COLLEGE OF INTENSIVE CARE MEDICINE
OF AUSTRALIA AND NEW ZEALAND

REPORT OF THE
INTENSIVE CARE PRIMARY EXAMINATION

SEPTEMBER / NOVEMBER 2011

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:	25
Number of candidates scoring >50% in the written:	6
Number of candidates scoring 45-50% in the written:	3
Number of candidates carrying a written score:	0
Total number invited to the Oral section based on written marks:	9
Total number of candidates successful at the CICM Primary:	8

Successful candidates:

Athavale	Vinit
Estensen	Kristine
McNamara	Robert
Murray	Andrew
Purvis-Smith	Michael
Richards	Stephen
Saxena	Anurag
Xu	Tina

WRITTEN SECTION: SAQ PAPER 1

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

It was expected answers would include a comment this would transiently increase the blood glucose and result in the stimulation of insulin production. Some detail was then required on the mechanism by which insulin production is increased and time frames over which this occurred, as well as mechanism by which glucose passes through to the urine. Other important points expected were the fate of glucose, once taken up by the cells, the inhibitory effects (and mechanisms) of insulin (ie inhibits glycogenolysis, triglyceride and protein breakdown, etc). A description of the physiological consequences of increased insulin production was required.

This question has been asked before, in it's current form. The pass rate on this occasion was higher. Candidates who did poorly did so due to a lack of detail and understanding of the topic.

14 (56%) of candidates passed this question.

Syllabus: N12b
Recommended sources: Textbook of Medical Physiology 11th ed Guyton and Hall, Chp 78.

2. Describe the factors contributing to inter-individual variability in drug response seen with an induction dose of an intravenous anaesthetic drug.

This was a broad question that required some structure and organisation to score well. The issues around variability in both pharmacokinetics and pharmacodynamics, of which there are many, needed be covered. For example variability due to differences in organ function, age, metabolism, body composition, were just a few of the factors expected to be mentioned.

15 (60%) of candidates passed this question.

Syllabus Ref: G2a 2.c,d,e,f,g
Recommended sources: Pharmacology and Physiology in Anaesthetic Practice / R K Stoelting – 4th ed, Chps 4,6.

3. Describe the role of the kidney in drug excretion and the factors affecting this (60% marks). Briefly outline how you would alter the dosing of a drug with high renal excretion in a patient with renal impairment (40% marks).

It was expected candidates would expand on the role of the kidney in the excretion of drugs and metabolites. A statement that referred to the classical features of drugs that undergo renal excretion (eg polarity, lipid solubility, size and protein binding, drug metabolites) and a definition of renal clearance was expected and would have provided an excellent introduction to any answer. It was then expected that candidates would mention, in some detail, the processes of filtration and secretion (both active and passive and both proximal and distal along the renal tubules). The question also asked for factors that affect renal drug excretion, for example, a reduction in GFR or alteration in protein binding. An approach to alterations of dosing would require some consideration of assessing degree of dysfunction (GFR estimation / calculation) then an understanding that it would not impact on loading doses but would influence subsequent dosing of renal cleared drugs. Plasma monitoring provides useful information for some drugs, particularly those with a narrow therapeutic index.

13 (52%) of candidates passed this question.

Syllabus: II2d, D12h
Recommended sources: Goodman and Gilman's the Pharmacological Basis of Therapeutics pgs10-14; Foundations of Anaesthesia Basic and Clinical Science, Hemmings pg107; Basic and Clinical Pharmacology, Katzung pgs 35, 48-49.

4. Describe the pharmacology of low molecular weight heparin (70% marks). Outline the pharmacology of hirudin (30% marks).

A question that asks for information of the pharmacology should mention pharmaceutical (only briefly), pharmacokinetics and pharmacodynamics of the drug or class of drugs requested. The term “describe” requests of the candidate a greater depth of information than “outline” and both are defined in the “Notes to Candidates” document. Candidates who did well, did so because they had sufficient knowledge of this commonly used class of drugs, and could structure a well organised answer.

10 (40%) of candidates passed this question.

Syllabus: J2a 2a
Recommended sources: Rang and Dale Pharmacology, Chp 21; Goodman & Gillman The pharmacological Basis of Therapeutics Chp 54.

5. Describe the essential components of an ECG monitor (60% marks). Outline the methods employed to reduce artefact (40% marks).

