



## **PAEDIATRIC FELLOWSHIP EXAMINATION REPORT**

**August / October 2010**

This report is prepared to provide candidates, tutors and their Supervisors of training with information about the way in which the Examiners assessed the performance of candidates in the Examination. Answers provided are not model answers but guides to what was expected. Candidates should discuss the report with their tutors so that they may prepare appropriately for the future examinations

The exam included two 2.5 hour written papers comprising of 15 ten-minute short answer questions each. Candidates were required to score at least 50% in the written paper before being eligible to sit the oral part of the exam. The oral exam comprised 8 interactive vivas and two separate hot cases.

The tables below provide an overall statistical analysis as well as information regarding performance in the individual sections. A comparison with the previous three examinations is also provided.

The Written section of the Examination was held in Auckland, Melbourne and Sydney. The Clinical and Viva sections of the examination were held in Auckland at the Starship Children's Hospital.

## STATISTICAL REPORT

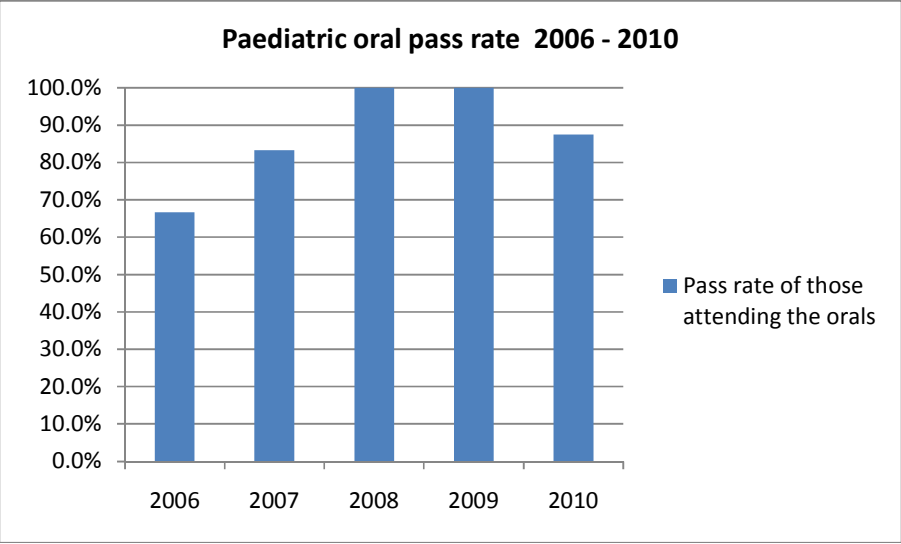
Overall pass rates	October 2010	October 2009	October 2008
Total number presenting (written + carry + OTS)	13	6	5
Total number invited to the oral section	8	5	4
Total number successful at orals	7	5	4
	88%	100%	100%
Overall pass rate	7/13	5/6	4/5
	54%	83%	80%

Sectional pass rates	October 2010	
	Pass rate	Highest individual mark
Hot Case 1	75%	77%
Hot Case 2	75%	87%

Total number successful in the Hot Case section	6
Overall Hot Case pass rate	75%

Sectional pass rates	October 2010	
	Pass rate	Highest individual mark
Viva 1	62%	90%
Viva 2	87%	90.0%
Viva 3	62%	75%
Viva 4	87%	90%
Viva 5	100%	90%
Viva 6	100%	100%
Viva 7	87%	100%
Viva 8	100%	90%

Total number successful in the Viva section	8
Overall Viva pass rate	100%



## WRITTEN EXAMINATION REPORT

### Question 1 (a)

*An infant with a large VSD is admitted to your ICU in severe heart failure at 6 weeks of age. He is intubated and mechanically ventilated. He has a history of poor feeding and is noted to be very thin.*

*How would you assess his nutritional state and requirements?*

#### Nutritional state:

- Anthropometry: Weight and height on growth charts. Evidence of failure to thrive on longitudinal data. Skin fold/arm circumference
- Laboratory      Serum proteins, inflammatory markers

#### Nutritional requirements:

- Estimation equations (eg Harris-Benedict) inaccurate. Requirement likely to be significantly higher in children with heart failure (Around 120- 150Cals/kg/day)
- Measurement - Indirect calorimetry if available. Depends on no leak around ETT and low FiO<sub>2</sub>

### Question 1 (b)

*How would you improve his nutrition?*

#### Intake - Aim to provide adequate calories as per above

- Enteral feeding is ideal. Nasogastric initially, but if problematic via nasojejunal tube.
- Appropriate feed for age
- Appropriate caloric density – may be increased if tolerated
- Bowel care. May need early implementation of laxative therapy.
- Avoid frequent interruptions to feeds (repeat fasting for possible extubation etc)
- Parenteral feeding if unable to tolerate enteral feeds

#### Expenditure

- Adequate treatment of heart failure (including correction of any anaemia)
- Adequate sedation
- Ventilation as treatment of heart failure and means of reducing caloric expenditure

### Question 1 (c)

*Discuss likely problems that may occur with feeding in this patient.*

- Excessive volume load with feeding.
- Feed intolerance due to increased caloric density of feed.
- Poor gut function – heart failure, opiates.
- Inability to provide adequate nutrition – may need surgery
- Dangers of overfeeding (CO<sub>2</sub> production, hyperglycaemia)
- Complications from TPN (line complications, sepsis, liver dysfunction)

### Question 2 (a)

*You are called urgently to the PICU in the middle of the night by a senior registrar who is having trouble intubating a 6 month old child with bronchiolitis. The infant was born at term, is thought to be deaf and has malformed ears. On your arrival the registrar is handbagging the moving patient. There is obvious respiratory distress, SpO<sub>2</sub> is 83%, HR 190, and the abdomen is grossly distended.*

*Outline your management of this patient's airway and breathing.*

- Airway and breathing need to be addressed urgently.
- Waking patient not an option because of respiratory failure
- Because of potential difficulty with intubation, inhalational induction safest option if patient is breathing.
- Prior to this - Assess for predictors of difficult intubation. If present or if not confident, call ENT, anaesthesia or both.
- Prepare for intubation including difficult airway kit
- Inhalational induction if breathing
- Ensure patient deeply anaesthetised before manipulating airway
- This will be slow if small tidal volumes
- Spray cords with local anaesthetic
- Intravenous induction if patient not breathing adequately/requires urgent intubation
- Use adequate dose of short-acting muscle relaxant
- Empty stomach
- If unable to intubate call for help
- If unable to ventilate –
- Secure airway immediately
- Airway adjuncts: Guedel, 2 main techniques, LMA
- Intubation using bougie, special scope
- Invasive airway - cricothyroidotomy
- Able to ventilate
- More time to use more advanced techniques (with appropriate help)
- Retrograde intubation
- Fiberoptic intubation

## Question 2 (b)

*How could this situation have been avoided?*

- Identification of potential difficulty with ventilation and intubation in syndromic patient
- Ideal intubation conditions including patient positioning, equipment etc
- Appropriate airway management plan including failed intubation algorithm
- Adequate training and practice for trainees

## Question 3.1

*Describe the underlying principles and mode of operation of a pulse oximeter.*

- Beer Lambert: absorption of light by a solute is proportional to solute concentration
- DeoxyHb and OxyHb absorb maximally at different wavelengths
- Both are estimated using absorption of light of 2 wavelengths (660nm, 940nm)
- Saturation estimated by ratio of OxyHb:(Oxy+DeoxyHb)
- Tissue (finger, earlobe, etc) interposed between photodetector and 2 LEDS
- LEDS flash several hundred times per second, allowing measurements during pulsatile and non-pulsatile flow
- Pulsatile saturation = arterial + venous + tissue saturation
- Non-pulsatile saturation = venous = tissue saturation
- These values are manipulated by an algorithm to display SpO<sub>2</sub> and waveform.

## Question 3.2

*List 8 factors which may affect accuracy of oximetry measurement.*

- Improper probe placement (contact, appropriate size etc)
- Motion artefact (seizures, shivering, transport, movement)
- Abnormal haemoglobins, carboxyhaemoglobin, methaemoglobin
- Poor perfusion (shock, low cardiac output)
- Severe vasoconstriction (cooling, pressors)
- Pulsatile venous congestion
- Skin pigmentation, nail polish
- Severe anaemia
- Intense ambient light
- Electromagnetic radiation

#### **Question 4 (a)**

*A three year old child with ascending weakness and respiratory difficulties is admitted to PICU with a provisional diagnosis of Guillain Barré Syndrome (GBS).*

*List five other common clinical findings of GBS.*

- sensory neuropathy
- pain
- hypo/ areflexia
- autonomic involvement: sinus tachycardia, hypertension, arrhythmia
- cranial nerve involvement
- ataxia

#### **Question 4 (b)**

*For GBS, briefly outline:*

##### **1. Mechanism of disease**

- Infection triggers autoimmune response with T cells, antibodies and complement directed against myelin proteins.
- Macrophages invade myelin sheath and denude axons.

