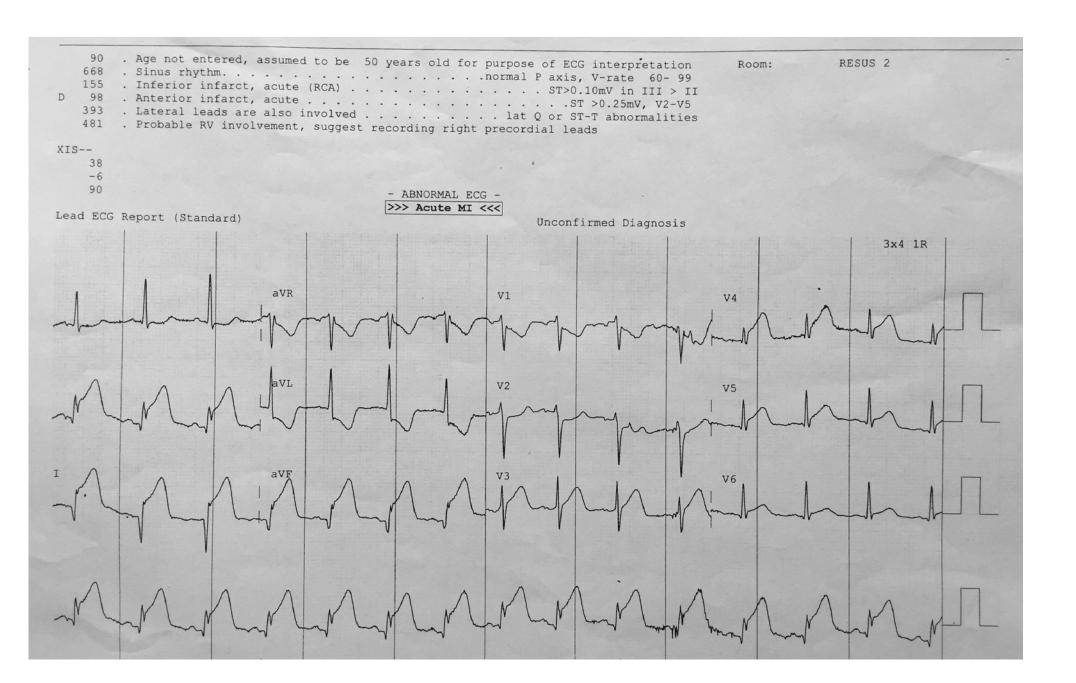
# QUIZ Feb 21<sup>st</sup> 2018 (answers below)

1. Lis	t 6 causes of intraparenchymal cerebral haemorrhage.
2. W	hat are the indications for head CT prior to lumbar puncture?
3. W	hy is hyperventilation harmful in cardiac arrest?
	hat clinical features would suggest massive pulmonary embolus as a use of cardiac arrest?
5. De	escribe and interpret the following ECG.



# QUIZ answers Feb 21st 2018

#### 1. List 6 causes of intraparenchymal cerebral haemorrhage.

Trauma

Hypertensive bleed (hypertension, ecclampsia, drug use)
Tumour (primary or secondary)
Haemorrhagic transformation of cerebral infarction
Rupture of aneurysm or AVM
Venous obstruction (venous sinus thrombosis)

Necrotic infection (abscess)

Cerebral amyloid angiopathy

Coagulopathy (thrombolysis, DIC, thrombocytopaenia)

# 2. What are the indications for head CT prior to lumbar puncture?

Age >60 years
Immunocompromise
Known CNS lesions
Seizure within a week of presentation
Signs of raised intracranial pressure (papilloedema, Cushing's sign)
Altered mentation
Focal neurological signs
Suspected subarachnoid haemorrhage

HASBUN R ET AL <u>COMPUTED TOMOGRAPHY OF THE HEAD BEFORE LUMBAR PUNCTURE IN</u> ADULTS WITH SUSPECTED MENINGITIS N ENGL J MED. 2001 DEC 13; 345(24): 1727-33

# 3. Why is hyperventilation harmful in cardiac arrest?

Cardiac arrest patients with an endotracheal or supraglottic airway do not need a pause in chest compressions in order to deliver ventilations. Chest compressions should be continuous ( $100 - 120/\min$ ) with a superimposed ventilation rate of only 6-10/min. It may be tempting to hyperventilate these patients, but there is clear evidence of harm caused by doing so.

Hyperventilation increases intrathoracic pressure which:

- Impedes venous return
- Reduces coronary artery blood flow
- Increases electrical impedence
- Causes barotrauma

Convinced?

# 4. What clinical features would suggest massive pulmonary embolus as a cause of cardiac arrest?

Risk factors for thromboembolic disease

Known DVT/PE, Immobilization, Active malignancy, Recent surgery

Pre-arrest presentation with signs/symptoms suggesting PE Respiratory distress, hypoxia, haemoptysis, normal CXR Tachycardia, hypotension

Arrest is PEA rather than VF/VT

ECHO findings during arrest
Right heart dilation (although not specific to PE)
Embolus visualized

No other obvious cause is identified for the cardiac arrest

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

Regular rhythm 90/min

P waves All conducted

Normal morphology and upright in II (so likely from SA node)

PR interval Normal

QRS Narrow, Normal axis ~0 degrees, Normal R wave progression

Inferior pathological q waves (II, III, aVF)

ST segments ST elevation inferiorly up to 6mm in lead III

ST elevation anteriorly V4-6 up to 3mm in V5

Reciprocal ST depression V1-2 of 2mm

Reciprocal ST depression aVL 3mm and aVR 2mm

T waves Pathological T wave inversion in aVL

Hyperacute T waves associated with ST elevation

QT interval 490msec – slightly prolonged

# → Sinus rhythm

Acute STEMI involving large area of infero-lateral territory Likely dominant RCA culprit as ST elevation in III>II Posterior infaction — ST depression in V1-2

# → Cardiac catheter findings:

LMCA widely patent

LAD widely patent with mild irregularities LCx widely patent with mild irregularities RCA large calibre dominant artery occluded proximally Subsequent mid vessel RCA lesion 70% also found

PTCA/stenting of proximal and mid RCA performed successfully!