Monitoring and monitors are essential to Intensive Care practice, and is the reason why it is included in the syllabus. Unfortunately candidates have performed poorly in this question, as they have in previous measurement and monitoring questions. Future candidates need to be aware that such questions WILL get asked again.
For a good answer it was expected that mention would be made of what an ECG monitors does (ie detects and amplifies the small electrical changes on the skin that are caused when the heart muscle depolarizes), how (ie use of 2 or more electrodes, typically being made of silver or silver chloride), the type of leads (ie unipolar and bipolar, and a description of the latter), the way the signal is handled (isolation, amplification, gain, filtering) and displayed. Methods to reduce artefact and improve signal:noise ratio, should have included skin conductive measures, minimising external interference (filters, earthing), common mode signal artefact rejection, high input impedance amplification and mention of diagnostic and monitoring modes.

2 (8%) of candidates passed this question.

Syllabus: S2a
Recommended sources: Davis and Kenny pgs 160-178, also Sykes & Vickers Principles in measurement and monitoring in Anaesthesia and Intensive Care, Chps 4, 5, 6, 23.

6. Describe the physiology of the Renin and Angiotensin system.

Renin and angiotensin are core components in the regulation of plasma volume and blood pressure regulation. It is unfortunate that many candidates presenting to this examination are not able to provide sufficient information for a pass. For a good answer, candidates were expected to mention what renin and angiotensin are and what they do, as well as briefly mention the place of angiotensin converting enzyme (converts Angiotensin I to Angiotensin II and inactivates bradykinin). Renin is a proteolytic enzyme cleaves angiotensinogen to angiotensin I, secreted by the juxtaglomerular cells of the kidney which are located in media of afferent arteriole and in close proximity to the glomerulus and the distal convoluted tubule (macula densa). Angiotensin II acts on cell surface AT_1 and AT_2 receptors. Major functions being to preserve of GFR & enhanced Na/H_2O reabsorption in the setting of reduced renal blood flow (candidates expected to outline the mechanism by which this occurs), vasoconstriction, stimulate aldosterone secretion and increase thirst and ADH secretion. The better candidates also mentioned that it decreases sensitivity of baroreceptor reflex, increases secretion of ACTH and facilitates noradrenaline release from sympathetic nervous system as well as its fate (metabolized by blood/tissue peptidases). A good response for regulation would have been mentioning principally regulated via renin release (which in itself is influenced by renal sympathetic nervous system activity, intrarenal baroreceptors and macula densa sodium chloride delivery, ADH and intra-renal prostaglandins), negative feedback from angiotensin II.

15 (60%) of candidates passed this question.

Syllabus: N2h
Recommended sources: Textbook of medical Physiology, Guyton, Chp 26.

7. Define lung compliance (30% marks). Describe how is it measured (70% marks).

This is a core area of physiology that relates to everyday Intensive Care practice, thus it was expected that more than the observed number of candidates would have scored well. Candidates performed poorly purely from a lack of sufficient knowledge. Easy marks were to be gained purely by mentioning that compliance is defined by $\Delta V/\Delta P$, the ΔP being the gradient from alveolar – intrapleural, normal values, static and dynamic compliance. Good answers would then include a mention of how static and dynamic compliance is measured (specifically how volume and pressures are measured.

9 (36%) of candidates passed this question.

Syllabus: B1d 2b
Recommended sources: Nunn Applied Respiratory Physiology, 6th Ed, Pgs 35-36.

8. Explain the physiological factors that prevent gastro-oesophageal reflux.

For a good answer candidates were expected to give a description of the lower oesophageal sphincter (the intrinsic and extrinsic sphincters and flap-valve), that it maintains a resting pressure of 15-25 mmHg above gastric pressure which prevents gastro-oesophageal reflux, which relaxes after swallowing, that the resting tone is maintained by myogenic and neurogenic mechanisms and the effects of hormones upon the sphincter (ie gastrin, motilin and α adrenergic stimulation increases and secretin, glucagon, VIP and GIP decrease tone).

3 (12%) of candidates passed this question.

Syllabus: Q1 2f
Recommended sources: Ganong Review of Medical Physiology Chp 26.

