Joint Faculty of Intensive Care Medicine

Australian and New Zealand
College of Anaesthetists
ABN 82 055 042 852

The Royal Australasian
College *of* Physicians

# REPORT OF THE INTENSIVE CARE PRIMARY EXAMINATION

## SEPTEMBER/NOVEMBER 2008

*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, each comprising of twelve short answer questions and twenty short fact questions. Candidates were required to perform at a satisfactory level in the written before being eligible to sit the oral part of the exam. The oral was comprised of eight, ten-minute Viva stations.

## OVERALL STATISTICS

| | |
|---|---|
| Total number of candidates presenting for the written examination | 5 |
| Number of candidates scoring >50% in the written | 3 |
| Number of candidates scoring 45-50% in the written | 0 |
| Total number invited to the Oral section based on written marks | 3 |

Successful candidates: Dr Shailesh Bihari, Dr John Moore, Dr Hemal Vachharajani

## WRITTEN SECTION

### PAPER 1

1. **Outline three (3) factors that alter the pharmacodynamic response of non-depolarising neuro-muscular blocking drugs and describe the mechanism by which they may occur.**

   There were a number of possible factors that candidates could have selected. Examples include drug interactions (anticholinesterases, aminoglycoside antibiotics, local anaesthetics, steroids, antiarrhythmic drugs, anticonvulsants (phenytoin), diuretics, magnesium, lithium), hypothermia / hyperthermia, acidosis, [$k^+$], burn injury, allergic reactions, gender, altered elimination due to renal or hepatic dysfunction and disease states (adrenocortical dysfunction, myasthenia, myopathies, denervation injury). Also

extremes of age and pregnancy. Areas of weakness for the candidates were failure to include sufficient factual knowledge for their selected factors, and as a result, a failure to illustrate sufficiently the mechanisms by which the pharmacodynamic response of non-depolarising neuro-muscular blocking drugs may be affected.

Syllabus: H2a 2 (c)
Reference Text: Pharmacology and Physiology in Anaesthetic Practice / R K Stoelting

**2 ( 40%) candidates passed this questions**

2. **A published trial shows an Odds Ratio for effect of 0.88 [95% CI, 0.62 – 1.26]. Discuss your interpretation of this statistical statement.**

This question asked for an interpretation of a statistical statement. There was good understanding by candidates of the terms odds ratio and confidence intervals. The main points expected for a pass included a discussion of how the Odds Ratio is more appropriate than Risk Ratio in a retrospective case-control study where the outcome is a rare event (<10%) and the denominator uncertain; if the outcome occurs commonly, Odds Ratio tends to overestimate risk; an Odds ratio of 1 implies no difference.

| Risk Factor (treatment) | Outcome (mortality) Yes | No |
|---|---|---|
| Yes | a | b |
| No | c | d |

Risk Ratio = $\frac{a/(a+b)}{c/(c+d)}$ Odds Ratio= $\frac{ad}{bc}$

Odds Ratio ≈ Risk Ratio if the outcome (cells a,c) is sufficiently uncommon.

The use of the formula to illustrate how to calculate the confidence interval and the Standard Error assisted with explaining these terms

A weakness amongst the candidates was a failure to provide a sufficient information on the interpretation of the data. The good answers mentioned the point estimate of 0.88 implied a reduction in the occurrence of the measured end point (eg mortality, a commonly measured end point). However, the trial failed to reach statistical significance because the 95% CI includes the value of 1.0 for the Odds Ratio. Candidates did not mention that the power of the study was not given and therefore the data could not be properly interpreted and there was no discussion on sampling errors or systemic error or bias possibly affecting the result.

Syllabus: EBM 1, 2b, c, d, g, h.
Reference Text: Statistical Methods for Anaesthesia and Intensive Care / P S Myles and T Gin – 2001. Pages 8-11, 24-28, 74-75.

**1 (20%) candidate passed this question**

3. **Compare and contrast the body's bicarbonate, phosphate and protein buffer systems.**

This question was generally well answered. Successful candidates illustrated their answer through the use of a table. For a good pass candidates were expected to include a definition of a buffer; mention the buffering capabilities of the differing buffering systems (bicarbonate, phosphate and protein buffers) in relation to the pKa; the area in the body where they are most effective and whether it was an open or closed system.
The protein buffer system was the least well conveyed by candidates, despite approximately 60 to 70 per cent of the total chemical buffering of the body fluids being inside the cells, and most of this from the intracellular proteins. For a good answer it was expected that candidates also mention that, except for the red blood cells, the slowness with which $H^+$ and $HCO_3^-$ move through the cell membranes often delays for several hours the maximum ability of the intracellular proteins to buffer extracellular acid-base abnormalities. In addition to the high concentration of proteins in the cells, another factor that contributes to their buffering power is the fact that the pKas of many of these protein systems are fairly close to 7.4.

**3 (60%) candidates passed this question**

Syllabus: F2b
Reference Text: Guyton Chp 30

4. **Define basal metabolic rate and describe factors that influence it (70% of marks). How could you measure metabolic rate? (30% of marks)**

Metabolism is all the chemical reactions in all the cells of the body. Metabolic rate is usually described in terms of rate of heat liberation. Candidates often confused *basal* with metabolism during *activity*.

A good answer was expected to outline conditions under which basal metabolic rate is measured (no food for 12 hours, after a night of restful sleep, person is at rest in reclining position for 30 minutes, all psychic or physical factors that cause excitement eliminated, at a controlled comfortable temperature), and the mention of and an explanation of the methods by which metabolic rate is measured (eg direct and indirect calorimetry). Any validated technique of determining the BMR was rewarded with marks, with oxygen consumption methods being the easiest to explain. No candidate mentioned any 'normal' value for the BMR.

