"List" = 1-3 words
"State"= short statement/ phrase/ clause

UNIVERSITY HOSPITAL, GEELONG
FELLOWSHIP WRITTEN EXAMINATION
WEEK 13– TRIAL SHORT ANSWER QUESTIONS Suggested answers

PLEASE LET TOM KNOW OF ANY ERRORS/ OTHER OPTIONS FOR ANSWERS

Please do not simply change this document - it is not the master copy !

Question 1 (18 marks)

a. What is the role of serum procalcitonin levels in the diagnosis of meningitis? List three (3) points. (3 marks)

- **Bacterospecific marker**
- **Rises early (<4/24) following an endotoxin challenge**
- **Useful in paediatric? meningitis**
- **Differentiate between ? viral vs bacterial**
- **Consensus yet to be reached on Dx value**
  - **Sensitivities > 99% in small studies**

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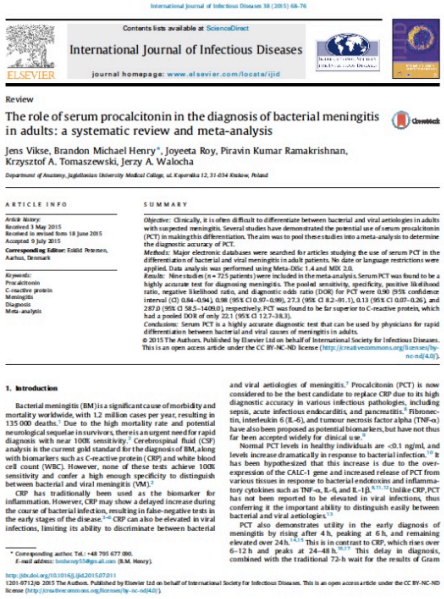

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Review

The role of serum procalcitonin in the diagnosis of bacterial meningitis in adults: a systematic review and meta-analysis

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ARTICLE INFO

SUMMARY

1. Introduction

A 25 year old presents with a severe headache.

b. Complete the reference table below regarding expected CSF findings. (10 marks)

	Normal	Bacterial meningitis	Viral meningitis	Fungal (eg Cryptococcal)	Sub arachnoid Haemorrhage
Opening pressure	**50- 200 mmH20**	**↑**	**↑**	**↑**	**↑**
Colour	**Clear**	**Turbid**	**Turbid**	**Turbid/ clear**	**Xanthochromia**
WCC	**0- 5**	**> 1000** **> 500 PMN**	**100-1000** **Lymphocyte predominance**	**0-200** **(Lower in HIV 0-50)**	**1:500** **WBC:RCC**
RBC	**0- 5**	**0-5**	**0.5**	**0-5**	**>1000 (usually > 10,000)**
Protein	**0.2- 0.5**	**↑**	**Normal**	**↑**	**↑**
CSF Glucose	**60-80% serum**	**↓ < 60%**	**Normal**	**↓ < 60%**	**Normal**

c. List five (5) contraindications to performing a lumbar puncture prior to a CT Brain in the setting of suspected meningitis. (5 marks)

- **Abnormal conscious state**
- **Focal neurological deficit**
- **Signs of raised ICP eg papilloedema**
- **Immunocompromise**
- **Seizure in preceding 1 week**

Question 2 (12 marks)

A 2 year old girl presents with a suspected febrile convulsion.

a. List six (6) criteria that must be met for the patient to be safely discharged. (6 marks)

Must be a "simple seizure"

- **Febrile**
- **< 10 min**
- **Tonic clonic seizure (ie not focal)**
- **Focus identified**
- **Normal conscious state after post ictal period**
  +
- **Adequate social environment/ parental understanding**

b. List six (6) pieces of advice that you would give to the parent on how to deal with a possible future convulsion. Include three (3) indications to call an ambulance. (6 marks)

- **The most important thing is to stay calm - don't panic**
- **Time how long the convulsion lasts**
- **Place your child on a soft surface, lying on his or her side or back**
- **Do not put anything in their mouth, including your fingers. Your child will not choke or swallow their tongue**
- **Try to watch exactly what happens, so that you can describe it to the doctor later**

(Do not put a child who is having a convulsion in the bath)
(Do not restrain your child)

Three (3) indications to call an ambulance:

- **Convulsion lasts more than five minutes**
- **Your child does not wake up when the convulsion stops**
- **If your child looks very sick when the convulsion stops**

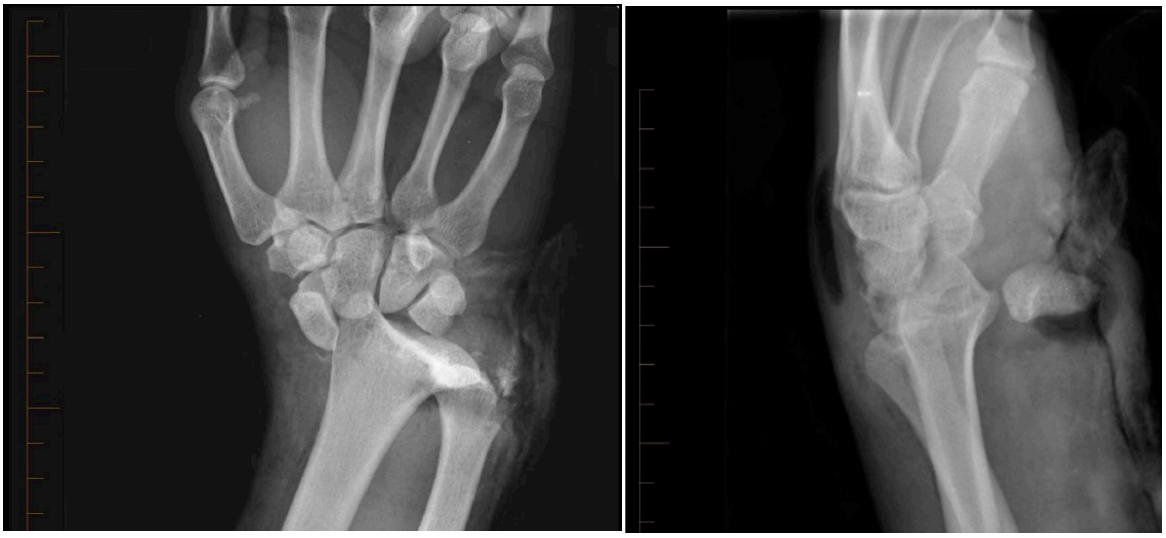
Additional Q:

