QUIZ 23rd May 2018 (answers below)

1. **What is your clinical definition of anaphylaxis?**

2. **What is the management of rocuronium anaphylaxis?**

3. **List all the potential causes of a raised troponin level.**

4. **How would you investigate viral myocarditis?**

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

RADIOMETER ABL800 FLEX

ABL827 Emergency
PATIENT REPORT

Syringe - S 250uL

Sample # 23578

Identifications

Patient ID	
Patient Last Name	
Patient First Name	
Sex	
Sample type	Not specified
T	37.0 °C
$FO_2(I)$	21.0 %
PEEP	cmH2O
Pressure Support	cmH2O
SIMV	Rate
Liter Flow	L/min
Note	
Operator	
Accession No.	

Blood Gas Values

↑ pH	**7.459**		[7.350 - 7.450]
↑ pCO_2	**62.4**	mmHg	[32.0 - 45.0]
↓ pO_2	**28.3**	mmHg	[75.0 - 105]

Oximetry Values

*c*tHb	**151**	g/L	[115 - 165]
↓ sO_2	**47.2**	%	[95.0 - 99.0]
*F*COHb	**0.9**	%	[0.0 - 1.5]
*F*MetHb	**0.5**	%	[0.0 - 1.5]

Electrolyte Values

↓ cNa^+	**134**	mmol/L	[137 - 146]
↓ cK^+	**2.2**	mmol/L	[3.5 - 5.0]
↓ cCa^{2+}	**1.09**	mmol/L	[1.15 - 1.30]
↓ cCl^-	**84**	mmol/L	[98 - 106]

Metabolite Values

*c*Glu	**5.0**	mmol/L	[3.0 - 7.8]
*c*Lac	**1.5**	mmol/L	[0.0 - 2.2]
*c*Crea	**59**	µmol/L	[40 - 90]

Calculated Values

ABE_C	**16.1**	mmol/L	[-]
$cHCO_3^-(P)_C$	**43.7**	mmol/L	[-]

Notes

↑	Value(s) above reference range
↓	Value(s) below reference range
c	Calculated value(s)
	0293: Warning: HbF detected and compensated for

QUIZ answers 23rd May 2018

1. **What is your clinical definition of anaphylaxis?**

In 2005, there was an international symposium that put together a universally accepted definition of anaphylaxis, and established clinical criteria for diagnosis. They published the following:

Clinical criteria for diagnosing anaphylaxis
Anaphylaxis is highly likely when any one of the following 3 criteria are fulfilled:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (eg, generalized hives, pruritus or flushing, swollen lips-tongue-uvula)
   AND AT LEAST ONE OF THE FOLLOWING
   a. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
   b. Reduced BP or associated symptoms of end-organ dysfunction (eg, hypotonia [collapse], syncope, incontinence)

2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):
   a. Involvement of the skin-mucosal tissue (eg, generalized hives, itch-flush, swollen lips-tongue-uvula)
   b. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
   c. Reduced BP or associated symptoms (eg, hypotonia [collapse], syncope, incontinence)
   d. Persistent gastrointestinal symptoms (eg, crampy abdominal pain, vomiting)

3. Reduced BP after exposure to known allergen for that patient (minutes to several hours):
   a. Infants and children: low systolic BP (age specific) or greater than 30% decrease in systolic BP*
   b. Adults: systolic BP of less than 90 mm Hg or greater than 30% decrease from that person's baseline

   *Low systolic blood pressure for children is defined as < 70mmHg from 1 month to 1 year, <(70 mmHg + 2xage) from 1 to 10 years, and less than 90mmHg from 11 to 17 years.

J Allergy Clin Immunol 2006;117:391 - 7

2. What is the management of rocuronium anaphylaxis?

A New Zealand study published Anesthesiology. 2015 Jan;122(1):39-45 found the incidence of anaphylaxis to rocuronium and suxamethonium seem to be much the same at around 1:2000 – 2500. In contrast, the incidence of anaphylaxis to atracurium was 10 times lower at 1:22000.

Adrenaline is the only drug recommended as first line therapy in all published national anaphylaxis guidelines. Australian guidelines (ASCIA - Australian Society of Clinical Immunology and Allergy) recommend intramuscular injection of adrenaline (1:1000) into outer mid thigh (0.01mg per kg up to 0.5mg per dose). Dosing can be repeated every 5 minutes, moving on to an adrenaline infusion if needed.

Other measures include stopping administration of the offending agent, calling for help, maintaining the airway, administering oxygen, Trendelenburg position and IV saline 20mL/kg for hypotension. Persistent hypotension despite adrenaline and adequate volume, may require vasopressors.

Glucocorticoids have been thought to decrease protracted or biphasic reactions by down-regulating the late eosinophilic inflammatory response, not the early phase of the reaction. There is some published evidence that they might be ineffective and there are no RCTs that demonstrate benefit.
Glucocorticoids for the treatment of anaphylaxis: Cochrane systematic review. Choo KJ, Simons E, Sheikh A Allergy. 2010 Oct; 65(10):1205-11.

