QUIZ 31st Jan 2018 (answers below)

1. **How does Targin work?**

2. **What are the indications for thrombolysis in pulmonary embolus?**

3. **What is the management of traumatic hyphaema?**

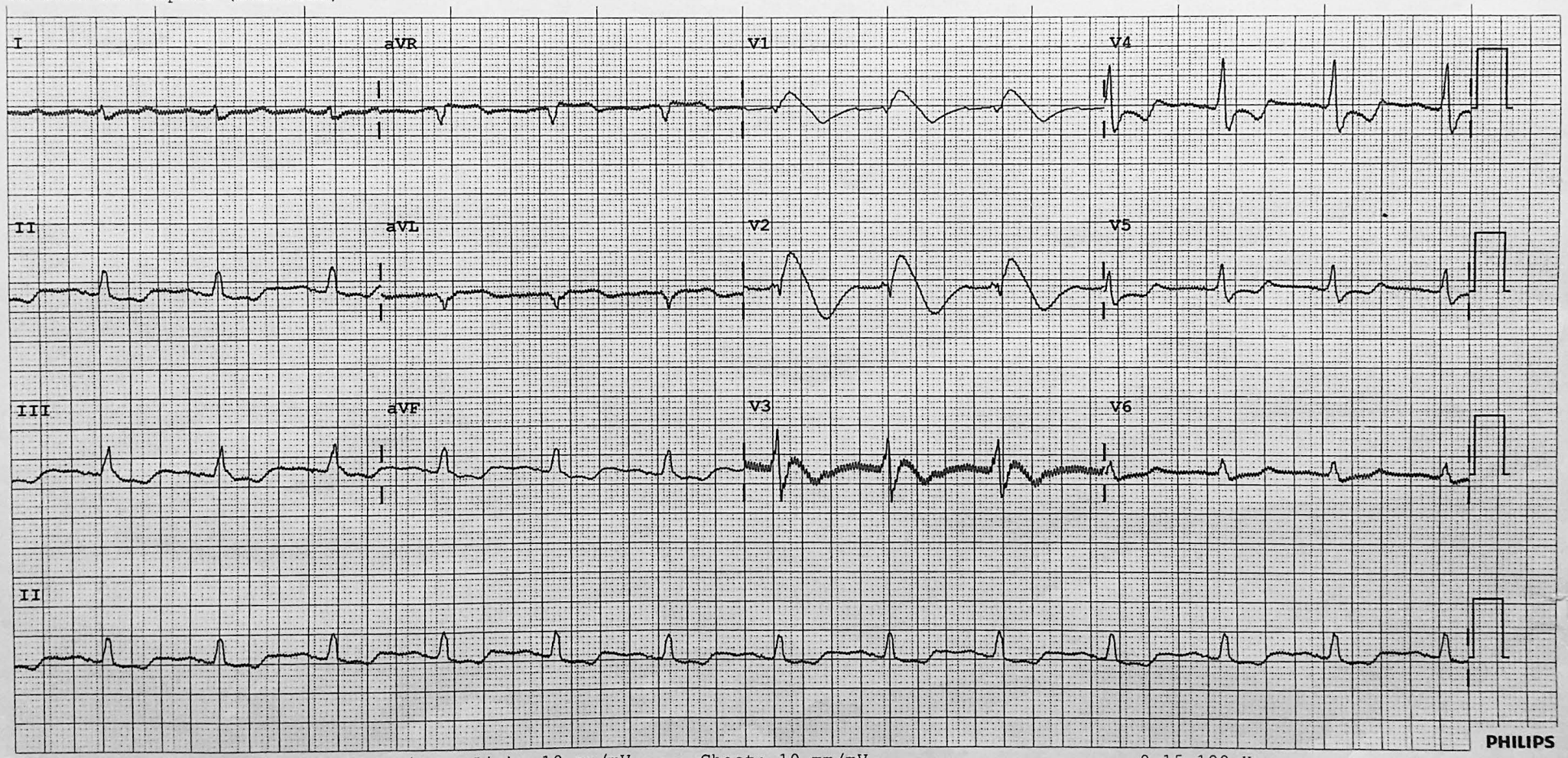
4. **What are the clinical features of funnel web spider envenomation?**

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

ST VINCENTS HOSPITAL

HR	79 bpm	ST-I	0.1 mm	ST-V1	2.0 mm
PVC	0 /min	ST-II	-0.6 mm	ST-V2	4.7 mm
		ST-III	-0.8 mm	ST-V3	1.0 mm
		ST-aVR	0.2 mm	ST-V4	-0.6 mm
		ST-aVL	0.4 mm	ST-V5	-0.3 mm
		ST-aVF	-0.7 mm	ST-V6	-0.2 mm

12 Lead ECG Report (Standard)



Device: RESUS 1 Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10 mm/mV 0.15-100 Hz

QUIZ answers 31st Jan 2018

1. **How does Targin work?**

Targin is the commercial brand name for a fixed dose combination of controlled release oxycodone and naloxone and is available in a variety of strengths.

Oxycodone is semisynthetic opioid with action similar to morphine and principle therapeutic action is analgesia. It is a full opioid receptor agonist. By binding opioid receptors in the CNS, oxycodone produces analgesia, sedation, respiratory depression, cough suppression and miosis. Oxycodone acts on smooth muscle that can result in constipation, reduced GIT secretions and sphincter of Oddi spasm. Oxycodone can also cause histamine release just like morphine, causing pruritus, and flushing and orthostatic hypotension.