Candidates were expected to briefly mention and explain how factors affecting metabolic rate (eg exercise, specific dynamic action of protein after a meal is ingested,, age, thyroid hormone, sympathetic stimulation, fever, gender, hormonal, climate, sleep, chronic malnutrition, trauma, inflammatory response, etc).

Questions with more than one part will have an indication of the proportion of the total mark that will be allocated to each part. It is important that candidates apportion their allocated time and content accordingly.

Syllabus: K 2a, b
Reference Text: Guyton Chp72

**3 (60%) candidates passed this question**

5. **Outline the pharmacology of amiodarone.**

Successful candidates applied, a systematic approach/format to answer questions that refer to outlining pharmacology of select drugs. A number of useful mnemonics are suggested in the recommended texts for use when answering such a question. All candidates correctly stated what amiodarone is used for but most were not structured methodically and thus suffered from significant omission. Amiodarone is an important class III anti-arrhythmic (with some characteristics of all 4 Vaughan-Williams classes). For a good pass candidates were expected to actions of amiodarone (eg blocks inactivated Na channels, decreases Ca current, non-competitive adrenergic blocking effect, blocks myocardial K channels which contributes to slowing of conduction and prolongation of refractory period in AV node, prolongs refractory period in all cardiac tissues, prolongs cardiac action potential duration) and it's pharmacokinetics (eg bioavailability, large volume of distribution, high protein binding, complex metabolism and long elimination half life – 29 days)

Syllabus: C2c
Reference Text: Goodman and Gillman's The Pharmacological basis of Therapeutics 11$^{th}$ ed 2006 and Pharmacology and Physiology in Anaesthetic Practice / Stoelting 4$^{th}$ ed 2006

**2 (40%) candidates passed this question**

6. **Describe the formation, circulation and functions of cerebrospinal fluid.**

To achieve a pass in this question, candidates needed to state where and how CSF was formed, where it flows to after formation followed by a list of its functions. Additional credit was given for knowledge of rates of production and basic CSF composition. The fact that CSF production is constant whilst its absorption is pressure dependant was often overlooked. Thus candidates were expected to mention that there is ~ 150 ml of CSF in the adult, half within the cranium; about 60-70% of the CSF is formed by the choroid plexuses, the remaining 30-40% by the cerebral vessels lining the ventricular walls; in humans the CSF turns-over ~ 4 times/day; composition is essentially brain ECF; brain ECF normally occupies ~ 15% of brain volume; CSF flows out through the foramina of Magendie and Luschka and is absorbed through the arachnoid villi into the cerebral venous sinuses; absorption, being largely by bulk flow, is proportional to ventricular pressure [at normal pressure ~ 7.0-18.0 $cmH_2O$ (mean ~ 11), filtration = absorption, when pressure falls below ~ 7 $cmH_2O$ absorption ceases] and CSF Functions [buoyancy, constant metabolic environment, buffers CSF against rapid plasma changes in $K^+$, $Ca^{++}$, $Mg^{++}$, transport of chemical messengers, sink for waste disposal].
A number of candidates embarked on long discussions of how CSF pH affects physiology to the exclusion of what was asked for in the question. Many answers did not adequately cover the three components asked for in the question.

Syllabus: CNS 2d
Reference Text: Guyton Chp 61

**4 (80%) candidates passed this question**

7. **Describe five (5) potential mechanisms by which poisoning can be fatal and provide one specific example for each mechanism.**

For a good answer candidates were expected to select and describe 5 of a large number of possible mechanisms and provide definite examples for each.

Examples of potential mechanism which candidates could select from included any of the following –
CNS – Obtundation / Seizures, ( benzodiazepines , Tricyclic antidepressants, Lithium)
CVS – Hypotension, Arrhythmia's (Theophylline, Beta Blockers, Calcium channel blockers, Lithium)
Respiratory failure – Hypoventilation, hypoxia (organophosphates due to muscle weakness , any CNS depressant), paraquat with pulmonary fibrosis
Hyperthermia – Selective Serotonin Reuptake Inhibitors, Monoamine oxidase inhibitors, Malignant Hyperthermia triggers
Cellular hypoxia – Cyanide (Sodium Nitroprusside), Carbon monoxide
Massive hepatic necrosis – paracetamol / mushrooms
Behavioural changes – Excess Ethanol-> Motor Vehicle Crashes etc , PCP -> flying, self harm
Bone marrow / cell death -> colchicine (arrests cells in metaphase)

Syllabus: General Pharmacology
Reference Text: Multiple sections from standard pharmacology texts

**3 (60%) candidates passed this question**

8. **Describe the clinical findings you would expect to see in a patient who underwent acute hemi-section of the spinal cord at the upper thoracic level.**

The clinical condition that results from this lesion is the so called Brown-Sequard syndrome. However only a very small proportion of points were given to mention of the latter, with the majority of points allocated to knowledge relating to spinal cord anatomy and physiology.

The expected 4 main clinical features that are associated with this lesion are -
1. There is loss of pain and temperature sensation on the contralateral side below the level of the lesion due to interruption of ascending fibres in the crossed lateral spinothalamic tract.

2. There is loss of vibration, joint position and 2 point discrimination on the ipsilateral side below the level of the lesion due to interruption of ascending fibres in the posterior [dorsal] columns.

