CASE-BASED DISCUSSIONS 19th May 2020

CASE ONE

A 23yo mane has been found collapsed on his bedroom floor by friends. He was last seen well over 48hours ago while at a party. He has been transported into the ED with the Ambulance service. He has received no pre-hospital treatment so far.

Vitals: HR 40, BP 100, 34.5 C, BGL 4.5, GCS 12 (M5), 92% o/a, RR 20

1. **Discuss important further history and information you would like?**

- Background medical history if able
  - Concerto/records
  - Friends/family
  - Scene information from ambulance crew
  - Any mental health hx
  - Tox history
  - Past medical history
  - Medications

- Information surrounding party
  - Drug and alcohol use
  - Mental state
  - Trauma

He is placed in the Resuscitation bay. There are no external signs of trauma, and he is calmly agitated remaining settled if not stimulated.

2. **What are your differential diagnoses?**

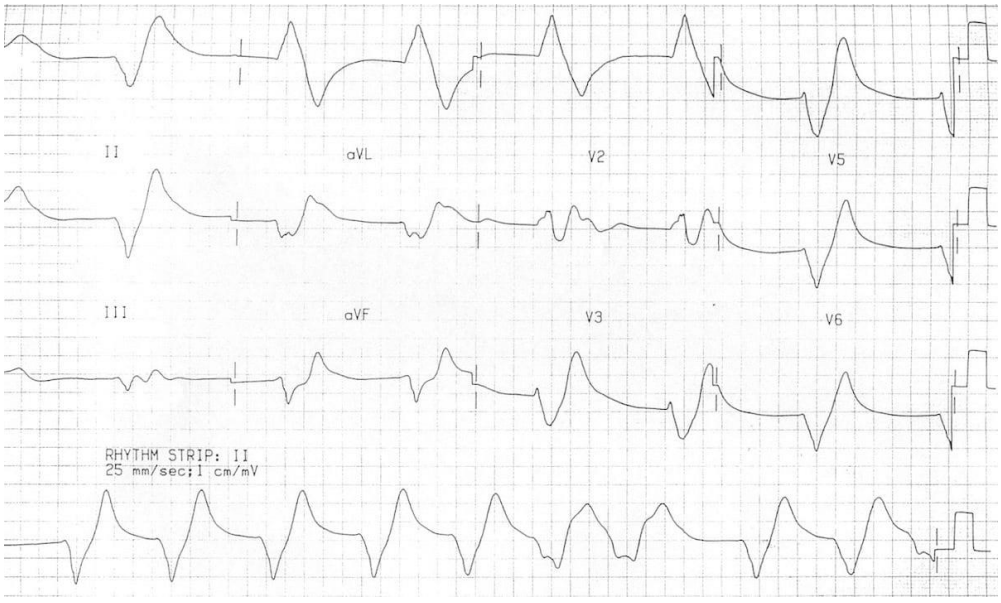
- Broad range
  - Medical event CNS, infection
  - Trauma
  - Toxicology
  - Environmental

3. **What investigations do you want at this stage?**

- Bedside
  - ECG (bradycardia)
  - BGL
  - MSU (MC+S, colour ? myoglobinuria)
- Labs
  - VBG – electrolytes, metabolic state, perfusion
  - Septic screen
  - TFTs, cortisol
- Radiology
  - CXR
  - CT head

4. **Below is his VBG result and ECG. Describe and discuss findings and concerns?**

pH 7.06, PCO2 4, HCO3 16, Na 129, K 7.2, AG 22, Lactate 4, Gluc 3.5



Severe mixed metabolic acidaemia, severe life threatening hyperkalaemia, raised anion gap, hypoglycaemia

Broad complex bradycardia, no p waves likely due to hyperkalaemia

5. **Outline your management for this man?**

- Supportive cares
  - Maintain airway, oxygenation as required
  - Thermal (external warming measures)
  - Correct hypoglycaemia
  - U/O with IDC
- Treat hyperkalaemia
  - Stabilise cardiac membrane Ca Gluconate 20ml (2g) or Ca chloride (1g)
  - Drive K into cells with insulin and dextrose, B agonists (?)
  - Eliminate K with IVF if hypovolaemia, Fruesmide if hypervolaemia, dialysis if severe/refractory/renal failure or oliguria
  - Sodium bicarbonate indicated if unstable, renal failure, peri arrest/arrest in the setting of acidaemia.
- Determine cause of hyperkalaemia
- Consider cover for sepsis
- Consider antidotes as appropriate

