A 63 year old woman is admitted from the ward to intensive care for respiratory support following an emergency laparotomy for an acute abdomen eight days previously. The findings upon examination include:

A clinical photograph of a pressure sore

a) What complication has developed?

Pressure area ulcer

b) What are the risk factors for this complication?

Deregulated Physiology

College Answer

a) What complication has developed?
Pressure area ulcer

b) What are the risk factors for this complication?
Duration of surgery, faecal incontinence and/or diarrhoea, low albumin concentrations, disturbed sensory perception, obesity, moisture of the skin, impaired circulation, use of inotropic drugs, diabetes mellitus, too unstable to turn, decreased mobility, and high APACHE II score. Waterlow’s score, or other valid scores.

c) What is the management of this complication?
Remove all pressure from area, appropriate wound management, plastic surgical review, and adequate nutrition. Wound nurse team.

d) What are the major preventative strategies for this complication in intensive care patients?
Maintaining clean and dry skin, visualise skin integrity twice a day, regular pressure relief, pressure relief mattresses.

Discussion

This question closely resembles a part of Question 2 from the first paper of 2003. To simplify revision, the answer is reproduced here.

**Risk factors for pressure ulcers in ICU**

A good article from 2000 has an exhaustingly long table (Table 1).

Highlights from this article include the following:

- Prolonged immobility
- Use of neuromuscular junction blockers
- Age over 60
- Severe illness (APACHE II score over 13)
- Hemodynamic instability preventing pressure area care
- Diabetes
- Incontinence
- Low albumin
- Poor nutrition
- Oedema
- Peripheral vascular disease
- Steroid use

**Prevention of pressure ulcers in ICU**

- Risk assessment and monitoring
- Mobility (may be unreasonable in this context)
- Minimise sedation and restraints to allow for self-repositioning (may not be relevant in this context)
- Management of incontinence
- 2 hourly repositioning
- Air mattress or specialised foam (evidence is not strong)
- Adequate skin care

**Management of pressure ulcers in the ICU**

- Engagement of a multidisciplinary wound care team
- Debridement
- Antibacterial (silver sulfadiazine) dressings
- Frequent dressing changes
- Exudate-absorbing dressings
- Promote wound healing:
  - Adequate nutritional supplementation, particularly of protein (2g/kg/day)
  - Control of diabetes
  - Avoidance of corticosteroids
Optimisation of tissue perfusion
Avoidance of oedema

References:


List the ultrasound features of a pneumothorax.

[Hide Answer]

College Answer

- Loss of comet tails and “marching ants” appearance
- Ribs and pleura move together
- “Lung point” – motionless horizontal lines are replaced by normal lung appearance moving from non-dependent to dependent region and also seen with inspiration and the probe held stationary.
- Loss of “waves on the beach” appearance in M-mode

Discussion

This is one of those things which is better explained with pictures.

In dry boring words, there is a list of features which one can easily memorise:

1. Absence of "Seashore sign" on M-mode
2. Absence of "B-lines" or "comet-tails" moving in synchrony with the pleura
3. Absence of sliding lung
4. Lung point sign - the point where the pneumothorax transitions into normal lung

References:

Examine the clinical photograph.

a) List the abnormalities visible in this patient’s hands.

b) What is the most likely diagnosis?

c) List 4 other clinical features associated with this condition?
College Answer

a)  
- Evidence of Raynaud’s or ischaemia with pallor of digits,
- Sclerodactaly
- Calcinosis,
- Multiple amputations of distal digits
- Distal ulceration with gangrene
- Fixed flexion deformity of ring finger right hand.

b) Systemic sclerosis or Scleroderma

c)  
- Telangiectasia,
- Fine creps bibasally due to lower lobe fibrosis,
- Beaking of the nose
- Limitation of mouth opening
- Thickening of the skin and pigmentation changes
- Features consistent with Pulmonary H/T,
- Sjogrens syndrome
- Oesophageal involvement
- Renal failure

Discussion

Scleroderma is also the subject of Question 28 from the first paper of 2012, but the information requested there is rather different.

Who cares if the hands don’t demonstrate all the signs in the college answer? The cutaneous manifestations of scleroderma are as follows:

- Skin thickening
- Oedema of the digits
- Sclerodactyly
- Pitting ulceration, particularly at the fingertip
- Calcinosis over joints
- Telangiectasia
- Raynauds phenomenon

Other clinical features:

- Sjogren syndrome
- difficult intubation (limited mouth opening)
- Pulmonary fibrosis, restrictive lung disease, pulmonary hypertension
- Cardiac arrhythmias, myocardial fibrosis, pericardial stricture
- Corticosteroid-associated psychosis; chronic fatigue
- hyponatraemia and fluid retention due to corticosteroid therapy
- renal failure, renal artery stenosis
- Oesophagitis, poor gut motility, decreased feed tolerance, risk of aspiration
- Difficult vascular access
- Immunosuppressive therapy leading to increased infection risk

References:

Farber, Harrison W., Robert W. Simms, and Robert Lafyatis. "Analytic Review: Care of Patients With


In order to replace the college photograph, I have had to misappropriate a series of images from various sites:

The first set of (ulcerated) hands are from *Health in Plain English*;

The hands with digital pallor and telangiectasia are from... I can't remember where. Google?