Naloxone is a competitive opioid antagonist so can prevent or reverse the effects of oxycodone. Oral administration of naloxone results in local competitive antagonism of the opioid receptor in the GIT, hence reducing the constipating effect of oxycodone. Naloxone has such significant first pass metabolism (<3% bioavailability), that oral naloxone does not usually result in significant systemic opioid antagonism although acute withdrawal symptoms have occurred in some opioid dependent patients.

Targin is indicated for chronic moderate-severe pain (unresponsive to non-narcotic analgesia) where opioid induced constipation is refractory to optimised regular laxatives.

Targin is also indicated as second line symptomatic treatment in patients with severe restless legs syndrome who have failed dopaminergic treatment.

2. What are the indications for thrombolysis in pulmonary embolus?

Thrombolytic therapy in acute pulmonary embolus (PE) results in early haemodynamic improvement, but at the cost of increased major bleeding where the consequences can be devastating.

Haemodynamic compromise due to acute PE is the only widely accepted indication for systemic thrombolysis. The few trials that exist are part of a meta-analysis that showed a drop in mortality from 19% to 9.4%.

Thrombolysis is not recommended in most patients with PE that are not haemodynamically compromised. The most controversial of this group is patients with severe or worsening right ventricular dysfunction. They are at an increased risk of pulmonary hypertension and mortality but randomised controlled trials of thrombolysis in these patients have not shown a mortality benefit. These trials didn't stratify for the degree of RV impairment – something for the future maybe. Case by case consideration of thrombolysis in these patients may be considered.
Other situations where thrombolysis for PE may be considered in patients without haemodynamic compromise are:

a) *Extensive clot burden*
b) *Free floating RA or RV thrombus*
c) *Patent foramen ovale*

Most guidelines also recommend catheter directed therapies in patients with high bleeding risk or as rescue therapy when systemic thrombolysis has failed. Catheter directed thrombolysis is not faster than systemic administration.

In cardiac arrest, there is no evidence for routine use of thrombolysis. There are at least 3 prospective trials that failed to show any benefit. The largest of these was the European TROICA trial (NEJM 2008;359(25):2651) where over 1050 patients involved failed to show any benefit in routine thrombolysis. ILCOR concedes that there may be a role for thrombolysis in patients where pulmonary embolus is known or suspected to be the cause. ERC/AHA/ARC guidelines make this same vague statement.

Subsequently, there is no clear guidance in dosing of thrombolysis in cardiac arrest. In non-arrested patients, guidelines are tPA 100mg over 2 hours. SVH protocol is alteplase 10mg as a bolus with the remaining 90mg infused over 2 hours. A study published in American Journal Emergency Medicine last year looked at 23 patients in PEA due to PE that were administered tPA 50mg as a bolus and found an astounding 87% survival to 2 years and no bleeding complications. I am not convinced that their electrical activity was all that pulseless, but nonetheless, I think it offers some preliminary guidance to the tPA dosing in PE patients in cardiac arrest.

UpToDate Fibrinolytic (thrombolytic) therapy in acute pulmonary embolism and lower extremity deep vein thrombosis last updated May 2017

3. What is the management of traumatic hyphaema?

- *Exclude immediate threats to vision*
  - *Open globe*
  - *Orbital compartment syndrome*
- *Head elevation 30 degrees*
  - *Facilitates inferior settling of blood away from visual axis*
- *Eye shield*
  - *A cupped shield, not padding, to avoid any pressure on the eye*
  - *Prevents further trauma to eye*
- *Bed rest*
  - *Strict – with toilet privileges only!*
  - *Dim lighting to keep pupil dilated*
  - *Avoid reading as accommodation is thought to stress blood vessels*
- *Analgesia*
  - *Avoiding NSAIDS because of platelet inhibiting properties*
- *Treat nausea*
  - *To avoid the raised intraocular pressure from vomiting*
- *Reverse coagulopathy (may be risk:benefit decision)*

- *Ophthalmology consultation at time of presentation for:*
  - *Full eye assessment*
    - *Looking for associated injuries eg vitreous bleed, retinal tear*
  - *Assess need for surgery eg large hyphaemas, high IOP*
  - *Cycloplegic drops*
    - *Facilitates examination*
    - *Provides analgesia*
    - *Long term prevents development of posterior synechiae*
  - *Steroid drops*
    - *Lower the risk of rebleeding*
  - *Intraocular pressure measurement*
    - *Raised intraocular pressure can cause optic nerve atrophy*
    - *Daily IOP measurements are required initially*
  - *Long term follow up as risk of traumatic glaucoma*

4. **What are the clinical features of funnel web spider envenomation?**

Funnel web spider toxin prevents inactivation of sodium channels causing a massive increase in autonomic activity and neuromuscular excitation. Severe systemic envenoming develops rapidly, usually within 30 minutes and almost always within 2 hours in the absence of pressure bandage immobilisation.

General – agitation, vomiting, headache, abdominal pain.
Autonomic – sweating, salivation, piloerection, lacrimation
Cardiovascular – hyper/hypotension , tachycardia, bradycardia, pulmonary oedema
Neurological – fasciculations, oral parasthesia, spasm, coma
Toxicology Handbook 3rd Ed. Murray et al

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

Regular rhythm 79/min

P waves	*Very flat, difficult to see, but can be seen in at least lead I*
PR interval	*Upper limit of normal at 0.2sec*
QRS	*Wide 0.12 in limb leads, 0.16 in V4-6*
	Axis to the right at just over 90 degrees
	Terminal rightward shift in frontal plane axis
	Small terminal R wave in aVR
	Terminal rightward forces in all chest leads
ST	*Brugada type pattern in V1 – 5*
T waves	*Widespread TWI*

➔ *Sodium channel blockade*

In this case, severe tricyclic antidepressant toxicity
AFTER $NaHCO_3$ 8.4% 50mL