



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

# Joint Faculty of Intensive Care Medicine



The Royal Australasian  
College of Physicians

## REPORT OF THE INTENSIVE CARE PRIMARY EXAMINATION

JULY/SEPT 2007

*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 12 short answer questions and 20 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 comprised 8, 10-minute VIVA stations.

### OVERALL STATISTICS

Total number of candidates presenting for the written examination	7 (1 candidate withdrew)
Number of candidates scoring >50% in the written	1
Number of candidates scoring 45-50% in the written	3
Total number invited to the Oral section based on written marks	4

Successful candidate:  
Dr. Corrine Balit

### WRITTEN SECTION

#### Short Answer Paper 1

**Question 1:** Explain how oxygen supply is maintained to the tissues in chronic anaemia.

Candidates were expected to base their answer around the variables involved in the equations that describe oxygen content in blood and oxygen delivery. Although most candidates mentioned changes in haemoglobin that increase oxygen carriage, a more complete discussion of the changes that influence cardiac output and the peripheral circulation was often omitted

**1 candidate (14%) passed this question.**

**Question 2:** Outline the sites and mechanisms of action of diuretics. Give one example of drug acting at each site and list two side effects for each drug.

Good answers to this question were those that had a tabular format to the structure of the answer – for example columns headed mechanism, sites, drug and side effects. Most common omissions were not to further describe how the different mechanisms of action of diuretics increased urine output, e.g. “disruption of the counter current multiplier

system by decreasing absorption of ions from the loop of Henle into the medullary interstitium, thereby decreasing the osmolarity of the medullary interstitial fluid". There was often little mention of increased urine solutes and the effect the electro chemical effect had in promoting a diuresis. Examples of drugs were well done.

**4 candidates (57%) passed this question.**

**Question 3:** Describe the physiological effects and principles of management of a tricyclic antidepressant overdose.

Good answers to this question were those that gave an accurate account of the physiological effects, e.g. inhibition of the fast sodium channels in the His-Purkinje system as well as the atrial and ventricular myocardium, decreasing conduction velocity (differential conduction inhibition of RBB being more susceptible) and increasing duration of repolarization, and the absolute refractory periods. Once having done that the rest of the answer would have flowed more easily, e.g. ECG changes and conduction disturbances. The effects of tricyclic antidepressants on Na channels and as a consequence the cardiovascular conduction abnormalities were often omitted. Anti cholinergic (e.g. slowing GIT motility) and antihistamine effects (e.g. obtundation) were often overlooked. Also frequently overlooked were basic pharmacology relevant to treatment, e.g. lipophilic, large volume of distribution, systemic acidosis reduces the extent of protein binding and increases unbound (active) drug. Additional points were available to those who not only mentioned sodium bicarbonate, but also mentioned the principles behind its use for this circumstance

**1 candidate (14%) passed this question.**

**Question 4:** Describe the Principles of ultrasound imaging – including the Doppler Effect.

It was expected candidates would outline the underlying principles of ultrasound imaging (reflection, scattering, refraction, and attenuation) and discuss that the basic image is the result of reflection of the transmitted ultrasound wave. Most candidates appreciated that the amplitude of the reflected echo is a function of the acoustic mismatch of the tissues and the angle of incidence and many candidates provided details mathematical descriptions concerning these principles.

While high levels of technical details were not required the answer should include a mention of the use a piezoelectric transducer and that an ultrasound beam has 3 dimensions – Axial, Elevation and Lateral. Some comment of the modes of Display (A= Amplitude, M = Time Motion, 2D, etc) was expected.

Extra credit was given for answers that included details regarding limits of depth of penetration (longer wavelength penetrate deeper, but loose image quality with longer wavelengths) and the varying properties of human tissue regarding refraction and attenuation (little refraction (path deviation) in human tissue and air attenuates).