The final set of digits with amputations and calcinosis are from *Pathguy*. Thank you, Pathguy!
A 64-year-old man with a past history of CLL presents with fever and malaise after an 8-day illness for which he was originally prescribed celecoxib.

The photographs below are of his forehead (figure 1) and back (figure 2).

What is your differential diagnosis?

Give two investigations that would aid diagnosis.

[ Hide Answer ]

College Answer

a)

- Severe drug hypersensitivity reaction
- Erythema multiforme
- Stevens Johnson/TENS
- Viral infection
- Herpes simplex
- Varicella
- EBV/CMV
- Bacterial infection – staphylococcal scalded skin syndrome
- Autoimmune bullous disease
- Urticarial vasculitis
b) 
- Skin biopsy
- HSV and varicella PCR
- Viral serology
- Mycoplasma serology

Discussion

The college did not provide us with a picture of the patient. Given the breadth of differential diagnosis offered, it is difficult to Google a satisfactory rash picture for this question. The picture of the back is a shot of somebody with Stevens-Johnson syndrome, stolen from Medscape; the face is apparently also SJS but it is from the slightly less reputable studyblue.com, where it is featured as a flashcard.

Without reliable pictures from the college paper, it is difficult to generate a sensible list of counter-differentials to match the college answer.

However, at this stage the author of these notes must humbly confess that even if the rash picture was available in high resolution, he would likely still be powerless against its mysteries.

In view of this, here is a generic list of differentials one could spout, for the question "name that rash". It is so generic that it is applicable even if one cannot see the skin.

**Vascular causes:**
- Shower of emboli
- Vascular insufficiency

**Infectious causes:**
- Toxic epidermal necrolysis
- Staphylococcal scalded skin syndrome
- Viral exanthem/manifestations of viral infection
- Fungal infection eg. candida

**Neoplastic causes:**
- Cutaneous lymphoma/leukaemia

**Drug-related causes**
- Stevens Johnson syndrome
- Red Man syndrome (vancomycin)

**Autoimmune cause**
- Allergic reaction
- Vasculitis
- Erythema multiforme
- Graft-versus-host disease

**Traumatic causes**
- Burns

The standard work-up:
- FBC for eosinophilia
- Viral serology for HSV, VZV, HIV, EBC, CMV
- Culture of exudate
- Mycoplasma serology
• Vasculitic screen
• Skin biopsy

References:


List 2 causes of clubbing (apart from cardiovascular and respiratory causes)

College Answer

° Inflammatory bowel disease
° Thyrotoxicosis
° Idiopathic
° Familial
° Cirrhosis
° Celiac
° Pregnancy

Discussion

There are numerous causes of clubbing.

Here is an unreasonably long list:

Causes of bilateral clubbing in both hands and feet

Cardiac

° Congenital heart disease, particularly cyanotic defects
° Congestive cardiac failure
° Aortic aneurysm
° Subacute bacterial endocarditis
° Arteriovenous fistula (and it would have to be a major one)

Respiratory

° Chronic lung disease of any sort, really;
  ◦ Bronchiectasis, which makes you think of cystic fibrosis
  ◦ COPD, asbestosis
Pulmonary fibrosis of any aetiology
Empyema which has been going on for a while
Lung carcinoma (according T and O’C, usually not the small-cell variety)

**Gastrointestinal**

- Cirrhosis; for some reason, particularly primary biliary cirrhosis
- Coeliac disease
- Inflammatory bowel disease
- Malignancy of the colon

**Uncommon causes of clubbing**

- Thyrotoxicosis
- Pregnancy
- Syringomyelia
- Hyperparathyroidism
- Pregnancy
- Inherited familial (some people are just born that lucky – it’s a Mendelian dominant trait)

**Causes of clubbing in feet only**

- Coarctation of aorta
- Abdominal aortic aneurysm

**Causes of unilateral clubbing**

- Any sort of arterial aneurysm leading into the clubbed limb
- Apical lung tumour
- Hemiplegia

---

**References:**

Clinical Examination of the Critically Ill Patient, 3rd edition by L.I.G. Worthley - which can be ordered from our college [here](#).


List 2 causes (apart from cardiovascular or respiratory) of cyanosis.

[ Hide Answer ]

College Answer

List 2 causes (apart from cardiovascular or respiratory) of cyanosis.

- Severe methemoglobinemia
- Sulfhemoglobinemia
- Hemoglobin mutation
- Polycythaemia
- Hypothermia / cold
- High altitude

Discussion

The college presents us with a long and inventive list.

To this list, one can still add a few (obscure) differentials.

Here we go:

- Methylene blue or indocyanine gree dye injection
- Hemosiderosis
- Excess consumption of colloidal silver
- Chronic massive doses of amiodarone
- Chlorpromazine

And, the causes suggested by the college:

- Severe methemoglobinemia (though the blood in these cases is supposed to be chocolate brown)
- Sulfhemoglobinemia
- Hemoglobin mutation (eg. Haemoglobin Beth Israel, etc)
- Polycythaemia
- Hypothermia
- High altitude
References:


The following image is of the blood sample tubes into which a specimen of blood from a critically ill patient had been drawn by the phlebotomist.

(a) What does this image show?

(b) List three (3) causes for this appearance in blood samples from critically ill patients.

(c) If the condition causing this appearance in the blood tubes were to be long standing, what clinical signs specific to this condition may be found in this patient?
College Answer

(a) What does this image show?
A creamy supernatant in blood tubes (serum and plasma) due to severe hypertriglyceridaemia (lipaemic serum).