##### **2. Diagnostic test findings**

- CSF: raised protein concentration in 80%
- Motor conduction studies: identify demyelination, earliest studies to be abnormal
- Sensory conduction studies: abnormal later

##### **3. Clinical course**

- Acute onset, nadir at 2-4 weeks, plateau phase, then recovery phase.
- 4-15% mortality overall, children very unlikely to die, recovery more rapid and complete than adults
- Up to 20% disabled, residual weakness and fatigue
- Artificial respiration adverse prognostic indicator

##### **4. Treatment**

- Monitor VC: Intubate if necessary
- Monitor for cardiac arrhythmia
- Multidisciplinary care.
- Management of symptoms: pain, retention, ileus
- Occupational therapy
- Physiotherapy
- Plasma exchange, equal efficacy to:
- IVIG five days of 4g/kg
- (No evidence supporting steroid use.)

References:

Lancet 2005;366:1653-1666

J Paediatr Child Health 2005;41:237-241

Corticosteroids for GBS: Cochrane Database of Systematic Reviews 2010

Intravenous immunoglobulin for GBS: Cochrane Database of Systematic Reviews 2010

Plasma exchange for GBS: Cochrane Database of Systematic Reviews 2008

**Question 5**

*A three year old girl with lower limb injuries following a motor vehicle accident develops oliguria and dark urine. Laboratory findings are as follows:*

	Result	Normal range
K <sup>+</sup>	6.5	3.5-5.2 mmol/l
Calcium	1.4	2.1-2.8 mmol/l
Phosphate	3.5	1.00-2.00 mmol/l
Uric acid	1.2	0.14-0.36 mmol/l
AST	660	0-60 U/l
ALT	580	<45 U/l
Creatinine	120	20-60 umol/l
Urea	10	1.4-5.4 mmol/l
CK	50 000	60-220 U/l
APTT	70	25-37 seconds
INR	3.0	0.8-1.2
Urine:	Myoglobinuria, protein casts, uric acid crystals	

**Question 5 (a)**

*What is the most likely diagnosis?*

- rhabdomyolysis

**Question 5 (b)**

*Outline the pathophysiology.*

- Destruction or disintegration of striated muscle, characterised by muscle breakdown and necrosis resulting in leakage of intracellular muscle constituents into circulation and extracellular fluid



### Question 5 (c)

*Outline an appropriate management plan.*

- ABC resuscitation
- Preservation of renal function with vigorous hydration
- Bicarbonate/ alkalinising agents and mannitol/ osmotic diuretics (unproven benefit over aggressive volume replacement with saline alone)
- Renal replacement therapy with haemodialysis or hemofiltration (PD inadequate/ plasma exchange no additional benefit)
- Treat underlying cause
- Measurement of compartment pressures
- Fasciotomy may be indicated

### References

Critical Care 2005;9(No 2).

Critical Care Clinics 2004;20:171-192

### Question 6

*A two year old child is admitted following a week-long febrile illness. She has been treated with regular paracetamol for the duration of the illness. Her results are shown:*

	Results	Normal range
Sodium	141	135-145 mmol/L
Potassium	4.5	3.5-5.2 mmol/L
Urea	4.0	1.8-5.4 mmol/L
Creatinine	60	20-60 µmol/L
Glucose	2.5	3-11 mmol/L
APTT	40	25-37 seconds
INR	3.3	0.8-1.2
AST	7000	0-80 U/L
ALT	9618	<45 U/L
SBR	123	0-24 µmol/L
Paracetamol level	10 on day 2	<40 µmol/L

### Question 6 (a)

*What is the recommended maximum daily dose of paracetamol for this child?*

- 15 mg/kg (180 mg) 4 hourly to max of
- 90 mg/kg/day (1080mg) for 48 hours then max of
- 60mg/kg/day (720mg) thereafter

**Question 6 (b)**

*Outline the mechanism of paracetamol toxicity.*

- Principal toxic metabolite (NAPQI) produced by hepatic cyto P450.
- Glutathione stores in liver detoxify this metabolite
- Glutathione depletion results in accumulation of NAPQI, causing hepatocellular necrosis and damage to other organs.
- Greater production of NAPQI from overdose further depletes glutathione stores and toxicity.

**Question 6 (c)**

*List 4 risk factors for chronic paracetamol toxicity.*

- unintentional multiple overdosing (>150 mg/kg/day if no other risk factors or >75 mg/kg/day if other risk factors)
- fasting/ dehydration/ vomiting: (causes glutathione depletion)
- renal failure
- hepatic failure
- inborn error of metabolism
- enzyme-inducing or hepatotoxic drugs
- rectal administration (levels vary widely)
- delays in treatment (N-AC)

**Question 6 (d)**

*Outline your management plan for this child.*

- ABC/ resuscitation
- history - determine amount ingested.
- Apply paracetamol level to nomogram
- Commence N-Acetylcysteine 150mg / kg over first hour then 10mg/kg/h for 20 hours.
- Supportive measures as required: ventilation, clotting factors, glucose
- Monitor liver function tests
- Cysteamine and methionine have also been used but more adverse effects than N-acetylcysteine and less effective
- (Charcoal/ lactulose not indicated)

Reference

Pediatrics 2001;108(4):1020-1024

### Question 7 (a)

*A newborn infant is admitted to your ICU with profound cyanosis. A diagnosis of total anomalous pulmonary venous drainage (TAPVD) is made by echocardiography. Surgery is planned within six hours but the SpO<sub>2</sub> remains in the low 60s.*

*Outline your approach to management and optimisation prior to surgery.*

- ABC: Resuscitate if shocked: oxygen, intubate, inotropes
- Sedation and paralysis
- Diuretics
- Treat pulmonary hypertension: alkalosis, nitric oxide. There is a risk of making things worse by increasing pulmonary blood flow.
- Transfuse
- prostin to open duct in obstructed lesion. This may improve cardiac output, but will further decrease saturation.
- Contact surgical team to expedite surgery.
- exclude other abnormality: cardiac lesions (pulmonary atresia, dextrocardia, ASD), lung problems (pulmonary hypoplasia) and spleen problems (asplenia, polysplenia)
- ECMO if surgery cannot be brought forward

### Question 7 (b)

*Outline the role and actions of prostaglandin E1 (prostin/ alprostadil) in unrepaired TAPVD.*

- No contraindications
- If stable but cyanosed, wait for echo
- Start prostaglandin if unwell and cyanosis, murmur, abnormal pulses
- PDA allows R to L shunt and may increase cardiac output
- If obstruction:
  - + may relax sinus venosus, reduce resistance and help arterial saturation
  - may worsen congestion, increase systemic at expense of pulmonary flow and worsen saturation
- may make no difference, suggests diagnosis

### Question 7 (c)

*List 3 particular issues seen in ICU following surgical correction of this lesion.*

- Pulmonary hypertension: Paralyse, sedate, alkalinise, iNO
- Respiratory failure from preoperative oedema and bypass (dialysis, diuretics)
- Dysrhythmias: nodal, SVTs (pacing, antiarrhythmics)
- Residual venous obstruction (surgery)

### Question 8

*The anatomical and physiological immaturity of children (relative to adults) predisposes them to certain traumatic injuries.*

*In table form list 10 such features and the injuries that are more likely to occur as a result of them.*

Anatomic Difference	Injury
Head: large relative to body	Brain: Shearing injuries, head takes impact
Weak neck muscles  Ligamentous laxity and cartilaginous spine	Cervical injuries less common but more likely to be higher level  SCIWORA
Chest: Ribs compliant/unossified	Pulmonary contusion, haemopneumothorax may occur without rib fractures Fewer penetrating injuries Few flail chests Aortic transection unlikely
Low FRC and high metabolic demand	Hypoxic injury common
Liver and spleen less protected	More likely to have solid organ injury
Small blood volume	Haemorrhage from scalp laceration or other site may be life threatening
Bladder abdominal not pelvic	Increased risk of bladder injury
Limbs cartilaginous	Greenstick fractures
Small and dependent	Non accidental causes: shaking injuries
Floppy upper airways	Increased risk of hypoxic injury

## Question 9

*A 6 year old boy who was resuscitated following drowning is intubated and ventilated in PICU. Brain death has been confirmed and his family has consented to organ donation. Organ retrieval is scheduled in 12 hours time.*

*Outline the principles of management of a brain dead donor prior to organ retrieval.*

### 1. Maintain organ perfusion

- Initial sympathetic storm with hypertension – use short-acting agents as this is followed by loss of vasomotor tone
- Hypotension (especially if fluid restricted) - give fluid boluses.
- Vasopressors (usually noradrenaline) after adequate filling.
- Inotropes if there is also impaired cardiac function.
- Aim to keep vasopressor and inotrope doses to a minimum to avoid organ ischaemia. Normotension may not be achievable – follow indicators of organ perfusion.