Below are his initial blood results

Cr 420, Na 128, K 6.9, CK > 50000, AST 320, ALT 290, Bili 35
Hb 175, WBC 22
INR 1.8
TFT normal, cortisol 300

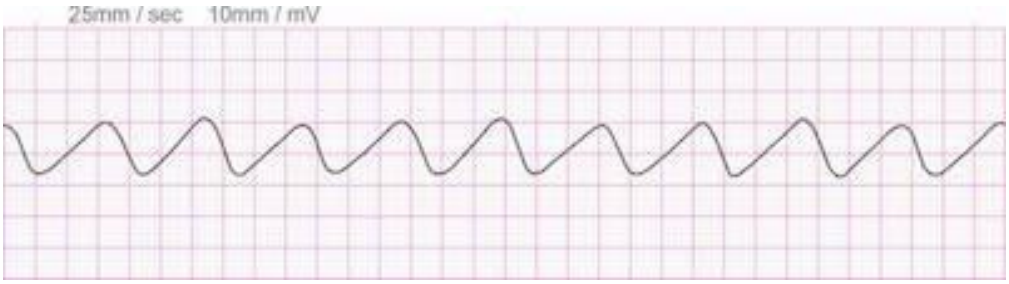
6. Discuss the potential causes for his hyperkalaemia?

- Wide potential cause but substance use or toxicologic cause likely, with probable renal failure stemming from prolonged period on floor, dehydration and resulting rhabdomyolysis.

- Other causes

1. **Medications:** ACEi, Potassium sparing diuretics, B-Blockers, NSAIDs, Trimethoprim (Septra) and Non-prescription salt substitutes
2. **Renal Failure**
3. **Cell death:** Secondary to rhabdomyolisis, massive transfusion, crush or burn injuries.
4. **Acidosis:** Consider Addisons crisis, primary adrenal insufficiency and DKA.
5. **Pseudohyperkalaemia (haemolysis)**

7. He deteriorates despite receiving treatment 1L saline, 20ml calcium gluconate, 10IU actrapid + 100ml DW50. His current ECG and vitals are below, outline your management?



- BP unrecordable, no palpable pulse, agonal breaths

- **Hyperkalemia in Cardiac Arrest**
- Based on the principles of treatment and indications discussed above, our experts recommend the following approach to suspected hyperkalemia (based on patient history and rhythm strip) or confirmed hyperkalemia (based on a point of care blood gas) in cardiac arrest in addition to usual ACLS measures:

- **Push 1 amp calcium chloride in well running peripheral IV or central line and repeat until the QRS is <100ms**

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- **Epinephrine 5-20 mcg q2-5 minutes (shifts K intracellularly)**

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- **Sodium Bicarbonate 1 amp IV (if suspect severe acidosis)**

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- **Bolus IV NS**

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- **Shift potassium with Insulin and Glucose followed by B-agonist**

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- **Dialysis**

- **Rebound Hyperkalemia**
- In cases of cardiac arrest due to hyperkalemia, perform CPR until the hyperkalemia is corrected. This may be a much longer time than usual. When ROSC is achieved, it will be primarily due to the effects of calcium rather than decreased potassium levels. The effect of calcium can last 20-30min. Since the stabilizing effects of calcium will wear off, you must promptly work on shifting the potassium and enhancing its elimination as described above. Consider repeating the calcium bolus if there are any worsening ECG changes. Repeat serial potassium measurements to monitor for rebound hyperkalemia, which occurs more often than we'd like.

- ***PEARL:*** *the patient in cardiac arrest with hyperkalemia should not be pronounced dead until their potassium level is normalized*

CASE TWO

A 17yo woman has been found collapsed in the streets. She was found with a plastic bag with strong smelling chemical odour next to her. She has been transported to the ED. Below are her vitals. She is floppy and has reduced tone and has shallow breathing.