Specific comment on the Doppler Effect was required. It was expected candidates would described that it refers to the change in frequency of a sound wave reflected by a moving target and that the reflected frequency differs if moving toward or away. Correctly stating that the reflected Frequency is Higher Towards and Lower Away scored additional marks. Comments concerning obtaining the best Doppler images with lower frequencies (opposite to ultrasound) and colour Doppler attracted additional marks.

**4 candidates (57%) passed this question.**

**Question 5:** Compare and contrast the spectrum of activity and the mechanisms of microbial resistance for the following penicillins; benzyl penicillin, flucloxacillin and ampicillin.

It was expected candidates would specifically address both the spectrum of activity and mechanism of resistance.

Benzyl penicillin is highly active against Gram positive organisms, particularly streptococci but also effective against Meningococcus / Clostridia and other anaerobes, *Listeria* and is used as treatment for syphilis (*treponema*). It is readily hydrolysed by penicillinases or beta lactamases so any organisms that produce these are resistant ie. most staphylococci.

Flucloxacillin contains a modified beta lactam ring so is not susceptible to hydrolysis by staphylococcal penicillinases, therefore the spectrum is Staphylococci not resistant to methicillin (ie. not MRSA). Extra credit was given for comments that it won't cover *Listeria* or some other organisms covered by Benzyl penicillin and it is much less active than Benzyl penicillin on organisms sensitive to both.

Ampicillin is an alpha amino benzyl penicillin (aminopenicillin) and has a broader activity than Benzyl penicillin so covers the streptococci but also a variety of gram negative bacteria including some enterobacteriaceae and *Haemophilus influenzae*. It also covers *Helicobacter* and Enterococci (better than Benzylpenicillin). It is destroyed by beta- lactamase.

Additional credit was given for discussion of other mechanisms of bacterial drug resistance.

**4 candidates (57%) passed this question.**

**Question 6:** Explain the role of the skin in maintaining body temperature

It was expected candidates would describe that the maintenance of body temperature is a balance of heat loss and heat production and outline the series of controls that are important in this process.

An overview of the reflexes involved was expected with some comments on temperature receptors in skin for hot and cold, the hypothalamic integration and then the effector being skin blood flow (vasodilation / vasoconstriction and A-V shunts).

Answers were expected to include some comment on the fact that heat exchange occurs via radiation / conduction / convection/evaporation and requires a heat gradient and that skin blood changes can facilitate or impair this exchange. Using examples such as; if body needs to lose heat there is vasodilation and sweating often added clarity. Extra credit was given for discussing sweat production and the principle of how that will allow increased heat loss with further credit given for discussing the impact of humidity.

Credit was also given for mentioning piloerection, particularly if candidates went on to explain why this might be useful (even though not really applicable in man).

**4 candidates (57%) passed this question.**

Question 7: Compare and contrast the pharmacology of noradrenaline and vasopressin.

This was best answered using a table.

The main points expected for a pass were

- Both are naturally occurring substances
- Direct acting via receptors
- Mechanisms by which both increase mean arterial pressure
- Metabolism
- Uses in Intensive Care, septic shock, vasodilatory shock and diabetes insipidus.
- Side effects related to intense vasoconstriction and for vasopressin possible coronary ischaemia and sodium and water retention

**5 candidates (71%) passed this question.**

**Question 8:** Explain the role of the baroreceptors in the control of blood pressure

Good answers included the following

- description of, and types of, baroreceptors (e.g. stretch-receptors),
- their locations (e.g. walls of the aorta, carotid sinuses, the atria, etc),
- the stimulus they respond to (e.g. pressure, volume),
- short term and long term responses, alteration to set points, impulse frequency / pressure curve
- A brief description of the afferent and efferent pathways and the resultant efferent effects (e.g. alterations to heart rate, blood pressure, etc).

**5 candidates (71%) passed this question.**

**Question 9:** Classify the hypersensitivity reactions. Briefly describe the pathophysiological processes of each reaction. Give an example of each reaction.