3. There is paralysis of voluntary movement on the ipsilateral side below the level of the lesion due to interruption of descending fibres in the lateral corticospinal [pyramidal] tracts. Initially the paralysis is flaccid, later it becomes hypertonic and hyperreflexic with extensor plantar response [upper motor neurone lesion].

4. Finally there is segmental anaesthesia of the dermatome at the level of the lesion on the ipsilateral side due to damage of the nerve roots and anterior horn cells at this level.

Some candidates described the clinical features of complete section of the spinal cord which was not asked for.

Syllabus: G1 2b
Reference Text: Guyton Sections IX and XI

**1 (20%) candidate passed this question**

9. **Describe the physiological consequences that follow an intravenous bolus of 50mls of 50% glucose.**

This question was mainly about the metabolic consequences of an intravenous load of 25 grams of glucose. For a good pass the main areas that required description included:
The transient glycosuria.
The mechanism and time course of the biphasic release of insulin from pancreatic β cells.
The mechanism of the trapping of glucose within liver cells.
The multiple actions of insulin which include inhibiting glycogenolysis and promoting glycgogen synthesis in the liver, promoting the uptake of glucose by muscle, fat and other cells, increasing the metabolism of glucose to fatty acids and triglycerides, inhibiting triglyceride breakdown and promoting protein synthesis.
Additional marks were awarded for details of where these actions of insulin occurred and the enzymes that were stimulated or inhibited by insulin.
Some candidates stated that this bolus of hypertonic glucose would produce a significant and prolonged increase in plasma osmolarity and then went on to produce detailed descriptions of the thirst and ADH mechanisms. In fact with a normal insulin response glucose is rapidly cleared from the blood.

Syllabus: N2a,b
Reference Text: Guyton Chp 67

**1 (20%) candidate passed this question**

10. **Outline the pharmacological properties of an ideal agent for sedating patients undergoing mechanical ventilation in intensive care (50% of marks). Describe how propofol compares to the 'ideal' agent (50% of marks).**

Candidates can benefit by having a system by which they approach topics that involve a broad and general topic such as that of the pharmacology of a particular drug or ideal agent. A good answer included the following logical subheadings:

Desirable pharmacology – long shelf life, stable when drawn up and on exposure to light, cheap, mixes well with other agents in the central line lumen. Bacteriostatic.
Desirable pharmacokinetics – Low volume of distribution, rapid clearance (context-sensitive half-life), clearance not affected by either renal or hepatic dysfunction. Little inter-individual variation in pharmacokinetics. (Availability of an antagonist).
Desirable pharmacodynamics – Affects only CNS. Reliable dose – effect curve with little inter-individual variation in effect. Anxiolysis. (Analgesic properties). No effect on cardiovascular performance. Does not depress respiratory drive.
Minimal side effects – No incidences of allergy / anaphylaxis. No idiosyncratic reactions. No tachyphylaxis.

As indicated, 50% of the marks were allocated to mentioning how well propofol reflects these properties. Mention of 'propofol infusion syndrome' characterised by cardiac failure

which can occur when propofol is used at >4mg/Kg/Hr for more than 24 hours also attracted marks.

Syllabus: G2a 1&2
Reference Text: Pharmacology and Physiology in Anesthetic Practice / R K Stoelting

**4 (80%) candidates passed this question**

11. **Describe the adult coronary circulation (50% of marks). Describe the physiological control of the coronary circulation (50% of marks).**

For a good pass candidates were expected to cover at least the following areas -
Anatomy of the coronary arteries and their supply, variations in supply and venous circulation
Other unique features - coronary sinus saturation <30%, the diastolic aortic pressure, Tachycardia reduces the coronary blood flow through a reduction in diastolic time, left ventricle perfused mainly during diastole and right ventricle perfused mainly during systole, different pattern of left and right ventricular coronary perfusion (drawing a figure of Rt and Lt coronary blood flow), lack of capacity for the myocardium to increase its extraction ratio
Physiological control: The most important mechanism through which coronary blood flow can be changed is by autoregulation which changes the coronary vascular resistance to maintain constant flow in response to different coronary perfusion pressure and changing metabolic demand. Important mediators are adenosine, nitric oxide, and opening of the ATP-sensitive K+ channels, prostaglandins, carbon dioxide, lactic acid or hydrogen ion.
Sympathetic stimulation to heart increases coronary blood flow.
Overall the greatest deficiency by candidates was lack of detail, use of illustrations and clarity in their response. It is important that candidates take note of the distribution of marks given within the question.

Syllabus: C1f 2 a and b
Reference Text: Berne and Levy Cardiovascular Physiology Chapter 11 Coronary circulation.

**3 (60%) candidates passed this question**

12. **Describe the concept of autoregulation as it relates to the renal circulation.**

This question was concerned with a very important physiological principle and was generally well answered. A good answer included a definition of autoregulation. In relation to the renal circulation, the kidneys extract only 10% of the available $O_2$ supply and therefore the renal blood flow is high for the purpose of filtration and not metabolic demand, renal blood flow is autoregulated to remain constant against arterial blood pressures from 75 – 160 mmHg (an illustration helps explain this concept), Tubuloglomerular Feedback (including a description of the mechanism). A discussion about other mechanisms thought to play a role is important – eg intrinsic contractile response of smooth muscle to stretch (myogenic theory of autoregulation). Vasodilator substances tend to accumulate in active tissues, and these "metabolites" (decreases in $O_2$ tension, increased $CO_2$ tension and decreased pH) also contribute to autoregulation (metabolic theory of autoregulation). The sympathetic nerves innervate afferent and efferent arterioles. Renal autoregulation usually overrides mild to moderate degrees of sympathetic stimulation. Strong sympathetic stimulation however will constrict renal arterioles reducing flow to 10% of normal. GFR falls to a lesser extent than

renal blood flow owing to a differential effect of sympathetic stimulation constricting the efferent arteriole to a greater degree than the afferent arteriole.