Vasopressin may be used to reduce inotrope requirement. Several studies report no effect on graft survival of high inotrope infusion rates. The need for catecholamine support in the donor is not a contraindication to organ donation.

### 2. Diabetes Insipidus

- If suspected, check serum osmolality and electrolytes and urine specific gravity, osmolality and electrolytes
- Treat with vasopressin (AVP) infusion: 0.002 – 0.04U/kg/h. (DDAVP intranasal or IV 0.25 - 2µg (not/kg) sometimes used)
- ½ - 1 hourly urine output. Adjust Vasopressin dose/infusion
- Replace excess fluid loss with water-based solution with sodium and potassium in same concentration as in urine
- Alternatively 2-5U vasopressin may be added to 1 litre of hypotonic fluid to replace urine output +10% each hour.
- Check serum and urine electrolytes every 4 hours

### 3. Hypernatraemia

- No correlation between donor serum sodium and early liver graft function in children (unlike adults) – manage hypernatraemia gradually

### 4. Steroids

- No effect on haemodynamics but may improve pulmonary graft function

### 5. Thyroxine

- No evidence for routine use but may be useful in haemodynamically unstable patients (T3 at 0.05-0.2 micrograms/kg/hour)

## 6. Hyperglycaemia

- Catecholamine induced insulin resistance and inhibition of secretion. Treat with insulin if causing osmotic diuresis

## 7. Other electrolytes / Hypothermia / Anaemia, coagulopathy / Respiratory / treatment of bleeding etc

### References

ANZICS. The ANZICS Statement on Death and Organ Donation (3<sup>rd</sup> Edition). Melbourne: ANZICS, 2008  
Pediatr Clin North Am 2001 Jun;48(3):715-49

## Question 10

*Outline the effects of positive pressure ventilation on left and right ventricular performance.*

### Right ventricular preload

- Effect of PPV on ITP – pressure transmitted depends on airway resistance and lung compliance
- PPV decreases RA Ptm and increases RAP – decreasing venous return unless mean systemic pressure increases by compensatory sympathetic vasoconstriction
- As the diaphragm descends, intra-abdominal pressure increases and the transmbrane pressure for intra-abdominal venous capacitance vessels decreases – reducing the compliance – increasing IVC pressure – partially compensates for increases ITP on reducing venous return

### Right ventricular afterload

- PVR depends on lung volume – ↑ PVR at high and low volumes
- PVR also affected by acidaemia/alkalaemia
- Pulmonary hypoxic vasoconstriction
- PPV can increase RV afterload and reduce RV output

### Left Ventricular preload

- Dependent on RV output and PVR plus effect on LA Ptm – reduced venous return
- Reduced RV emptying – cause ventricular septal deviation and reduced LV filling –

### Left Ventricular afterload

- reduces LV afterload: increased pressure gradient between ITP and Extrathoracic vascular pressure potentially increasing cardiac output
- PPV reduces myocardial oxygen consumption by reducing cardiac work
- Effect of PPV on cardiac function also dependent on any cardiac dysfunction
- Eg Right Ventricular Diastolic dysfunction – PPV reduces cardiac output because of increased RV afterload has more impact than other factors
- Left Ventricular Systolic dysfunction – PPV increase cardiac output because effect on LV afterload predominates

(PPV – positive pressure ventilation; ITP – intrathoracic pressure; RA – right atrium; Ptm – transmembrane pressure  
RAP – Right atrial pressure)

#### Reference

Pediatric Critical Care Medicine 2009;10(3):313-322

### Question 11 (a)

*A 9 month old boy is admitted to your unit following a MVA where he was a restrained passenger in a forward facing child seat. He was intubated & ventilated at scene for apnoea. MRI reveals C1-C2 spinal cord contusion and an unstable lesion. There is no other major trauma.*

*Outline the clinical picture expected with this acute spinal cord injury.*

- C1/2 so diaphragm and intercostals affected if complete will not breathe
- Initial flaccid paralysis of all muscle groups below neck followed by hypertonia and hyperreflexia
- Bladder and bowel affected
- Autonomic paralysis followed by dysreflexia
- Spinal shock
  - Refers to transient paralysis that can rarely recover fully
  - Potassium mediated
  - Sometimes (incorrectly) used to refer to hypotension due to SCI
- Primary and secondary injury

### Question 11 (b)

*Outline your management in the first 24 hrs.*

- Steroids. Controversial. NASCIS I II and III. In most guidelines. Standard dose: methylprednisolone 30 mg/kg IV bolus, then infusion of 5.4 mg/kg/hour for 23 hours
- Ventilation. Maintain normal O<sub>2</sub> and CO<sub>2</sub>
- Circulation: fluids/ pressors if needed for SCI-related hypotension (exclude other causes)
- Analgesia
- Tertiary survey /serial exam
- Counselling. Complete vs partial. Spinal shock. Chances of recovery. Need for stabilization. Possibility of withdrawal. If no improvement after spinal shock resolution and complete <5% chance of improvement
- Team communication (radiology, spinal team, neurology, nursing staff, social work)
- Neck stabilization: stiffneck vs Philly vs sandbag and tapes. Discuss surgical stabilization with surgeon.

- Pressure sores; early and proactive
- Bladder catheter
- Ulcer prophylaxis (feed or PPI's)
- Temperature support.

#### References

Steroids for acute spinal cord injury. Cochrane Database of Systematic Reviews 2002

### Question 12 (a)

*A previously well 5 yr old with Human Metapneumovirus pneumonia has been mechanically ventilated for 5 days. Despite HFOV and inhaled Nitric Oxide oxygenation deteriorates and there is ongoing air leak syndrome. You institute V-V ECMO via the neck and groin using a centrifugal device. There is no other organ dysfunction.*

*List the important prognostic features for this child.*

- ELSO data: paediatric ECMO for viral pneumonia survival ~60 % (bacterial pneumonia 55 %).
- Superinfection would increase morbidity and mortality (especially in PVL - Staph. Aureus).
- Air Leak syndrome not specifically listed in ELSO paediatric data, would however certainly contribute further to mortality.
- Neonatal ECMO with air leak syndrome survival ~75 %.
- VV ECMO support likely to reduce mortality in this child (compared to VA ECMO support): pulsatile flow less invasive / lower ACT acceptable / "lung" as filter for emboli.
- Overall estimate risk of mortality ~ 50 %.

### Question 12 (b)

*Outline assessment of respiratory function while on V-V ECMO.*

#### 1. Lung compliance

- Various approaches
- Rely on dynamic compliance calculated by ventilator
- Observe change in tidal volume with fixed PIP and PEEP at either resting settings or predetermined settings (eg 30/5) using either the ventilator or a calibrated pneumotachograph.

#### 2. Oxygenation

- Testing oxygenation goes hand in hand with weaning of VV ECMO support.
- While set on a fixed ventilation setting FiO<sub>2</sub>, fixed (preferable minimal VV ECMO support: 80 ml/kg/min, with fixed settings on oxygen blender and gas flow), an improving arterial oxygenation



(PaO<sub>2</sub> and SaO<sub>2</sub>) with an increasing discrepancy of measured mixed venous Sats and SaO<sub>2</sub> indicates lung improvement.

- An “oxygen challenge test” can reveal some more information: increasing ventilation settings to conventional settings and increasing FiO<sub>2</sub> to 100 %, the difference of paO<sub>2</sub> and calculated pAO<sub>2</sub> – paO<sub>2</sub> over time give also a reasonable good indication for lung function.

3. CXR and or CT assessed resolution of lung injury

### **Question 12 (c)**

*List possible complications of VV ECMO in this child.*

- Bleeding – local or intracerebral
- Infection
- Mechanical
- Embolic (reduced as VV)

### **Question 13 (a)**

*You are asked to admit a 9 month old girl who presents to your Emergency Department in cardiac failure. She has been progressively less active and more tachypnoeic over the last 3 weeks.*

*An echocardiogram shows:*

*Structurally normal heart with gross biventricular dilation*

*Left Ventricular Ejection Fraction (LVEF) 18% (normal range >50% )*

*Left ventricular Shortening Fraction (SF) 10% (normal range >30% )*

*List 6 possible causes for this child's myocardial dysfunction.*

- Idiopathic
- Myocarditis
- Missed ALCAPA or other structural lesion
- Kawasaki disease
- Cardiomyopathy (syndromic or metabolic – Carnitine, Selenium, Barth)
- Incessant arrhythmia
- Extracardiac L to R Shunt (eg vein of Galen)
- Endocardial Fibroelastosis (EFE)

### Question 13 (b)

*Outline your management priorities over the next 4 days.*

#### Support LV

- CPAP or BIPAP
- If need to intubate consider ECLS on Stand-By
- Commence  $\beta$ -agonists as indicated. Dopamine, Dobutamine or Adrenaline up to 0.05  $\mu\text{g/kg/min}$
- Commence Milrinone if tolerated
- Consider Levosimendan
- Consider biventricular pacing
- Early use of ECLS if patient is deteriorating or not improving
- Consider ECLS bridge to recovery or transplant.

