In-Patient Medicine for Redeployed Doctors

Essential Facts

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In-Patient Medicine for Redeployed Doctors: The Essential Facts

Introduction

In the middle of March 2020, the UK government declared its strategic response to the crisis posed by the Covid-19 pandemic. This announcement was followed by an immediate response from the NHS which enacted profound changes in the provision of all clinical services and a complete upheaval in the way doctors practice medicine. As it became clear that the numbers of extremely ill Covid-19 patients would swamp the existing acute services, a plan was launched to mobilise clinicians from non-acute sectors to reinforce the frontline.

Part of the response at King's College Hospital in London was to create a new, temporary category of consultant clinician: the Tier 2 Physician. Members of this group (dermatologists, rheumatologists, genito-urinary physicians *et al*) were redeployed to Covid wards to act as deputies to the general medicine physicians. Each of us was given the assistance of 1 or 2 junior doctors and the responsibility for one-half of a ward of patients. And so, at extremely short notice, the Tier 2 Physicians found themselves working on the wards, helping to manage Covid pneumonia and the myriad other problems which comprise general internal medicine.

For many of us, the practice of ward-based medicine was last undertaken 20+ years ago, and so the initial days of redeployment were full of anxiety. However, thanks to the generosity of our acute medicine colleagues, and an appetite to (re-)learn bedside skills, the Tier 2 Physicians not only survived but contributed to the care of in-patients. Over these periods 'on the wards' my colleagues and I collected a significant body of clinical know-how, practical knowledge which underpinned our day-to-day work as temporary general physicians. The material assembled for this document, ***In-Patient Medicine for Redeployed Doctors,*** is drawn largely from notes made by my dermatology colleagues during periods of redeployment in 2020 and 2021.

Each topic is presented in a uniform 5 bullet points: the essential facts are heavily compressed with a great deal of ground being covered in a small space. The bullet points are little more than headings, cited to remind the clinician of the salient features of the subject. There are lots of resources for clinicians to turn to for more detailed information, including advice about drug doses and management guidelines. Our document should be used as a prompt to signpost the scope of common ward-based problems encountered on redeployment.

We hope that the material covered in ***In-Patient Medicine for Redeployed Doctors: Essential Facts*** may be useful to doctors who, at short notice, find themselves working in the unfamiliar territory of a general medical ward.

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Use was made of websites and guidelines and general medical textbooks. In particular, I owe a major debt of gratitude to the ***Oxford Handbook of Clinical Medicine*** (Oxford University Press) which has been shamelessly plundered for our Essential Facts. The Oxford Handbook is a giant among pygmy-sized texts: the first edition got me through my houseman's year in 1990, the tenth edition got me through redeployment in 2020/21. Thank you.

Dr Daniel Creamer

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In-Patient Medicine for Redeployed Doctors: Essential Facts

PATIENT ASSESSMENT

National Early Warning Score 2 (NEWS2)

- NEWS2 is a scoring system used to identify clinical deterioration in hospitalised patients
- Physiological parameters measured in NEWS2: resp rate; O_2 sats; supplemental O_2; temp; systolic BP; heart rate; level of consciousness
- NEWS2 generates a score from 0 to 23 via aggregation of weighted values; a threshold of >5 is a trigger for immediate clinical review
- Relative underscoring of hypoxaemia by NEWS2 may compromise its utility in COVID-19, however:
- Longitudinal monitoring of NEWS2 in COVID-19 appears to be sensitive in identifying those at risk of clinical deterioration

Fluid balance assessment

- Normal fluid requirements for 70kg person = 2.0-2.5 L/24 hrs. Normal fluid losses: urine = 1500 ml, stool = 200 ml, insensible = 800ml
- Sources of additional fluid loss: diarrhoea, surgical drains, fever, tachypnoea, erythroderma
- Signs of 'underfill': tachycardia, BP postural drop, decreased urine output, decreased capillary refill time, dry mucous membranes
- Signs of 'overfill': raised JVP, pitting oedema at sacrum, tachypnoea, bibasal crackles, pulmonary oedema on CXR
- Accurate fluid balance assessment: daily weight, volume of fluids in, volume of urine output, fluid balance chart

Glasgow coma scale

- Records conscious state from 3 types of response. Best score = 15, worst score = 3
- *A. Best motor response:* obeys commands (6); localises pain (5); withdraws from pain (4); flexes to pain (3); extends to pain (2); no response to pain (1)
- *B. Best verbal response:* orientated in TPP (5); confused speech (4); inappropriate speech (3); random sounds (2); no sounds (1)
- *C. Eye opening:* spontaneous (4); in response to sounds (3); in response to pain (2); no eye opening (1)
- Simple coma scale is AVPU: Alert; Voice responsive; Pain responsive; Unresponsive