(b) List three (3) causes for this appearance in blood samples from critically ill patients.
- Familial hyperlipidemia
- Propofol infusion
- TPN use
- Pancreatitis from hyperlipidemia

(c) If the condition causing this appearance in the blood tubes were to be long standing, what clinical signs specific to this condition may be found in this patient?
- Eyes
  - Lipaemia retinalis
  - Corneal arcus senilis
  - Xanthelasma
- Skin
  - Xanthomata
  - Tendon
  - Eruptive

Discussion

Of the ICU trainees, I am sure very few would have seen such a thing as this.

The specific "chicken fat" supernatant demonstrated in the college photograph is characteristic - that "cream" is all chylomicrons. In fact, this finding had in the olden days formed part of the classification of hyperlipidaemias- they used to observe "standing serum" to see if a supernatant would form. In the most severe forms of hyperlipidaemia, this fatty impurity can cause the blood to look milky and turbid.

There is little one can add to the college answer.

References:


On clinical examination of patient with abdominal pain, you find a mass in the left hypochondrium. List 4 clinical features will you use to distinguish between a palpable spleen and the left kidney?

[ Hide Answer ]

**College Answer**

- Presence of a notch – spleen
- Spleen moves inferomedially on inspiration
- Not ballotable or bimanually palpable
- Usually no band of resonance over s splenic mass
- Spleen has no palpable upper border
- Dullness over ribs 9, 10, 11

**Discussion**

This is another question which reaches into a deep dark recess of Talley and O’Connor.

The differences between spleens and kidneys are mobility, ballotability, and edge palpation.

- Thus, the spleen is mobile with respiration, whereas the kidneys is not.
- The kidney is "ballotable" whereas the spleen is not.
- The spleen has a notch on the anterior surface, and the kidney does not
- The spleen should be dull to percussion, where the kidney can be resonant due to overlying gas
- The spleen enlarges diagonally, towards the umbilicus and the RLQ, whereas the kidney enlarges inferiorly, to the ipsilateral pelvis.
- There is no palpable upper border to the spleen, whereas the kidney should have one

The reference for the above wisdom, shamefully, is Wikiversity.
"The genetic make up of the patient influences severity of sickness and recovery in a variety of disease states" – Outline a few examples in support of this statement in critical illness.

College Answer

1) Sepsis – It is now believed that genetic predisposition influences the risk of serious infection and outcome from severe injury. These genetic variations are thought to be the result of single nucleotide polymorphisms (SNP). These are thought to influence the severity of injury by controlling the induction of TNF, NF kappa B and toll receptors. Some examples include polymorphisms in TLR 2, 4 and 5 genes, CD14 and mannose binding lectin genes.

2) Acute lung injury The genetic susceptibility to the development of and variable outcomes in acute lung injury/acute respiratory distress syndrome (ALI/ARDS) has become a topic of great interest in the pulmonary and critical care community. Published studies of variable genetic susceptibility to ALI/ARDS already have identified some important candidate genes and potential gene-environment interactions. Some examples include variant alleles in Mannose binding lectin genes and surfactant protein B gene polymorphism.

3) Head injury – There is now data to suggest that the presence of certain Apo Lipoprotein genes may have an adverse outcome in head injury.

4) Pharmacogenomics: Response to and adverse effects of a drug are thought to have a genetic basis

5) IHD, CVA also have some genetic basis.

Discussion

In brief, the following specific associations between genotype and response to critical illness have been found:

- Risk of developing MODS from acute pancreatitis is influenced by TNF-α gene variants (Bishehsari et al, 2012)
- Risks of developing ARDS in community acquired pneumonia is related to genes encoding proteins A and D of pulmonary surfactant (Garcia-Laorden et al, 2011)
- Susceptibility to sepsis (and to death from sepsis) seems to be related to a whole host
of genetic variations, specifically in the genes encoding interleukin (IL)-1 receptor antagonist gene, the heat shock protein gene, the IL-6 gene, the IL-10 gene, the CD-14 gene, the Toll-like receptor (TLR)-4 gene, and the TLR-2 gene (Holmes et al, 2003)

- Delirium in critical illness is associated with a apolipoprotein E4 polymorphism (Ely et al, 2007)
- Outcome in brain injury seems to be worse for people featuring the apolipoprotein E-ε4 genotype (Friedman et al, 1999)

In addition, the following comorbidities feature significantly in ICU outcomes, and have a known genetic basis:

- NIDDM
- IHD
- Emphysema (α-1 antitrypsin deficiency)
- Cerebrovascular disease
- Various genetic disorders and sporadic mutations / chromosomal abnormalities; of which some notable examples are Down syndrome, which results in congenital cardiac defects, and Prader-Willi, which is associated with OSA and obesity hypoventilation syndrome.

References:


What are the age related factors which adversely affect outcome in the elderly (>65 years) critically ill patient?

[ Hide Answer ]

College Answer

Multisystem issues;

CVS: High prevalence of cardiac disease, CAD, silent ischemia, less responsive to sympathetic stimulation and therefore lesser response to catecholamines, greater diastolic dysfunction and conducting system disease, likelihood of being on cardiac drugs. (3 marks)

RS: Swallow dysfunction- risk of aspiration

Decreased ventilatory response to hypoxia and hypercapnia

Decreased chest wall compliance, muscle strength and increase in closing volume.