#### Treat / monitor complications

- Fluid management
- Fluid and Water Restriction to 25 % maintenance
- Frusemide (?infusion), add Spironolactone.
- Avoid hypovolaemia

#### Thrombosis

- consider heparin/warfarin

#### Control arrhythmias / HR

- Antiarrhythmic therapy. Consult cardiology. Do not use  $\beta$ -blocker when on  $\beta$ -agonists.

#### General

- Ensure appropriate nutrition.
- Supplemental therapy with carnitine, co-enzyme Q10, vitamins as indicated.

#### Investigate/monitor

- Serial cardiac ultrasound to monitor function and exclude clots
- Monitor renal function and electrolytes
- Investigate cause

#### Reference

European Journal of Pediatrics, 2010, 169, 269 – 279

### Question 14 (a)

A four year old boy newly diagnosed with diabetes is admitted to your PICU with diabetic ketoacidosis. He is responding to voice but lethargic. He has received 30ml/kg 0.9% NaCl as fluid resuscitation in the Emergency Department.

Examination findings are as follows:

Weight 15kg  
Pulse 140 bpm, BP 90/60 mmHg  
Capillary refill time 4 seconds  
Respiratory rate 60 per minute, no recession,  
Normal abdominal examination,  
Pupils equal and reactive to light  
No focal neurologic signs

Initial investigations are shown below:  
Arterial Blood Gas

	Value	Normal range
pH	7.01	7.35 – 7.45
PaCO <sub>2</sub>	91	80 - 100 mmHg
PaO <sub>2</sub>	18	40 - 44 mmHg
HCO <sub>3</sub>	3	22 - 26 mmol/l
Base Excess	-17	-2 to +2

Biochemistry

	Value	Normal range
Sodium	166	135 – 145 mmol/l
Potassium	4.7	3.5 – 4.5 mmol/l
Chloride		98 - 110 mmol/l
Urea	7.1	2.1 – 6.5 mmol/l
Creatinine	75	20 - 60 mmol/l
Glucose	66	4 - 6 mmol/l

Outline your management and goals for the next 24 hours.

Assess airway/breathing : airway appears to be safe, but should be prepared for intubation. Supplemental O<sub>2</sub> if SpO<sub>2</sub> declines.

Circulation : Establish 2 large bore IVs.

Intra-arterial line for monitoring blood pressure & biochemistry.

2nd hourly ABG, electrolytes; hourly glucose, Mg, Po<sub>4</sub>. Urine/blood ketones

Disability : Monitor HR, BP, CRT, SpO<sub>2</sub>, GCS hourly

Watch out for signs of raised ICP

Fluid management :

Assume 5% dehydration in this pt, maintenance + dehydration fluid for 48 hrs  
= 2400 (maintenance) + 750 (5% dehydration) - 450 ( fluid given as boluses)  
= 57 ml/hr.

Commence as NS + 20 mmol K<sup>+</sup>/lit.

(estimate corrected Na = Na (measured) + 0.3 \* Glucose + 6)

Insulin : Start insulin infusion @ 0.05 – 0.1 u/kg/hr. Monitor biochemistry q 1 hrly

Once insulin infusion is started BSL & K<sup>+</sup> may fall rapidly. If higher than 12.5% conc of glucose or K<sup>+</sup> ( more than 0.2 mmol/kg/hr) needed patient will need central venous access either CVL or PICC.

In this case hypernatremia indicates severe dehydration and is considered neuro-protective. When BSL starts declining with treatment Na levels should rise. Monitor corr Na. Also calculate measured osmolality. As long as corr Na rises with treatment there will be no sudden changes in osmolality. Change IV fluid to 0.45% NaCl .

Correct hypomagnesemia if Se Mg < 0.8 mmol/lit by supplementing MgSo4 @ 0.2 mmol/kg & PO4 as KPo4 in fluids (2/3 Kcl + 1/3 KPo4).

In view of increased risk of cerebral edema : young age, altered LOC at presentation keep mannitol, 3% Saline at bedside.

Nil by mouth

Broad spectrum antibiotic coverage for suspected infection. rationalise or cease depending on investigations.

Endocrinology consult.

#### **Question 14 (b)**

*Outline the risk factors and pathophysiology of cerebral oedema in diabetic ketoacidosis.*

Cerebral oedema : Mortality & morbidity ~ 20%

Prevent - limiting fluid replacement to < 3000 ml/m2

Using isotonic fluid as replacement

avoid shifts in osmolality

Treatment:

Mannitol 0.25-0.5 gm/kg 20% over 20 min

3% Saline : 3-5 ml/kg over 20 min

intubate/ventilate. Maintain CO<sub>2</sub> between 35-40 mm Hg.

maintain low normal temperatures

Normal to high Se Na

Head end elevated 30° & midline.

Sedate/paralysis if required

Neuroimaging once resuscitation is completed

Consider ICP monitoring.

### Hypokalemia:

Insulin deficiency causes serious total body potassium depletion. Extracellular K<sup>+</sup> maybe normal or high in presence of severe intracellular K depletion.

Management wise : If Se K<sup>+</sup> > 5.5 , monitor closely

4-5 : add 10-20 mmol/lit to fluids

3-4 add 40 mmol/k

< 3 : will need central access for higher K<sup>+</sup> delivery.

### Hypoglycemia:

Once insulin infusion is commenced, Blood Glucose levels tend to fall rapidly.

Once BSL falls below 13 mmol/L , add dextrose to maintenance fluid.

If more glucose is needed to maintain BSL this can either be done by increasing glucose concentration in fluid or running a dextrose 50% infusion along with fluid.

Ensure dextrose concentration does not exceed 12.5% thru peripheral vein.

Do not reduce insulin infusion till ketoacidosis corrected.

### Infection:

DKA may either be precipitated or may worsen a pre-existing infection.

Commence broad spectrum antibiotics unless source of infection obvious.

Blood/urine MCS, FBC, CRP, CXR to identify source.

Rationalise & narrow the spectrum as soon as organism identified. Persistent acidosis despite adequate insulin maybe an indication of septic shock.

## Question 15

*Outline the uses of portable ultrasound in the paediatric ICU.*

- Portable, simple technology. Basic training only needed for some applications. Others much more specialised
- Cardiac
  - Diagnosis/exclusion of CHD
  - Function, fluid collection
  - Estimate of CO (filling and function) in the absence of measurement
- Cranial
  - Important tool in infants with open fontanelle
  - Diagnosis of abnormalities
  - Excellent for ICH
  - Poor views of Posterior fossa
- Diaphragmatic screening
  - Diagnosis of phrenic nerve palsy
- Chest
  - Diagnosis of effusion (distinction from collapse/consolidation)
- Abdomen
  - Fluid collections
  - Inspection of individual organs
    - Liver (parenchyma, biliary tract including GB, flow patterns in hepatic portal vessels)
    - Spleen (presence, parenchyma)
    - Kidneys (echogenicity, flow pattern. Diagnosis of ATN vs ACN, renal vein thrombosis, resistance to flow)
- Bladder
  - confirmation of urine in bladder (SPA, ? urine production)
- Vascular
  - Diagnosis of thrombosis, patency of vessels
  - Doppler confirmation of arterial supply
- Vascular access
  - Venous (and arterial)

### Question 16.1

*List 4 conditions for which use of inhalational anaesthetic agents may be indicated in the PICU.*

1. Induction agent for difficult airway intubation
2. Procedural sedation and analgesia
3. Status asthmaticus
4. Status epilepticus

### Question 16.2

*Compare and contrast the benefits and disadvantages use of inhalational anaesthetic agents versus intravenous agents in the PICU.*

	inhalational anaesthetic agents	intravenous agents
advantages	<ul style="list-style-type: none"><li>• Rapid onset/offset</li><li>• Minimal accumulation</li><li>• Maintenance spontaneous breathing during induction</li><li>• Potential efficacy in status epilepticus and status asthmaticus after other agents tried and ineffective</li></ul>	<ul style="list-style-type: none"><li>• Ease of drug delivery and monitoring</li><li>• Staff familiarity with agent</li></ul>
disadvantages	<ul style="list-style-type: none"><li>• Delivery difficulties in the PICU (ventilator compatibility, scavenging)</li><li>• Staff unfamiliarity with agent and delivery</li><li>• Monitoring difficulty</li><li>• Cardiovascular depression</li><li>• Occasional specific adverse reaction eg malignant hyperthermia</li></ul>	<ul style="list-style-type: none"><li>• May not be effective in status epilepticus and status asthmaticus</li><li>• Cardiorespiratory depression</li></ul>

### References

Pediatric Critical Care Medicine 2008;9(2):169-179

Rogers' Textbook of Pediatric Intensive Care 4<sup>th</sup> Edition 2008, Ch 35, p540.