Syllabus: C1f 2a, d D2i
Reference Text: Guyton Chp 26

**4 (80%) candidates passed this question**

**PAPER 2**

1. **Classify antiarrhythmic drugs, including their mechanisms of action, and give an example of one drug from each group.**

This question again highlighted the importance of candidates utilising a predetermined format or structure to their questions. Well structured responses were less likely to overlook important details, which was the predominate weakness for some candidates. A table format was one useful way of displaying a good answer, for example -

| Vaughan Williams' | | |
|---|---|---|
| **Class** | Electrophysiology | Examples |
| I. **Na-Channel Blockers** | | |
| Ia. | ↓ phase 0<br>↓↓ conduction *v*<br>↑ repolarisation<br>↑ APD | quinidine<br>disopyramide<br>procainamide |
| Ib. | ↔,↓ phase 0<br>↓ conduction *v*<br>↓ repolarisation<br>↓ APD | lignocaine<br>phenytoin<br>tocainide, mexiletine |
| Ic. | ↓↓↓↓ phase 0<br>↓↓↓↓ conduction *v*<br>± repolarisation<br>↔ APD | flecanide<br>ecainide, lorcainide |
| II. **β–blockers** | | propranolol<br>atenolol, esmolol |
| III. Prolong Repolarisation | K-channel blocker | amiodarone<br>bretylium, sotalol |
| IV. **Calcium Entry Blockers** | | verapamil<br>diltiazem |
| **NB: Many agents have effects in more than 1 class. Various agents do not fit into this classification, therefore some add a 'Class V' = other, e.g. digoxin.** | | |

Syllabus: C2c
Reference Text: Goodman and Gillman Chp 34

**3 (60%) candidates passed this question**

2. **Compare and contrast the pharmacology of midazolam and dexmedetomidine when used for sedation.**

This question was also well suited to be answered in a preset format. For example a tabular format that had headings such as mechanism of action, preparations, dosing, pharmacokinetics, metabolism and excretion, pharmacodynamics, drug interactions and side effects.

A good answer was expected to include the following points. Under mechanism of action, mention that both drugs produce sedation by hyperpolarizing CNS nerve membranes and act on different receptors (Midazolam binds the benzodiazepine receptor and dexmedetomidine being selective for the a2 receptor). Also mention of other effects for each drug, eg anxiolytic, anticonvulsunt, analgesia, etc. A similar approach would be required for other key areas such as metabolism and excretion,(including alterations with age, organ failure, disease, etc), drug interactions, pharmacodynamics, particularly in relation to important physiological effects (eg CNS and CVS effects).

A brief summary of the similarities and differences which influence the clinical use of these agents gained more marks and showed the candidate had applied knowledge of these drugs. The common omissions were lack of explanation of mechanism of action and failure to mention pharmacodynamic effects, drug interactions and specific advantages for each agent.

Syllabus: G2b
Reference Text:

**1 (20%) candidate passed this question**

3. **Outline the physiological consequences of hyperthyroidism in an adult.**

This question sought an understanding of the physiological effects of thyroid hormones. The major area of weakness for candidates was a lack of detailed understanding of the physiological actions of thyroid hormones and/or providing an answer that predominately listed clinical manifestations. A good answer would have included the following points -

Stimulation of Carbohydrate Metabolism **-** all aspects of carbohydrate metabolism, including rapid uptake of glucose by the cells, enhanced glycolysis, enhanced gluconeogenesis, increased rate of absorption from the gastrointestinal tract, and increased insulin secretion

Stimulation of Fat Metabolism **-** all aspects of fat metabolism are also enhanced lipids are released from fat stores and increased oxidation of free fatty acids by the cells - *decreases* the concentrations of cholesterol, phospholipids, and triglycerides in the plasma, even though it *increases* the free fatty acids

Increased Basal Metabolic Rate – increased CMRO2

Increased Requirement for Vitamins

Increased vasodilatation, Cardiac Output, heart rate (not BP), contractility

Increased Respiration – secondary to increased metabolism

Increased Gastrointestinal Motility and secretions

Excitatory Effects on the Central Nervous System, seizures and insomnia

Effect on the Function of the Muscles **–** stimulates contractility and metabolism, but too much leads to the muscles become weakened because of excess protein catabolism. Also muscle tremor by increased reactivity of the neuronal synapses in the areas of the spinal cord that control muscle tone

Effect on Other Endocrine Glands - . increases the rate of glucose metabolism everywhere in the body and therefore causes a corresponding need for increased insulin and glucagon secretion by the pancreas.

Also, increase bone formation and, as a consequence, increases the need for parathyroid hormone. Thyroid hormone also increases adrenal glucocorticoid metabolism by the liver.

Syllabus: N2e
Reference Text: Guyton Chp 76

**0 (0%) candidates passed this question**

4. **Classify bacteria according to the Gram stain system and the shape of the bacteria, and give two examples for each classification (40% of marks). Outline the different mechanisms of bacterial antibiotic resistance and an antibiotic for which that mechanism may apply (60% of marks).**

This question also highlighted the importance of candidates noting the way marks were proportioned. A good answer required the following points –
Classification and examples - gram-positive cocci (*Staphylococcus aureus, Streptococcus pyogenes* or *pneumoniae* or *agalactiae*), gram-negative cocci (*Neisseria meningitidis, N. gonorrhoeae*), gram-positive bacilli (*Bacillus anthracis, Listeria, corynebacteria, Clostridium difficile*, etc), gram-negative bacilli (*Escherichia Coli or E. Coli, Proteus, Yersinia, Salmonella, Shigella, Pseudomonas aeruginosa, Klebsiella pneumoniae, Legionella*).

