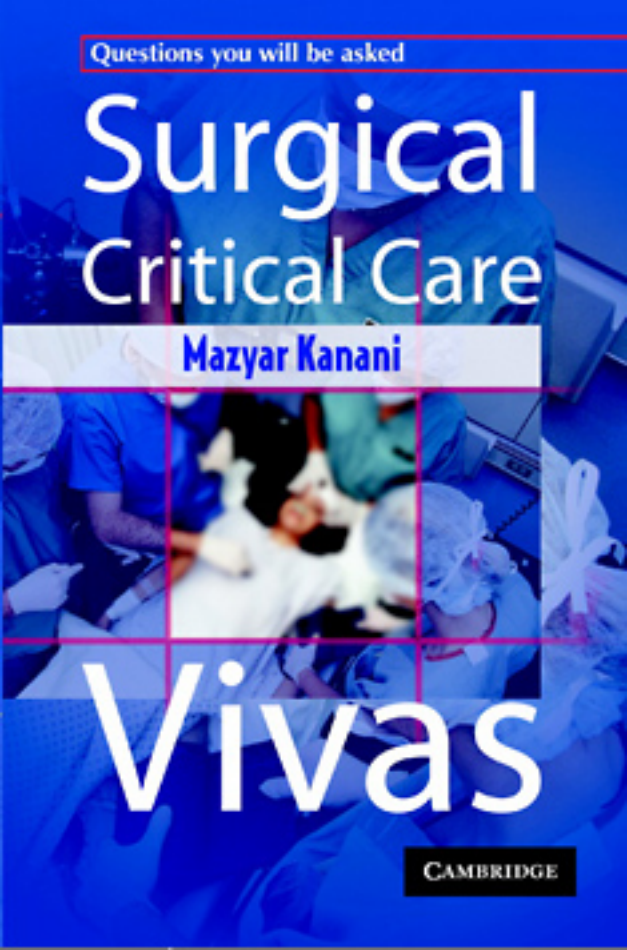
Questions you will be asked

Surgical Critical Care Vivas

Mazyar Kanani



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For my wife, Pauline Cornelia O'Keeffe

Surgical Critical Care Vivas

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CONTENTS

LIST OF ABBREVIATIONS

ACTH	Adrenocorticotropic hormone
ADH	Anti diuretic hormone
ADP	Adenosine diphosphate
ALI	Acute lung injury
AMP	Adenosine monophosphate
APTT	Activated partial thromboplastin time
ARDS	Acute respiratory distress syndrome
ATLS	Advance trauma life support
ATN	Acute tubular necrosis
ATP	Adenosine triphosphate
ATPase	Adenosine triphosphatase
AV	Atrioventricular
BBB	Blood-brain barrier
2,3 BPG	2,3 Bisphosphoglycerate
CAPD	Citrate, Adenine, Phosphate, and Dextrose
cGMP	Cyclic guanosine monophosphate
CMV	Cytomegalovirus
CO	Cardiac output
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CSF	Cerebrospinal fluid
CVP	Central venous pressure
CXR	Chest radiograph
DIC	Disseminated Intravascular Coagulation
DKA	Diabetic ketoacidosis
DPL	Diagnostic peritoneal lavage
DVT	Deep venous thrombosis
ECF	Extracellular fluid
ECG	Electrocardiogram
ELISA	Enzyme linked immunosorbent assay
ESR	Erythrocyte sedimentation rate
FFA	Free fatty acids
FFP	Fresh frozen plasma
FiO_2	Fraction of inspired oxygen

FRC	Functional residual capacity
GCS	Glassow coma score
GFR	Glomerular filtration rate
HITS	Heparin-induced thrombocytopenia syndrome
HIV	Human immunodeficiency virus
HLA	Human leucocyte antigen
HMSO	Her Majesty's Stationery Office
HRT	Hormone replacement therapy
I:E RATIO	Inspiratory:Expiratory ratio
ICF	Intracellular fluid
ICP	Intracranial pressure
IgA	Immunoglobulin A
IL	Interleukin
IMV	Intermittent mandatory ventilation
INR	International normalised ratio
IPPV	Intermittent positive pressure ventilation
ITU	Intensive therapy unit
JVP	Jugular venous pulse/pressure
MAP	Mean arterial pressure
MI	Myocardial infarction
MODS	Multi-Organ dysfunction syndrome
MPAP	Mean pulmonary artery pressure
MRI	Magnetic resonance imaging
MRSA	Methicillin resistant *Staph. aureus*
NG	Nasogastric
NJ	Nasojejunal
NSAIDs	Non-steroidal anti-inflammatory drugs
PA	Pulmonary artery
PAF	Platelet activating factor
PAOP	Pulmonary artery occlusion pressure
PCA	Patient-controlled analgesia
PCC	Prothrombin complex concentrate
PE	Pulmonary embolus
PEEP	Positive end-expiratory pressure
PSV	Pressure support ventilation
PTH	Parathormone
PVR	Pulmonary vascular resistance