Q: List three (3) risk factors for recurrence of febrile convulsions in an individual. (3 marks)

- **Onset < 1 yr of age**
- **Repetitive seizures**
- **Focal features**
- **Brief duration between fever onset and seizure**
- **FHx FC**

Question 3 (12 marks)

A 23 year old man presents following a fall onto his outstretched right hand from a height of three metres.



a. State four (4) abnormal findings shown in these xrays. (4 marks)
   - **Lunate dislocation**
   - **Dislocation of the carpus (proximal row)**
   - **Distal ulnar styloid #**
   - **Marked soft tissue swelling**
   - **Air in soft tissue suggests open injury**

b. List four (4) complications of this injury in the first week following injury. (4 marks)
   - **Median n compression**
   - **Radial/ ulnar artery injury- ischaemic digits**
   - **Compartment syndrome**
   - **Infection**
   - **POP complications**
   - **Post op complications (post anaesthetic)**

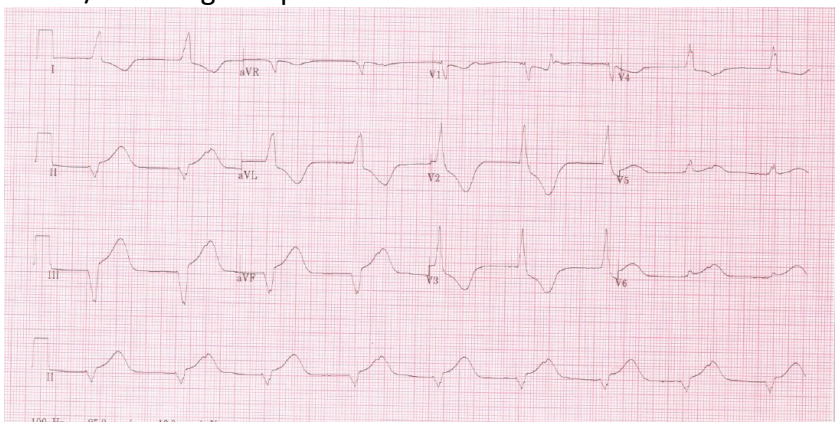
A manipulation is to be performed in the emergency department.

c. List two (2) sedative/ analgesic options to facilitate this manipulation. Define the drugs and doses that you would use. He is a 70kg male. (4 marks)

Sedative /analgesic option	Drug/ dose
Deep sedation	**Propofol 0.5-1 mg/kg** (provided no sig. amount opioids already and fasted) **Ketamine 1-3 mg/kg**
GA	**Propofol 2-3 mg/kg**
LAMP	**Prilocaine 0.5% 0.5ml / kg**
Interscalene n block	**Bupivocaine 0.5% maximum dose 2mg/kg**

Question 4 (12 marks)

A 54 year man presents with chest pain. An initial ECG reveals an inferior STEMI. Fifteen minutes after receiving intravenous thrombolysis a further ECG is taken.
His observations are BP150/80mmHg Temperature 36°C nO2 saturation 98%on room air



a. State five (5) abnormal findings shown in this ECG. (5 marks)
   - **Ventricular/ idioventricular escape rhythm rate 54**
   - **No p waves**
   - **LAD**
   - **Qs II, III, aVF**
   - **STD- V2 3mm, V3 3mm, V4 1mm, and high lateral leads: I 1mm, aVL 2mm**
   - **STE- 2mm II, III, aVf, 1mm V5-6**
   - **TWI I, aVL, V2-V4**

Junctional are QRS < 120 msec

Junctional bradycardia	≤ 40 bpm.
Junctional escape rhythm	= 40-60 bpm.
Accelerated junctional rhythm	= 60-100 bpm.
Junctional tachycardia	≥ 100 bpm.

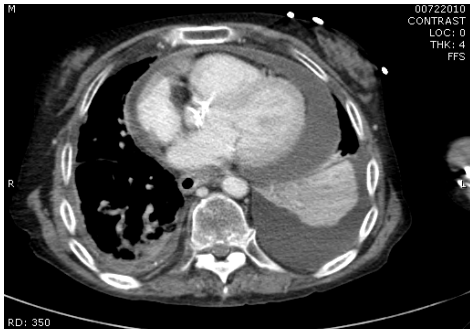
b. What is the significance of this ECG? State three (3) points of significance. (3 marks)
   - **Rhythm:**
     - **Usually well tolerated/ benign**
     - **Usually self limited**
     - **Marker of reperfusion - "reperfusion arrhythmia"**
     - **May indicate further likelihood of needing rescue PCI**
     - **May imply imminent significant bradycardia**
   - **Widespread STE and deep STD V2-V3:**
     - **Marker of extensive myocardial damage**
   - **Inferior q waves- marker of completed infarct**

10 minutes after this ECG is taken, his blood pressure drops to 60 mmHg.

c. List four (4) likely causes for this change in blood pressure. (4 marks)
   - **CHB/ bradycardia**
   - **Cardiogenic shock- RV infarct**
   - **Anaphylaxis to thrombolysis**
   - **Bleeding from thrombolysis- major site**
   - **Bleeding from thrombolysis - Pericardial tamponade**
   - **VT**

Question 5 (12 marks)

A 59 year old man presented following a motor vehicle accident via ambulance to your regional emergency department.



a. State four (4) abnormal findings shown in his CT. (4 marks)
   - **Moderate pericardial effusion**
   - **Large L pleural effusion- likely haemopneumothorax**
   - **L collapsed lung**
   - **Small pleural effusion**
   - **R airspace opacification- collapse/ contusion/ aspiration**
   - **AVR**
   - **L anterior thorax haematoma/ small R side haematoma**

b. What is the role of hypotensive resuscitation in this patient? State three (3) points in your answer. (3 marks)
   - **No high level evidence to support its use in blunt multitrauma** (well defined role in penetrating trauma)
   - **Hypotension will worsen ischaemia in traumatised vascular beds**
   - **Avoid overresuscitation- may precipitate cardiac tamponade**
   - **CI if CHI or spinal injury**