Mechanisms of resistance include: a) Enzyme inactivation, beta-lactamase or Extended Spectrum Beta-lactamase b) Enzyme addition: enzyme produced by bacteria that add a chemical group to the antibiotic to inhibit its activity, aminoglycoside resistance by *Staphylococcus aureus* or *Pseudomonas*. c) Impermeability, anaerobes have no oxygen-dependent transport mechanism which stops the penetration of aminoglycosides into the bacteria. d) Efflux mechanisms by acquisition of an inner member protein which actively pumps antibiotics out of the cell, *E.Coli* becomes resistance to tetracycline by this mechanism. e) Alternative pathway to circumvent the metabolic block impose by antibiotic, Some *Staphylococcus aureus* are resistant to methicillin by developing or acquiring the gene mecA which produces an alternative penicillin binding protein and hence they are not inhibited by methicillin. f) Alteration of the target site, rifampicin resistance by point mutations, insertions, or deletions in RNA polymerase gene.

Syllabus: M2a, b, c
Reference Text: Microbiology and Infection at a Glance by Gillespie & Bamford 3rd Ed, 2007 page 8-9,21.

**3 (60%) candidates passed this question**

5. **Describe the respiratory changes that occur in morbid obesity.**

Obesity is an increasing problem in the broader community and in Intensive Care practice. Hence it is important candidates understand the physiological and pharmacological consequences of obesity. This question confined its scope towards obesity and the respiratory system. Major area of weakness of candidates was a lack of depth and breadth in knowledge of this topic and in applying basic physiology. A good answer required the following points -
Definition of morbid obesity (>200% ideal body weight or body mass index > 35)
Upper airway effects: fat infiltration of pharyngeal soft tissues → difficult airway, prone to airway obstruction eg OSA
↑ O2 consumption and CO2 production: due to ↑ total body fat, requires ↑ cardiac output and ↑ alveolar ventilation
↓ FRC mainly via ↓ ERV: due to mass loading and splinting of diaphragm, upright obese, closing capacity > FRC → small airway closure, ↑ V/Q mismatch, ↑ venous admixture and arterial hypoxaemia, ↓ O2 stores
↓ total respiratory system compliance:↓ chest wall compliance , subcutaneous and intra abdominal fat excess, ↓ lung compliance, ↑ airways resistance, ↑ work of breathing, ↓ resp muscle efficiency
Altered ventilatory control: Obstructive sleep apnoea, Obesity hypoventilation syndrome

Syllabus: B1k B1d2k
Reference Text: Nunn's Applied Respiratory Physiology / A B Lumb & J F Lunn - 6th ed

**2 (40%) candidates passed this question**

6. **Describe the physiology of bilirubin production, metabolism and clearance (70% of marks). Outline the changes in blood and urine of the products of bilirubin metabolism with intra and post hepatobiliary disease (30% of marks).**

A good answer for this question required the description of the pathway of bilirubin production beginning with the breakdown of haemoglobin breakdown, then haem to biliverdin by biliverdin reductase, biliverdin to bilirubin, bilirubin transported to liver bound to plasma proteins, bilirubin monoglucuronide and diglucuronide by conjugation, secreted into bile. Secretion into bile is dependent on active transport and is the first to be impaired in inflammation of the liver. Bilirubis metabolized to stercobilinogen which is absorbed and excreted in urine as urobilinogen. The remaining part to this question flowed on from this point, ie Intrahepatic disease – increased conjugated bilirubin, urobilinogen and urine bilirubin, Posthepatic disease – increased conjugated bilirubin, no urobilinogen, increased urine bilirubin

Syllabus: I2c
Reference Text: Guyton Ch 70

**3 (60%) candidates passed this question**

7. **Classify the hypersensitivity reactions, give an example for each reaction and describe the pathophysiological processes of each reaction.**

A tabular format is a very desirable format for a response to this question. Again candidates who struggled to provide an organised response also failed to provide sufficient and succinct detail in their answer. A good answer would have included the following features.

| Mechanisms of Immunological Injury | | |
|---|---|---|
| Mechanism | Pathophysiology | Disease types |
| Type I<br>Immediate hypersensitivity<br>IgE mediated | • basophil & mast cell degranulation<br>• histamine, SRSA, ECFA, NCF<br>• immediate weal & flare | • anaphylaxis<br>• atopy |
| Type II<br>cell cytotoxicity<br>IgG, IgM mediated | • direct phagocytosis or cell lysis<br>• activation of complement, classical<br>• tissue deposition of complement | • blood transfusions<br>• Goodpasteur's syndrome<br>• autoimmune cytopaenias |
| Type III<br>Immune complex<br>IgG, IgM, IgA mediated | • tissue deposition of Ag-Ab complexes<br>• accumulation of PMN's, macrophages & complement | • SLE<br>• serum sickness<br>• necrotising vasculitis |
| Type IV<br>Delayed hypersensitivity<br>T-cell mediated | • T-cell induced mononuclear cell accumulation<br>• release of lymphokines & monokines<br>• often with granuloma formation | • TB, sarcoid<br>• Wegener's granulomatosis<br>• granulomatous vasculitis |