Mechanisms of Immunological Injury		
Mechanism	Pathophysiology	Disease types
Type I Immediate hypersensitivity IgE mediated	<ul style="list-style-type: none"> <li>• basophil &amp; mast cell degranulation</li> <li>• histamine, SRSA, ECFA, NCF</li> <li>• immediate weal &amp; flare</li> </ul>	<ul style="list-style-type: none"> <li>• anaphylaxis</li> <li>• atopy</li> </ul>
Type II cell cytotoxicity IgG, IgM mediated	<ul style="list-style-type: none"> <li>• direct phagocytosis or cell lysis</li> <li>• activation of complement, classical</li> <li>• tissue deposition of complement</li> </ul>	<ul style="list-style-type: none"> <li>• blood transfusions</li> <li>• Goodpasture's syndrome</li> <li>• autoimmune cytopaenias</li> </ul>
Type III Immune complex IgG, IgM, IgA mediated	<ul style="list-style-type: none"> <li>• tissue deposition of Ag-Ab complexes</li> <li>• accumulation of PMN's, macrophages &amp; complement</li> </ul>	<ul style="list-style-type: none"> <li>• SLE</li> <li>• serum sickness</li> <li>• necrotising vasculitis</li> </ul>
Type IV Delayed hypersensitivity T-cell mediated	<ul style="list-style-type: none"> <li>• T-cell induced mononuclear cell accumulation</li> <li>• release of lymphokines &amp; monokines</li> <li>• often with granuloma formation</li> </ul>	<ul style="list-style-type: none"> <li>• TB, sarcoid</li> <li>• Wegener's granulomatosis</li> <li>• granulomatous vasculitis</li> </ul>

Key points:

1. "Anaphylaxis" may be:
  - ◆ **true anaphylaxis:** a symptom complex following exposure of a *sensitised* individual to an antigen, produced by a type I hypersensitivity reaction, associated with IgE mediated mast cell degranulation
  - ◆ **anaphylactoid reactions:** indistinguishable from true anaphylaxis, however the immune nature of the reaction is either unknown, or not due to a type I hypersensitivity reaction, □ **immediate generalised reaction** a better term
    - i End-organ effects, e.g. H<sub>1</sub> and H<sub>2</sub> receptors

**2 candidates (28%) passed this question.**

**Question 10:** Describe the control of gastric emptying.

The main points expected for a pass were

- An appreciation that the aim of controlling gastric emptying is to present the food to the small bowel for absorption in a controlled manner
- That there are both gastric neural and hormonal mechanisms e.g. Gastric volume and the hormone gastrin
- There are duodenal neural and hormonal mechanisms e.g. composition of the chyme, secretin and cholecystokinin and the influence of duodenal distension.
- Extra points were given for mentioning the effect of sympathetic stimulation and pregnancy on gastric emptying

Common problems were not enough knowledge, naming hormones but not saying what their action was and including drugs.

**2 candidates (28%) passed this question.**

**Question 11:** Describe the acid-base changes that occur in acute hypoxaemia

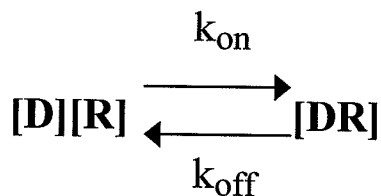
The main points expected for a pass were

- Definition of hypoxaemia
- Physiological causes of hypoxaemia
- Formation of a lactic acidosis
- Development of hypocarbia secondary to stimulation of respiration from hypoxia and acidosis
- Explanation of the decreased bicarbonate concentration and negative base excess
- Extra points were given for more detailed explanation of the lactic acidosis, commenting on the anion gap and some consequences of hypoxaemia.

A common problem was to incorrectly state the relationship between pH and hydrogen ion concentration.