9. Define a Portal System. Describe the anatomy and function of three portal systems in the body.

A portal system is an arrangement by which blood collected from one set of capillaries passes through a large vessel or vessels, to another set of capillaries before returning to the systemic circulation. The three portal systems are the -

1) system of blood vessels that link the hypothalamus and the anterior pituitary in the brain, which allows endocrine communication between the two structures.
2) within the liver, whereby venous blood from the GI tract drains into the superior and inferior mesenteric veins; these two vessels are then joined by the splenic vein to form the portal vein which enters the liver, drains into the hepatic sinusoids and then eventually into the hepatic veins which join the inferior vena cava, with the purpose of defending against by breaking down and metabolising most of what has been absorbed from the gastrointestinal tract (including an immunoprotective action).

3) within the kidney, whereby blood from the afferent arterioles enters the glomerulus (first capillary network), followed by the efferent arterioles, then the peritubular network (second capillary network) and eventually the venous system, with the purpose of stronger re-absorptive capacity for water from within long Loops of Henle that go deep within the renal medulla.

8 (32%) of candidates passed this question.

Syllabus: N1 2c, I2d, D1 2a
Recommended sources: Ganong Review of Medical Physiology Chps 18, 38, 29

10. Describe the pharmacology of magnesium sulphate.

Magnesium is a commonly administered agent within Intensive Care practice. Candidates performed poorly because of a lack of sufficient knowledge and a failure to structure their answer. For a good answer candidates were expected to mention that Magnesium comes as an inorganic sulphate, acts as a cofactor for a vast number of reactions, can be given orally (poor absorption) as well as intravenously, renal excretion with a low threshold.

1 (4%) of candidates passed this question.

Syllabus: O2 2c
Recommended sources: Rang and Dale Pharmacology Pg 388; Katzung Basic and Clinical Pharmacology, Pg 244.

11. Outline protein metabolism, including the effects of starvation.

For a good answer candidates were expected to mention that there were “essential” and “non-essential proteins”, that protein synthesis draws on amino acids sourced from the common amino acid pool (because there is no cellular storage of amino acids) which is the combination of amino acids derived from gut absorption of digested dietary protein and also the continuous turnover of endogenous protein, amino acids filtered via the glomerulus are reabsorbed within the renal tubule and that in health losses are minor, but increases with illness. Protein synthesis is a semicontinuous activity, the rate of which varies considerably between cells under the influence of factors neuro- endocrine factors, substrate availability and energy availability. Proteolysis occurs within nuclear and cytoplasmic proteasomes, via peptide bond disruption catalysed by proteases, which are then processed further into amino acids being then available for release into the amino acid pool or utilised immediately for protein synthesis.

In relation to starvation candidates were expected to mention that it results from a severe ongoing restriction of protein, energy and nutrient intake. That initially, glycogen stores from the liver and skeletal muscle are utilised after conversion to glucose and provide energy, thus sparing of fat and protein stores from use as an energy source for a period of less than a day, following which fats are increasingly utilised and finally protein catabolism accelerates, especially from the liver and skeletal muscle, with relative sparing of the heart and brain.

1 (4%) of candidates passed this question.
Syllabus: K2c,f
Recommended sources: Ganong Review of Medical Physiology Chp 2.

12. List the functions of the liver (60% marks). Discuss the metabolism of paracetamol in toxicity and the pharmacologic management of this overdose (40% marks).

A good response to this question required an ordered and well structured response. There are numerous important functions that the liver undertakes (eg bile formation, immunological, protein, lipid, glucose metabolism, storage, endocrine, etc), yet most candidates could not generate a sufficient list. In relation to paracetomol toxicity, a good response required candidates to mention that normal conjugating reactions in the liver are saturated and metabolism diverts to mixed function oxidases, which generate toxic metabolites. These in turn are inactivated by conjugation with glutathione. However when glutathione is depleted, toxic metabolites react with cellular nuclear material, thus causing liver necrosis. Toxic compound depletes sulphydril groups and also causes direct damage via lipid peroxidation. Regeneration of sulphydril groups and glutathione depends on availability of cysteine, thus the need to administer acetylcysteine.

5 (20%) of candidates passed this question.

Syllabus: I2a, G2e2c
Recommended sources: Gannong Review of Medical Physiology pg 485; Power and Kam Principles of Physiology for the Anaesthetist pg185; Stoelting Pharmacology and Physiology in Anesthetic Practice 4th edition pg 285; Rang & Dale Pharmacology 5th Ed pg 244.

SAQ PAPER 2

13. Discuss the regulation of cardiac output. Illustrate your answer by using a graph to describe the important physiological relationships.