### Question 17

*List current treatment strategies for acute peri- and post-operative pulmonary hypertension in infants and children.*

1. Anaesthesia techniques
2. Ventilatory strategies / blood gas manipulation
3. Drug treatment targeting pulmonary vascular resistance (sedation, inhaled nitric oxide, cGMP related drugs (sildenafil, prostacycline)
4. Support Right Ventricle
5. Extracorporeal Life Support
6. Surgical management

#### References

Pediatric Critical Care Medicine 2010;11(2 Suppl 1):S27-29

Rogers' Textbook of Pediatric Intensive Care 4<sup>th</sup> Edition 2008, Chapter 70A, p1166.

### Question 18.1

*Draw, label and explain the components of a normal left atrial pressure waveform corresponding to the ECG tracing on the blank grid provided below.*

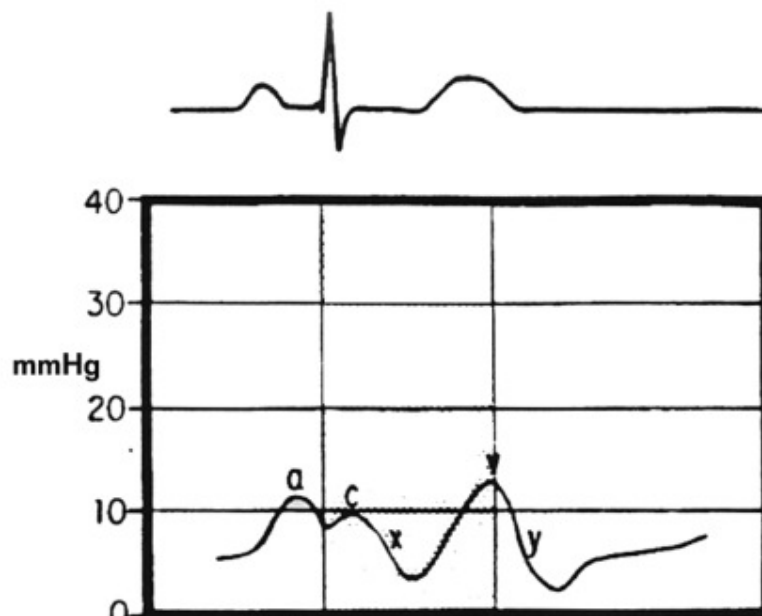


Figure 2. Typical left atrial pressures: a-wave = 4.0- 16 mmHg; v-wave = 6.0-21 mmHg; Mean pressure = 2.0-12 mmHg



- a wave          atrial contraction
- c wave          displacement of M valve (in early systole)
- x descent      fall in atrial pressure during systole
- v wave          atrial filling against closed M valve (late systole)
- y descent      fall in atrial pressure as Tr valve opens in diastole

### Question 18.2

*List 5 likely causes of an elevated left atrial pressure following an AVSD repair.*

1. Abnormal placement of line
2. Decreased ventricular function
3. Arrhythmia
4. Fluid overload
5. Left AV valve regurgitation
6. Tamponade

### Question 19 (a)

*A screening test for a disease that has a prevalence in the general population of 1 in 1,000 has a 5% false positive rate and a 0% false negative rate.*

*To the nearest 1%, calculate the sensitivity, specificity, positive predictive value and negative predictive value of the test.*

- True Positives (TP)
- False Positives (FP)
- True Negatives (TN)
- False Negatives (FN)
- Sensitivity =  $TP/(TP+FN) = 100\%$
- Specificity =  $TN/(TN+FP) = 95\%$
- PPV =  $TP/(TP+FP) = 2\%$
- NPV =  $TN/(TN+FN) = 100\%$

### Question 19 (b)

*1,000 people are selected at random from the population and screened.*

*What is the probability that an individual that tests positive for the disease actually has the disease (to the nearest 1%)?*

- 2% (ie the positive predictive value of the test)

Reference

Practical Statistics for Medical Research, Altman DG. Chapter 14.

## Question 20

*Outline the use of unfractionated heparin in ICU with particular attention to:*

- a) *Mode of action*
- b) *Indications*
- c) *Dosing and administration*
- d) *Monitoring*
- e) *Risks*

### **(a) Mode of Action**

- Negatively-charged sulphated glycosaminoglycan. All UFH preparations have range of MWts
- Acts predominantly by inhibition of IIa and Xa
- These mechanisms are Antithrombin (ATIII)-dependant
- Others:        ↑TFPI by endothelium; ↑ Heparin Cofactor II inhibition of IIa

### **(b) Indications**

- Anticoagulation
- Thrombus     Arterial or venous
- Extracorporeal support - ECMO/VAD/RRT
- Prophylaxis
- DVT prophylaxis
- Intracardiac stasis
- Post cardiac surgery – shunts, prosthetic valves
- Flushes Art, central line flushes (1U/ml)
- Heplocking of indwelling lines
- For all central lines – No evidence to support use 10U/kg/hr (PCCM 2010)

### **(c) Dosing and administration.**

- Usually IV
- 75-100U/kg bolus, 20-30 U/kg/hr by infusion.
- Neonates need higher doses (up to 40 U/kg/hr)
- T<sub>1/2</sub> – shorter in newborns
- Can be used subcutaneously (rare): 12 hourly dosing.

### **(d) Monitoring**

- APTT traditionally used, aiming for 1.5-2.5 x normal. Surrogate for heparin concentration or effect. Not accurate measure of either in clinical setting.
- ACT crude, but easiest bedside test. Traditionally in CPB, has been extended into ICU for ECMO, CRRT etc. Test of whole blood clotting, not heparin activity.
- Anti-Xa specific test of (one) heparin effect. Usual aim for 0.3-0.7U/ml. More expensive, difficult.
- Protamine titration - heparin concentration. Slow, expensive, rarely done clinically. Does not measure heparin effect.

### (e) Risks

- Haemorrhage. Significant (although poorly quantified) risk.
- Overdose/drug errors. Mention multiple concentrations available. Mention antidote - protamine (positively charged, basic, binds heparin to form stable complex with no anticoagulant activity)
- HIT II – antibody (anti-PF4/heparin complex) mediated
- Most common in surgical patients (adults)
- Lower incidence in children seems genuine
- Difficulties with testing: presence of Ab  $\neq$  HIT II
- Osteoporosis/osteopenia – associated with prolonged exposure. Less well described in children

### References

Pediatrics 2009;123:e510-518

Pediatric Critical Care Medicine 2007;8(4):404-405

Pediatric Critical Care Medicine 2010;11:489-495

### Question 21

*A newborn is admitted to your ICU from a local maternity hospital. The infant was born 6 hours ago at a gestational age of 38 weeks following an emergency caesarean section for foetal tachycardia. The infant is poorly perfused and grunting. Respiratory rate 65 breaths per minute. Heart rate 210 beats per minute. Blood pressure 42/29 (mean 35) mmHg.*

#### *Arterial Blood Gas:*

	Value	Normal range
pH	7.30	7.35 – 7.45
PaCO <sub>2</sub>	32	80 - 100 mmHg
PaO <sub>2</sub>	88	40 - 44 mmHg
HCO <sub>3</sub>	18	22 - 26 mmol/l
Base Excess	-6	-2 to +2
Lactate	4.6	1.0 – 1.8 mmol/l

*The rhythm strip below is taken using an oesophageal electrode connected to ECG upper limb leads*

*A rhythm strip was provided to candidates in the examination paper.*

**Question 21 (a)**

*What rhythm is shown on the ECG?*

Atrial flutter/IART with 2:1 block

**Question 21 (b)**

*Outline your management.*

- Initial assessment and resuscitation: Does the child need intubating now? Probably yes - relative hypotension and high lactate. (may be reasonable to hold off if going to overdrive pace imminently)
- Ensure adequate IV access and administer intravenous Fluid Bolus – may improve BP, no effect on rate
- Urgent echo - congenital heart defect (CHD)/assess function
- Examination - Evidence of hydrops? – might indicate duration of arrhythmia
- Treat arrhythmia
- Do not give Adenosine (diagnosis has been made and may precipitate severe bradycardia)
- attempt trans-oesophageal overdrive pacing if available
- DC cardioversion if unstable
- Digoxin if stable
- Amiodarone if resistant to Digoxin
- Supportive therapy
- Inotropes if poor function in SR – try to avoid catecholamines as may precipitate arrhythmia
- May need ongoing digoxin (amiodarone) if recurrent. Recurrence unlikely if no CHD
- Treatment of CHD if identified

### Question 21 (c)

*What other investigations would you perform?*

ECG in sinus rhythm (pre-excitation, abnormal p waves etc)

Echo in sinus rhythm to look at function

Thyroid function test, full blood count (anaemia)