**1 candidate (14%) passed this question.**

**Question 12:** The binding of a drug (D) to a receptor (R) is illustrated by the following expression: –



where [D] and [R] are the concentrations of the drug and receptor, respectively, and k is the rate constant for the particular reaction. Using the equation given, explain the following terms: –

- the ratio of  $k_{\text{off}} / k_{\text{on}}$
- the implications of a low value for the ratio above
- the term affinity
- the clinical implications of a high value for affinity
- two physiological factors that affect the rate constant k

The main points expected for a pass were

- The ratio of  $k_{\text{off}}/k_{\text{on}}$  is the dissociation constant
- A low value indicates that less drug is required to bind to the receptors
- Affinity is the reverse of the dissociation constant
- Clinical application of high affinity include large effect at lower concentrations
- Physiological factors could include temperature

The main problem with this question was lack of knowledge.

**1 candidate (14%) passed this question.**

### Short Answer Paper 2

**Question 1:** Briefly describe the factors that affect lung compliance.

Main points/concepts expected in answer.

- **Surfactant** ▪ increases lung compliance ▪ decreases surface tension at alveolar air - water – interface ▪ prevents small alveoli from collapsing ▪ accounts for most of hysteresis in intact lung
- **Lung elastic recoil** ▪ lung compliance changes in disease states
- **Lung volume** ▪ lung compliance greatest around FRC ▪ lung compliance reduced at low and high lung volumes ▪ gravitational effects on regional lung compliance
- **Pulmonary blood volume** ▪ pulmonary venous congestion reduces lung compliance
- **Lung size** ▪ specific compliance = lung compliance / FRC
- **Dynamic lung compliance** ▪ influenced by airways resistance ▪ lung compliance measured during normal breathing ▪ less than static lung compliance ▪ frequency dependence

**1 candidate (14%) passed this question.**

**Question 2:** Describe the term second messenger. Give an example of a drug that manifests its action via a second messenger.

Second messenger – Hormone/drug - receptor binding is coupled to a subsequent series of intracellular biochemical events that precipitate the ultimate hormone/drug effect.

Examples are G proteins an energy dependent process by which there is hydrolysis of G protein-associated GTP to GDP. There are both stimulating and inhibitory proteins which subsequently act to increase or decrease activity of the enzyme adenylyl cyclase, resulting in increased levels of cyclic adenosine 3',5'-monophosphate (cAMP) in the cell which in turn activates protein kinases that phosphorylate various proteins, ion channels, and second messenger enzymes.

Also G proteins stimulate hydrolysis of phosphatidyl-inositol-4,5-bisphosphate (PIP<sub>2</sub>) generating inositol-1,4,5-trisphosphate (IP<sub>3</sub>) and 1,2-diacylglycerol (DAG). Both systems increase intracellular calcium.

**3 candidates (43%) passed this question.**

**Question 3:** Compare and contrast ibuprofen and tramadol as analgesic agents in intensive care.

Ibuprofen - inhibition of the cyclooxygenase (COX) and synthesis of prostaglandins, which are important mediators for peripheral sensitization and hyperalgesia. Act peripherally and spinal COX – non selective. Oral and PR only Associated with a number of side effects, including decreased haemostasis, renal dysfunction, gastrointestinal haemorrhage, and effects on bone healing and osteogenesis

Tramadol - is a synthetic opioid that exhibits weak  $\mu$ -agonist activity and inhibits reuptake of serotonin and noradrenaline. Analgesic effects primarily through central mechanisms, it may exhibit peripheral local anaesthetic properties.

Tramadol is comparable in analgesic efficacy to ibuprofen. Common side effects (overall incidence of 1.6% to 6.1%) include dizziness, drowsiness, sweating, nausea, vomiting, dry mouth, and headache. Tramadol should be used with caution in patients with seizures or increased intracranial pressure and in those taking monoamine oxidase inhibitors. IV and oral preparations. No bleeding, GIT and renal complications. More expensive

Both have advantage of lack of respiratory depression, major organ toxicity, and depression of gastrointestinal motility and a low potential for abuse.