Syllabus: M2g
Reference Text: Goodman and Gillman Chp 64

**3 (60%) candidates passed this question**

8. **Compare and contrast the pharmacology of sodium nitroprusside and glyceryl trinitrate.**

It was expected candidates would address specific aspects of pharmacology such as action, mechanism of action, half life and duration of effect, route of administration, potential toxicity and special precautions. These agents lend themselves to comparison and contrast as several distinct similarities and differences exist and credit was given for highlighting these. Specific comments should include that both agents result in blood vessel dilation with extra credit given for detailing the differences in the balance of arterial versus venous effects between them. For both agents the effect is mediated through nitric oxide and it was expected candidates would identify that nitroprusside releases NO spontaneously and GTN requires enzymatic degradation with the resultant effects on smooth muscle mediated via c GMP. They are both short acting agents when used intravenously and require careful titration to measured blood pressure for effect.

Extra credit was given for mentioning that routes other than IV are available for GTN (topical / oral) but not for nitroprusside. Comments on special precautions such as Nitroprusside should be protected from light and GTN given via non PVC giving sets gained additional marks. In addition to the well described adverse effects of each agent, it was expected

candidates would mention the potential for cyanide toxicity with nitroprusside and extra marks were awarded for an indication of usual doses.

Syllabus C2b 2e
References Katzung 10th edition, Goodman and Gillman Chp 31 & 32

**4 (80%) candidates passed this question**

9. **Outline the pathophysiological basis for the use of angiotensin converting enzyme inhibitors (ACE-I) and angiotensin receptor blockers (ARB) in congestive cardiac failure.**

The renin-angiotensin system plays a central role in the pathophysiology of heart failure. Thus this question required integration of knowledge of the renin-angiotensin system and how pharmacological agents affect it in the treatment of cardiac failure. Candidates were expected to describe the pathway and the influence of these drug groups on cardiac failure and to recognise underlying basic physiological principles such as the interaction between $AT_1$ and $AT_2$ receptors along with awareness of production of Ang II by ACE-independent enzymes.

A good answer was expected to contain the following points: Angiotensinogen is cleaved by kidney-derived renin to form the decapeptide angiotensin I (Ang I); ACE converts Ang I to Ang II; Ang II is a potent arterial vasoconstrictor and an important mediator of $Na^+$ and water retention through its effects on glomerular filtration pressure and aldosterone secretion; Ang II potentiates neural catecholamine release, is a secretagogue for catecholamine release from the adrenal medulla, promotes vascular hyperplasia and pathologic myocardial hypertrophy.

ACE inhibitors suppress Ang II and aldosterone production, decrease sympathetic nervous system activity, and potentiate the effects of diuretics in heart failure. ACE is identical to kininase II, which degrades bradykinin and other kinins that stimulate production of NO, cyclic GMP, and vasoactive eicosanoids; these vasodilator substances seem to oppose the effects of Ang II on the growth of vascular smooth muscle and cardiac fibroblasts and on production of extracellular matrix. Thus, the increased levels of bradykinin that result from ACE inhibition may play a role in the hemodynamic and anti-remodeling effects of ACE inhibitors.

An alternative means of attenuating the haemodynamic and vascular impact of the renin-angiotensin system is through inhibition of angiotensin receptors. Most of the known clinical actions of angiotensin II are mediated through the $AT_1$ angiotensin receptor. $AT_1$ receptor antagonists may provide more potent reduction of the effects of angiotensin II than do ACE inhibitors.

Syllabus C2d 1 and part C2b 2f
Reference Goodman and Gilman 561-7, Guyton Chp 22

**0 (0%) candidates passed this question**

10. **Describe how gas exchange is facilitated across the placenta near the end of pregnancy.**

Candidates were expected to cover the basic principles of gaseous diffusion across the placenta, with reference to: Both oxygen and carbon dioxide; Fick's Law, including placental area and thickness, and relative gas tensions and solubilities; Changes in maternal and foetal blood flow approaching term; Approximate values for maternal and foetal gas tensions and content; The differences between foetal and maternal haemoglobin, quantitative and qualitative; The double-Bohr and double-Haldane effects; Relative maternal hyperventilation and it's effects on arterial gas tensions and how these influence foetal transfer.

A good answer would also include a description of the physical arrangement of maternal sinuses and foetal capillaries; labelled dissociation curves for $O_2$ and $CO_2$ detailing the differences between maternal and foetal Hb; a placental gas exchange diagram, showing foetal and maternal arterial and venous gas tensions and content values.

Syllabus ref: O1 2d
Suggested Reading: Review of Medical Physiology / W F Ganong – 22nd ed Chapter 32. Nunn's Applied Respiratory Physiology / A B Lumb & J F Lunn - 6th ed

**3 (60%) candidates passed this question**

11. **Describe the factors which contribute to inter-individual variability in drug response seen with an induction dose of an intravenous anaesthetic drug.**

This question required the candidate to provide some detail why a given dose of intravenous anaesthetic administered for the purpose of induction may result in a variable response between individuals. A logical division into pharmacokinetic and pharmacodynamic factors pertaining to the drug and how these are impacted by patient physiology, citing appropriate examples, was expected. Extremes of age, pregnancy, low and high cardiac output states, sympathetic tone, body habitus and factors impacting on drug redistribution and elimination should have been addressed. Extra marks were awarded for relevant discussion of pharmacogenetics. Discussion of the "bolus effect" versus slow infusion of an induction drug, relative to these factors should have been included. Mention of drug interactions, idiosyncratic reactions and relevant pathophysiology impairing organ function reserve was appropriate. The consequences of variability in drug response in terms of over- / under-dosing, haemodynamic compromise, respiratory depression, delayed recovery and allergic reactions needed emphasis to demonstrate understanding of the significance of predisposing factors.

