Inferior ST elevation

Pericarditic changes

Electrical alternans (tamponade)

Classification by Stanford;

- Type A - Involves the ascending aorta. Surgery usually indicated
- Type B - Involves the aorta beyond left subclavian artery. Managed medically

Typical presenting symptoms;

- Retrosternal chest pain -anterior dissection
- Interscapular pain - descending aorta
- Severe pain ("worse ever" described in 90% of presentations)
- Sudden onset (90%)
- Sharp (64%) or tearing (50%)
- Down the back (46%)
- Migrating pain (16%)
- Maximal in onset (not crescendo build up, as in AMI)

Risk factors;

- HTN, smoking, hyperlipidaemia
- Previous CV surgery
- Structural abnormalities (eg bicuspid AV, aortic coarctation)
- Iatrogenic (eg recent cardiac catheterisation)
- Infection
- Arteritis
- Aortic dilatation / aneurysm
- Wall thinning
- 'Crack' cocaine (abrupt catcholamine-induced HTN
- Inherited diseases (eg Marfans, Ehlers-Danlos)
- Age

Spontaneous coronary artery dissection (SCAD)

Rare, sometimes fatal traumatic condition, with 80% of cases occurring in women.

One of the coronary arteries develops a tear, causing blood to flow between the layers which forces them apart. Mortality may be as high as 70%.

A primary cause of MI in young, fit, healthy women (and some men) with no obvious risk factors. These can occur during pregnancy, postpartum and peri-menopausal periods.

Coronary angiogram is the most common method to form diagnosis, typically using intravascular ultrasound (IVUS).

Spasm of the coronary arteries (Prinzmetal's/variant angina)

Pattern of ST elevation very similar to acute STEMI - localised ST elevation with reciprocal ST depression occurring during episodes of chest pain. Unlike acute STEMI, changes are transient, reversible with vasodilators and not usually associated with myocardial necrosis.

It may be impossible to differentiate these two conditions based on the ECG alone.

Myocardial infarction recently (Leading to ventricular aneurysm)

Seen in patients with previous/recent MI. Mechanism is thought to be related to incomplete reperfusion and transmural scar formation following AMI.

Typical ECG pattern;

- Residual/persistent STE>2 weeks post MI
- Well formed Q or QS waves
- T wave flattening or inversion

Device (Ventricular paced rhythm)

Ventricular pacing causes identical changes to those seen in LBBB. There is appropriate discordance with the ST segment and T wave directed opposite to the main vector of the QRS complex.

Raised intracranial pressure (Such as in SAH or haemorrhagic stroke)

Raised intracranial pressure (eg due to intracranial haemorrhage or traumatic brain injury) may cause ST elevation or depression that simulates myocardial ischaemia or pericarditis. More commonly, raised ICP is associated with widespread, deep T wave inversions.

Electrolytes (Hyperkalaemia)

Although not the most common ECG abnormality caused by hyperkalaemia, there is a large body of case studies and anecdotal evidence of ST segment elevation in hyperkalaemia. The tall/peaked T waves very typically seen in this context can also complicate the evaluation of the ST segment.

Serum potassium > 5.5 mEq/L
- Peaked T waves (usually the earliest sign of hyperkalaemia)

Serum potassium > 6.5 mEq/L
- P wave widens and flattens
- PR segment lengthens
- P waves eventually disappear

Serum potassium > 7.0 mEq/L
- Prolonged QRS interval with bizarre QRS morphology
- High-grade AV block with slow junctional and ventricular escape rhythms
- Any kind of conduction block (bundle branch blocks, fascicular blocks)
- Sinus bradycardia or slow AF
- Development of a sine wave appearance (a pre-terminal rhythm)

Serum potassium level of > 9.0 mEq/L
- Asystole
- Ventricular fibrillation
- PEA with bizarre, wide complex rhythm

Temperature (Hypothermia)

Common ECG changes due to hypothermia;
- Bradyarrhythmia's
- Osborne waves (J wave) – these may give the appearance of STE due to J point elevation/positive deflection
- Prolonged PR, QRS and QT intervals
- Shivering artefact
- Ventricular ectopics
- Cardiac arrest due to VT, VF, or asystole