**3 candidates (43%) passed this question.**

**Question 4:** Describe the physiological response to hypoglycaemia.

To pass this question, the candidate only needed to state the range of normal blood glucose, define hypoglycaemia then give an overview of the body's response to hypoglycaemia (control of blood glucose – sensors, integrators, effectors). Additional credit was given for a more detailed description of the various responses. Few candidates mentioned the role of the sympathetic nervous system. Much time was wasted in giving detailed descriptions of metabolic pathways to the exclusion of an overview of the body's responses.

**4 candidates (57%) passed this question.**

**Question 5:** List the potential clinical uses of an alpha 2 adrenoceptor agonist. Outline the limitations of clonidine for each use.

Producing a list that included the following was required:

To treat hypertension and substance withdrawal.

To provide anxiolysis, sedation, analgesia, and sympatholysis.

A brief discussion of the abilities of clonidine in each of these areas would have rounded off a good answer.

Many candidates listed only a couple of uses of these agents, and then followed this with a comparison of clonidine and dexmedetomidine. Most answers did not include sufficient information to achieve a pass mark.

**2 candidates (29%) passed this question.**

**Question 6:** Describe the autoregulation of renal blood flow.

To achieve a pass in this question, candidates needed to briefly define autoregulation and state the range of Mean Arterial Pressure over which this occurs, and why it occurs, then provide a more detailed discussion about the mechanisms thought to be responsible for this.

The main site of autoregulation in the kidney is the afferent arteriole. There are two main factors that affect vascular tone in the afferent arteriole, these are stretch-activated constriction of vessels (myogenic mechanism) and tubulo-glomerular feedback (TGF).

Both of the above mechanisms are important to maintenance of near-constant blood flow. Stretch results in membrane depolarisation, increased intra-cellular concentrations of calcium ions, and ultimately, vasoconstriction.

In tubulo-glomerular feedback, complex signals pass from the macula densa to the afferent arteriole, regulating its tone. The fundamental theme of TGF is that increased delivery of fluid and/or NaCl to the distal tubule causes vasoconstriction, thus limiting the flow (negative feedback).

The major weakness in answers was again the failure to include sufficient information to achieve a pass mark

**3 candidates (43%) passed this question.**

**Question 7:** Describe the effects of a tachycardia on myocardial oxygen supply and demand in a normal heart.

The main points expected were the determinants of myocardial oxygen supply. These include arterial oxygen content and coronary blood flow. Coronary blood flow depends on coronary perfusion pressure and coronary vascular resistance and that most left coronary blood flow occurs in diastole. Tachycardia reduces diastolic time and hence left coronary blood flow. In comparison blood flow in the right coronary artery is continuous both in systole and diastole and is little affected by heart rate. A correctly labelled diagram of left and right coronary blood flow attracted extra marks. Unfortunately most diagrams were inaccurate, not labelled and had no units on the axes. Systolic compression particularly reduces blood supply to the left ventricular subendocardium which is most susceptible to ischaemia. Extra marks were given for describing metabolic autoregulation, the high oxygen extraction, explaining that oxygen supply cannot be increased by increasing oxygen extraction in the coronary circulation and describing the driving pressure differences in both coronary arteries in systole and diastole.



A description of the determinants of myocardial oxygen demand was also required (e.g. left ventricular, preload, contractility, afterload and tachycardia). This part of the question was particularly poorly answered.

**2 candidates (29%) passed this question.**

**Question 8:** Describe the determinants of serum potassium. Outline the consequences of acute hyperkalaemia.

Most candidates did not appreciate that serum potassium is a function of two variables:

1. Total body potassium
2. Distribution between the extracellular and intracellular fluid compartments.

Approximately 98% of total body potassium is intracellular due to the action of Na<sup>+</sup>/K<sup>+</sup> ATPase. Potassium is important in the electrophysiology of excitable cells and changes in serum potassium can affect their function. Hence the importance of keeping the serum potassium within a narrow normal range.