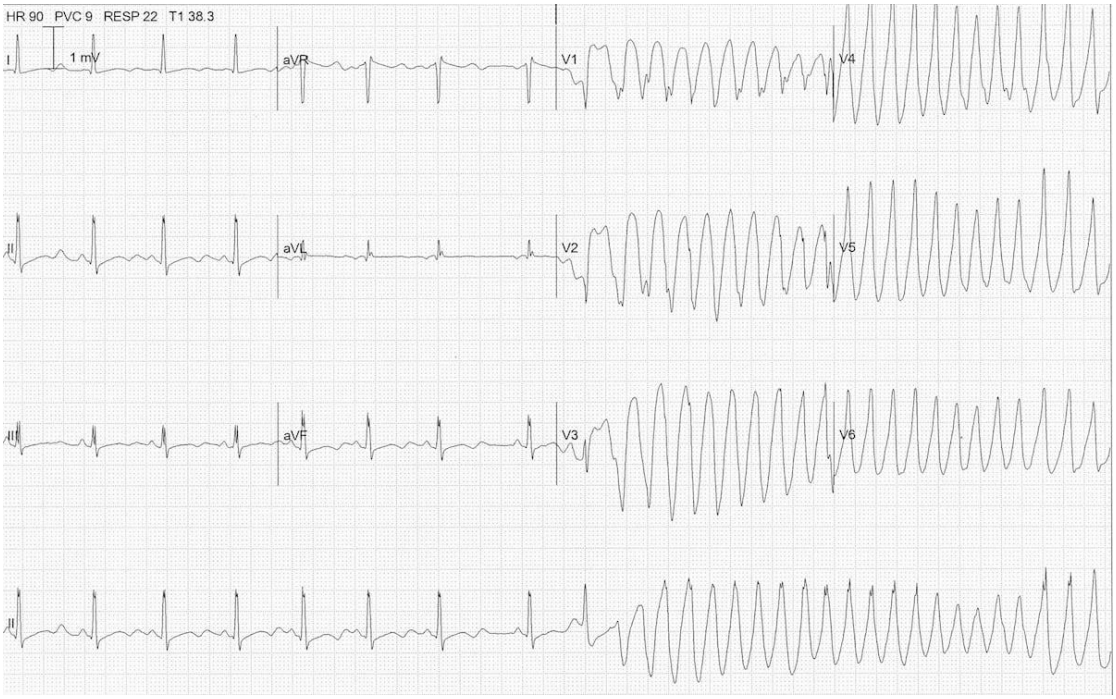
HR 100, BP 80, 35 C, RR 24, Sats 92%, BGL 5, GCS 10 (M5)

1. **What is your concern for this woman?**

- Probably solvent induced presentation
  - Concern for metabolic disturbance in particular hypokalaemia and arrhythmia and hypokalaemic paralysis risk impaired ventilation.
  - Consider seizures
  - Potential traumatic injury
  - Other substance use
  - May have other ingestions

2. **Below is her VBG result and initial ECG. Discuss your findings?**

pH 7.1, PCO2 3.5 Na 128, K 1.2, HCO3 12, AG 14, Lactate 2.2, Gluc 5.2



- **Severe normal anion gap metabolic acidaemia, with severe hypokalaemia**
- **Prolonged QT sinus rhythm with run of polymorphic VT (Torsades)**

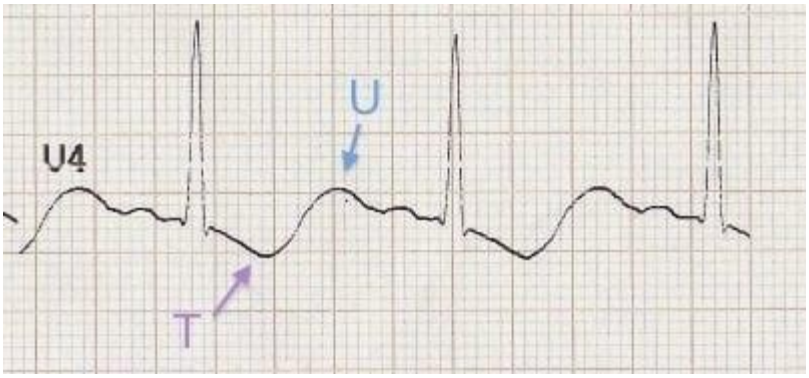
3. **Discuss the changes in the ECG you expect with hypokalaemia?**