RAA	Renin-angiotensin-aldosterone
SAMG	Saline, Adenine, Mannitol, and Glucose
SaO_2	Arterial oxygen saturation
SIADH	Syndrome of inappropriate ADH
SIMV	Synchronised intermittent mandatory ventilation
SIRS	Systemic inflammatory response syndrome
SLE	Systemic lupus erythmatosus
SVC	Superior caval vein
SvO_2	Mixed venous oxygen saturation
SVR	Systemic vascular resistance
SVT	Supra-ventricular tacycardia
TB	Tuberculosis
TNF	Tumour necrosis factor
TPN	Parenteral nutrition
TT	Thrombin time
TURP	Trans-urethral resection of the prostate
V/Q RATIO	Ventilation/perfusion ratio
VA	Alveolar ventilation
VSD	Ventricular septal defect

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ABDOMINAL TRAUMA: INVESTIGATIONS

What are the two major types of abdominal trauma?

The two types of injury are blunt and penetrating. The abdomen may be considered as being composed of five parts:

- *Abdominal wall:* front and back
- *Subcostal portion:* containing the stomach, liver, spleen and lesser sac
- *Pelvic portion:* containing the rectum, internal genitalia and iliac vessels
- *Intraperitoneal portion* in between the above: containing the small and large bowel
- *Retroperitoneum:* containing the kidneys, urinary tract, great vessels, pancreas and the rest of the colon

Which abdominal organs are most commonly injured?

The three most commonly injured organs are the liver, spleen and kidneys.

How may suspected injuries be investigated?

The initial investigations performed to assess the abdomen as a whole are

- *Plain radiography:* also assesses the bony pelvis
- *Ultrasound:* particularly good for the presence of free fluid in the abdomen, or haematoma around solid organs. There is a 10% risk of missing a significant injury
- *Diagnostic peritoneal lavage (DPL):* this is 98% sensitive for intra-peritoneal bleeding
- *CT scanning:* this can be used if the results of the DPL are equivocal, and may also be performed at the same time as a brain scan. Very good for retroperitoneal injury, less so for hollow viscus injury such as the bowel

Under which circumstances would you perform a diagnostic peritoneal lavage (DPL)?

Some of the indications are

- A suspicion of abdominal trauma on clinical examination
- Unexplained hypotension: with the abdomen being the source of occult haemorrhage
- Equivocal abdominal examination because of head injury and reduced level of consciousness
- The presence of a wound that has traversed the abdominal wall, but there is no indication for immediate laparotomy, e.g. a stab wound in a stable patient

When is DPL contraindicated?

The most important contraindication for DPL is in the situation which calls for mandatory laparotomy, e.g. frank peritonitis following trauma, abdominal gunshot injury or a hypotensive patient with abdominal distension.

How is DPL most commonly performed?

Performance of a DPL by the open method

- Requires an aseptic technique
- The abdomen is decompressed by insertion of a urinary catheter and nasogastric tube
- Local anaesthetic is administered to the subumbilical area in the mid-line
- An incision is made over this point. If a pelvic fracture is suspected, then a supraumbilical incision is made to prevent haematoma disruption
- Dissection is performed down to the peritoneum and the cannula is inserted under direct vision, guiding it towards the pelvis
- One litre of warmed saline is infused. Tilting and gently rolling the patient helps distribution
- The bag of saline can be left on the floor to siphon off the sample fluid from the abdomen

What are the positive criteria with DPL?

- Lavage fluid appears in the chest drain or urinary catheter
- Frank blood on entering the abdomen
- Presence of bile or faeces
- Red cell count of >100,000/μl
- White cell count of >500/μl
- Amylase of >175 U/ml

A

ACCESSING THE THORAX

In which major ways may the thorax be accessed?