Again most candidates did not provide the overview that serum potassium levels reflect a balance between intake, output and transcellular distribution. Normal dietary intake is highly variable. Transcellular distribution by the mechanisms of insulin and glucagon, catecholamine's and  $\beta_2$  activity and acid base changes all work to rapidly restore changes in serum potassium levels back towards the normal range. Many candidates did not provide any details on the long term renal regulation of serum potassium involving distal tubule potassium secretion and aldosterone and also the effect of distal tubular flow and sodium excretion.

The effects of hyperkalaemia were better described than the first part of the question. Most candidates concentrated on the cardiac effects where most marks were awarded. The effects of an increased potassium on the cardiac action potential earned extra marks. The correlation between actual serum potassium level and ECG changes is variable and depends on many factors including how acute or chronic the hyperkalaemia is.

Treatment of hyperkalaemia was mentioned by a few candidates but attracted no extra marks.

**3 candidates (43%) passed this question.**

**Question 9:** Compare and contrast the cardiovascular effects of an induction dose of propofol and ketamine.

The key words in this question were "compare and contrast", "cardiovascular effects" and "induction dose". Some candidates described aspects of both drugs other than the cardiovascular effects but gained no marks for this. Better answers used a combination of a table plus some explanation to contrast the cardiovascular effects of the two drugs concentrating on aspects such as heart rate, cardiac output, vascular resistance and blood pressure.

Many candidates were confused by the direct versus the indirect cardiovascular effects of both drugs.

Propofol probably has no direct negative inotropic effect. Ketamine has a direct myocardial depressant action but this effect is overridden by the centrally mediated sympathetic action of the drug. The effect of both drugs on the baroreceptor response alone is a difficult area as there is significant interplay between the direct cardiovascular effects of the drugs and

their effect on the baroreceptor reflex. Allowance was made for this in the marking. Propofol resets the baroreceptor reflex producing a slower heart rate for a given level of blood pressure. Overall both drugs depress the baroreceptor reflex.

Comparison of the effects on cerebral, coronary, renal and hepatic blood flow earned extra marks.

**2 candidates (29%) passed this question**

**Question 10:** Describe the formation, flow and absorption of cerebrospinal fluid

The main points expected for a pass were

- CSF is formed by ultra filtration and secretion
- CSF volumes and turnover
- Flow through the ventricles and subarachnoid spaces
- Absorption through the arachnoid villi
- Relationship between absorption and pressure

This is not a question about intracranial pressure so no points were given for Munroe Kellie doctrine etc, Also no points were given for the functions of CSF. Diagrams need to have the axes labelled correctly.

**2 candidates (29%) passed this question**

**Question 11:** Describe the mechanisms of action of drug groups commonly used to treat acute severe asthma.

Common drugs listed were

- Beta 2 agonists salbutamol and adrenaline
- Steroids
- Magnesium
- Phosphodiesterase inhibitors

In order to obtain marks for that class of drug the mechanism of action had to be described e.g. for theophylline; acts as a bronchodilator by inhibiting the breakdown of cyclic AMP and cyclic GMP.

There were several excellent answers to this question.

**3 candidates (43%) passed this question.**

**Question 12:** Explain the causes of the differences between measured end tidal and arterial partial pressures of carbon dioxide.

The main points for a pass were

Patient factors:

- Normal value for the difference between end tidal and arterial CO<sub>2</sub>
- Physiological factors e.g. alveolar dead space, failure to plateau of the capnograph trace
- Pathological factors e.g. cardiac arrest, air embolism, asthma.

Equipment factors:

- Leaks
- Occlusion
- Sampling site errors etc

A common mistake was to state that dead space caused the difference between end tidal and arterial carbon dioxide but not which type of dead space. The Bohr equation was not required.