Most candidates approached this question by defining cardiac output as stroke volume × heart rate and then discussing the determinants of cardiac output - preload, contractility, afterload and heart rate rather than focusing on the regulation of cardiac output. Under preload a brief description of the Frank Starling mechanism was required. Important was the concept that at rest cardiac output is controlled almost entirely by peripheral factors that determine venous return. These concepts were best illustrated by graphing vascular function (venous return vs right atrial pressure) and cardiac function (cardiac output vs right atrial pressure) curves. Then demonstrating on these curves the factors that affected preload, contractility and afterload such as changes in blood volume, sympathetic and parasympathetic stimulation and exercise as examples. Also important to demonstrate on these curves was the fact that venous return and cardiac output are equal at steady state. Most candidates tried to illustrate these cardiovascular concepts with a series of left ventricular pressure volume loops rather than use the vascular and cardiac function curves. They then went on to demonstrate via these pressure volume loops the effects of changes in preload, contractility and afterload on stroke volume. Candidates who took this approach were not penalised, if there were clear, correct diagrams with explanations indicating comprehension of these concepts. On the whole graphs were poorly drawn and were not well integrated into the answer. Some candidates also wasted time by unnecessarily describing excitation-contraction coupling and sympathetic nerve reflex pathways.

11 (44%) of candidates passed this question.

Syllabus: C1c 2a,c,e,f,g
Recommended sources: Guyton Textbook of Medical Physiology 11th edition pgs 241-243.

14. Compare and contrast the pharmacology of dobutamine and milrinone

Many candidates presented their information in a tabular form and this worked well as it allowed direct comparison between the two drugs. Most candidates did not mention that dobutamine was a racemic mixture of [+] and [-] isomers. Also that the [+] isomer was a potent β1 agonist and α1 antagonist, while the [-] isomer was an α1 agonist. The administration of the racemic mixture results in the overall β1 agonism responsible for its activity and also its mild β2 agonist effect. While most candidates stated that milrinone was an inodilator details on its mechanism of action as a selective phosphodiesterase type III inhibitor were on the whole vague. Within the cytoplasm of the cardiac myocyte milrinone inhibits the enzyme PDE 3 which results in the inhibition of the breakdown of cyclic AMP which in turn results in elevated cellular levels of cAMP. These elevated levels of cAMP in turn activate cAMP dependant protein kinases with a resultant increase in the influx of Ca2+ into the cell via the sarcolemma. Also uptake of Ca2+ by the sarcoplasmic reticulum is increased. The overall effect is an increase in intracellular Ca2+ which increases myocardial contractility. Milrinone also has lusitropic action, inducing left ventricular relaxation. This probably occurs as a result of the inhibition of SR membrane bound PDE3. Milrinone also cause peripheral vasodilatation by inhibiting PDE3 in vascular smooth muscle cells which again results in elevated cAMP levels.In vascular smooth muscle cAMP normally inhibits myosin light chain kinase the enzyme that is responsible for phosphorylating smooth muscle myosin and causing muscle contraction.

Many candidates did not emphasize how very different the pharmacokinetics of these two drugs were. Milrinone has a much longer half life than dobutamine and because of its predominant renal excretion accumulates in renal failure.

14 (56%) of candidates passed this question.

Syllabus: C2d 2a
Recommended sources: Goodman & Gillman The pharmacological Basis of Therapeutics Chp 33.

15. Outline the motor and sensory pathways involved in withdrawing the lower limb from a painful stimulus.

A good answer required a description of the sensory pathway(s) (eg nociceptors, Aδ and C fibres, spinal dorsal horn, spinothalamic, thalamic and cortical pathways), reflex arc (nocioceptive sensory fibres synapse with spinal inter-neurons that in turn synapse with peripheral motor neurons supplying the lower limb as well as inter-neurones that also synapse with motor neurons on the contra-lateral lower limb producing a crossed extensor response), central integration and the motor pathways (fibers from the contra-lateral motor (and pre-motor) cortex pass through the posterior internal capsule forming the lateral and ventral cortico-spinal tracts, the cortico-spinal tracts pass through the anterior brainstem, the lateral tract decussating in the caudal medulla, continue to synapse with spinal motor neurons in the ipsilateral lumbosacral anterior horn cells, passage via peripheral nerves and flexor muscle stimulated, extensors inhibited, resulting in withdrawal of the limb) Common mistakes included errors in naming nerve pathways and receptors. Another error was to confuse the polysynaptic pain response pathways with the monosynaptic stretch reflex. Very few candidates mentioned feedback regulation via cerebellar input and at spinal level from muscle spindles and Golgi tendon organs

5 (20%) of candidates passed this question.

