

**QUIZ 17<sup>th</sup> May 2017 (answers below)**

1. What is bidirectional ventricular tachycardia?
2. What are the indications for thrombolysis in pulmonary embolus?
3. What is extra axial haemorrhage?
4. What are the common sites for intracranial aneurysms?
5. Describe and interpret the following blood gas analysis.

# RADIOMETER ABL 700 SERIES

ABL725 Emergency

PATIENT REPORT

Syringe - S 195uL

Sample #

## Identifications

Patient ID

Patient Last Name

Patient First Name

Sample type

Venous

temp

37.0 °C

FO<sub>2</sub>(I)

21.0 %

PEEP

cmH<sub>2</sub>O

Pressure Support

cmH<sub>2</sub>O

SIMV

Rate

Liter Flow

L/min

Note

Operator

## Blood Gas Values

↓ pH	7.127		[ 7.350 - 7.450 ]
↑ pCO <sub>2</sub>	57.4	mmHg	[ 32.0 - 45.0 ]
↓ pO <sub>2</sub>	33.7	mmHg	[ 75.0 - 105 ]
cHCO <sub>3</sub> <sup>-</sup> (P) <sub>c</sub>	18.2	mmol/L	
cBase(Ecf) <sub>c</sub>	-9.5	mmol/L	
p50 <sub>c</sub>	29.14	mmHg	
Anion Gap <sub>c</sub>	4.9	mmol/L	

## Oximetry Values

↓ ctHb	84	g/L	[ 115 - 180 ]
↓ sO <sub>2</sub>	59.7	%	[ 95.0 - 99.0 ]
FCOHb	0.8	%	[ 0.0 - 2.0 ]
FMetHb	1.3	%	[ 0.0 - 1.5 ]

## Electrolyte Values

cNa <sup>+</sup>	139	mmol/L	[ 137 - 146 ]
↑ cK <sup>+</sup>	7.0	mmol/L	[ 3.5 - 5.0 ]
↑ cCa <sup>2+</sup>	1.38	mmol/L	[ 1.15 - 1.30 ]
↑ cCl <sup>-</sup>	116	mmol/L	[ 98 - 106 ]

## Metabolite Values

cGlu	4.8	mmol/L	[ 3.0 - 7.8 ]
cLac	0.8	mmol/L	[ 0.5 - 2.2 ]

## Calculated Values

ctO <sub>2c</sub>	6.9	Vol%	
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## Temperature Corrected Values

pH(T)	7.127		
pCO <sub>2</sub> (T)	57.4	mmHg	
pO <sub>2</sub> (T)	33.7	mmHg	

## Notes

↑	Value(s) above reference range
↓	Value(s) below reference range
c	Calculated value(s)

Printed

## QUIZ answers 17<sup>th</sup> May 2017

### 1. What is bidirectional ventricular tachycardia?



*Bidirectional ventricular tachycardia (VT) is a really cool, but rare arrhythmia. The QRS axis (and/or morphology) is alternating in the frontal plane leads with both morphologies meeting criteria for ventricular in origin (in contrast to ventricular bigeminy where one beat originates from the sinus node).*

*The proposed mechanism is triggered activity arising alternately from the left anterior and posterior fascicle. This results in an alternating left and right axis, and the typical “bidirectional” appearance.*

*Bidirectional VT was initially described and associated with the increased automaticity of digitalis toxicity but an important other cause is catecholaminergic polymorphic ventricular tachycardia. The clinical picture of exercise or stress-induced tachycardia in a usually young individual without structural heart disease points to the correct diagnosis.*

Richter S. Bidirectional Ventricular Tachycardia JACC Volume 54 Issue 13 Sept 2009

### 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 extra axial haemorrhage?**

*Extra axial haemorrhage is intracranial bleeding that is outside the brain parenchyma. It includes extradural, subdural and subarachnoid haemorrhage.*

### **4. What are the common sites for intracranial aneurysms?**

*Cerebral aneurysms typically occur at branch points of large vessels. Approximately 90% are from the anterior circulation and 15-30% of these patients have multiple aneurysms.*

*Anterior circulation: ~90%*

- Anterior Cerebral Artery/ Anterior Communicating Artery complex: 30-40%*
- Internal Carotid Artery/ Posterior Communicating Artery junction: ~30%*
- Middle Cerebral Artery bifurcation: 20-30%*

*Posterior circulation: ~10%*

- Basilar artery bifurcation 5%*
- Superior Cerebellar Artery and Posterior Inferior Cerebellar Artery 5%*

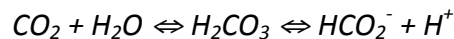
## 5. Describe and interpret the following blood gas analysis.

*The immediate concern in this blood gas analysis is the K 7.0 mmol/L and should prompt immediate clinical assessment and treatment. Having done that, we can proceed to look at the rest of the result.*

*The patient is acidotic at 7.127*

*The pCO<sub>2</sub> is raised at 57.4mmHg indicating a respiratory acidosis.*

*A rise in pCO<sub>2</sub> drives the carbonic acid dissociation equation to the right:*



*This results in an expected acute rise in HCO<sub>2</sub><sup>-</sup>.*

*This is known as cellular buffering. Acutely, HCO<sub>2</sub><sup>-</sup> rises by 1mmol/L above 24mmol/L for every 10mmHg pCO<sub>2</sub> above 40mmHg.*

*So in this case, HCO<sub>2</sub><sup>-</sup> should be 25.7mmol/L. But it is 18.2mmol/L = a fall of 7.5mmol/L. So there is a metabolic acidosis, i.e. an acid (H<sup>+</sup>) is driving the above dissociation equation to the left.*

*Base excess is calculated as the concentration of base in excess if the pCO<sub>2</sub> is returned to 40mmHg and temperature is 37°C for a pH of 7.4. The base excess is -9.5 (base is deficient, as there is acidosis) consistent with the fall of 7.5 in HCO<sub>2</sub><sup>-</sup>.*

*p50 is 29.1mmHg indicating a slight shift of the oxyhaemoglobin dissociation curve to the right, consistent with acidosis.*

*Anion gap is 4.9 mmol/L = the lower end of normal range (4-12mmol/L).*

*Causes for a low anion gap include decrease in unmeasured anions (low albumin, low phosphate) or an increase in unmeasured cations (high Ca, Mg, K, or cationic IgG paraprotein in multiple myeloma). Here there is raised K and slightly raised iCa.*

*Anaemia with Hb 84g/L*

➔ *Hyperkalaemia – potentially life threatening level*

*Mixed respiratory and normal anion gap metabolic acidosis*

*Anaemia*

*(See QUIZ answers 3<sup>rd</sup> May 2017 for causes of normal anion gap metabolic acidosis)*