Frailty assessment

- The Clinical Frailty Scale (CFS) is a judgement-based frailty tool that evaluates domains including comorbidity, function, and cognition. It can be used to identify patients who may not benefit from critical care interventions
- Grade 1: fit, regular physical activity. Grade 2: well, occasional physical activity
- Grade 3: well but not active. Grade 4: vulnerable but independent, symptoms are limiting. Grade 5: mildly frail, needs help with some home tasks
- Grade 6: moderately frail, needs help with all home tasks. Grade 7: severely frail but stable, completely dependent. Grade 8. very severely frail, completely dependent, approaching end of life
- Grade 9. terminally ill, life expectancy < 6 months, without being evidently frail

Treatment escalation, levels of care and resuscitation

- For every in-patient, a ceiling of care and treatment escalation plan needs to be discussed and documented. Levels of care are:
- Level 1: Patient's needs can be met on an acute ward with, if needed, additional advice and support from critical care team. Patient would not be for admission to a HDU or ITU setting - 'ward-based ceiling of care'
- Level 2: If needed, patient requires HDU management with intervention including support for 1 or 2 failing organ system, eg haemofiltration / inotrope support / NIV ('level 2 bed on ITU'). However, the patient is not for intubation
- Level 3: If needed, patient requires ITU admission for intubation and full supportive therapy but not resuscitation in the event of a cardiac arrest
- DNAR: the decision to attempt CPR if warranted during an admission needs to be discussed with the pt. If the pt does not have capacity, DNAR decision must be discussed with the NOK. A Consultant or SpR must approve the DNAR form

<u>CHEST MEDICINE</u>

Acute exacerbation of asthma

- Features of severe asthma attack: resp rate >25/min; PR >110 beats/min; peak exp flow <50% of predicted; unable to complete sentences
- Clinical signs: wheeze, hyperinflated chest, hyper-resonant percussion, increased work of breathing, use of accessory muscles
- Investigations: decreased peak exp flow; expanded lung fields on CXR; ABG showing normal/low PaO_2 and low $PaCO_2$. Check FBC, U&E, CRP, blood cultures, sputum culture

- Give salbutamol 5mg/4 hr nebulised with O_2, maintaining sats at 94-98%. Steroids: prednisolone 40mg po, or hydrocortisone 100mg IV
- If peak expiratory flow remains <75%, repeat salbutamol neb and add ipratropium neb 500mcg/6 hr. If $PaCO_2$ is raised (signifying falling respiratory effort) --> transfer to HDU

Acute exacerbation of COPD

- Features of COPD exacerbation: increased cough & sputum; worsening SOB; decreased exercise tolerance
- Differential diagnosis: asthma; pulmonary oedema; pulmonary embolism; pneumothorax; LRTI
- Investigations: FBC (increased PCV); CXR (hyperinflated, flat diaphragm); lung function tests (obstructive FEV1<80%); ECG (R atrial and R ventricular hypertrophy); ABG shows P_aO_2 decreased with increased PaCO2 (can also be low); send U&E, CRP, blood cultures, sputum culture
- Give salbutamol neb 5mg/4 hr + ipratropium neb 500mcg/6 hr. Controlled O_2 therapy, aim for sats of 88-92%. If evidence of infection, give co-amoxiclav / doxycycline / clarithromycin
- If poor response, consider non-invasive positive pressure ventilation (NIPPV) --> transfer to HDU

Covid-19 pneumonia: diagnosis

- COVID-19 is caused by SARS-CoV-2 (confirmed with RT-PCR on a nasopharyngeal swab). 15% patients develop severe pneumonia, 5% suffer a critical syndrome of respiratory failure, shock, multi-organ failure
- Symptoms: fever, new & continuous cough, dyspnea, fatigue, loss of appetite, anosmia (loss of smell), ageusia (loss of taste)
- Signs: fever, tachycardia, tachypnea, widespread crackles throughout chest
- Tests indicating severe COVID-19: low 0_2 sats, lymphopenia, high CRP, elevated D-dimers, elevated troponin-1, raised urea & creatinine, cytokines
- Imaging: CXR: asymmetric peripheral ground-glass opacities without pleural effusions. CT chest: bilateral multi-lobar ground-glass opacities with a peripheral, asymmetric and posterior distribution. If concerns about PE: order CTPA