ECG changes when K+ < 2.7 mmol/l

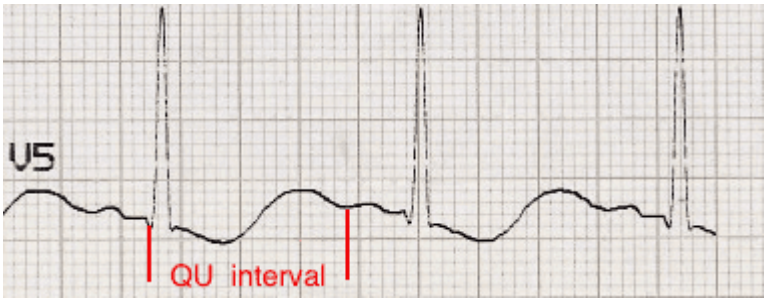
- Increased amplitude and width of the P wave
- Prolongation of the PR interval
- T wave flattening and inversion
- ST depression
- Prominent U waves (best seen in the precordial leads)
- *Apparent* long QT interval due to fusion of the T and U waves (= long QU interval)

With worsening hypokalaemia…

- Frequent supraventricular and ventricular ectopics
- Supraventricular tachyarrhythmias: AF, atrial flutter, atrial tachycardia
- Potential to develop life-threatening ventricular arrhythmias, e.g. VT, VF and Torsades de Pointes



T wave inversion and prominent U waves in hypokalaemia



Long QU interval in hypokalaemia

4. **What is the likely cause for her metabolic disturbance, and how are you going to manage her?**

- Likely Toluene toxicity
  - RTA1 distal tubular acidosis typically with hyperchloraemic, hypokalaemic, normal AG metabolic acidosis.
  - Risk of hypokalaemic paralysis, Rhabdomyolysis from hypokalaemia and hypophosphataemia, other organ injury
  - Altered mental status common
  - Arrhythmia due to low K

- Urgent replacement of Magnesium and K required
  - 10-20mmol Mg SO4 (2 amps)
  - Stable 20mmol K over 60 minutes
  - 10-20mmol diluted over 15-20min if recurrent arrhythmia then review (Preferable via central access, large bore)
  - In refractory hypokalaemia arrest VT/VF/asytole bolus 20mmol over 2-3minutes and consider repeating if persistent refractory arrest/ventricular arrhythmia.

CASE THREE

A 42 yo man has been brought into ED by his family due to confusion and drowsiness. He has been unwell for the last 2 weeks during COVID-19 lockdown, with abdominal pain, frequent loose motions.

Vitals: BP 140/70, Afebrile, HR 120, GCS 13 (M6), RR 20, Sats 90% O/A

1. **What are your differential diagnoses and investigations you want?**

- Broad range of causes
  - GIT illness volume and electrolyte disturbance (Pancreatitis?)
  - Sepsis
  - Metabolic such as DKA or HHS , Withdrawal ETOH
  - Medical ACS
  - Trauma SDH
  - COVID ?

- Investigations
  - ECG and BGL
  - VBG and sepsis bloods/abdo
  - Trop, TFT
  - MSU
  - CXR and CT head

2. **Below is his VBG result, his ECG only shows a sinus tachycardia. Discuss your concerns and initial management?**