- Percutaneous methods
  - *Needle thoracostomy:* to drain fluid, air or for biopsy of tissue
  - *Tube thoracostomy ('chest drain'):* for drainage of air or fluid
  - *Thoracoscopic surgery:* permits procedures such as lung/pleural biopsy, lobectomy, pleurodesis, pleurectomy, sympathectomy, pericardiocentesis and pericardial window
- Thoracotomy
  - *Median sternotomy:* from the top of the manubrium at the jugular notch, passing longitudinally through the sternum to the xiphisternum. It permits access to the pericardium, great vessels, and both hemithoraces
  - *Posterolateral thoracotomy:* the most common approach in thoracic surgery. The incision runs from a point mid-way between the medial scapular edge and the thoracic spine, following a curve that runs 2 cm below the inferior scapular angle, to the mid-point of the axilla
  - *Anterior thoracotomy:* from the sternal edge, curving laterally along the intercostal space below the nipple to the axilla. It allows lung, pericardial and lung access, and also to lymph nodes in the aorto-pulmonary window
  - *Posterior thoracotomy:* the line of the incision is similar to that of a posterolateral thoracotomy, but starts at a more posterior point, encroaching on to the trapezius and erector spinae muscles. It allows access to the lung and great vessels for some paediatric cardiac procedures
  - *Bilateral anterior sternotomy ('clamshell' incision):* this incision runs from below one nipple to the contralateral side, dividing the body of the sternum in-between. It permits emergency access to the

pericardium and simultaneous exposure of both pleural cavities

- *Thoraco-laparotomy:* the incision runs like that of a posterolateral thoracotomy, but continues anteriorly to cross the costal margin at the junction of the sixth and seventh ribs. The line runs for another 5 cm into the abdominal wall. It is extended inferiorly as a para-median or mid-line laparotomy. It permits access to posterior mediastinal structures, such as the aorta or oesophagus as they run into the abdomen

- Mediastinoscopy: the incision runs across the anterior neck, two fingers-breadth above the jugular notch. Allows access to the sub-carinal lymph nodes for disease diagnosis and staging

Which important piece of anaesthetic equipment is required for thoracotomy, and why?

The double-lumen endobronchial tube. This permits the use of one-lung anaesthesia where one lung may be collapsed and inflated at will for the purposes of surgery. This is particularly important for thoracoscopy where one lung has to be collapsed to permit the safe passage of the instruments through the thoracic wall.

What is the important pre-requisite to closure of all thoracotomies?

Chest drain insertion. Post cardiac surgery, one or two drains may be inserted into the mediastinum/posterior pericardium, exiting through the skin subcostally. Other drains are placed into any opened pleural space, e.g. during internal mammary artery harvest. After thoracotomy, one apical and one basal chest drain may be placed, both exiting sub-costally.

Briefly mention some important local complications of thoracotomy.

Wound complications

- Early:
  - Immediate dehiscence from poor technique

  - Haematoma formation
  - Poor pain control leading to atelectasis, retention of secretions, hypoxia and infection
- Intermediate:
  - Infection, leading to wound dehiscence
- Late:
  - Post-thoracotomy neuralgia

Pulmonary complications

- Early:
  - *Air leak:* seen as continuous bubbling from the drains when placed on suction. May be due to parenchymal injury or a leak from the suture-line of a bronchial stump
  - *Bleeding:* producing haemothorax. May be from the raw parenchymal surface, or from a larger vessel
- Intermediate:
  - *Pneumonia:* can lead to a lung abscess
  - *Pulmonary oedema:* seen particularly in the contralateral lung following pneumonectomy. May also occur following re-expansion of a chronically collapsed or compressed lung from effusion
- Late:
  - Chronic broncho-pleural fistula
  - Empyema

ACID-BASE

Define the pH.
The pH is $-\log_{10}[H^+]$.

What is the pH of blood?
7.36–7.44.

Where does the acid load (H^+) in the body come from?
Most of the H^+ in the body comes from CO_2 generated from metabolism. This enters solution, forming carbonic acid through a reaction mediated by the enzyme carbonic anhydrase.

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

Acid is also generated by

- Metabolism of the sulphur-containing amino acids cysteine and methionine
- Anaerobic metabolism, generating lactic acid
- Generation of the ketone bodies acetone, acetoacetate and β-hydroxybutyrate

What are the main buffer systems in the intravascular, interstitial and intracellular compartments?
In the plasma the main systems are

- The bicarbonate system
- The phosphate system ($HPO_4^{2-} + H^+ \rightleftharpoons H_2PO_4^-$)
- Plasma proteins
- Globin component of haemoglobin

Interstitial: the bicarbonate system

Intracellular: cytoplasmic proteins

What does the Henderson–Hasselbalch equation describe, and how is it derived?
This equation, which may be applied to any buffer system, defines the relationship between dissociated and undissociated

acids and bases. It is used mainly to describe the equilibrium of the bicarbonate system.