   Problems with normotensive resuscitation:
   ↑ perfusion to bleeding site, dislodge thrombus, loss vascular spasm, PC not as good as what is lost
   Hypotensive resuscitation:
   Studies underway → most benefit in young with single penetrating injury
   Avoid unnecessary IV fluids, inotropes, V/D, short acting β blockers, early Rx to control haemorrhage
   ? how hypotensive → SBP 60-80, MAP 40 suggested in adults (higher in older, CHI, pregnant)
   ∴ role unclear → likely for single, penetrating injury
   → ? non penetrating trauma, GIT, Ectopic, APH/PPH
   → ? role for reduction of normal BP
   Contraindications:
   - **Blunt trauma**
   - **controlled haemorrhage**
   - **uncontrolled haemorrhage when unable to be stopped**
   - **evidence of serious endorgan hypoperfusion → neurotrauma, RF, MI**

   Resus with avoidance of hypertension:
   AAA rupture, TAD, penetrating truncal/ extremity trauma, epistaxis

His CT brain and entire spine CT are reported as normal. His CT Pelvis shows an open book pelvic fracture. After referral to the nearest trauma service, it is decided to transfer the patient via road to the nearest tertiary facility 2 hours away. You are to accompany the patient.

c. Assuming the department has adequate staffing, state five (5) key steps in preparation for the transfer of this patient. (5 marks)
   - **Stabilise pelvis- pelvic binder**
   - **L ICC, consider R side if rib fractures or pneumothorax**
   - **Pericardiocentesis if signs of tamponade- take equipt. Be prepared to use**
   - **Blood for ongoing resuscitation**
   - **Analgesia**
   - **Warfarin reversal- AVR suggests warfarin likely- care not to reverse too aggressively**
   - **Communication- family/ receiving hospital**
   - **Monitoring- IABP**
   - **Documentation including imaging**

   +/- portable US / ETT / secure 2x functioning IV lines / check Equipment

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The role of hypotensive resuscitation in the management of trauma

K Jackson, J Nolan

The primary objective of trauma care is to minimise or reverse shock thus saving life. Aggressive fluid resuscitation may be harmful in these patients because the resulting increased blood pressure and circulating volume may cause clot disruption, dilution of clotting factors and/or the reversal of the body's natural response to haemorrhage. The concept of hypotensive resuscitation has evolved where small aliquots of fluid are infused, with hypovolaemia and hypotension tolerated as a necessary evil until definitive haemorrhage control can be achieved. This review outlines the animal and human data to support the strategy of hypotensive resuscitation.

Key words: hypotensive resuscitation; shock, haemorrhagic; shock, traumatic; head injuries; fluid therapy

Introduction

Trauma is the leading 'killer' of young people in the United Kingdom (UK).[1] Death is commonly caused by hypovolaemic shock secondary to haemorrhage, 'shock' being defined as circulatory failure leading to inadequate perfusion and oxygenation of tissues. This may ultimately cause irreversible organ failure and death. The primary objective of trauma care is to minimise or reverse shock, thus saving life. The American College of Surgeons' Committee on Trauma teaches that increasing the circulating volume and blood pressure will improve and maintain organ perfusion, thereby improving patient outcome and survival.[2] This review outlines the animal and human data to support the strategy of hypotensive resuscitation. Three subgroups of trauma will be considered – penetrating trauma, blunt trauma and head injury.

Fluid resuscitation

Most of the perceived benefits of fluid resuscitation were established by animal experimentation using controlled haemorrhage (CH) animal models in the 1950s and 1960s. The Wiggers' preparation involved the insertion of an intravenous (IV) catheter from which the animal was bled and maintained at a predetermined level of blood pressure (hypotension) for varying periods before resuscitation was initiated.[3] A marked extracellular fluid (ECF) deficit was observed, which could only be corrected with isotonic crystalloid 2-3 times the volume of the estimated blood loss, hence the traditional fluid-replacement regimen of 3:1, crystalloid: blood.

Recommending aggressive volume replacement based on these animal model experiments is problematic. Firstly, the Wiggers' model does not accurately reproduce the pathophysiology of the acutely exsanguinating trauma patient. The maintenance of blood pressure (BP) is controlled by the investigator rather than being a reflection of the animal's physiological response to haemorrhage. Furthermore, the animals are bled slowly from the catheter, which can be turned off instantaneously; again, this is not representative of the modern trauma patient who usually dies from rapid exsanguination or central nervous system (CNS) injury, not from protracted hypotension. Secondly, there is a lack of randomised controlled trials (RCTs) investigating aggressive volume replacement in trauma patients with ongoing uncontrolled haemorrhage.

In an attempt to improve the physiological modelling of trauma patients, models of haemorrhage from uncontrolled vascular injury were developed in the 1980s. The resulting haemorrhage volume and duration were dependent on the animal's physiological response to haemorrhage, ie thrombus formation and vasoconstriction. The experiments with these models highlighted the fact that fluid resuscitation was harmful.[4-6] Several mechanisms of harm were hypothesised, for example that increased blood pressure and circulating volume resulting from fluid resuscitation, caused clot disruption, dilution of clotting factors from crystalloid administration, and/or the reversal of the body's natural response to haemorrhage, ie vasoconstriction, which diverts blood from the peripheries and maintains perfusion of vital organs.

Solid-organ injury models of uncontrolled haemorrhage (UCH) were developed to simulate blunt trauma. In these, aggressive resuscitation with isotonic crystalloid increased blood loss and increased blood pressure abruptly before reducing it to less than or equal to the blood pressure of control animals and, most importantly, did not reduce mortality.[7,8]

Difficulties with trauma research

Trauma patients are a heterogeneous group. In the USA, deaths occur commonly from both penetrating and blunt trauma, whereas in the UK deaths result primarily from blunt trauma and head injury. It is therefore difficult to devise a single resuscitation strategy that is optimal for all healthcare

The Journal of TRAUMA® Injury, Infection, and Critical Care

Hypotensive Resuscitation during Active Hemorrhage: Impact on In-Hospital Mortality

Richard P. Dutton, MD, MBA, Colin F. Mackenzie, MD, and Thomas M. Scalea, MD

Background: Traditional fluid resuscitation strategy in the actively hemorrhaging trauma patient emphasizes maintenance of a normal systolic blood pressure (SBP). One human trial has demonstrated improved survival when fluid resuscitation is restricted, whereas numerous laboratory studies have reported improved survival when resuscitation is directed to a lower than normal pressure. We hypothesized that fluid resuscitation titrated to a lower than normal SBP during the period of active hemorrhage would improve survival in trauma patients presenting to the hospital in hemorrhagic shock.

Methods: Patients presenting in hemorrhagic shock were randomized to one of two fluid resuscitation protocols: target SBP > 100 mm Hg (conventional) or target SBP of 70 mm Hg (low). Fluid therapy was titrated to this endpoint until definitive hemostasis was achieved. In-hospital mortality, injury severity, and probability of survival were determined for each patient.