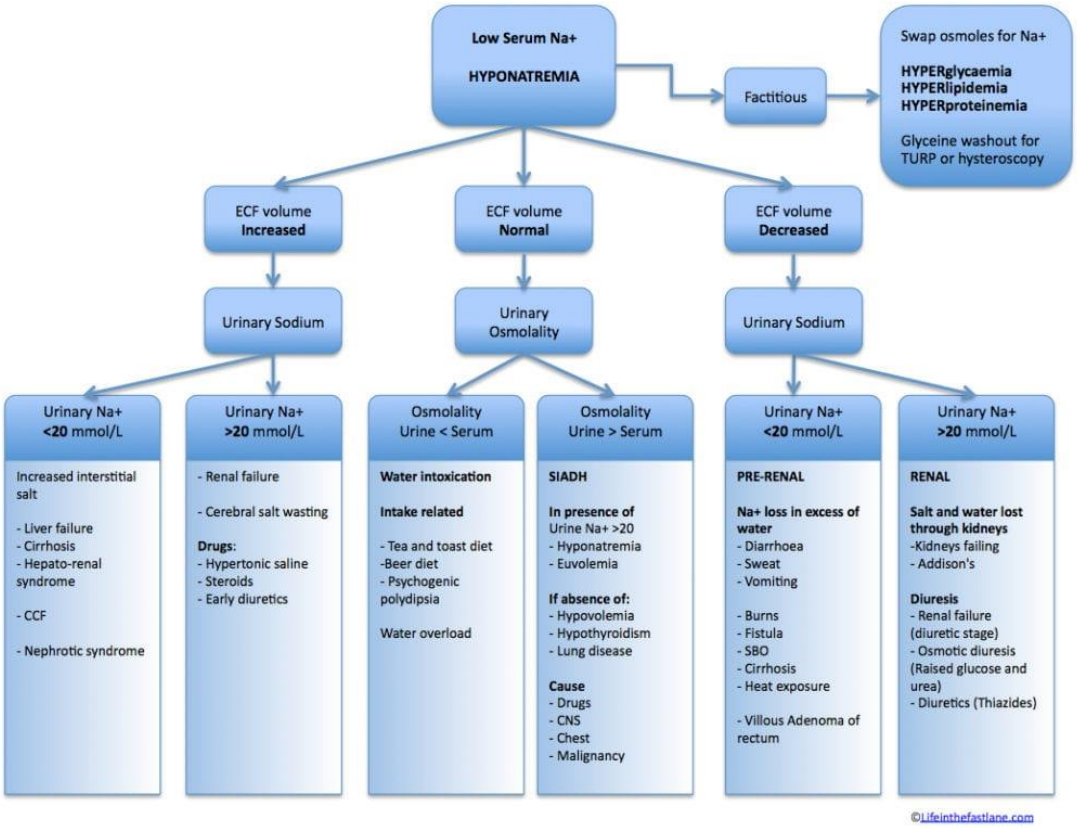
pH 7.28, HCO3 14, PCO2 4, Na 110, K 3.0, Gluc 10, lactate 3.2

- PPE consideration contact and droplet

- Severe hyponatraemia with metabolic acidaemia
  - Likely on basis of hypovolaemia and GIT losses given history
  - Altered mental state with risk of seizures

- Need to assess volume state
  - 3% saline 1-2ml/kg up to 150ml over an hour for altered mental state/seizures
  - If hypovolaemic 0.9% saline to replace volume carefully 250-500ml bolus only and careful review
  - Avoid rapid correction (0.5-1 mmol per hour, <10mmol in 24hrs)

3. Discuss how to determine causes of hyponatraemia?



4. Despite initial 150ml 3% saline he develops persistent seizure activity with obstructive airway due to Trismus. His repeat VBG shows a Na of 112 and Gluc 8. How will you manage this man?

- Further bolus 150ml 3% saline (sodium 513mmol/L) or 10ml 4molar (23.4% sodium 80mmol/20ml)
- Airway control BMV PEEP O2
- Ensure Glucose and K okay
- Look at ECG/rhythm for any abnormality
- Benzodiazepine for persistent seizure if above fails
  - Unusual on going seizures once Na increased 1-3mmol consider alternative or additional causes
- RSI propofol infusion/ benzo infusion
- **Repeat VBG for Na, K, Gluc**
- **Abs**
- **CT head exclude intracranial cause**
- **Monitor and correct Na slowly once seizures ceased**