Effective answers to this question utilised either clear headings, or a tabular format, dividing what is a large topic into discrete areas. The lack of a structured approach to this question was invariably unsuccessful.

Syllabus Ref: G2a 2.c,d,e,f,g
Suggested Reading: Pharmacology and Physiology in Anaesthetic Practice / R K Stoelting – 4th ed - 2006. Chapters 4,6.

**2 (40%) candidates passed this question**

12. **Describe the gravity dependent processes which affect pulmonary blood flow (70% of marks). Describe the changes that result from an acute increase in pressure in the pulmonary vessels (30% of marks).**

Most candidates quite correctly approached this from the perspective of "West's zones of the lung. A clear description of the relationship between pulmonary arterial, venous and alveolar pressures producing the classical 3 zones was expected, along with situations which may alter the normal balance between the 3 zones, e.g. changing posture or airway pressure. Additional points were awarded for candidates describing 'zone 4' or alternate theories of V/Q distribution.

Whilst most candidates described recruitment and distension with respect to changing pulmonary arterial pressure, candidates were also expected to correctly state that an increase in pulmonary artery pressure is only observed when these processes are exhausted, or in the setting of pulmonary vascular disease, then describing the subsequent effects of pulmonary hypertension on the heart and circulation.

The vascular tree is distensible, that is, a change in pressure will produce a corresponding increase in dimension of the blood vessels:

$$\text{Vascular distensibility} = \frac{\text{increase in volume}}{\text{Increase in pressure} \times \text{original volume}}$$

Increased pressure delivered to the arterioles causes dilation and decreases resistance to flow, increasing flow by as much as twice what would be expected due to pressure alone
The veins are 6 to 10 x as distensible as arteries owing to the structural differences in their respective walls. A notable exception is the pulmonary circulation where arteries are approximately ½ as distensible as veins; this buffers pressure changes transmitted to the alveolar capillaries and also permits the arteries to adopt a reservoir function.

Pulmonary vascular resistance is also a function of lung volume. At extremes pulmonary capillaries are linearly stretched and collapse as may occur with hyperinflation. At very low lung volumes, extra-alveolar blood vessels become compressed and flow reduces (Zone 4).

Use of appropriate graphs to illustrate some of the above points would have been desirable..

Syllabus Ref: B1i 2, B1k 2. a,c,i
Suggested Reading: Nunn's Applied Respiratory Physiology / A B Lumb & J F Lunn - 6th ed - Chapters 7,8

**4 (80%) candidates passed this question**

**PAPER 1 and 2 CLOZE QUESTIONS**

**5 (100%) candidates passed these questions**

**PAPER 1 and 2 RANK QUESTIONS**

**3 (60%) candidates passed these questions**

**PAPER 1 and 2 CLOZE QUESTIONS**

**4 (80%) candidates passed these questions**

## ORAL SECTION

3 candidates were invited to attend the oral section based on their written marks.

**Viva 1**
The initial information given to the candidates was -
*In this station you will be asked to draw and discuss a capnograph tracing.*
*Draw a graph of partial pressure of $CO_2$ versus time, from a ventilated patient, i.e. a 'capnograph tracing'.*

This viva explored the candidates' knowledge in relation to the following points
- Capnograph trace and information that can be ascertained from it
- Methods used to measure partial pressure of CO2 and the underlying principles
- ETCO2 and PaCO2 relationships and factors that influence it
- Capnograph and deadspace

Examination feedback: General there was a lack of depth in the understanding of the capnograph. Specifically what it measures, principles of measurements and an understanding of deadspace.

**2 ( 66%) candidates passed this questions**

**Viva 2**
The initial information given to the candidates was -
*What is normal cerebral blood flow?*

This viva explored the candidates' knowledge in relation to the following points
- Cerebral Blood Flow, it's measurement and factors that alter it
- Cerebral Blood Flow and acute changes in altitude
- Morphine, typical dose, CNS effects and metabolites

Examination feedback: There was good understanding of cerebral blood flow, and most candidates used figures to good effect during the viva. Areas of weakness were an understanding of the methods of measuring CBF and differential flow to the white and gray matter. Candidates also were less confident with the different CNS effects of morphine. Adapting factual knowledge to define what would happen in states of altered physiology was also handled poorly.

**2 ( 66%) candidates passed this questions**

**Viva 3**

The initial information given to the candidates was -
*You were asked by an intensive care nurse whether you would like to start some anti-hypertensive treatment for an ICU patient who had a very high arterial blood pressure (systolic 180mmHg, diastolic 60mmHg) displayed on the invasive haemodynamic monitor at the bedside. The ICU nurse said that the non-invasive oscillometric blood pressure monitor (systolic 140mmHg and diastolic 80mmHg) was much lower than the invasive blood pressure.*

*Can you explain how a non-invasive oscillometric blood pressure monitor works?*

This viva explored the candidates' knowledge in relation to the following points
Principles of oscillometric blood pressure measurements, factors affecting accuracy
Principles of invasive blood pressure measurement, factors affecting accuracy, resonance and damping

Examination feedback: All candidates had a good understanding of the principles behind oscillometric blood pressure monitoring and its pitfalls. A good understanding on invasive blood pressure monitoring was also essential to pass this viva.