Results: One hundred ten patients were enrolled over 20 months, 55 in each group. The study cohort had a mean age of 31 years, and consisted of 79% male patients and 51% penetrating trauma victims. There was a significant difference in SBP observed during the study period (114 mm Hg vs. 100 mm Hg, $p < 0.001$). Injury Severity Score (19.65 ± 11.8 vs. 23.64 ± 13.8, $p = 0.11$) and the duration of active hemorrhage (2.97 ± 1.75 hours vs. 2.57 ± 1.46 hours, $p = 0.20$) were not different between groups. Overall survival was 92.7%, with four deaths in each group.

Conclusion: Titration of initial fluid therapy to a lower than normal SBP during active hemorrhage did not affect mortality in this study. Reasons for the decreased overall mortality and the lack of differentiation between groups likely include improvements in diagnostic and therapeutic technology, the heterogeneous nature of human traumatic injuries, and the imprecision of SBP as a marker for tissue oxygen delivery.

Key Words: Resuscitation, Hemorrhage, Hypotension, Trauma, Shock.

J Trauma. 2002;52:1141–1146.

Hemorrhage is a leading cause of death after trauma, and identification and management of hemorrhage is at the core of the American College of Surgeons Advanced Trauma Life Support (ATLS) curriculum.[1] Conventional emergency department protocols and ATLS call for rapid fluid resuscitation in all hemorrhaging trauma patients, beginning with the administration of up to 2 L of crystalloid and continuing with packed red blood cells and plasma as needed to maintain a normal systolic blood pressure.

This approach has been challenged by a number of authors, on the grounds that aggressive fluid administration in animal models leads to increased bleeding because of increased arterial and venous pressure, dilution of clotting factors, and decrease in blood viscosity.[2–10] Models of uncontrolled hemorrhage in swine,[3–6] dogs,[7] sheep,[8] and rats[9,10] have demonstrated increased hemorrhage when a normal systolic blood pressure is used as the target for fluid resuscitation. The majority of these studies have also documented a decrease in survival in animals targeted to a normal systolic blood pressure.[3–7,9,10] Several trials have identified a decrease in tissue oxygen delivery (largely because of hemodilution) when hemorrhaging animals are resuscitated to normal baseline blood pressure.[3,5,10]

Clinical study of deliberate hypotension in the resuscitation of trauma patients has been confined to one prospective trial completed in Houston in the early 1990s.[2] Hypotensive victims of penetrating torso trauma were randomized in the field to either receive intravenous fluids or not, and this therapy was continued until the end of the patient's stay in the emergency department. Although this study showed a survival advantage in the no-fluid group, it was subject to a number of statistical and methodologic shortcomings. The results were limited to penetrating trauma, although the majority of hemorrhagic shock seen in the United States is the result of blunt injury. Second, limited resuscitation occurred only during prehospital and emergency department care; all patients were aggressively resuscitated in the operating room, even if still hemorrhaging. Finally, the "all-or-none" nature of the protocol ignored the titration of fluid administration to the patient's vital signs and clinical condition, the normal standard of care.

The Houston study sparked controversy but has done little to change the standard practice of resuscitation in hem-

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Poster presentation at the 60th Annual Meeting of the American Association for the Surgery of Trauma, October 11–15, 2000, San Antonio, Texas.
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Question 6 (12 marks)

A 65 year old woman with a history of osteoporosis and depression presents with two weeks of increasing confusion and malaise. Her observations are: BP130/80 mmHg HR 100/ min Temp. 36°C GCS13 E4/V4/M5

			Reference Range
Na^+	144	mmol/L	134-146
K^+	4.2	mmol/L	3.4-5
Cl^-	98	mmol/L	98 - 106
$HCO3^-$	38	mmol/L	22-32
Urea	17.2	mmol/L	3-8
Creatinine	258	micromol/L	45-90
Glucose	5.4	mmol/L	3.5-5.5
Calcium	4.47	mmol/L	2.1 – 2.5
Phosphate	0.92	mmol/L	0.75 – 1.4
Albumin	40	g/L	35 - 50

a. Provide one (1) calculation to help you to interpret these results. (1 mark)
   - Derived value 1: **Se Osmo= 310 (↑)**

b. List three (3) significant abnormal findings in these results. (3 marks)
   - **Severe hypercalcaemia**
   - **A on Cr RF**
   - **High bicarbonate suggesting alkalosis (expect ↓ with degree of renal impairment)**

c. List four (4) likely differential diagnoses for this presentation. (4 marks)
   - **Dehydration secondary to vomiting**
   - **Milk alkali syndrome**
   - **1° hyperparathyroidism eg parathyroid adenoma**
   - **1° Malignancy- eg myeloma**
   - **2° Malignancy- bony mets**
   - **Drugs eg Vit D (for osteoporosis)**
   - **Immobilisation due to toxic ingestion**

d. Complete the following table demonstrating three (3) key treatment tasks. State how you would achieve each of these tasks. (6 marks)

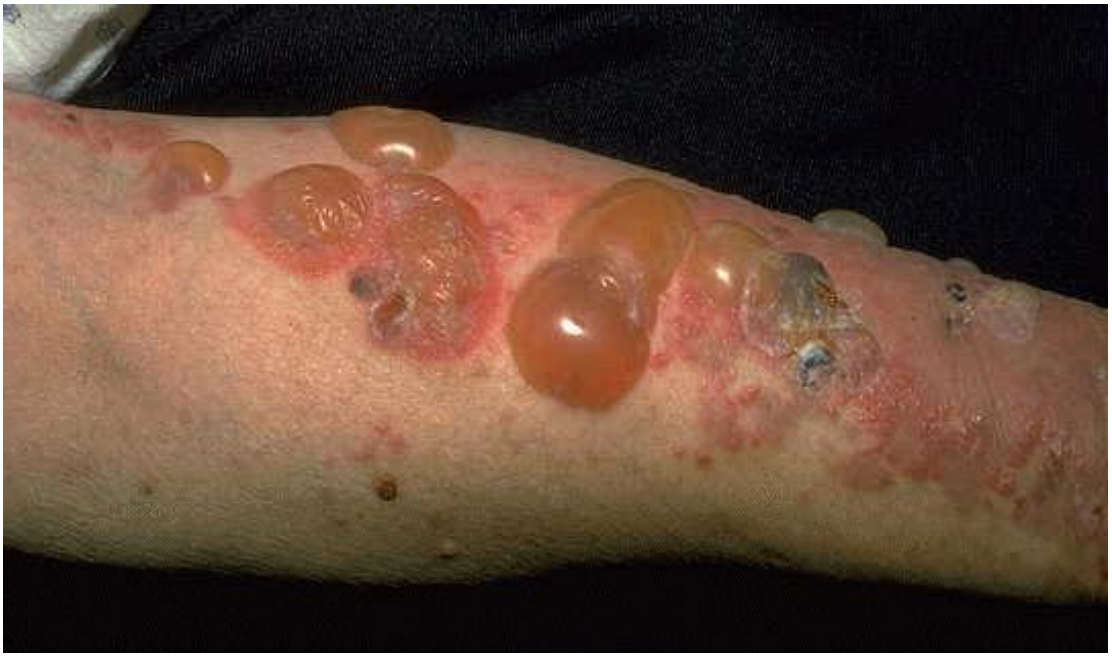
Key treatment task (2 marks)	How will you achieve it? (2 marks)
Rehydrate to Rx ↑Ca and ARF	• **NS** • **Aim U/O > 0.5 ml/kg/ hr**
Rx hypercalcaemia	• **Bisphosphonates**
Rx other 1° illness	• **Eg. UTI** • **Toxic ingestion**