Covid-19 pneumonia: extra-pulmonary features

- Extra-pulmonary manifestations are caused by direct viral injury, uncontrolled inflammation ('cytokine storm'), activation of coagulation, and activation of

complement. Coagulopathy + endothelial dysfunction ('thrombo-inflammation') underlies susceptibility to PE and other thrombotic complications
- Kidneys: AKI from pre-renal causes and renal thrombotic microangiopathy
- Gut & Liver: diarrhoea, abdo pain, vomiting, acute liver injury (direct viral cytopathic effect / uncontrolled immune reaction)
- Heart: myocarditis, myocardial ischaemia, ventricular dysfunction
- Skin: perniosis on fingers and toes ('Covid toes'), urticaria, papulo-vesicular exanthem

Covid-19 pneumonia: management

- Deliver oxygen to maintain O_2 sats at 92-96%
- Dexamethasone 6mg od for 10 days. IV Remdesevir can be used depending on COVID severity (contra-indicated if ALT is >5x ULN, or eGFR is <30ml/min). Dose = 200mg day 1, then 100mg daily for next 4 days.
- IV fluids often needed to avoid dehydration. Thromboprophylaxis in all patients (unless contra-indicated). Treatment dose enoxaparin if documented PE or other thrombus.
- Proning can improve ventilation/oxygenation. Regimen of positions: lying fully prone, lying on right side, sitting up, lying on left side, and repeat. Each position for 0.5 – 2 hours
- Increased work of breathing indicates disease deterioration. Consider referral for noninvasive ventilation or endotracheal intubation & mechanical ventilation

Pneumonia (non-Covid-19)

- Up to 40% adults with community acquired pneumonia require hospital admission which is associated with a mortality rate of 5-14%
- *Strep pneumoniae* and respiratory viruses are commonest causes. *Mycoplasma pneumoniae* and *Legionella spp*. cause atypical pneumonia
- Symptoms: fever, productive cough, dyspnoea, pleuritic chest pain, confusion (in elderly). Atypical pneumonia: insidious onset + extrapulmonary manifestations
- Investigations: FBC, U&E, CRP, LFT, ABGs, blood & sputum culture, urinary pneumococcal and legionella antigens. CXR: consolidation, cavitation, effusion
- Management: IV fluids, oxygen, antibiotics according to local policy (eg po amoxicillin or clarithromycin or doxycycline)

Pulmonary embolism (PE)

- Symptoms: acute dyspnoea, pleuritic chest pain, haemoptysis, syncope
- Signs: tachycardia, tachypnoea, cyanosis, hypotension, pleural rub, raised JVP

- Investigations: FBC, U&E, clotting, D-dimer (raised), ECG (right ventricular strain), CXR, ABGs, CTPA (sensitive & specific)
- Management: oxygen 10-15L/min (if hypoxic); IV morphine 5-10mg (if ++pain / distress); start IV low mol weight heparin
- If there is haemodynamic instability, consider thrombolysis

Respiratory failure

- Respiratory failure (RF) occurs when gas exchange is inadequate. Defined as hypoxia with PaO_2 < 8 kPa
- Type I RF: PaO_2 < 8 kPa with normal or low $PaCO_2$. Examples: pneumonia, pulmonary oedema, PE, asthma, Covid-19 pneumonia
- Management of type I RF: Treat underlying cause; give O_2 via nasal cannulae or mask; consider assisted ventilation
- Type II RF: PaO_2 < 8 kPa with $PaCO_2$ > 6 kPa. Examples: COPD, obstructive sleep apnoea, sedative drugs, end-stage ILD, neuromuscular disease
- Management of type II RF: Treat underlying cause; controlled O_2 therapy *(O_2 must be given with care since respiratory centre is insensitive to CO_2 & ventilation is driven by hypoxia*); consider assisted ventilation

Oxygen delivery systems

- Titrate the percentage or fraction of O_2 (FiO_2) delivered according to the patient's sats & clinical condition
- Nasal cannulae: flow rate 1-4 L/min = 24-30%
- Venturi Blue: 2-4 L/min = 24%; White: 4-6 L/min = 28%; Yellow: 8-10 L/min = 35%
- Red: 10-12 L/min = 40%; Green: 12-15 L/min = 60%
- Non-rebreathe mask: 15 L/min = 90%