Syllabus: G12b
Recommended sources: Ganong Review of Medical Physiology Chps 11 & 16

16. Using a diagram, explain the effect of PaO2, PaCO2 and MAP (mean arterial pressure) on cerebral blood flow (60% marks). Outline the effects of propofol and ketamine on cerebral blood flow (CBF), cerebral metabolic requirement for oxygen (CMRO2), and cerebral venous oxygen saturation (40% marks).

Graphical depictions of the effect of Mean Arterial Pressure, oxygen tension and carbon dioxide tension on cerebral blood flow were common and in general accurate. Mention of factors that affected, and regulation of, the MAP vs CBF graph was expected in order to pass this question well.

The effect of propofol and ketamine on the CBF was well answered. Propofol and ketamine have an opposite effect on cerebral haemodynamic and metabolic rate. Propofol produces a dose dependent reduction in CBF with proportionate reduction in CMRO2, and thus a minimal change in cerebral venous O2 Sat. Propofol doesn't affect the autoregulatory curve of CBF and the PaCO2 response. Ketamine produce a dose dependent increase in CBF and a mild increase in CMRO2.

12 (44%) of candidates passed this question.

Syllabus: C1f 2c, G2a,2a.
Recommended sources: Guyton Textbook of Medical Physiology Chp 61; Goodman and Gilman The pharmacological basis of therapeutics 11th edition pgs 350-352.

17. Classify the calcium channel blockers and provide one example of a drug for each class (20% marks). Compare and contrast the pharmacology of nimodipine and verapamil (80% marks).

Most candidates were able to classify the calcium channel blockers well (Type I : Phenylalkylamines eg verapamil, Type II : Dihydropyridines eg nimodipine and Type III : Benzothiazepines eg diltiazem) However, the comparison of the pharmacology of nimodipine and verapamil was in general answered poorly. Few candidates demonstrated an organised approach to this part of the question. The two drugs' presentation, routes of administration, indications and dosing were poorly answered considering that nimodipine in particular is used frequently in intensive care units. Mode of action was well answered, but important principles relating to pharmacokinetics (such as a basic outline of protein binding, bioavailability, and metabolism) were expected, but common omissions. More knowledge than 'metabolism in the liver' is required. Few candidates mentioned interactions, adverse effects, or predictable effects of over dosage of these drugs.

9 (36%) of candidates passed this question.

Syllabus: C2b 2d
Recommended sources: Peck Hill and Williams Pharmacology for Anaesthesia and Intensive care 3rd Ed pgs 252-255.

18. Outline the abnormalities in pulmonary function testing of a person with severe obstructive lung disease (40% marks). Describe the physiological changes that explain these abnormalities (60% marks).

The majority of candidates were able to state that obstructive lung disease resulted in a reduction in the FEV1 and the FVC. Very few gave any indication of how much these parameters might be reduced in *severe* obstructive lung disease or that FVC is usually only slightly reduced, whereas the FEV1 is markedly reduced and therefore so is the FEV1/FVC ratio. Candidates were also expected to indicate that peak expiratory flow rate is also reduced (by 50% or more in severe disease), that carbon monoxide diffusing capacity is also reduced, and residual volume and total lung capacity are increased. A reasonable proportion of candidates commented that the forced expiratory flow (FEF) 25-50% being reduced and this results in a 'scalloped' flow volume loop. Accurately reproducing a normal flow volume loop and a flow volume loop of a patient with severe obstructive lung disease on the same graph could have depicted most of the above points and scored the majority of marks available.

The second part of this question was poorly answered. Whilst the majority correctly stated that an increase in airways resistance was present, few stated what changes gave rise to this. Dynamic airway compression, was mentioned by many, but why this occurred was explained poorly. The effect of closing capacity rising above the FRC was seldom mentioned and why the FRC increased again was seldom mentioned or explained. A detailed knowledge of what is a core subject in intensive care medicine was expected.

0 (0 %) of candidates passed this question.

Syllabus: B1a 2a, b; B1j 2a
Recommended sources: Respiratory Physiology: The Essentials. West 6th Ed pgs 26, 96-99, 131-142.