Pass criteria
Examiners for this question sought all of the following to pass:
Describe:
Hypercalcaemia
Normal phosphate
Significant renal impairment
Elevated HCO3 or probable metabolic alkalosis
Interpret:
Have at least hyperparathyroidism and malignancy in the differential diagnosis
Note dehydration as a potential cause for the renal impairment or note that the degree of hypercalcaemia would explain her altered mental state

Fail criteria
Any one of the following:
Osteoporosis / osteomalacia given as a cause for hypercalcaemia
Hypoalbuminaemia as a cause for the elevated calcium
Failure to meet all of the mandatory pass criteria

Question 7 (12 marks)

A 72 year old male presents with a painful arm for the last 1 week.



a. List four (4) differential diagnoses for this appearance. How would you confirm each diagnosis? (8 marks)

Diagnosis	Method of confirmation
Bullous impetigo	**Clinical- golden crust** **Swab - +ve for S Aureus**
Bullous pemphigus	**+ve Nickolsky sign, biopsy**
Bullous pemphigoid	**-ve Nickolsky sign, biopsy**
Burns	**History**
H zoster with 2°bacterial	**Clinical, PCR**

b. How would you dress these lesions? State four (4) points of explanation. (4 marks)

- **Non adhesive dressing- Vaseline impregnated dressing**
- **Absorptive layer**
- **Crepe bandage**
- **Aseptic technique to prevent secondary bacterial infection**
- **Leave blisters intact unless interfering with dressings**
- **(If interfering- drain with sterile needle)**
- **Remove crusting if impetigo**

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Question 8 (12 marks)

A 45 year old man presents unwell after eating mushrooms.

a. What is/are the usual initial symptoms of toxic mushroom ingestion? (1 mark)
   - **GIT upset- D's & Vs**

b. Other than accurate species identification, which feature on history most accurately predicts a serious from a benign ingestion? (1 mark)
   - **Timing of onset of symptoms- benign usual symptoms < 3/24, sinister > 6/24**

c. Which mushroom is associated with the most number of fatal ingestions? (1 mark)
   - **Amanita Phalloides**

d. List the two (2) most common life threatening effects of mushroom ingestion. (2 marks)
   - **Liver failure**
   - **Renal failure**

c. List four (4) key management steps in suspected serious mushroom toxicity. (4 marks)
   - **Early and aggressive gastric decontamination**
     - **Induce emesis if < 2/24 or if GIT onset symptoms onset > 6/24 from ingestion**
     - **MDAC**
   - **Supportive care**
   - **Consultation with a Toxinologist (all cases)**

e. List three (3) antidotes that may be used in toxic mushroom ingestions. (3 marks)
   - **Atropine**
   - **NAC**
   - **Penicillin**
   - **Silibinin**
   - **Cimetidine**
   - **Alphalipoic acid (Thioctic acid)**
   - **Pyridoxine**

   NB: non are supported by RCT, anecdotal reports only

Mushroom Poisoning

Key points

- Contrary to popular belief, there are no easy 'rules of thumb' that will distinguish toxic from non-toxic species.
- Cooking will **not** detoxify a poisonous species.
- Only an experienced mycologist with a microscope can reliably identify many particular species!
- Toxic/ non-toxic sp co-exist side by side in wild ∴ species shown to you from the wild may not be the species that was ingested!
- 95% of fatal ingestions worldwide are due to **Amanita Phalloides. (Death cap)**
- Amanita muscara

Clinical Features of mushy munching

- The most important feature is the clinical presentation and time of ingestion
  → usually more important than attempts at accurate identification of the sp ingested
- Regardless of species, **initial symptoms** of mushroom poisoning will be **GIT upset**
  - ∴mushroom poisoning should be in DDx of acute GIT upset of uncertain causation)
- Clinical course of symptoms can be used as a guide to the likely offending species
  - Time of onset of symptoms from ingestion is the most important feature in this regard.

Ingestion

Non serious mushies, psychodelic, magic etc

Early onset symptoms symptoms (<3hrs)

1. GIT upset
2. Generally follows:
   i. autonomic disturbances, (muscaninic or sympathom)
   ii CNS disturbances, esp. confusion, hallucinations.

Generally follows benign self-ltd course over 6 hrs.

Amanita phalloides

Initial latent phase
Late onset of symptoms (>6hrs)

1. GIT upset (amatoxins may be delayed up to 12hrs) →watery diarrhoea
2. A latent phase where patient may seem well.
3. At 3-4 days onset of severe liver failure, ARF

Treatment:

Treatment:

- **Early and aggressive gastric decontamination**
  - If very early presentation (<2hrs) ipecac may be considered.
  - Later presentation (>3hrs), charcoal may be given, if vomiting is not a prominent feature.
  - Early charcoal hemoperfusion may be useful in cases of amanita phalloides ingestion
- **Treatment is otherwise supportive**
- **Many specific treatments have been advocated but not proven**
  - Cimetidine
  - Penicillamine
  - NAC
- **Enquiry into possibility of other people having ingested same mushrooms is important.**
- **Education re not eating field mushies**
- **Disposition**
  - Do not discharge patient without seeking advice of toxicologist
    - Need to watch for late development of severe liver failure, ARF
    - Toxicology Unit at the Austin Hospital should be contacted for further advice
    - Consider d/w liver transplant unit in case of huge OD