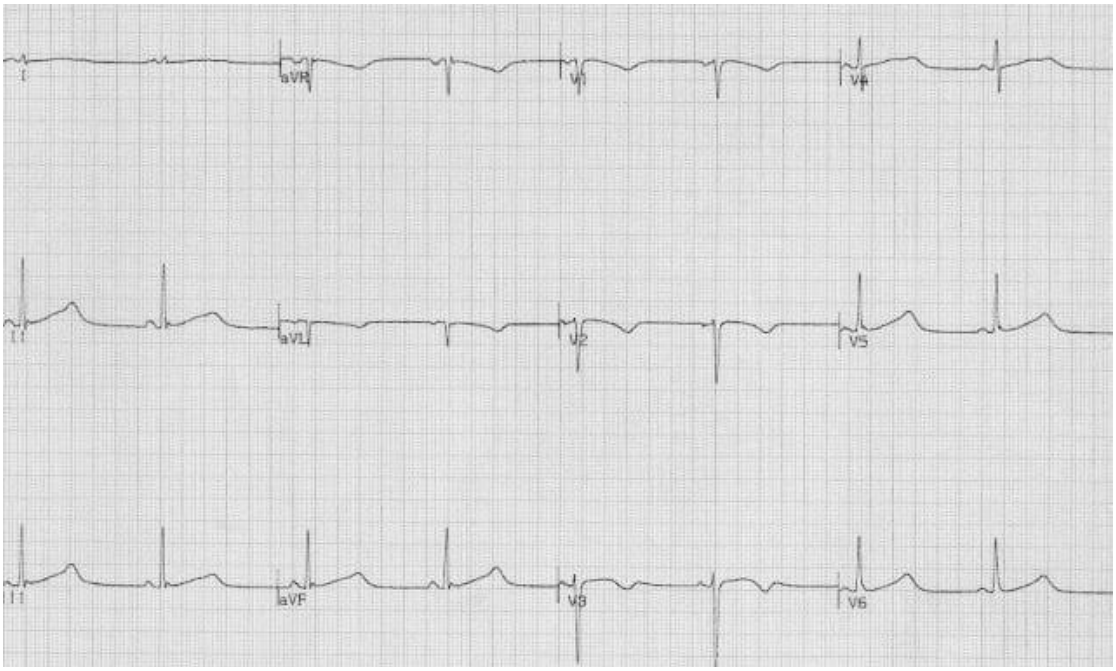
CASE FOUR

22yo male presents with two episodes of witnessed syncope during the morning, with brief LOC each time and collapse to the floor. He has no history of chest pain, SOB, and no family history of note. He denies and seizure history. He is on Erythromycin for a URTI started 5 days ago; he is not on any other medication. He has had some diarrhoea since starting the antibiotic.

1. **What are your differential causes for his syncope?**

- Concerning syncope history
  - Need to consider Cardiac cause
  - On macrolide may prolong QT interval
  - Possible congenital cardiac cause
  - Potential K/Mg depletion from GIT loss

2. **Below is his baseline ECG, interpret findings?**



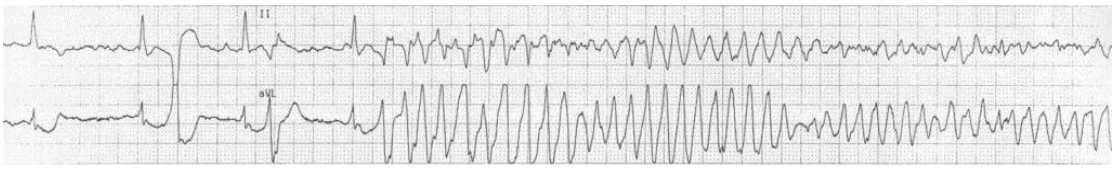
Sinus rhythm, prolonged QT interval, TWI anterior

3. **Outline potential causes of long QT intervals?**

Causes of a prolonged QTc (>440ms)

- Hypokalaemia
- Hypomagnesaemia
- Hypocalcaemia
- Hypothermia
- Myocardial ischemia
- ROSC Post-cardiac arrest
- Raised intracranial pressure
- Congenital long QT syndrome
- Medications/Drugs

4. **The patient suddenly loses consciousness while on his bed in the ED monitored area. He regains consciousness but his ECG shows an abnormal rhythm, interpret and outline your treatment?**