The common weaknesses were 1) the effects of the intra-arterial catheter size or bore, catheter length, and compliance of the tubing on resonance and damping; 2) how to assess the damping factor at the bed side; and 3) what is the minimal requirement in terms of natural frequency of the measuring system in relation to the frequency of the blood pressure waveform to prevent resonance. Most candidates found it difficult to convert a heart rate of 60 per minute into frequency in Hz.

**3 ( 100%) candidates passed this questions**

**Viva 4**
The initial information given to the candidates was -
*This station will explore the pharmacology related to analgesic medications.*
*How will you classify non opioid analgesic agents?*

This viva explored the candidates' knowledge in relation to the following points
Classification of non opioids, mechanism of actions of non opioids
Classification of opioids, mechanism of action, some aspects of their general pharmacology, contrasting fentanyl and morphine, tolerance to opioids

Examination feedback: Generally candidates performed better at classifying non opioids in comparison to the opioids. There was also a lack of depth and breadth in knowledge related to opioid receptors

**3 ( 100%) candidates passed this questions**

**Viva 5**
The initial information given to the candidates was -
*This station is mostly concerned with the pharmacology of drugs acting on the gastrointestinal tract.*
*What is a prokinetic?*

This viva explored the candidates' knowledge in relation to the following points
Prokinetics, examples of prokinetics, mechanism of action, general pharmacology of metoclopramide

GIT secretions
Laxatives, mechanism of action of lactulose
Octreotide, mechanism of action, general pharmacology, indications for use

Examination feedback: This is an important, and potentially overlooked, area of clinical ICU practice. Candidates breadth and depth of knowledge for this important topic was lower than expected. In particular was mechanism of action of prokinetics, laxatives (of which most of the discussion centred around lactulose) and octreotide.

**1 ( 33%) candidates passed this questions**

**Viva 6**
The initial information given to the candidates was -
*This viva will mostly cover pharmacology and physiology of insulin.*
*Describe the primary function of insulin.*

This viva explored the candidates' knowledge in relation to the following points
Structure, mechanism of action and physiology of natural insulin
Insulin preparations
Pathophysiology of re-feeding syndrome

Examination feedback: Candidates knowledge in relation to the physiology of insulin was good and much stronger than their knowledge in relation to the pharmacology of the different insulin preparations. It is important that candidates have a good understanding of the pharmacology of insulin preparations as well as remain up to date with changes in those preparations. Candidates also struggled with describing the basic elements, in particular the changes to electrolytes, associated with the re-feeding syndrome.
**3 ( 100%) candidates passed this questions**

**Viva 7**
The initial information given to the candidates was -
*This station will explore your knowledge of the pharmacology and physiology of noradrenaline.*

*Discuss the pharmacologic effects of noradrenaline on the human heart in-vivo.*

This viva explored the candidates' knowledge in relation to the following points
Pharmacology of noradrenaline
Pressure-Volume Loop of the cardiac cycle
Mark on the Pressure-Volume Loop the changes you would expect for an adult patient infused noradrenaline at 10 mcg/min.
Explain the consequences of a noradrenaline infusion on myocardial oxygen consumption, work and efficiency.
Dobutamine infusion of 5 mcg/kg/min and affect the Pressure-Volume Loop

Examination feedback: This viva tested knowledge related to cardiac cycle, P-V loop, inotropes and most importantly a candidates ability to integrate those topics and then discuss them. Generally candidates knowledge in describing the basic P-V loop were sound, but were weaker in explaining how the P-V loop changed with changes in physiology and pharmacological intervention. Candidates were expected to generate discussions relating to the impact in changes to afterload, pre load and contractility as well as concept of potential energy as it elated to the P-V loop.

**3 ( 100%) candidates passed this questions**

**Viva 8**
The initial information given to the candidates was -
*This Station will explore aspects of respiratory physiology and related measurement.*

*This Chest Xray shows complete collapse of the Left Lung.*

*What patho-physiological processes might contribute to Hypoxia in this patient?*

This viva explored the candidates' knowledge in relation to the following points
- Describe ventilation and perfusion to the lung and illustrate their relationship
- Significance of high and low V/Q
- Shunt and mechanisms to limit hypoxia, effect of supplemental oxygen
- Pulmonary Vascular Resistance
- Pulse Oximetry, principles of measurement, limitations

(A digital reproduction of a CXR was displayed – but candidates were not expected to make any clinical interpretation of the CXR and were provided with the CXR findings and diagnosis)

Examination feedback: Candidates used graphs to illustrate their discussions with good effect. Areas of weakness was in understanding and explaining the limitations of the pulse oximetry.

**3 ( 100%) candidates passed this questions**

***A detailed syllabus has been developed and forms the foundation for the knowledge base for the JFIC Primary Examination. All questions are sourced directly from that syllabus and candidates should have a sound understanding of those topics, and confidence to express their understanding of the subject material in both written and oral form. The candidates should be able to integrate and express basic physiological and pharmacological principles as to how the relate to various scenarios relevant to Intensive Care practice. Candidates would find it valuable to develop strategies and standard formats to answer typical questions, eg compare and contrast the pharmacology of Drugs X and Y. Candidates are also strongly encouraged to use accurate and labelled figures and tables wherever possible to help display their knowledge and to describe basic principles.***

**Dr Arthas Flabouris**
**Deputy Chair**
**Primary Examination Committee**

Circulation:
Board of Joint faculty
Supervisors of Intensive Care Training
Regional Education Offices
Panel of Examiners
Course Supervisors
Registered Trainees