NOTABLE CASES

Fatal muscarinic syndrome after eating wild mushrooms

Question 9 (18 marks)

A 25 year old woman is brought in by ambulance after a T-bone car collision.
She was the driver of the car that was hit in the drivers' side at high speed. She is 32 weeks pregnant and is otherwise well. She is complaining of severe abdominal pain only.
Her observations: GCS 15 HR 140/min BP 100/60mmHg RR28/min O2 saturations 98% on RA
Temperature 36.8°C

a. How would you assess foetal viability in this patient? List three (3) points. (3 marks)
   - **Antenal Hx- prior US ? single/ multiple any abnormalities detected**
   - **Vaginal exam / speculum ? vaginal bleeding/ ROM/ Show- sign of 1st stage of labour**
   - **US FHR/ Mvts/ Evidence of abruption**
   - **Continuous CTG monitoring > 4/24**

NB: Fundal height and signs of peritonism unreliable

b. State four (4) key treatment principles for this patient. (4 marks)
   - **Management of 2 patients- Maternal resuscitation is the best method of foetal resuscitation (best for mum= best for baby)**
   - **Nurse in L lateral or wedge R hip (Pressure off aorta)- whilst maintaining spinal immobilisation**
   - **Early consultation with Obstetrician & surgeons**
   - **Theatre if significant abdominal trauma identified**
   - **Analgesia required ("severe pain")**
   - **Rh Isoimmunisation prevention- Ig as indicated**
   - **Admit for observation**

NB: Limit radiation is strictly speaking assessment.

The general surgical registrar suggests a "pan scan".

c. State two (2) possible appropriate arguments for pan scan in this patient. (2 marks)
   - **High risk mechanism** (be careful- mechanism has been shown to NOT be a good predictor of need for pan scan)
   - **If to OT- ongoing spinal immobilisation required and potential occult injury remain undefined**
   - **May improve directed Sx management**
   - **A diagnostic modality necessary for maternal evaluation should not be withheld o basis of potential hazard to foetus**

d. List two (2) possible appropriate arguments against pan scan in this patient. (2 marks)
   - **Large radiation dose and certain scans may not be indicated eg CTB**
   - **Will delay definitive Rx if this is indicated on clinical grounds/ +ve eFAST**
   - **Other screening plain XR may be sufficient eg CXR**

e. List three (3) pieces of information gained from this monitoring. (3 marks)
   - **Uterine contractions- 2 minutely**
   - **Late decelerations**
   - **FHR between 140-160**

f. In general, list four (4) signs of foetal distress that you may see in this type of monitoring . (4 marks)
   - **Lack of beat to beat variability**
   - **Resting tachycardia > 160 bpm**
   - **Extensive depth to decelerations (< 100)**
   - **Late decelerations**
   - **Prolonged decelerations (> 90 sec)**
   - **Variable decelerations**

Monitoring in labour

Normal FHR pattern on continuous monitoring has > 95% probability of foetal well- being

US

→ Doppler → FHR
→ Uterine size 1) > expected (placental abruption) 2) < expected (uterine rupture) →eg in trauma
→ confirm foetal movement
Combined with external strain gauge over abdo for recording motion of uterus during contractions
Limitations limited ability to determine ST variability
Strength of uterine contractions cannot be quantified

Internal monitoring

Greatest amount and accurate information
Electrode to presenting part (usu head)
ECG impulses amplified → transmitted to cardiotachometer
Filter converts foetal ECG into discrete electrical impulses
Standard calibration 1min/ cm (square) → 20 minutes between 2 numbers

Normal

- Basal FHR → 120- 160 beats/ min
- Normally small, rapid, rhythmic fluctuations 5- 15 bpm → sign of good autonomic activity, foetal well being
- Accelerations→ physiological, usually 2 per 20 minutes
  ∴ reassuring

Signs of foetal distress

- Lack of beat to beat variability
- Resting tachycardia > 160 bpm
- Extensive depth to decelerations (< 100)
- Late decelerations
- Prolonged decelerations (> 90 sec)
- Variable decelerations

Decelerations→ transient ↓ FHR 2° to uterine contractions
amplitude deceleration in bpm is difference from basal FHR and lowest FHR

Early during normal labour especially latter stages
contractions compress foetal skull → reflex bradycardia at commencement of contraction
FHR normal post contraction
uniform shape to decelerations
more common post ROM
rarely < 100bpm or > 90 seconds duration

Late ↓ FHR **after** beginning of contraction
uniform
FHR does not return to normal until well after contraction
caused by ↓ uteroplacental gas exchange
< 90 sec
baby may be born with ↓ Apgars

Variable compression of umbilical cord
decelerations at odd times
most common pattern associated with foetal distress
not uniform in shape or amplitue (wide variation)
relieve by turning mother from back to side or from one side to other

CTG explained:

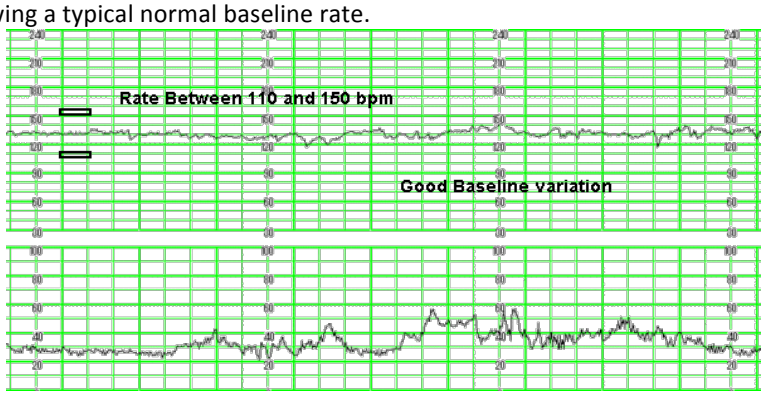
A Cardiotocograph (CTG) is a record of the foetal heart rate (FHR) either measured from a transducer on the abdomen or a probe on the foetal scalp. In addition to the foetal heart rate another transducer measures the uterine contractions over the fundus.

The interpretation of a cardiotocograph is complicated but this site will aim to demonstrate some of the more straightforward characteristics a CTG may display. The CTG trace generally shows two lines. The upper line is a record of the foetal heart rate in beats per minute. The lower line is a recording of uterine contractions from the toco. The vertical scale of this trace depends on how the transducer is picking up the contractions so interpretation needs to be in relation to the rest of the trace. The trace may also have markings on it that are indications that the mother has felt a foetal movement (operated by a switch given to the mother). Each big square represents 1 min on the X axis.