Renal: Decrease in renal function, lower muscle mass so a serum creatinine at the upper end of normal may indicate renal failure

Metabolic: Reduced BMR, risk of overfeeding

CNS: Higher incidence of delirium, age related loss of cerebral volume

Drug dosing: Altered pharmacokinetics, reduced renal and hepatic reserve, need dose adjustment, increased sensitivity to sedation and analgesia

Greater operative morbidity and mortality

Discussion

This question closely resembles a part of Question 9 from the first paper of 2012. To simplify revision, parts of that answer are reproduced below. However, the college answer does not seem to answer the college question. The question asked for age related factors which adversely affect outcome; the college answer instead went on to discuss age-related changes in physiology, and how these influence intensive care management.
Let us talk about the outcomes first.

**Influence of age:**

- Mechanical ventilation: in-hospital mortality among octogenarians = 70%
- If the reason for ventilation was pneumonia, in-hospital mortality for the over-65s = 62%
- In hospital mortality for octogenarians admitted with sepsis = 85%
- In brain injury, risk of death or disability is doubled in the elderly.

**Influence of functional status and co-morbidities:**

- Functional dependence: in-hospital mortality = 30% (vs. 7.8% if independent)
- Dementia: mortality = 55.9% versus 8.2% in those without cognitive impairment.
- Delirium: independent predictor of reintubation, prolonged hospital stay and mortality.
- Malnutrition: low BMI increases mortality in the elderly.

**Expected functional outcome:**

- Only 14% of patients aged 85 years or older went home without home health care.
- After discharge, mortality occurred predominantly during the first 3 months. If you survive ninety days, you're probably going to be ok.
- Many elderly patients do not want intensive care. *In a population of patients with limited life expectancy and aged 60 years or older, 74% stated that they would not choose treatment if the burden of treatment were high and the anticipated outcome survival with severe functional impairment*.

Now, how does old age influence intensive care management?

**Changes in airway management:**

- Intubation may be easier due to the patient being edentulous
- Intubation may be more difficult due to C-spine and TMJ arthritis
- Bag-mask ventilation may be more difficult because of missing teeth and wasting of facial soft tissues
- Swallowing may be impaired and aspiration is more likely
- Mechanisms maintaining airway patency are impaired, and extubation failure is more likely
- Greater risk of post-induction cardiovascular collapse

**Changes in respiratory management and ventilation**

- Decrease in expectations: SpO\textsubscript{2} goals may be lowered (~ 92%, due to “senile emphysema”)
- Chest physiotherapy becomes more important (decreased respiratory muscle strength)
- Early extubation is favoured (to prevent deconditioning)
- There is decreased sensitivity of respiratory centres to hypoxia and hypercapnia, which must be considered.

**Changes in approach to cardiovascular support**

- Responsiveness to β-adrenergic receptor stimulation is decreased; higher doses of vasopressors may be required
- Baroreceptors and chemoreceptors are less reactive
- Levels of circulating catecholamines are increased
- An increased blood pressure goal is therefore appropriate when titrating vasopressors: chronic hypertension is almost assured, and with this, organ bloodflow autoregulation is impaired (i.e. more closely tied to pressure).

**Changes in assessment of neurological function**

- Increased risk of delirium (therefore, greater vigilance in screening for delirium)
• Decreased expectation of neurological performance, e.g. when assessing for extubation (pre-existing dementia)
• Parkinson disease (unusual response to antidopaminergic drugs)
• Pre-existing weakness may produce a "difficult to wean" scenario

Changes in approach to the support of renal function

• Age-related decrease in GFR is to be expected
• Renal blood flow decreases; the kidneys are more susceptible to fluctuations of blood pressure
• Renal blood flow autoregulation undergoes a left-shift; thus a higher perfusion pressure may be required (e.g. a MAP of 75-80)
• Because of these factors, old kidneys are more susceptible to dialysis-associated renal dysfunction. This influences the decision as to whether one does or does not offer dialysis.

Changes in approach to nutrition

• Albumin is expected to be low (due to age-related decrease in albumin synthesis)
• Decreased glycogen reserve means greater vigilance in monitoring for hypoglycaemia
• Decreased metabolic rate means nutritional requirements may be lower than predicted by crude approximations of caloric requirements
• Likelihood of premorbid malnutrition is greater - thus, more susceptible to refeeding syndrome

Changes in approach to blood transfusion

• Age-related changes in haematological function influence your expectations: these patients are more likely to be chronically anaemic, and well adapted to anaemia.
• Decreased marrow cellularity results in a diminished response to anaemia and EPO.
• Chronic anaemia of malnutrition may pre-date ICU admission

Changes in interpretation of the clinical features of sepsis

• There is diminished immune response, both cellular and humoral.
• The elderly may not mount a febrile response, and may actually be hypothermic with sepsis
• Decreased synthetic function of the liver may result in diminished synthesis of CRP

Changes in pharmacology

• Drug levels may not represent the effective "free" fraction due to changed in protein binding and volume of distribution
• Dose adjustments need to be made to account

References:


List the clinical features associated with Systemic Lupus Erythematosus, and outline how they would influence your management of a patient in Intensive Care.

