QUIZ 11th April 2018 (answers below)

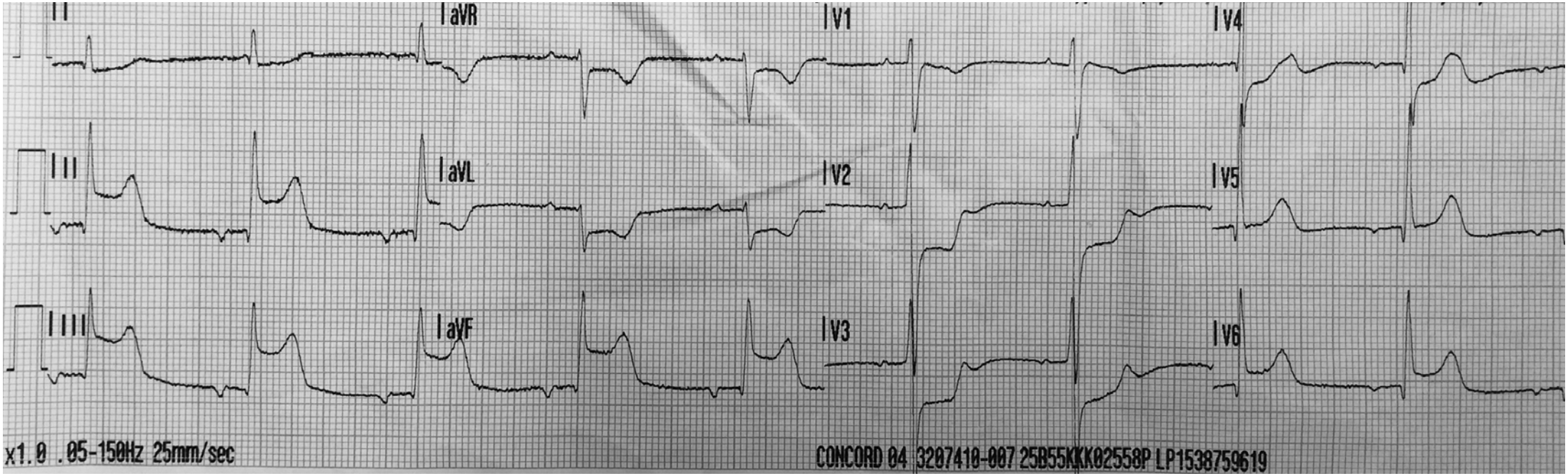
1. **What is HIET?**

2. **What are the indications for HIET?**

3. **What are potential adverse affects of HIET?**

4. **Why do we have intralipid in ED?**

5. **Describe and interpret the following ECG.**


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
x1.0 .05-150Hz 25mm/sec
CONCORD 04 3207410-007 25B55KKK02558P LP1538759619

QUIZ answers 11th April 2018

1. **What is HIET?**

   High-dose Insulin Euglycaemia Therapy

   High dose insulin is thought to have an inotropic effect by increasing lactate oxidation while eliminating fatty acid oxidation in myocardial cells. Glucose is administered simultaneously to maintain normal blood glucose levels. This combination produces increased myocardial contraction, increased intracellular glucose transport and vasodilation. Peak inotropic response is usually seen within an hour of therapy.

2. **What are the indications for HIET?**

   Moderate to severe cases of calcium channel blocker or beta blocker poisoning with haemodynamic compromise refractory to initial fluid resuscitation. It is most successful if commenced as soon as haemodynamic compromise becomes evident, rather than waiting for poor response to other measures. It takes up to an hour to have full effect, so other measure should be simultaneously instituted (volume, calcium, adrenaline, noradrenaline).
   HIET has been advocated in other toxin-induced shock states, but clinical experience is limited so is not currently recommended.

3. **What are potential adverse affects of HIET?**

   <u>Hypoglycaemia</u>
   - *BSL should be checked 30 minutely during initiation and titration of insulin and then hourly*
   - *Target range is 5.6 – 11.1 mmol/L*
   - *BSL will need to continue to be check hourly for 6 hours post cessation of insulin infusion, then 2-6 hourly after that*

   <u>Hypokalaemia</u>
   - *note that total body stores of potassium are not depleted (unlike in DKA); potassium is just being pushed into cells, so potassium should just be maintained to 2.8 - 3.2 mmol/L to avoid hyperkalaemia once insulin is ceased*
   - *Potassium should be checked hourly during initiation and titration of insulin*

   <u>Hypomagnesaemia</u>

   <u>Hypophosphataemia</u>

4. Why do we have intralipid in ED?

The only current indication for intralipid in SVH ED is severe refractory cardiovascular toxicity from a local anaesthetic agent.
The threshold for use is controversial.
Intralipid 20% 500mL bottles are kept in the ED drug cupboard
Give 100mL stat and then run remaining 400mL over 20 minutes

5. Describe and interpret the following ECG.

Rhythm	*Regular 58/min*
P waves	*All conducted*
	Inverted in II, III and aVF
PR	*Normal interval*
QRS	*Narrow*
	Normal frontal axis
	Normal R wave progression across precordium
	Normal morphology
ST segment	*ST elevation*
	Inferiorly - 6mm in III, 5mm in aVF and II
	Laterally - 2mm in V6, 1mm in V5
	ST depression
	Anteriorly - 6mm V2/V3 and 3mm in V4
	Superiorly - 3mm in aVL and 2mm in aVR
T wave	*Inversion in aVL*
QTc	*432 msec = normal*

➔ *Large infero-postero-lateral STEMI*
Reciprocal antero-superior ST depression
ST elevation in III >II suggests RCA as culprit

Ectopic atrial focus (inverted P waves inferiorly)
= coronary sinus rhythm (as pointed out by Prof Preisz)
Also consistent with RCA as culprit causing ischaemic SA node

➔ *Acute proximal right coronary artery occlusion*

This ECG is from a 58 year old man.
Cardiac catheterisation:
Acute thrombus in proximal RCA with 70% stenosis in distal RCA.
Successful PCI to proximal and distal RCA.
Subsequent ECG is sinus rhythm with inferior q waves.