**0 candidates passed this question.**

### **ORAL SECTION**

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

#### **Viva 1**

#### **Heparin as Prophylaxis against DVT after Acute Stroke**

	<b>Heparin Treatment</b> N= 16	<b>No Treatment</b> n= 16
<b><u>Baseline Variables</u></b>		
Age (yrs) #	78.9 ( $\pm$ 8.0)	78.2 ( $\pm$ 7.4)
Stroke Severity Score#	3.4(1.8)	4.3(1.8)
Female	10	11
<b><u>Outcome Variables</u></b>		
Positive DVT Leg Scan	2	12
Mortality	3	5
# mean (SD)		

From this table, what types of data are represented?

**1 candidate (25%) passed this question**

#### **Viva 2**

A 30 year old brittle asthmatic presents to the emergency department in extremis and requires immediate intubation to facilitate ventilation.

In this station you will be asked to review the features of the neuromuscular junction and the characteristics of the neuromuscular blocking drug that you might choose to maintain paralysis in this patient.

**2 candidates (50%) passed this question**

**Viva 3**

Please explain the physiological basis of the electrocardiograph.

**All 4 candidates (100%) passed this question**

**Viva 4**

Describe the physiology of bilirubin production and clearance.

**All 4 candidates (100%) passed this question**

**Viva 5**

How would you measure gas flow?

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

**Viva 6**

A 55 year old male presents with cardiogenic shock.

BP = 90/60 mmHg

CI = 1.8 L/min/m<sup>2</sup>

SVR = 1500 dyne.sec.cm-5

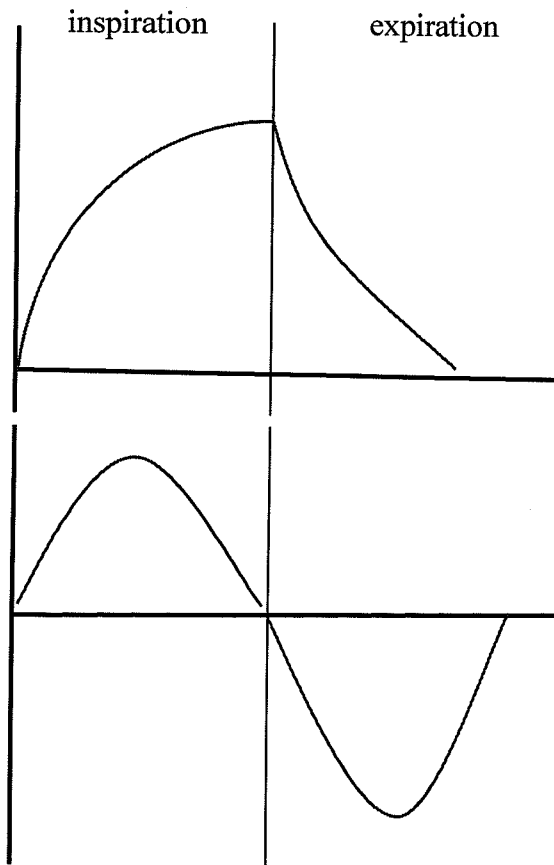
What drugs would you use to increase blood pressure in this situation?

**1 candidate (25%) passed this question**

**Viva 7**

What are the causes of a low P<sub>a</sub>O<sub>2</sub>?

**2 candidates (50%) passed this question**

**Viva 8**

These are graphical representations taken from a ventilator that is connected to a paralysed, ventilated 23 year old male. (Unconscious from an overdose).

What are the axis of the 2 graphs?

**3 candidates (75%) passed this question.**

**This was the first JFICM Primary examination. A detailed syllabus has been developed and forms the foundation for the knowledge base for the JFICM. All questions are sourced directly from that syllabus and candidates should have a sound understanding, and confidence to express their understanding, of the subject material in both written and oral form.**

**Dr. Gillian Bishop**  
Chair

**Dr. Arthas Flabouris**  
Deputy Chair

**Primary Examination**

Circulation:

Board of joint faculty  
Supervisors of Intensive Care Training  
Regional Education Offices

Panel of Examiners  
Course Supervisors  
Registered Trainees