Torsades de Pointes

- Mg 1-2g IV slow bolus
- Attach defib – persistent or unstable immediate cardioversion (may need unsynchronised)
- Look for cause
  - Correct low K/Mg/Ca
  - Remove ppt drugs
  - Avoid QT prolonging drugs
  - Review history for any triggers (medications/overdose)

5. **His electrolytes are normal along with his VBG. Despite initial bolus of Mg 2g he has a further episode of above rhythm with LOC. Discuss your approach?**

- ACLS approach
- Further Mg bolus and DC cardioversion
- Prevention – ensure Mg level 1.5-2mmol/L (rpt bolus/infusion)
- Refractory – lidocaine 1.5mg/kg bolus (+/- infusion)
- Prevention with increasing HR for acquired prolonged QT
  - Increase rate (chemical or electrical)
  - Relatively contraindicated in congenital prolonged QT syndromes

Occasional patients will have recurrent episodes of torsades ("Torsades storm"). Each individual episode may be treated with magnesium or defibrillation, if needed (Treatment step #1 above). However, additional therapies are required to stop recurrence and end the storm.

re-load magnesium PRN

- Recurrent torsades may reflects inadequate magnesium dosing (e.g. patient is bolused with 2-4 grams, without an infusion). The first step when managing recurrent torsades is therefore to ensure that the patient has truly received an adequate dose of magnesium.
- If the patient was bolused with magnesium a few hours ago without an infusion, re-load with 2-4 grams IV immediately (8-16 mM).
- If the patient is a candidate for magnesium infusion (GFR >30 ml/hr), this should be ordered.
- If the patient has renal failure and has already received 4-6 grams of magnesium (16-24 mM), then check magnesium levels and ensure that a high level is achieved. Note that a therapeutic level for torsades is roughly 3.5-5 mg/dL (1.5-2 mM) – not a "normal" level.
- More on magnesium above (Treatment step #2).

optimize the potassium

- The target potassium level here is probably >4.5-4.7 mEq/L.
- Giving potassium alone is unlikely to work, but it might help a bit.

speed up the heart

- Speeding up the heart rate will decrease the QT interval and reduce the risk of acquired torsades (but it may be contraindicated in some forms of congenital torsades).
- The usefulness of chronotropy depends on the patient's **baseline heart rate**.
    - Chronotropy is most beneficial for patients starting out with bradycardia.
    - If the patient is already significantly tachycardic, chronotropy is unlikely to provide benefit. The usual target heart rate is 100-110 b/m, but occasionally heart rates up to 140 b/m may be needed.[2] There's no high-quality data on this.
- **Medical chronotropy** is generally the easiest & fastest way to stabilize the patient. The ideal chronotrope depends on the patient's hemodynamics and baseline blood pressure:
    - Baseline severe hypotension: epinephrine infusion.
    - Baseline normotension or mild hypotension: dobutamine or isoproterenol infusion.
- **Electrical chronotropy** may be used if medical chronotropy fails:
    - Transcutaneous pacing may work, but this is painful for conscious patients.
    - Transvenous pacing is more comfortable, but this is more invasive and takes a bit longer to achieve.
    - Patients with a pacemaker may have the device rate increased.

lidocaine

- Lidocaine is the preferred anti-arrhythmic drug for torsades, although there isn't a ton of evidence supporting its use.

    - **Do not use amiodarone, procainamide, beta-blockers, or most other antiarrhythmics.** Most of these will stretch out the QT interval even further! Beta-blockers will slow down the heart rate, increasing the risk of torsades.
- Start with a loading dose of 1-1.5 mg/kg followed by a 1 mg/min infusion. For recurrent arrhythmias, re-load with another 1 mg/kg bolus and increase the maintenance infusion.

Consider an alternative diagnosis

- Torsades is generally fairly easy to control with a combination of high-dose magnesium, heart rate augmentation, and occasionally some lidocaine. Failure to respond to these interventions suggests an alternative diagnosis (e.g. polymorphic VT due to ischemia or catecholaminergic ventricular tachycardia).