CARDIOLOGY

Chest pain: causes & investigation

- Chest pain can be divided into cardiac-type chest pain and non-cardiac-type chest pain. Musculoskeletal chest pain is the most frequent diagnosis
- Cardiac-type chest pain: rapid-onset, heavy/tight, retrosternal, associated with sweating and pallor, exacerbated by exertion, relieved by GTN or rest: Causes: ST segment elevation MI (STEMI), non-ST segment elevation MI (NSTEMI), unstable angina. These three entities are collectively acute coronary syndrome (ACS). Pericarditis & aortic dissection cause non-ischaemic cardiac-type pain

- Investigations of cardiac-type chest pain: serial ECGs, bloods (FBC, U&E, LFT, lipids, serial troponin), CXR. Second-line investigations include: trans-thoracic echocardiogram, angiography, and exercise testing
- Non-cardiac-type chest pain - Causes: respiratory (pulmonary embolism, viral infection with pleurisy, pneumonia), musculoskeletal (costochondritis, chest wall injury), gastro-intestinal (GORD, cholecystitis, pancreatitis), neurogenic (shingles)
- Investigations of non-cardiac-type chest pain: bloods (FBC, U&E, LFT, CRP, troponin), ECG, CXR, ABG if respiratory cause suspected, CTPA (if PE suspected)

Acute coronary syndromes

- Symptoms of ACS: central chest pain >20mins, nausea, sweating. Silent ACS: no chest pain, but syncope, pulmonary oedema, acute confusion +/- epigastric pain
- ST elevation MI (STEMI) early ECG changes: ST elevation, tall T wave, new LBBB; late ECG changes: Q wave & T wave inversion
- NSTEMI / unstable angina ECG changes: ST depression & T wave inversion
- Cardiac troponin levels (T & I) are most sensitive and specific marker of myocardial necrosis. Troponin also up in myocarditis, pericarditis, ventricular strain
- Management of ACS**:** aspirin 300mg + ticagrelor 180mg, morphine 10mg IV + metoclopramide 10mg IV. Refer for percutaneous intervention (PCI)

Acute heart failure

- Acute heart failure is the leading cause of hospital admission in people 65+ years. It carries a 30-day mortality of 15% with NT-proBNP > 5000ng/L
- Investigations: CXR, FBC, U&E, LFT, NT-proBNP, troponin, ABGs, echocardiogram (with new diagnosis of heart failure)
- NT-proBNP levels: acute heart failure is likely if >450 ng/L in <50yrs; >900 in 50-75 yrs; >1800 in >75 yrs
- Treatment: O_2 to maintain SaO_2. IV furosemide 40-80mg bolus followed by infusion at 5-20 mg/hr (if required). Consider IV GTN infusion (10-200 micrograms/min)
- Monitor pulse, oximetry and blood pressure every 10 mins with continuous ECG. Maintain systolic BP > 100mmHg. If cardiogenic shock develops: urgent cardiology review

Atrial fibrillation

- Causes: heart failure, hypertension, IHD, PE, mitral valve disease, pneumonia, hyperthyroidism, caffeine, alcohol

- Symptoms: can be asymptomatic or cause chest pain, palpitations, faintness, dyspnea. Signs: Irregularly irregular pulse. ECG: absent P waves & irregular QRS complexes
- If patient is in shock → ABCDE and consider DC cardioversion +/- amiodarone. Correct electrolyte disturbances
- Patient stable and AF started <48 hrs ago: DC cardioversion / flecainide / amiodarone + anticoagulation. Patient stable and AF started >48 hrs ago: rate control with bisoprolol or diltiazem + anticoagulation
- Main risk of AF is embolic stroke. Use a DOAC (or warfarin: target INR 2-3) if high risk of emboli

DIABETES & ENDOCRINOLOGY

Diabetic ketoacidosis (DKA)

- Take blood: FBC, U&E, glucose, lactate, venous blood gas, blood culture. Contact the diabetes team & ITU
- Insert 2x iv cannulae & urinary catheter. Fluid resus: give 1 litre NaCl 0.9% (without potassium) over 15 mins. Then give another 1 litre NaCl 0.9% (without potassium) over 1 hour
- IV insulin infusion via 2nd cannula: Actrapid 50 units in 50ml 0.9% NaCl, infuse at 6 units/hr. If blood glu <14mmol/L, infuse 10% gluc at 100ml/hr as well as NaCl and reduce insulin to 3 units/hr. If pt was taking insulin beforehand continue long-acting but stop short- & intermediate-acting
- After the first 2 litres of fluid switch to 0.9% NaCl with 40mmol KCl (if serum K is NOT >5.5mmol/L). Administer 2 litres over the next 4 hours at 500ml/hr
- Hourly monitoring of: NEWS, blood glucose, fluid balance, electrolytes (esp K^+). Call HDU/ICU if any impairment of consciousness