19. With respect to a clinical trial, define Type 1 and Type 2 Error, the Point Estimate and Confidence Interval (40% marks). Discuss the relevance of Power to study design and interpretation of the results (60% marks).

Most candidates correctly distinguished between a type 1 and type 2 errors. Type I error is: known as α and is when you incorrectly reject the null hypothesis – that is say a difference exists when it actually doesn't. Type II error is: when you incorrectly accept the null hypothesis, is termed β, that is to say no difference exists when it actually does. The point estimate: Is a single value estimate of a population parameter. It represents a descriptive statistic for a summary measure, or a measure of central tendency from a given population sample. Confidence intervals: define a range of values that are likely to include a population parameter. They are derived from the standard error for a given population. The percentage given, eg. 95 % reflects the probability that the true value will be contained within that interval.

Few candidates gave a definition for power or a value. Power is the likelihood of detecting a specified difference if it exists. It is important as it is a key determinant of the Sample size required for a study and this is a vital aspect of experimental design and evaluation. A sample size can to be too small – so can't adequately rule in / out an effect (ie. the study will lack the precision to provide reliable answers). If too large, a study will enrol unnecessary subjects to an experiment and this wastes time uses excess resources. It is unethical to conduct studies in both these cases.

6 (24%) of candidates passed this question.

Syllabus: EBM 2d,c,h
Recommended sources: Myles & Ginn Statistical methods for anaesthesia and intensive care.

20. Describe the structure and function of platelets (50% marks). Outline the pharmacology of clopidogrel (50% marks).

Platelets are small cells in blood, approximately 150,000 – 300,000 / microliter of blood with a half life of approximately 4 days. They have important membrane receptors (for collagen, vWF and fibrinogen) and intracellular contents (Actin, Myosin, Glycogen, Lysosomes, Dense granules, Alpha granules) and are involved in forming a platelet plug and clotting. Common omissions were to not give a normal platelet count or half life. Diagrams of the clotting cascade were not required.

Clopidogrel is an oral, thienopyridine class antiplatelet agent (a prodrug activated in the liver by cytochrome P450 enzymes), used to inhibit blood clots in coronary artery disease, following stent placement, peripheral vascular disease, and cerebrovascular disease. Mechanism of action is: specifically and irreversibly inhibits the P2Y12 subtype of ADP receptor, which is important in aggregation of platelets and cross-linking by the protein fibrin and thus platelet aggregation, which is inhibited when binding blocks activation of the glycoprotein IIb/IIIa pathway. Elimination half-life of about 8 hours; rapidly absorbed after oral administration; undergoes rapid hydrolysis in liver and renal excretion; 95% protein bound. Important interactions/precautions include proton pump inhibitors, phenytoin, warfarin, heparin, danaparoid, enoxaparin and various thrombolytics. Greatest risk is bleeding. The best answers had a structured approach to describing a drug.

13 (52%) of candidates passed this question.

Syllabus: J1 2f, J2 2d
Recommended sources: Ganong Review of Medical Physiology pg 485; Goodman & Gillman The pharmacological Basis of Therapeutics Chp 54.

21. Outline the functional anatomy, and the physiological factors, that determine oxygen delivery to the renal medulla.

A good answer required mentioning factors that affect systemic oxygen capacity and delivery (eg Hb, cardiac output, PaO2, etc), Hb-HBO2 dissociation and a description of the anatomy and regulation of renal medullary blood flow. 20 – 30% of nephrons have glomeruli deep in the cortex and long Loop of Henle that go deep into medulla. Here blood supply differs – long efferent arterioles from glomeruli into outer medulla and inner cortex that then divide into vasa recta deep in medulla. These vessels carry post glomerular blood so have less serum, mostly plasma, ie more viscous and concentrated. Factors that influence medullary blood flow include: Sympathetic stimulation – decrease (via efferent arteriolar constriction), Angiotensin II (via tubuloglomerular feedback) – decrease (via efferent arteriolar constriction), Endothelin – decrease (via efferent arteriolar constriction), Prostaglandins – increase, Bradykinin – increase, high protein meal – increase, high glucose levels – increase. Candidates performed poorly due to a lack of knowledge of the topic and/or failure to logically present their answer. Common mistakes were to give no value for renal blood flow, to discuss the function of the medulla which was not required and to not describe the factors that influence medullary blood flow.