[ Hide Answer ]

College Answer

SLE is a chronic inflammatory disease, presumably auto-immune, which occurs predominantly in women, and can affect almost all organ systems. These can masquerade as many different conditions, and can make workup very complex. Clinical features (and examples of ways they would influence management) include:

- Fatigue (common and debilitating)- care with differential diagnosis
- Fever (episodic and related to activity of disease)- need to exclude sepsis, potential for unnecessary antibiotics
- Arthritis (painful, migratory and asymmetrical; rarely deforming)- care with positioning, may need analgesia
- Skin changes (butterfly rash, and hair loss)- care with handling
- Raynaud’s phenomenon- caution with vasoconstrictors, pulse oximeters, arterial lines
- Renal dysfunction (usually glomerulonephritis)- avoid nephrotoxins, adjust drug doses
- Pleurisy and pleural effusions- need to diagnose, exclude other conditions
- Pericarditis and Libman-Sacks (verrucous) endocarditis- may require TOE or surgery
- Increased incidence of coronary artery disease- need to be aware of problem in otherwise young females without risk factors!
- Delerium, psychosis and seizures- complex management and diagnostic problem
- Thrombosis in association with anti-phospholipid antibodies- need to diagnose, and may need treatment for pro-coagulant state
- Abnormal haematology (leukopenia, anaemia, thrombocytopenia)- may need further investigation
• Lymphadenopathy and splenomegaly- may make suspicious of alternative disease process; may need further investigation
• Auto-immune disease and immunosuppressive therapy (eg. corticosteroids, cyclophosphamide)- at particular risk of infections in immunosuppressed. Early and aggressive workup and initial treatment may be required. Aware of potential for adrenal suppression.

**Discussion**

This question would benefit from a structured tabulated answer.

<table>
<thead>
<tr>
<th>Topic Area</th>
<th>Manifestations</th>
<th>Influence on ICU management</th>
</tr>
</thead>
</table>
| Airway           | Cricoarytenoiditis                                  | • Need for intubation  
 • Difficult intubation  
 • Risk of post-extubation stridor                                                   |
| Respiratory      | Pulmonary fibrosis                                  | • Impaired gas exchange  
 • Restrictive ventilatory defect                                                      |
|                  | Pleuritis                                           | • Need to test pleural fluid for ANA                                                        |
|                  | Pulmonary hypertension                              | • Hypoxia  
 • Hypothension in response to high PEEP  
 • Right heart failure                                                                     |
| Circulatory      | Myocarditis                                         | • Need for inotropes, IABP or ECMO  
 • Potential need for RV biopsy for diagnosis                                                   |
|                  | Constrictive pericarditis                           | • Obstructive cardiac failure  
 • Danger of colchicine therapy  
 • Need for cardiothoracic surgical release                                                 |
|                  | Increased risk of coronary artery disease           | • MI is higher on the list of differentials for hemodynamic instability, even in young females |
| Neurological     | Cerebral vasculitis                                 | • A differential diagnosis of seizures and decreased level of consciousness  
 • Impaired blood brain barrier                                                               |
| Endocrine        | Cushing syndrome due to chronic steroid use         | • Hyperglycaemia  
 • Hyponatremia  
 • Fluid overload                                                                                |
| Renal            | Lupus nephritis                                     | • Pre-existing renal impairment  
 • Susceptibility to nephrotoxins is increased                                                  |
| Gastrointestinal | Gastrointestinal tract vasculitis (and/or ulceration)| • Vigilant monitoring for GI haemorrhage  
 • Malabsorption of feeds may occur                                                           |
## Hepatic
- Hepatic vein thrombosis due to APLS
  - A differential for ascites and hepatic failure in SLE patients
  - Anticoagulation will be required

## Haematological
### Anaemia of chronic disease
- Impaired haemopoietic response to haemorrhage
- Increased transfusion requirements

### Haemolytic anaemia
- MAHA needs to be considered in the anaemic SLE patient
- Vasculitic and haemolytic screening needs to be performed

## Antiphospholipid syndrome
- Risk of bleeding is increased
- Risk of thrombosis is increased
- Risks of anticoagulation need to be balanced with benefits

## Infectious
### Immunossuppression, and the risk of sepsis
- Need to consider opportunistic organisms in the selection of antibiotic cover

### References:


Critically evaluate the clinical value of daily routine chest radiographs in the ICU.

[ Hide Answer ]

College Answer

Daily “routine” CXR in (usually) intubated patients: controversial—evidence to support or refute practice, hard to study due to investigator bias, blinding problems and outcome assessment.

Generalisability may be an issue from often single specialty North American or European units to the usual multidisciplinary Australasian ICU. The consensus opinion of the Am. College of radiology is that daily routine CXR are indicated in patients who are mechanically ventilated. The evidence to date does not suggest that daily routine CXRs lead to changes in therapeutic decision making. Data suggest that length of stay and duration of mechanical ventilation are not adversely affected by elimination of daily routine CXR.