If CHD: look for associated abnormalities (cranial, renal ultrasound)

### Question 22

*Outline measures to reduce the incidence of ventilator associated pneumonia in ICU and the difficulties encountered specifically when trying to implement these in paediatrics. You may present your answer in table form.*

Intervention	Problems in PICU
General infection control (hand hygiene, barrier measures - gloves, gowns, masks)	Awake children may be frightened by barrier measures
Avoid intubation, use NIV	NIV technically difficult in very young children
Mouth care	Can be technically difficult in babies. Young children uncooperative, variety of sizes of toothbrush needed, fluoride-free toothpaste in younger kids
Bed position	Difficult to maintain elevation of head of bed, particularly in babies and younger children
Regular aspiration of stomach contents	None
Cuffed ETTs	Ongoing concerns about suitability in paediatric airway. Microcuff may be better
Dorsal lumen ETT	Not available in smaller sizes
Drainage of vent tubing condensation	Paediatric circuits are run wet to minimise chance of narrow diameter ETT blockage with thick secretions
Gastric protection	None (however, no evidence of benefit in children)
Transpyloric feeding in patients at risk of aspiration	NJT harder to insert in children, small-bore more likely to block.
Avoidance of muscle relaxants and deep sedation. 'Sedation vacation' with daily assessment of readiness to extubate	Risk of accidental extubation greater in children. Propofol not used for sedation in PICU
Continuous lateral rotational therapy	Expensive. Very limited equipment for and data in children

Reference

Pediatric Clinics of North America 2006;53:1231-51

### Question 23 (a)

*You are asked to assess a 6 year old girl in the emergency department of your hospital. She is known to have myasthenia gravis. Her usual treatment is pyridostigmine 20mg 4 times a day. She has had increasing weakness through the day and is now complaining of having difficulty breathing.*

*In table form compare and contrast the history, clinical features and treatment of myasthenic and cholinergic crises.*

	Myasthenic crisis	Cholinergic crisis
History	Exacerbating factor (Infection, aspiration, drug etc) Treatment non-compliance	No exacerbating factor ↑ dose/excessive administration of cholinesterase inhibitor
Features	Muscle weakness Respiratory failure	Muscle weakness Respiratory failure Cholinergic effects: increased secretions, bronchospasm, bradycardia, diarrhoea, miosis
Treatment	Support respiration if needed Pyridostigmine ?Steroid ?Plasmapheresis	Support respiration if needed Stop cholinesterase inhibitors

### Question 23 (b)

*Outline your initial assessment of respiratory function and treatment of this child's progressive respiratory failure.*

#### Assessment

- |                   |  |
|-------------------|--|
| 1. Airway         | Potentially Bulbar weakness                  |
| 2. Breathing      | Tachypnoea, Spirometry if able               |
| 3. General        | assess for fatigue                           |
| 4. Investigations | hypercapnia from arterial blood gas analysis |

#### Management

If no signs of respiratory failure

1. Observe
2. Dose of Cholinesterase inhibitor (consult neurology)
3. Steroid?

If worsening respiratory failure or rapidly progressing/tiring

1. NIV – only if swallowing not compromised
2. Intubate and ventilate if NIV not tolerated or effective

#### Intubation

- Appropriate assistance/advice from anaesthesia
- Prepare equipment (include equipment for potentially difficult airway)

- Inhalational or intravenous (propofol) induction. Spray cords with local anaesthetic.
- avoid muscle relaxant if possible or very careful use of small doses of non depolarising muscle relaxant. Unpredictable response to these.

### **Question 23 (c)**

*List 3 classes of drugs that should be avoided during this child's admission.*

1. Aminoglycosides and other antibiotics (Tetracycline, Ciprofloxacin Vancomycin)
2. Neuromuscular blockers
3. Beta blockers also Procainamide

#### **References**

Pediatric Emergency Care 2005;8:546-549  
Journal of Child Neurology 2009;24(5):548-590  
Australian Prescriber 2007;30:156-60

### **Question 24**

*Outline the use of, benefits, risks, dosing and pharmacokinetics of intravenous salbutamol in asthma.*

Limited evidence for use: only RCT in children is Gary Browne's study which looked at bolus of 15 mcg/kg in addition to standard inhalational treatment - did not look at infusion.

Potential side effects - all patients will experience increased O<sub>2</sub> consumption / CO<sub>2</sub> production - i.e. increased metabolic rate, lower mixed venous sat

IV salbutamol will substantially impair V/Q matching if there is any parenchymal lung disease.

Electrolyte abnormalities - especially K

Lactic acidosis secondary to metabolic effects in liver

Tachycardia

Diastolic hypotension, which together with tachycardia gives potential for myocardial ischaemia. Correct dose for infusion is probably around 1 mcg/kg/min - Shann recommends 5 mcg/kg/min for 1 hour then maintain level with 1 mcg/kg/min.

Adult data suggests IV alone is not as good as nebulised alone. Therefore IV salbutamol should only be used as an adjunct to (not replacement for) inhalational therapy. Half life is long - 2-3 hours, therefore need to wait 6-8 hours after discontinuing infusion before patient is free of drug. However, anecdotal intravenous salbutamol is useful in acute severe asthma because of guaranteed drug delivery.

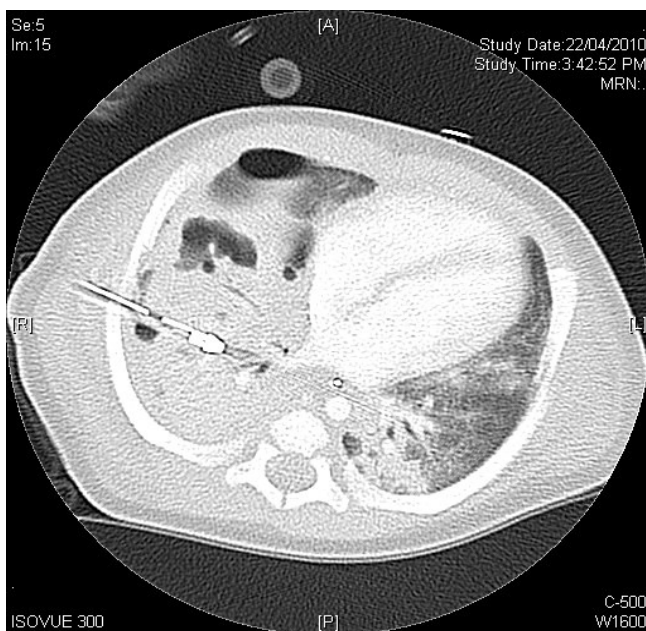
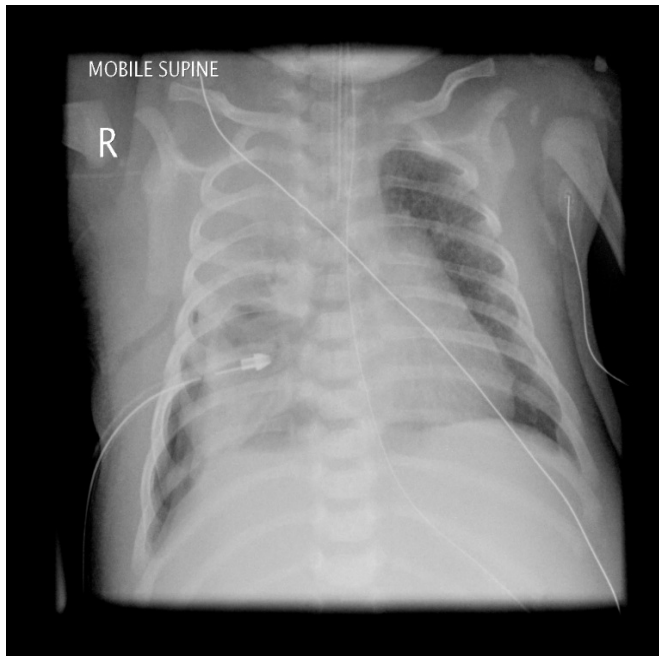
#### **References**

Critical Care Medicine 2002;30 (2):448-53, Chest 2002;122:1200-1207, Drug Doses (Shann)

### Question 25 (a)

*A previously well 6 week old male is admitted from home with acute pneumonia. On admission to ICU he requires intubation and ventilation with high pressures and a high  $\text{FiO}_2$ . He develops a right pneumothorax, which is drained. The chest drain is still bubbling continuously. 24 hours after insertion. Methicillin-resistant *Staphylococcus aureus* grows in blood cultures and chest drain fluid.*

*Describe the findings on CXR and CT chest.*





CXR:

- Endotracheal tube in good position
- Cannot visualize tip of nasogastric tube
- Right intercostal catheter in situ
- Incompletely drained right pneumothorax
- Consolidation throughout right lung field
- Right pneumatocoeles

CT Chest:

- Intercostal catheter in lung (or fissure)
- Dense consolidation right side
- Dependant atelectasis on left
- Right sided abscesses/cavities

### **Question 25 (b)**

*Outline your approach to management of this child's infection and complications.*

Treatment of MRSA:

- Vancomycin and gentamicin
- Minimum 14 days of intravenous treatment
- Consider testing for Panton-Valentine leukocidin (PVL)
- Some evidence for use of clindamycin (can be continued orally) or linezolid, particularly if PVL-producing strain

Treatment of severe pneumonia

- Ventilation
- HFOV may reduce air leak
- Poor outcomes in ECMO for necrotising *Staphylococcus aureus* pneumonia

Treatment of ongoing air leak/ICC malposition:

- Ensure chest drain on suction
- Consult surgeons
- VATS/mini thoracotomy removal of ICC, drainage of air, fluid, new ICC
- High likelihood of bronchopleural fistula
- May need surgical oversewing of fistula or lobectomy

References

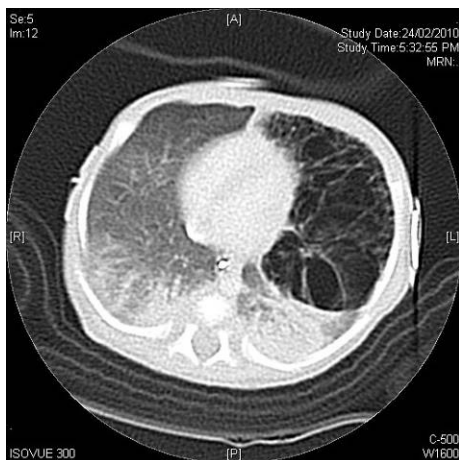
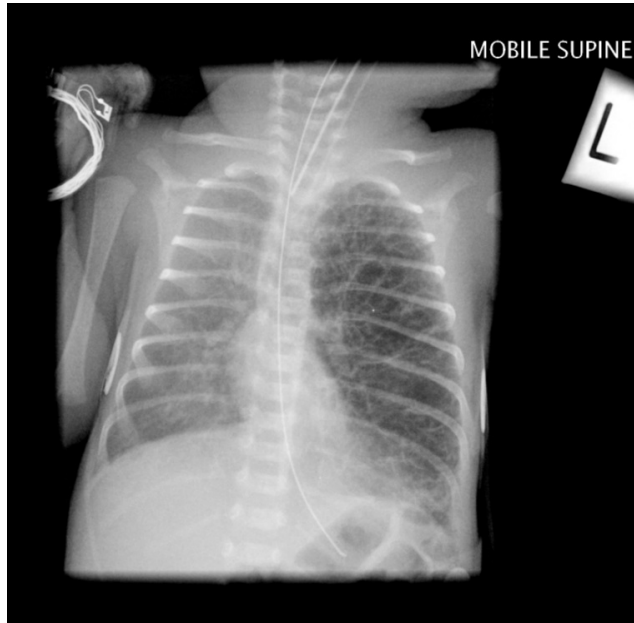
The Pediatric Infectious Disease Journal 2005;5:547-548

Journal of Antimicrobial Chemotherapy 2009;63:849-861

### Question 26 (a)

*A term newborn infant (normal antenatal history) requires intubation for respiratory distress a few hours after birth.*

*Describe the chest imaging findings.*



CXR and CT demonstrate a markedly emphysematous left upper lobe, with compression of left lower lobe and right lung, mediastinal shift.

**Question 26 (b)**

*What are the differential diagnoses?*

Congenital lobar emphysema or cystadenomatous malformation of the lung - more likely CLE because of location and appearance (cystadenomatous malformation more common in RLL).

**Question 26 (c).**

*Outline anticipated problems and your approach to management of ventilation.*

- Anticipate hyperinflation with further compression of normal lung and heart. Consequent impaired gas exchange and diminished cardiac output.
- Minimise inflation pressures
- Low rate with long expiratory time
- Tolerate hypercarbia
- ?HFOV
- Selective intubation of RMB
- Selective occlusion of LMB
- Surgical resection

### Question 27

*In table form, compare and contrast rotary wing and fixed wing vehicles for transport of critically ill children.(It is recognised there will be some variation depending on the model of aircraft used).*

	<b>Helicopter</b>	<b>Plane</b>
Speed of deployment	Faster	Slower
Speed of air travel	Slower	Faster
Access to hospitals	Can be hospital to hospital	Must be airport to airport
Range	Shorter	Longer
Flight restriction due to weather conditions	More prone	Less prone
Pressurisable cabin	No	Yes (not always)
Noise and vibration	More	Less
Space and access to patient	Generally less	Generally greater
Safety	Higher incidence of fatal crashes	Fewer crashes involving medical transport
Seating capacity	Less	Greater (more likely to be able to transport more team members or parent)
Load limit	Lower	Higher (as above)

### Question 28

*A 6 week old infant is brought into ED with coma. There is bruising over the head and the fontanelle is full. A single CT brain image is shown below.*



### Question 28 (a)

*List the major findings on the brain CT*

- Right occipital, temporal and posterior frontal lobe infarction.
- With mass effect and midline shift ~ 3mm.
- Right hyperdense subdural ~ 4mm thick.
- Right scalp haematoma and comminuted skull fracture on right
- Poor grey-white differentiation throughout.
- Lateral and 3<sup>rd</sup> ventricles are apparent.

## Question 28 (b)

*Outline your approach to further investigation and management*

- Resuscitation
  - ABC – intubate and ventilate.
  - Fluid as necessary
- Investigate and correct reversible problems
  - Coagulopathy – screen including vitamin C, factor 13, platelet function studies and routine PT, APTT, fibrinogen, FDP,
  - FBC, film.
  - U & Es, venous/ arterial blood gas, LFTS, lipase/amylase.
  - Bone mineralisation studies – vitamin D etc.
  - Dietary and family history (rickets/coagulopathy).
- Imaging
  - Chest and abdo X-rays (for #s)
  - CT neck. MRI of head and neck when stable.
  - Eye review and skeletal survey and bone scans at some stage
- NAI
  - immediate involvement of child protection services and social work, ensure other children safe.
  - Support all family members
- Management of brain injury
  - Neurosurgical consult for CSF pressure monitoring/ drainage.
  - Aim for CPP  $\geq 40$ mmHg and ICP  $< 20$ mmHg.
  - Treatment of low CPP
  - Treatment of elevated ICP
  - Seizure prophylaxis
- General care
  - Infection, feeds, skin etc.

### References

NEJM 1998;338(25):1822-29

Pediatric Critical Care Medicine 2009;10(4):517-523

### Question 29(a)

*A one year old with meningococcal disease has been fluid resuscitated and is mechanically ventilated. Ventilation has been with pressures of 32/15 (mean 26) with an  $F_iO_2$  of 1.0 for more than 6hrs. Bilateral parenchymal infiltrates are present on chest X-ray and  $SpO_2$  is 88%. Arterial blood gas result is shown below.*

	Result	Normal range
pH	7.22	7.34 – 7.43
pCO <sub>2</sub>	67	31 - 42 mmHg
pO <sub>2</sub>	46	80 – 105 mmHg
Bicarbonate	26.9	20 – 26 mmol/l
Base excess	-2.5	-5 to +5 mmol/l
Saturation	85%	
Lactate	2.3	1 – 1.8 mmol/l

*What is the respiratory condition or diagnosis? Justify your answer.*

- ARDS
- Acute onset
- Oxygenation, PaO<sub>2</sub>/F<sub>i</sub>O<sub>2</sub> <200
- Chest x-ray – bilateral diffuse pulmonary infiltrates
- Assume no evidence of left atrial hypertension (PAWP<18mmHg)

### Question 29(b)

*Briefly discuss the following treatment options in terms of clinical evidence, pathophysiology and relevance to this patient.*

*(1) Prone positioning*

No change in long-term outcome in adults or children. There is perhaps a brief temporary improvement in oxygenation.

Typically ventilation is more impaired posteriorly due to airspace loss/collapse – greater degree of “baby lung,” turning prone redistributes ventilation and can permit opening up of this atelectatic portion.

Assuming this patient is haemodynamically stable, has not imminent air leak on chest x-ray and is manageable from a sedation/muscle relaxation point of view – then prone positioning could assist for 12-24hrs and would be reasonable. Pressure area and nursing care are important considerations.

*(2) Inhaled nitric oxide*

Increased cost, temporary increase in PaO<sub>2</sub> potentially, but no proven benefit across age groups.

Particularly in indirect ARDS there is an important pulmonary microvascular congestion and pulmonary vasoconstriction – nitric oxide hence may have a role.

Potential for thrombotic or bleeding complications.