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

The dissociation constant,

$$K = \frac{[H^+][HCO_3^-]}{[H_2CO_3]}$$

Therefore

$$[H^+] = K\frac{[H_2CO_3]}{[HCO_3^-]}$$

Taking the log

$$\log[H^+] = \log K + \log\frac{[H_2CO_3]}{[HCO_3^-]}$$

Taking the negative log, which expresses the pH, and where $-\log K$ is the pK

$$pH = pK - \log\frac{[H_2CO_3]}{[HCO_3^-]}$$

Invert the term to remove the minus sign

$$pH = pK + \log\frac{[HCO_3^-]}{[H_2CO_3]}$$

The $[H_2CO_3]$ may be expressed as $pCO_2 \times 0.23$, where 0.23 is the solubility coefficient of CO_2 (when the pCO_2 is in kPa).

The pK is equal to 6.1.

Thus,

$$pH = 6.1 + \log\frac{[HCO_3^-]}{pCO_2 \times 0.23}.$$

Which organ systems are involved in regulating acid-base balance?

The main organ systems involved in regulating acid-base balance are

- *Respiratory system:* this controls the pCO_2 through alterations in alveolar ventilation. Carbon dioxide indirectly stimulates central chemoceptors (found in the ventro-lateral surface of the medulla oblongata) through H^+ released when it crosses the blood-brain barrier (BBB) and dissolves in the cerebrospinal fluid (CSF)
- *Kidney:* this controls the $[HCO_3^-]$, and is important for long term control and compensation of acid-base disturbances
- *Blood:* through buffering by plasma proteins and haemoglobin
- *Bone:* H^+ may exchange with cations from bone mineral. There is also carbonate in bone that can be used to support plasma HCO_3^- levels
- *Liver:* this may generate HCO_3^- and NH_4^+ (ammonia) by glutamine metabolism. In the kidney tubules, ammonia excretion generates more bicarbonate

How does the kidney absorb bicarbonate?

There are three main methods by which the kidneys increase the plasma bicarbonate

- Replacement of filtered bicarbonate with bicarbonate that is generated in the tubular cells
- Replacement of filtered phosphate with bicarbonate that is generated in the tubular cells
- By generation of 'new' bicarbonate from glutamine that is absorbed by the tubular cell

Define the base deficit.

The base deficit is the amount of acid or alkali required to restore 1 l of blood to a normal pH at a pCO_2 of 5.3 kPa and at 37°C. It is an indicator of the metabolic component to an acid-base disturbance. The normal range is −2 to +2 mmol/l.

ACUTE RENAL FAILURE

What is the definition of acute renal failure?

This is the inability of the kidney to excrete the nitrogenous and other waste products of metabolism and can develop over the course of a few hours or days. It is therefore a biochemical diagnosis

How are the causes basically classified?

The causes may be considered to be pre-renal, renal or post-renal.

What are the major 'renal' causes of acute renal failure?

- Acute tubular necrosis
- Glomerulonephritis
- Interstitial nephritis
- Bilateral cortical necrosis
- Reno-vascular: vasculitis, renal artery thrombosis
- Hepatorenal syndrome

What is acute tubular necrosis?

Acute tubular necrosis is renal failure resulting from injury to the tubular epithelial cells, and is the most important cause of acute renal failure. There are two types

- *Ischaemic injury:* following any cause of shock with resulting fall in the renal perfusion pressure and oxygenation
- *Nephrotoxic injury:* from drugs (aminoglycosides, paracetamol), toxins (heavy metals, organic solvents), or myoglobin (from rhabdomyolysis)

What are the major 'post-renal' causes?

- Acute obstruction from calculi
- Obstruction from tumours arising from the renal parenchyma or transitional epithelium of the pelvi-calyceal system
- Extrinsic compression from pelvic tumours
- Iatrogenic injury, e.g. inadvertent damage to the ureters during bowel surgery
- Prostatic obstruction
- Increased intra-abdominal pressure (>30 cmH_2O)

Which part of the kidney is the most poorly perfused?

The renal medulla is more poorly perfused than the cortex. This ensures that the medullary interstitial concentration gradient formed by tubular counter current multiplication is preserved and maintained.

Which part of the nephron is the most susceptible to ischaemic injury, and why?

The cells of the thick ascending limb are the most susceptible to ischaemic injury for two important reasons

- The cells reside in the medulla, which has poorer oxygenation than the cortex
- The active Na^+-K^+ ATPase pumps at the cell membrane have a high oxygen demand

What are the basic steps in the pathogenesis of acute renal failure?

The basic steps in the pathogenesis are

- Initially, there is renal parenchymal ischaemia: as part of the compensatory response to a fall in the renal perfusion pressure, there is vasoconstriction of the efferent arteriole. Thus, by reducing the pre to post capillary resistance ratio, the capillary filtration pressure is preserved at the expense of reducing the blood supply to the tubules from the efferent arteriole and vasa recta. This leads to worsening cortical and medullary ischaemia