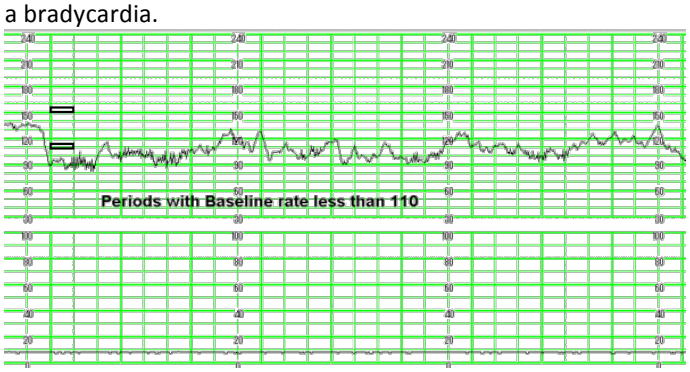
The following section describes the different patterns seen on a CTG.

Baseline Rate:- This should be between 110 and 150 beats per minute (BPM) and is indicated by the FHR when stable (with accelerations and decelerations absent). It should be taken over a period of 5 - 10 minutes. The rate may change over a period of time but normally remains fairly constant .

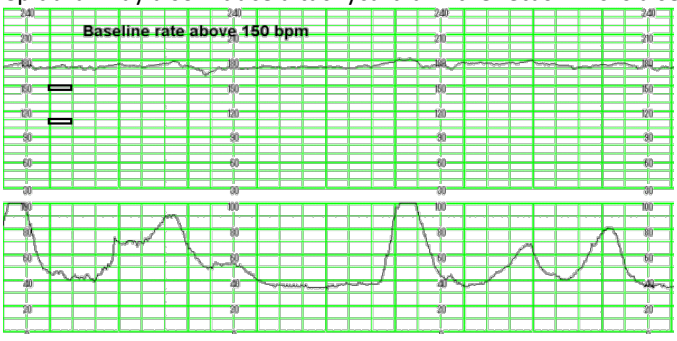
This is a section of CTG showing a typical normal baseline rate.



Bradycardia:- This is defined as a baseline heart rate of less than 110 bpm. If between 110 and 100 it is suspicious whereas below 100 it is pathological. A steep sustained decrease in rate is indicative of foetal distress and if the cause cannot be reversed the fetus should be delivered .
This is a section of CTG showing a bradycardia.

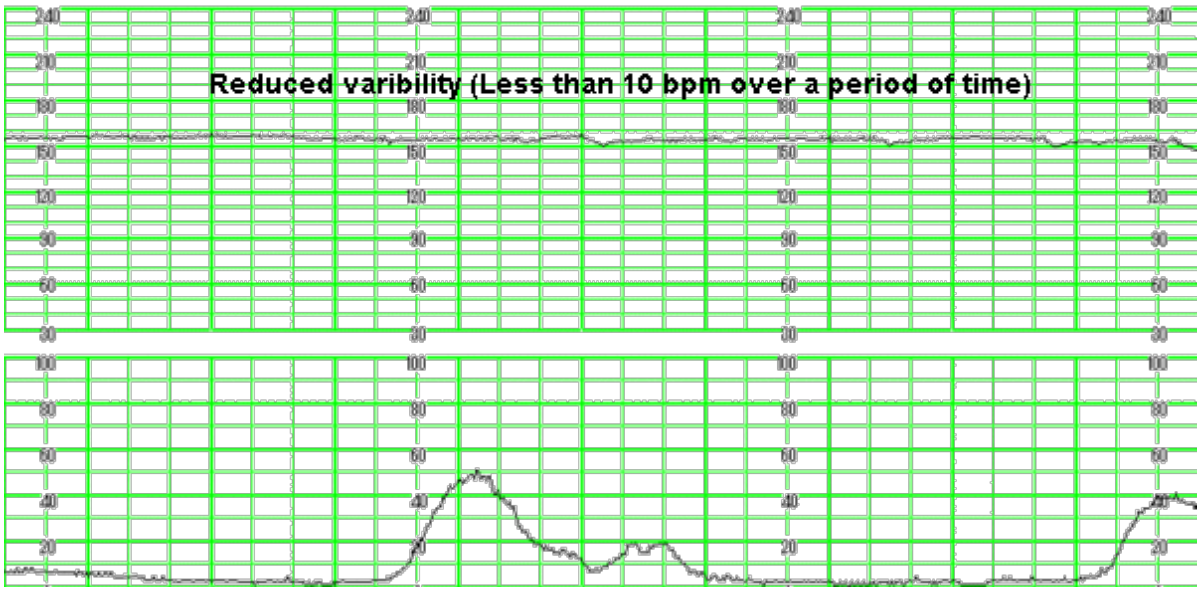


Tachycardia:- A suspicious tachycardia is defined as being between 150 and 170 whereas a pathological pattern is above 170. Tachycardias can be indicative of fever or foetal infection and occasionally foetal distress (with other abnormalities). An epidural may also induce a tachycardia in the fetus. This is a section of CTG showing a tachycardia.