Benefits:

a) Confirmation of placement of major lines / tubes / pipes / wires
detects expected/unexpected disease progression/complications requiring treatment

b) Reasonable assessment of hypervolaemia/LVF, new infiltrates accompanying fever, pleural complications, endotracheal tube displacement
Problems:

- Radiation exposure
- Staff/patients potential for line/tube displacement
- Cost
- False positive/false negative findings

Discussion

**Rationale for routine CXRs:**

- Critically ill patients may have rapidly evolving thoracic pathology
- This pathology may not be easily evaluated by clinical means
- Mechanically ventilated patients are especially prone to rapid changes
- Routine radiography may yield a management-influencing surveillance benefit

**Advantages:**

- Regular review of line and tube position
- Regular review of fluid balance as observed by pulmonary interstitial water
- Surveillance for VAP
- Assessment of the readiness for extubation
- Observation of changes in lung parenchyma in response to treatment (eg. resolution of pneumonia)

**Disadvantages:**

- Radiation exposure
- Risk of tube/line dislodgement with positioning
- Lack of association between radiological appearance and physiological performance

**Evidence and recommendations:**

- Only about 7% of CXRs result in a change in management
- In another study, only 2.3% of CXRs revealed new pathology
- 2012 meta-analysis: no harm associated with "restrictive" use of CXRs
- American College of Radiology recommends they are performed for clinical indications only

References:


Amorosa, Judith K., et al. "ACR appropriateness criteria routine chest


Physiotherapy as an "introduction" statement

This would have to be a brief fluffy motherhood statement. On offer is the college's own model answer introduction from Question 24:

Physiotherapists are part of the multidisciplinary team providing care to patients in the ICU.

Note how the first line of the model answer includes both the word care and the word multidisciplinary, both of which are important keywords for the examiner's intracranial SEO. Appropriate triggers are important. Marks are earned in this way.

Rationale for the use of physiotherapy in intensive care

- Functional performance of patients who recover from ICU stay is poor. In one representative longitudinal study, at one year after their ICU stay 69% of survivors were restricted in their ADLs and only 50% had resumed work. (van der Schaaf et al, 2009)
- This is thought to be due to muscular deconditioning, which in turn leads to increased risk of failed ventilator weaning, and prolonged intubation
- Worsened respiratory function due to poor secretion clearance contributes to morbidity and mortality
- Immobility contributes to muscle wasting and joint contractures
- Loss of tonic stimulus leads to hydroxyapatite resorption and bone density loss
- Ergo, therapies aimed at increasing mobility and muscle strength in critical illness should lead to improved functional outcomes and improved mortality/morbidity by addressing these problems.
Techniques and care involvement of ICU physiotherapists

Goals of routine ICU physiotherapy

- Optimisation of cardiopulmonary function
- Assistance in the weaning process utilising ventilatory support and oxygen therapy
- Instigation of an early rehabilitation/mobilisation program to assist in preventing the consequences of enforced immobility
- Advise on positioning to protect joints and to mini-mise potential muscle, soft tissue shortening and nerve damage
- Optimisation of body position to effect muscle tone in the brain-injured patient
- Optimisation of voluntary movement to promote functional independence and improve exercise tolerance
- Management of presenting musculoskeletal pathology
- Advise and education of family and carers
- Liaison with medical and nursing staff on the continuation and monitoring of ongoing physiotherapy-devised care plans.

A more structured list-like answer would resemble the following:

- **Respiratory optimisation**
  - manual hyperinflation, suction, inspiratory muscle training, positioning, percussion/vibration

- **Musculoskeletal optimisation**
  - Mobilisation, joint-protective positioning, tone-improving positioning

- **Orthopedic**
  - Management of immobilisation devices, application and removal of plaster casts, fitting of collars, braces and slings

- **Rehabilitation**
  - Exercise and education to improve function following a period of critical illness

Specific techniques used in ICU physiotherapy

Specific techniques are well discussed in the excellent recent article by Sommers et al (2015). I paraphrase their Table 2 in the following list:

**Passive techniques:**

- Passive joint exercises
- Stretching
- Passive cycling
- EMS (electrical muscle stimulation)
- CPM (continuous passive motion) - devices which continuously move a joint through a preprogrammed range of motion
- Splinting

**Active techniques:**

- Exercise therapy
- ADL (activity of daily living) training
- Out of bed mobilisation
- Cycling

Potential risks to critically ill patients from routine physiotherapy

- Airway trauma from suctioning
- Deterioration in gas exchange
- Barotrauma
• Paradoxically, chest physiotherapy can actually increase the duration of ventilation (Maie et al, 2007)
• Haemodynamic instability
• Increased cardiac output requirements
• Rise in ICP
• Increased patient pain, stress and anxiety
• Risk of falls
• Pressure areas from splints
• Wound dehiscence (eg. of laparotomy wounds)

Society recommendations for the use of physiotherapy in ICU

The ERS/ESICM Guidelines Statement from 2008 makes the following recommendations. Most of them were Level C, because at this stage there were few clinical trials available. Subsequent trials have actually retrospectively supported many of these recommendations.

• Chest physiotherapy: huff, cough, manual bag inflation - all supported by a level B recommendation.
• Oropharyngeal or nasopharyngeal suctioning is also recommended (but only on the basis of expert opinion) - but neither suctioning nor instillation of normal saline should be performed routinely.
• Active or passive mobilization and muscle training should be instituted early (C)
• Physiotherapists can be safely left in charge of ventilator weaning and spontaneous breathing trials (level A recommendation)

Evidence for the advantages of routine physiotherapy in the ICU

Mind you there is a massive amount of literature out there. A meta-analysis by Kaymabu et al (2013) identified 10 RCTs. In brief, physiotherapy was found to confer "significant benefit in improving quality of life, physical function, peripheral and respiratory muscle strength, increasing ventilator-free days, and decreasing hospital and ICU stay". There was no benefit in mortality. A good representative trial is by Burtin et al (2009) - single centre RCT, 90 patients got on to an exercise cycle ergometer as soon as their condition permitted. At intensive care unit discharge, quadriceps force and functional status were not different between groups, but at hospital discharge the 6-minute walking distance, isometric quadriceps force, and the subjective feeling of functional well-being were significantly higher in the treatment group.