1 (4%) of candidates passed this question.

Syllabus: B1h 2a,b; C1f 2d
Recommended sources: Guyton Medical Physiology Chp 26; Vander Renal Physiology Chps1, 5.

22. Outline the mechanism of action of ampicillin, gentamicin, vancomycin and ciprofloxacin. How does resistance develop to each of these antibiotics?

The question could have been answered succinctly using a table. Better answers included detail of how antibiotics inhibited enzymes, effects on ribosomal subunits, and impact on cell wall synthesis or inhibit DNA gyrase. The mechanism of resistance required an understanding of beta-lactam inhibition, gene mutation altering target enzymes and ribosomal subunits, and efflux mechanisms. Mention of Van A and Van B phenotypes was also sought.

9 (36%) of candidates passed this question.

Syllabus: M2a 2c,d
Recommended sources: Rang and Dale Pharmacology Chp 46, Katzung Basic and Clinical Pharmacolgy Chp 43, 45, 46.

23. Outline the production, release, and fate of noradrenaline at the sympathetic nerve terminal.

The question consisted of three parts (production, release and fate of noradrenaline). The context was the sympathetic nerve terminal. The synthesis of Noradrenaline from Tyrosine was expected. Better answers mentioned the roles of Tyrosine Hydrolase, DOPA Decarboxylase and DOPA Beta-hydroxylase. Candidates were expected to describe the storage of Noradrenaline in vesicles and Ca^{++}-mediated exocytosis in response to an action potential. Noradrenaline binds to post-synaptic and pre-synaptic receptors. Re-uptake, metabolism by MAO and COMT, and diffusion away from the synaptic cleft should have been discussed. An accurate diagram could be used to enhance the answer.

10 (40%) of candidates passed this question.

Syllabus: G3 2c
Recommended sources: Goodman & Gillman The pharmacological Basis of Therapeutics Chp 6.

24. Describe the pharmacology of corticosteroid drugs.

This question was very broad, requiring the discussion of corticosteroid pharmacology. This embraces the pharmacokinetics, pharmacodynamics and pharmaceutics of the drug class. Synthesis from cholesterol and hepatic metabolism was included in better answers. Comparison of mineralocorticoids versus glucocorticoids was expected. The mechanism of action through binding to intracellular receptors to affect protein synthesis should have been described. Additional marks were awarded for description of indications, preparations and routes of administration. In any pharmacology question, a list of common adverse effects is important. Of particular relevance is the suppression of the adrenal axis with prolonged administration.

5 (40%) of candidates passed this question.

Syllabus: N2 2c
Recommended sources: Stoelting, Pharmacology and Physiology in Anaesthetic Practice, pg 416; Peck & Hill Pharmacology for Anaesthesia and Intensive Care, pgs 355-8.

PAPER 1 and 2 CLOZE QUESTIONS

23 (92%) of candidates passed these questions.

PAPER 1 and 2 RANK QUESTIONS

20 (80%) of candidates passed these questions.

PAPER 1 and 2 MATCH QUESTIONS

21 (84%) of candidates passed these questions.

ORAL SECTION

9 candidates were invited to attend the oral section based upon their written marks.

Candidates were presented with the following information (shown in *Italics*) during the two-minute reading time.

VIVA 1

This Viva will examine your knowledge of the anatomy and physiology of the coronary circulation.

Q 1. Describe the anatomy of the coronary circulation.

Subsequent questions explored knowledge of factors that affect coronary artery blood flow (including diagrams), and physiology of beta-receptors.

8 (88.9%) of candidates passed this VIVA.

VIVA 2

This Viva will examine elements of the acute inflammatory response and serotonin physiology and pharmacology.

Q 1. Describe the mechanism of the acute inflammatory response to tissue injury.

Subsequent questions explored knowledge of what cytokines are, and what major roles they play in the body, the complement system, apoptosis, immune basis of rejection of allogeneic organs, and serotonin.

6 (66.7%) of candidates passed this VIVA.

VIVA 3

This Viva relates to the respiratory changes of pregnancy at term, hypoxaemia and the foetal circulation.

Q 1. Describe the respiratory changes in pregnancy at term.

Subsequent questions sought a description of ABG at term pregnancy, physiological mechanism to explain an increased A-a gradient, causes of a low PaO2 in a patient with a normal A-a gradient, the response of hypoxemia to supplemental O2 when the A – a gradient is normal and when abnormal, and a description of the foetal circulation and the changes that occur at birth.