There is no consensus around the use of antihistamines in anaphylaxis. According to ASCIA, antihistamines have no role in treating or preventing the respiratory or cardiovascular symptoms of anaphylaxis. Certainly, the use of IV first generation (sedating) H1 antagonists can cause hypotension.

It has been proposed that sugammadex could be an antidote to rocuronium anaphylaxis. Sugammadex is a rocuronium reversal agent that works by encapsulating the rocuromium. Unfortunately, molecular models seem to show that the allergenic ammonia group on the rocuronium is still exposed even when it is encapsulated by sugammadex. Additionally, clinical trials have not shown a benefit and there is also a relatively not insignificant incidence of anaphylaxis to sugammadex!
Takazawa T Sugammadex and Rocuronium-induced anaphylaxis J Anesth. 2016; 30: 290–297

ASCIA recommends observing all patients for at least 4 hours as there is a 3-20% incidence of biphasic reactions. However, these reactions would seem to be less severe.

All patients that have had an anaphylactic reaction should be referred for specialist follow up. If there is a risk of re-exposure (probably not for rocuronium in the community), patients should be given an Epipen and taught how to use it before discharge.

Patients that have had an allergic reaction to an anaesthetic agent should be referred to the anaesthetic allergy clinic in their local health district.

3. List all the potential causes of a raised troponin level.

Myocardial infarction
Heart failure
Cardiac contusion (trauma)
Myocarditis
Cardioversion
Heart biopsy
Aortic dissection
Tachy or brady arrhythmias
Cardiac surgery
Coronary artery stenting
Renal failure
Pulmonary embolus
Severe pulmonary hypertension
Sepsis
Severe critical illness
Burns
Extreme exertion
Amyloidosis or other infiltrative diseases
Stroke
Subarachnoid haemorrhage

4. **How would you investigate viral myocarditis?**

Troponins

Indicator of myonecrosis but level does not seem to correlate with degree or presence of cardiac dysfunction.

Myonecrosis occurs early in the illness. Persistent elevation suggests ongoing necrosis.

ECG

Can show changes of pericarditis in myopericarditis.

Can show arrhythmias.

May show localised ST changes and be difficult to distinguish from ACS.

Widening of QRS from myocarditis is associated with a worse prognosis

Viral antibody titres

Routine screening for various potential viral pathogens is not recommended due to the low yield, delay in rising titres and absence of influence on management. Specific viral diagnoses should be made, such as HIV infection.

ECHO

Not a diagnostic test but assesses for complications of myocarditis - Pericardial effusion, Ventricular function, and Wall thickness

MRI

Cardiac MRI can help confirm the diagnosis of myocarditis by detecting inflammatory hyperaemia, wall oedema and scarring as well as ventricular size, wall motion abnormalities and pericardial effusion

Cardiac angiography

May be necessary for cases that are indistinguishable from ACS.

Cardiac biopsy

Gold standard for diagnosis

Shows lymphocyte infiltration and myocyte necrosis

Not performed routinely

Cellular changes are patchy and may be missed on biopsy site

Invasive procedure

Does not change management in most cases

May be appropriate to investigate for giant cell arteritis or infiltrative diseases such as amyloid

5. Describe and interpret the following blood gas analysis.

pH 7.459 *Alkalotic side of normal*

pCO_2 62.4mmHg *This would cause a respiratory acidosis*
Here pH is alkalotic, so there must be a metabolic alkalosis

Respiratory compensation for metabolic alkalosis:
pCO_2 rises 0.7mmHg for every 1mmol/L rise in Bicarb
Compensation can go up to 50 – 55mmHg
pCO_2 above this suggests concurrent respiratory acidosis

In this case Bicarb is 43.7mmol/L, 19.7 above normal (24)
pCO_2 should rise to 40 + (19.7 x 0.7) = 53.8mmHg
Here pCO_2 has rise higher to 62.4 = respiratory acidosis

K 2.2 mmol/L *Hypokalaemia can be the cause of metabolic alkalosis, the result of metabolic alkalosis and can be the maintenance factor of metabolic alkalosis*

iCa 1.09 mmol/L *Ionised Ca goes down with alkalosis so this is expected*

Cl 84 mmol/L *Hypochloraemia maintains metabolic alkalosis as it increases bicarbonate reabsorption at the tubules*

➔ *Metabolic alkalosis*
Respiratory acidosis
Hypokalaemia and hypochloraemia that would maintain the alkalosis

➔ *This patient has anorexia nervosa and had lost 10kg over 2 months*
Diuretic misuse, purging, laxative misuse and malnourishment would all contribute to this blood gas result
It is unclear why there is a respiratory acidosis

Metabolic alkalosis

Metabolic requires an initiation factor. Then, because the kidneys are usually excellent at excreting excess bicarbonate, there needs to be something that impairs kidney function in order to maintain the alkalosis.

Initiation factors	Bicarbonate gain
	Endogenous - ketone metabolism
	Exogenous – antacids, sodium bicarbonate, citrate
	Acid loss
	Renal – diuretics, mineralocorticoid excess, hypercalcaemia
	GIT – vomiting gastric acids, NG suction
	Hyperventilation of patient with compensated hypercapnia
Maintenance factors	Chloride depletion
	Hypokalaemia
	Decreased GFR
	Mineralocorticoids