References

JAMA 2009;302(18):1977-1984  
JAMA 2005 Jul 13;294(2):229-37  
NEJM 2001;345:568-573  
PCCM 2007;8:317-323  
BMJ 2007;334-779

**Question 30**

*An infant is extubated the day after uncomplicated cardiac surgery and rapidly develops severe upper airway obstruction.*

*Outline your immediate and ongoing management.*

Preparation and procedure

- Prepare personnel and equipment
- Maintain airway and oxygenation with mask, positioning and suctioning
- Simultaneously obtain information on previous intubation
- Ensure access and haemodynamics prior to (& during & post) intubation.
- Use high flow O<sub>2</sub>, nebulised adrenaline, steroids and NIV as temporising measures prior to an
- induction of anaesthesia followed by oro-tracheal intubation with an ETT (half size smaller than used previously). Use of topical local anaesthetic solution to the cords.
- Consider intubation in operating room or in ICU depending on previous difficulties and need for adjuncts– if required obtain assistance from anaesthesia +/- ENT.
- Discuss potential risks if not fasted and need for oro or nasogastric tube.
- May need to minimise FiO<sub>2</sub> to ~0.4 if left to right shunt.



- Inhalational anaesthesia is recommended and preferred approach for induction of anaesthesia.
- Check position of ETT by visualisation, ETCO<sub>2</sub> and chest x-ray.

#### Ongoing management and investigation

- Sedation and analgesia
- Avoid accidental extubation
- steroids (Cochrane review 2009 suggested some benefits).
- Contributing factors? Trisomy 21, infection, airway abnormalities, vocal cord palsy.
- Further investigations

#### References

Corticosteroids for the prevention and treatment of post-extubation stridor in neonates, children and adults. Cochrane Database of Systemic Reviews 2009

## **ORAL SECTION**

### **THE CLINICAL SECTION**

The Clinical Section (2 clinical cases – 20 minutes per case) was conducted in the Paediatric Intensive Care Unit at Starship Children's Hospital, Auckland, New Zealand.

Candidates who approach the clinical examination of the patient and presentation of findings in an organized manner will impress the examiners. Candidates should approach the case discussion in a consultant like manner. 30% of the overall marks are allocated to the two clinical cases. Candidates should bear this in mind when preparing for the examination.

Candidates should listen carefully to the introduction given by the examiners and direct their examination accordingly. Cases were usually presented as problem solving exercises. For maximal marks, candidates should demonstrate a systematic approach to examination, clinical signs should be demonstrated, and a reasonable discussion regarding their findings should follow. The twenty minutes available for each case provides ample opportunity to discuss investigations and plans of management. Some candidates waste valuable time at the start of the case by spending more than a couple of minutes around the bedside before they actually commence examining the patient. Exposing the patients should be limited to those areas that are necessary for that component of the examination. Candidates must show appropriate courtesy and respect to patients and their families if present during the examination.

Cases encountered in the clinical component of the examination included:

A 13 month old child with hypoplastic left heart syndrome. The child had undergone a Norwood Procedure followed by a cavopulmonary shunt. Recent surgery to close the tracheostomy stoma was complicated by bilateral pneumothoraces. The candidate was asked to examine the child with regard to suitability for extubation.

A 14 month old ex 27 week premature child on Extracorporeal Life Support for RSV pneumonitis. The candidate was asked to examine the child and comment on the prospects for weaning off ECMO and also to advise on a ECMO weaning strategy.

A 4 month old child post surgical attempt to repair a congenitally dysplastic mitral valve. Severe mitral regurgitation persisted and the candidate was requested to examine the child with regard to the difficulties experienced with weaning from mechanical ventilation.

## STRUCTURED VIVA SECTION

There were 8 stations of ten minutes each for structured Vivas. There were two minutes provided to read an introductory scenario (which included the initial question) outside each viva room. This same information was also provided inside the viva room. Candidates should be able to demonstrate a systematic approach to the assessment and management of commonly encountered clinical problems. Candidates should also be prepared to provide a reasonable strategy for management of conditions that they may not be familiar with. Feedback from examiners suggested that common deficiencies encountered included ones related to knowledge deficits (and awareness of these deficits), questionable judgment, and poor exam technique. The range of material covered in the viva section can be deduced from the following illustrative examples of introductory cases with sample questions.

### Viva 1

A 12 year old girl presents with presyncopal episodes for 3 days. She is conscious. Her pulse is slow and irregular. (ECG is shown to candidate).

*What is your working diagnosis? What are your priorities for the management of this patient?*

### Viva 2

You respond to a trauma call in the emergency department where a 4 year old girl has been brought in by Ambulance. She was a pedestrian hit by a motor vehicle traveling at 40-60km per hour.

At the scene, she was described as having a GCS of 7. E1V2M4. She is orally intubated with a 5.0 tube and is being hand ventilated by the Ambulance Paramedic.

She has obvious bruising on her right side, particularly her right hip, which is markedly swollen. Her air entry is currently equal, she is tachycardic to 160 with a blood pressure of 85/50.

*Describe your approach to managing this child.*

### Viva 3

You are the intensivist working the weekend. Your unit suffers a power failure during the morning ward round. There are 14 patients; 7 ventilated and one on ECMO. The emergency generator (back up power system) fails to kick in.

*Describe the immediate steps you would take to ensure the safety of your patients.*

*What is UPS and how does it work?*

*Define microshock and describe how it can be prevented?*

*How can the risk of macroshock be minimized in ICU?*

*How does a floating or isolated circuit prevent macroshock in the ICU?*

*What advantage does a line isolation monitoring system have over a residual current detector?*

### Viva 4

A 4 year old girl with a history of Acute Myelogenous Leukaemia (AML) and a bone marrow transplant 2 weeks ago has been admitted to PICU because of a haematemesis and melaena. An endoscopy is scheduled in 4 hours time.

Hr 160, BP 74/30      Weight 15 kg

Full Blood Examination			
Parameter	Result	Unit	Normal Values
Hb	40	g/L	(105-135)
WCC	0.7		(6.0 - 18.0)
Platelets	16		(150 - 400)
Coagulation Profile			
INR	3.1	ratio	(0.8 – 1.2)
APTT	78	secs	(27 – 53)
Fibrinogen	0.6	g/L	(0.8 – 3.8)

*Discuss immediate transfusion strategy. What is the role of a TEG in this situation?*

*What is the role of recombinant factor 7a?*

## Viva 5

A 2 year old boy presents with a 25 minute seizure terminated by intravenous midazolam and a bolus of 10% dextrose in response to a bedside glucometer reading of "low". He has been lethargic for 2 days, and had diarrhoea and vomiting one week earlier with no fever. He is unimmunised with mild developmental delay. He is spontaneously breathing in air with a normal respiratory pattern and pulse oximetry. He is well perfused. Examination of chest and abdomen is normal. His Glasgow Coma Scale is 9 (E2V2M5). He does not appear to be fitting now. Pupillary responses are normal.

The following laboratory results collected at presentation are available as follows. In addition, full blood count, serum electrolytes, urea and creatinine are normal.

Test	Result	Normal Range
INR (International Normalised Ratio)	8.4	(0.9 – 1.2)
APPT (Activated Partial Thromboplastin Time)	47 sec	(30 – 45 sec)
Bilirubin Conjugated	64 umol/L	<5 umol/L
AST (Aspartate Transaminase)	12,940 IU/L	<40 IU/L
ALT (Alanine Transaminase)	10,850 IU/L	<10 IU/L
Ammonia	96 umol/L	<65 umol/L
Glucose	0.4 mmol/L	(3 – 6 mmol/L)

*Describe your immediate assessment and management.*

*What is the differential diagnosis for this child's presentation and name key initial investigations?*

*Discuss ongoing management.*

## Viva 6

A previously well 3 year old male presents with several weeks of increasing dyspnoea to the emergency department. The ED physician notes increased work of breathing and an expiratory wheeze, but the boy is well oxygenated. While having blood drawn in the ED, he desaturates to 60%, but recovers with 30 seconds of bag mask ventilation in 100% O<sub>2</sub>. A chest radiograph has been performed. *Review the CXR and list the possible causes.*

*The child undergoes a CT chest without an anaesthetic. Review the CT scan. Following admission to PICU, the patient develops increasing respiratory distress and becomes hypoxic. Describe your management of his airway.*

### **Viva 7 – Communication Station**

Your registrar tells you that a surgical colleague has been extremely rude to nursing and junior medical staff because the surgeon was not happy with the management of a patient. The patient is recovering well but had not been extubated as anticipated by the surgeon, because the patient was too sleepy. Enteral feeding had been commenced. The surgeon was not happy with either of these decisions. You have asked the surgeon to meet with you and discuss what has happened.

### **Viva 8 – Procedure Station**

You are accompanying a 14 year old boy to radiology for a CT head scan. He presented to the Emergency Department with an altered level of consciousness following a blow to the head during a rugby game and was intubated in the emergency department for the CT scan. He has just been moved onto the CT imaging platform. After transferring the patient onto the CT gurney he desaturates and the ventilator starts alarming.