8 (88.9%) of candidates passed this VIVA.

VIVA 4

The candidates were shown a diagram of a neuromuscular junction and asked to label the various components.

Subsequent questions sought knowledge of the processes occurring at the prejunctional / junctional / post junctional nerve terminal, to classify neuromuscular blockers, their mechanisms of action, and the pharmacokinetic properties that influence speed of onset and potency.

9 (100%) of candidates passed this VIVA.

VIVA 5

This Viva will examine your knowledge of drugs and drug delivery systems used in the treatment of asthma.

Q 1 Classify drugs used in the treatment of asthma.

Subsequent questions sought knowledge of mechanism of action of beta 2 agonists in the treatment of asthma, important side effects of beta 2 agonists, actions of muscarinic anti-cholinergic antagonists, corticosteroids and aerosol drug delivery systems.

8 (88.9%) of candidates passed this VIVA.

VIVA 6

This viva is about principles of electrophysiology in relation to the heart.

Q 1 Draw the cardiac action potential for a ventricular muscle cell.

Subsequent questions sought knowledge of what is the Resting Membrane Potential and how is it created, to describe ionic mechanism for each of the phases of the ventricular action potential, to draw the SA node action potential, compare and contrast the ventricular and SA node action potentials, and which pharmacological agents affect the different phases of the pacemaker potential.

9 (100%) of candidates passed this VIVA.

VIVA 7

This Viva will test your knowledge of the endocrinology of the thyroid gland.

Q 1 What is a hormone? How is it defined?

Subsequent questions asked about chemical types of hormones, hormones secreted by the anterior pituitary, the principal biological role of the thyroid hormones, production of thyroid hormones, advantages of thyroxine for thyroid replacement in comparison to triiodothyronine, and mechanism of action of propylthiouracil.

9 (100%) of candidates passed this VIVA.

VIVA 8

This Viva will discuss the pharmacology and the physiology related to the pulmonary artery pressure.

Q1. *What is the normal pulmonary artery pressure, and which factors will increase the pulmonary artery pressure?*

Subsequent questions asked about factors that increase pulmonary artery pressure, drugs used to treat pulmonary hypertension and their mechanism of action, advantages of NO over other drugs, how pulmonary artery pressure may be measured at the bed side, how to determine Pulmonary Vascular Resistance.

8 (88.9%) of candidates passed this VIVA.

Summary of the Examination

The CICM Primary Examination explores the knowledge of the basic sciences that forms the basis to Intensive Care practice. A detailed syllabus has been developed and forms the foundation for the knowledge required for this Examination. All questions are sourced directly from that syllabus. Following each examination a detailed report, such as this one, is produced which outlines the level of understanding that is expected.

The Syllabus reflects the basic sciences as they apply to intensive care practice. It is important that Candidates follow the Syllabus closely, and in its entirety. The Primary Examination will be based from within any section of the Syllabus. The level of understanding required to be successful at this Examination, can be ascertained from the past Examination reports, the syllabus, the examination guides to candidates and the suggested texts. To succeed Candidates must read widely, beyond any one textbook, and develop a level of knowledge that allows them to accurately discuss, explain, translate and illustrate essential aspects of the basic sciences. This will require time. Although the amount of time required may vary amongst candidates, as a guide, candidates should plan for approximately 12 months to study, and to prepare, for this Examination.

There are still candidates who are presenting for this examination, very unprepared and with a poor knowledge of core areas. Such candidates will not, and have not, succeeded. Candidates are strongly encouraged to discuss their level of preparedness, and to trial written and oral questions, with their Supervisor of Training and other CICM Fellows, prior to undertaking the CICM Primary Examination. Supervisors of Training are encouraged to regularly monitor their trainee's preparation and test their readiness to sit.

In contrast candidates with a level of understanding that enables them to do sufficiently well in the written section so as to be invited to the Vivas, are very likely to go on and achieve an overall pass in the exam.

On behalf of the Examination Panel, I would like to once again congratulate the successful candidates at this CICM Primary Examination and wish them success in their future training in Intensive Care, and in their future preparation to complete the College of Intensive Care Medicine Fellowship examination.

A/Prof Arthas Flabouris
Chair, Primary Examination Committee
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Circulation:	Board of College	Panel of Examiners
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