Some of the more recent trials which were not included in this review. Weirdly, these more recent trials are all negative studies.

ACT-ICU trial, Brunnel et al (2014) - 87 patients

• Randomised to either usual care, early once-daily physical therapy, or early once-daily physical therapy plus a novel, progressive, twice-daily cognitive therapy protocol.
• At 3-months, the investigators assessed cognitive, functional and health-related QOL outcomes.
• These did not differ among the groups.
• The authors consoled themselves with the finding that early cognitive and physical rehabilitation is at least feasible.

Goll et al (2015) - 50 ICU patients subjected to electro-torture to investigate the effects of daily EMS-therapy on muscular strength.

• EMS for 20 minutes 7 days/week to 8 bilateral muscle groups
• No benefit from this was found.
Kayambu et al (2015) - 50 patients with sepsis, randomised to either early physical rehab or routine care.

- Self-reported function was much better at 6 months, but objective measures of function were no different.

EPICC trial, Thomas et al (2015) - still in recruitment phase; 308 patients to be randomised to receive one of two different intensities of physiotherapy.

- The hypothesis is that "physiotherapy aimed at early and intensive patient mobilisation" will improve "improve physical outcomes and the mental health and functional well-being of survivors of critical illness"
- The primary outcome measure is physical health at 6 months, as measured by the SF-36 Physical Component Summary.

References

Oh’s Manual (7th ed) Chapter 5 (pp.38) Physiotherapy in intensive care by Fiona H Moffatt and Mandy O Jones


Outline the potential benefits and risks of the provision of physiotherapy to the critically ill patient.

[Hide Answer]

College Answer

There is still significant debate about the role of routine physiotherapy, but areas of physiotherapy with their potential benefit include:

- Optimization of ventilation/respiratory function
- Assistance in weaning
- Advice on positioning of chest for improving ventilation
- Joint protection
- Minimise muscle damage, soft tissue injury
- Muscle tone maintenance particularly in the neurological ICU patient
- Early rehabilitation/mobilization
- Liaison with medical and nursing staff

Potential risks/complications of physiotherapy techniques listed above include:

- Deterioration in gas exchange
- CVS instability
- Barotrauma
- Rise in ICP
- Increased patient pain, stress and anxiety

Discussion

The role of physiotherapy is discussed in Question 24 from the second paper of 2013.
Critically evaluate the role of a daily interruption of sedation for mechanically ventilated patients in the ICU.

[ Hide Answer ]

College Answer

Introduction / Rationale

A daily interruption of sedation is a strategy designed to reduce exposure to sedative agents, allow assessment of neurological status and assess readiness for extubation and to reduce duration of mechanical ventilation.

Evidence

Initial trials showed a marked reduction in duration of mechanical ventilation, and decreased duration of intensive care length of stay (e.g. Kress et al, NEJM 2000). It was notable that no sedation target nor protocol was used in the control group, thus this group may have been oversedated, analogous to the 12ml/kg TV group in the ARDSNET low TV trial.

Subsequent studies have been somewhat conflicting:

- ABC study (Girard et al, Lancet 2008) showed improved outcomes (mortality, less time on mechanical ventilation, reduced ICU length of stay) in patients treated with a paired daily interruption of sedation and a spontaneous breathing trial compared to usual care plus a spontaneous breathing trial.
- SLEAP study (Mehta et al JAMA, 2012) showed no difference in outcomes comparing protocolised sedation to protocolised sedation plus daily interruption of sedation.

Disadvantages / Adverse effects / Limitations

Potential adverse effects of daily interruption of sedation:

- Patient discomfort and risk of PTSD and other long term psychological issues
- Dislodgment of ETT, CVC, arterial lines etc.
- Increased nursing workload.
- Cessation of sedation could lead to agitation which can be associated with physiological instability, hypertension, tachycardia, ventilator dysynchrony and hypoxaemia, which could be associated with exacerbation of primary disease in certain conditions, e.g. myocardial ischaemia, brain injury.
- I.e. interruption of sedation contra-indicated in above patient groups.

Own Practice
Any reasonable justifiable approach acceptable.

**Summary**

Daily interruption of sedation may have a role in physiologically stable patients in ICUs that do not routinely use protocolised sedation.

**Discussion**

**Rationale**

- Exposure to sedating agents is undesirable, given that most agents have unpleasant side-effects
- Interruptions in sedation may decrease the total dose of sedation over the course of one's ICU stay

**Proposed advantages**

- More rapid weaning from ventilation
- Reduction in ICU length of stay
- Reduced need for vasopressors
- Reduced need for fluid boluses

**Expected disadvantages**

- Greater risk of self-extubation
- Greater psychological distress; potential for PTSD
- Increased nursing workload
- Increased patient-ventilator dyssynchrony

**Evidence:**

- **Kress et al (2000)** - small single centre RCT; \( n = 128 \)
  - length of stay in ICU and duration of ventilation was reduced
- **Kress et al (2003)** - small single centre RCT; \( n = 108 \)
  - reduced symptoms of PTSD; no adverse psychological outcome
- **Girard et al (2008)** - multicentre trial, \( n = 336 \)
  - Reduced mortality at 1 year, but increased risk of self-extubation
- **Mehta et al (2012)** - multicentre randomised controlled trial, \( n = 430 \)
  - No reduction in length of ICU stay or duration of ventilation
- **Cochrane verdict (2014)** - meta-analysis, \( n = 1282 \); no strong evidence in support of this practice. Tracheostomy may be performed less often.