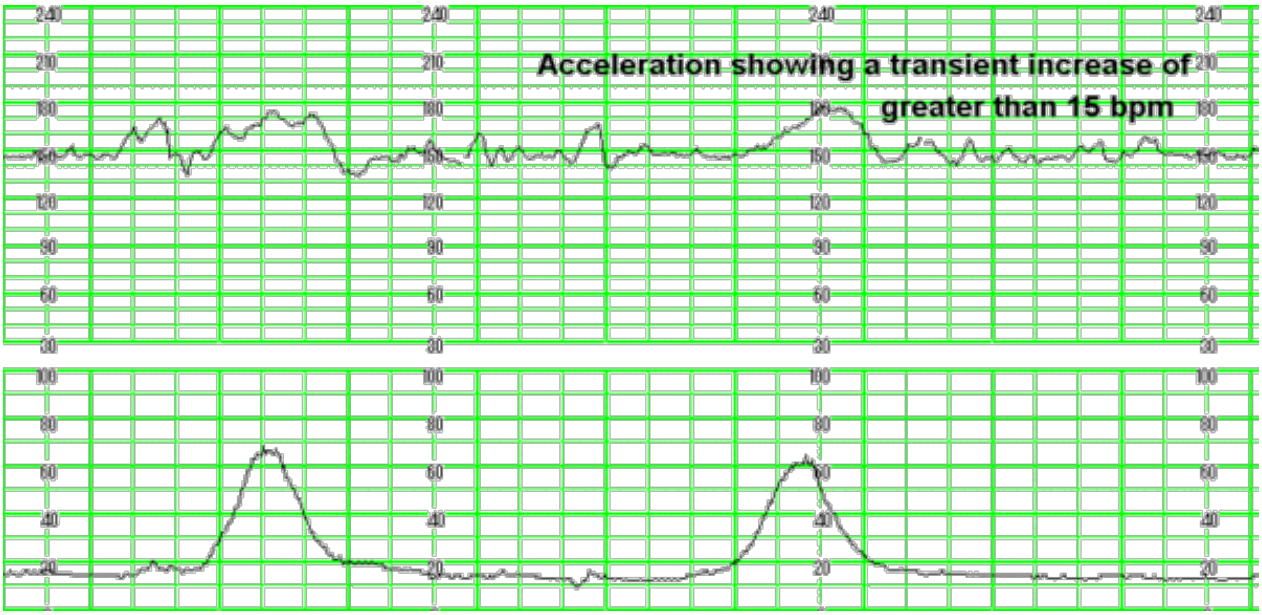
Baseline variations:- The short term variations in the baseline should be between 10 and 15 bpm (except during intervals of foetal sleep which should be no longer than 60 minutes). Prolonged reduced variability along with other abnormalities may be indicative of foetal distress.

This is a section of CTG showing decreased baseline variability.



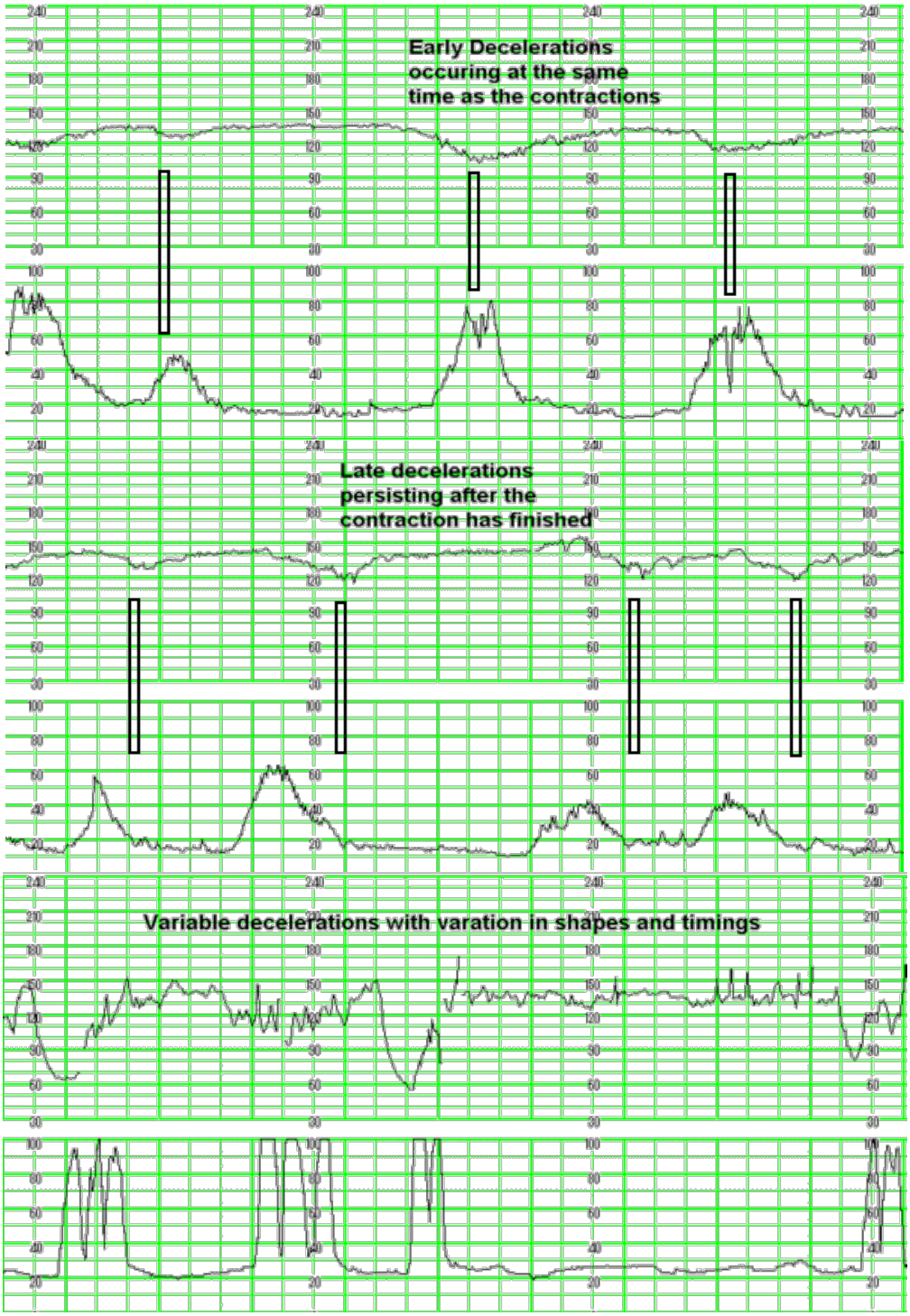
Accelerations:- This is defined as a transient increase in heart rate of greater than 15 bpm for at least 15 seconds. Two accelerations in 20 minutes is considered a reactive trace. Accelerations are a good sign as they show foetal responsiveness and the integrity of the mechanisms controlling the heart.

This section of CTG shows a typical acceleration in response to stimulus.



Decelerations:- These may either be normal or pathological. Early decelerations occur at the same time as uterine contractions and are usually due to foetal head compression and therefore occur in first and second stage labour with decent of the head. They are normally perfectly benign. Late decelerations persist after the contraction has finished and suggest foetal distress. Variable decelerations vary in timings and shape with respect to each other and may be indicative of hypoxia or cord compression.

The following CTGs show examples of early, late and variable decelerations.



A normal CTG is a good sign but a poor CTG does not always suggest foetal distress. A more definitive diagnosis may be made from foetal blood sampling but if this is not possible or there is an acute situation (such as a prolonged bradycardia) intervention may be indicated.