**References:**


A 30 year old man has been admitted to hospital with severe multiple injuries following a motor vehicle accident.

On day 2, his intracranial pressure has stabilised and his head CT shows scattered punctate haemorrhages with subarachnoid blood, with no mass lesion requiring evacuation. His pelvic fracture and right tibia / fibula fracture have been managed with external fixation and a left leg femoral fracture has undergone open reduction and internal fixation.

He has been in good health, but had a DVT 3 years ago and is not on any regular medication.

Outline your approach to prophylaxis for venous thrombo-embolism in this patient.

[ Hide Answer ]

College Answer

Risk of VTE is high based on:
- Major trauma with pelvic and lower limb injury and operative intervention
- Possibility of a pro-thrombotic disorder

Therapy also has potential risks:
- Risk of intracranial haematoma expansion with unfractionated or LMW heparin

Management options:
- Quantify potential pro-thrombotic disorder: ancillary history, previous investigations etc
- Unilateral mechanical prophylaxis
- Discuss timing of pharmacological prophylaxis
- Clinical and imaging surveillance
- IVC filter

Discussion

One can approach this question systematically:

The risk factors for PE are discussed in a separate chapter, but it is a general outline - not specific to ICU patients. The table of undifferentiated risk factors is reproduced below:
Risk Factors for Pulmonary Embolism

**Inherited risk factors**
- Antithrombin III deficiency
- Protein C deficiency
- Protein S deficiency
- Factor V Leiden mutation
- Hyperhomocysteinaemia

**Acute risk factors**
- Surgery
- Trauma
- Pregnancy
- Burns
- CVC, Swan-Ganz
- Spinal injury with paralysis
- Immobility

**Chronic risk factors**
- Smoking
- Obesity
- Oral contraception
- Hormone replacement therapy
- Malignancy
- Heart failure
- Lupus anticoagulant
- Increasing age

Thus, according to these generic risk factors, the patient in the question has several reasons to develop a venous thromboembolism: surgery, trauma, immobility, and a CVC. On top of that, he (a 30 year old man) has already had a DVT at the age of 27, which rings alarm bells. Does he have a weird prothrombotic diathesis? Who knows.

In view of this, he sounds like a candidate for some sort of prophylactic therapy.

If one were to answer this question like an adult, one would produce an answer which resembles the following:

**Method of anticoagulation**
- Initially, mechanical thromboprophylaxis only (one leg)
- Chemical anticoagulation with unfractionated heparin, after a second CT demonstrates no change in the punctate haemorrhages and the SAH.

**Rationale for chemical anticoagulation**
- This patient has had surgery, trauma, immobility, and a CVC as risk factors.
- The likelihood of VTE is high.
- Chemical anticoagulation improves survival
- Chemical anticoagulation is more effective than mechanical thromboprophylaxis
- There is currently a contraindication to adequate mechanical thromboprophylaxis (the leg fracture should not have a calf compressor on it)
- Unfractionated heparin is inferior to low molecular weight heparin in the context of ICU thromboprophylaxis, but in this case it would be preferred to LMWH because it can be easily reversed with protamine in case of bleeding.

**Advantages**
- May improve mortality by decreasing risk of VTE
- Reversible anticoagulation

**Disadvantages**
- The risk of thrombosis may not be reduced
- The risk of bleeding may increase
- The presence of existing intracranial haemorrhage (and no mention of an ICP measuring device) means catastrophic intracranial bleeding may result.
- Vigilant monitoring of clinical and radiological features of increased intracranial pressure may be required
- Advantages of protection from VTE must be weighed against the risk of intracranial bleeding (it is only day 2 post accident)

**Alternatives to chemical anticoagulation**
An IVC filter is an option, but it is not without its various adverse effects. Purely mechanical thromboprophylaxis is an option, but would be limited to one leg, and would be less effective.

And what if this patient is coagulopathic from his massive transfusion? Should you anticoagulate him, or is he already "auto-anticoagulated"? Well, it turns out, it doesn't matter what you do, these people clot away. For some reason, coagulopathic surgical patients seem resistant to the effects of thromboprophylaxis.

Realistically speaking, what is this guy's risk of having a major extension of his traumatic subarachnoid bleed? Generally one must say that we really don't know. It is known, however, that haemorrhagic stroke patients who have an unchanged second CT brain don't tend to suffer from any extension of their bleeding. Small scale studies of traumatic brain injuries complicated by haemorrhage also failed to detect any significant increase in the rate of bleed extension due to routine DVT prophylaxis.

References:


Edwards, Meghan, et al. "Venous thromboembolism in coagulopathic surgical intensive care unit patients: is there a benefit from chemical prophylaxis?" Journal of Trauma and Acute Care Surgery 70.6 (2011): 1398-1400.