Hyperosmolar hyperglycaemic state (HHS)

- Occurs in unwell patients with type 2 DM. Blood glu >30mmol/L + severe dehydration. No ketones, no acidosis
- Patients are typically ~10L of fluid in deficit
- Patients are at risk of vaso-occlusive events: give prophylactic low mol weight heparin
- Rehydrate slowly with 0.9% saline over 48 hrs. Replace K^+ when urine starts to flow
- Only use insulin if a) blood glucose not falling by 5mmol/L/hr, or b) if there is ketonaemia

Addisonian crisis

- Most commonly occurs in patients on long-term steroids. Causes: infection, trauma, surgery, missed medication
- Patients present in shock (tachycardia, postural hypotension, oliguria, confusion, coma) and hypoglycaemia
- Urgent bloods: cortisol, ACTH, U&E (low Na, high K), glucose (often low – correct it and monitor)
- Treatment: hydrocortisone 100mg IV stat, and thereafter 100mg IV or IM 8-hourly
- Fluid bolus of 500ml of IV 0.9% saline, and repeat as necessary

RENAL MEDICINE

Acute kidney injury (AKI): causes

- **Pre-Renal** (most common): reduced renal perfusion - periods of hypotension or shock, hypovolaemic states (haemorrhage, burns, GI losses), reduced cardiac output
- **Intrinsic Renal:** nephrotoxic insults (medications, IV contrast media), glomerulonephritis, acute interstitial nephritis (driven by drugs or infection), autoimmune nephritis, small vessel vasculitis, tubular pathology (acute tubular necrosis or rhabdomyolysis)
- **Post-Renal** (least common): obstructive states, eg hydronephrosis, urinary retention
- Symptoms of AKI include nausea, vomiting, reduced urine output, confusion, fatigue and altered consciousness
- *Stage 1*: Creatinine rise 1.5-2 fold from baseline or ≥26.4µmol/L and/or urine ≤0.5ml/kg/hr > 6h. *Stage 2*: Creatinine rise 2-3 fold from baseline and/or urine ≤0.5ml/kg/hr > 12 h. *Stage 3:* Creatinine rise >3 fold from baseline or ≥354µmol/L and/or urine ≤0.3ml/kg/hr > 24 h

Acute kidney injury (AKI): assessment & principles of management

- Assess stage of AKI (above). Assess intravascular volume status (capillary refill / JVP / thirst / pulse / BP / urine output). Exclude sepsis. Management is supportive to normalize renal perfusion & renal recovery whilst screening for the cause
- Investigations: urine dip (leu & nitrates = infection; blood & protein = nephritis); send urine MC+S; send urine protein: creatinine ratio; send renal screen: FBC, U&E, creatine kinase, bone profile, Igs & electrophoresis, ANA, ENA, ANCA, ds-DNA, complement, HIV, Hep B/ C, anti-GBM abs; send venous blood gas for pH status (metabolic acidosis). Exclude an obstructive cause: US KUB
- Stop nephrotoxic drugs, eg ACE inhibitors, ARBs, NSAIDs, aminoglycosides, diuretics
- Give IV fluids to establish normal fluid status. Fluid bolus = 250ml of 0.9% NaCl IV, repeat until clinically euvolaemic

- Involve the renal team early. Indications for emergency haemodialysis: refractory pulmonary oedema, refractory hyperkalaemia, uraemic encephalopathy, pericarditis

CLINICAL CHEMISTRY

Arterial Blood Gases

- pH < 7.35 = acidosis; pH > 7.45 = alkalosis
- Metabolic acidosis pH: low $PaCO_2$: N/low HCO_3: low
  Causes: lactic acid (eg shock, sepsis), urate (eg renal failure), ketones (eg DKA)
- Respiratory acidosis pH: low $PaCO_2$: high HCO_3: N/high
  Causes: Type 2 respiratory failure (eg COPD)
- Metabolic alkalosis pH: high $PaCO_2$: N/high HCO_3: high
  Causes: vomiting, K depletion (eg diuretics)
- Respiratory alkalosis pH: high $PaCO_2$: low HCO_3: N/low
  Causes: hyperventilation (eg stroke, asthma, anxiety)

Hyponatraemia

- Defined as serum Na <135 mmol/L. Symptoms vary: Na >125 asymptomatic; Na 115-125 confusion, lethargy, nausea, vomiting; Na <115 weakness, seizures, coma
- Causes include: excessive water/beer intake, SIADH, hypergylcaemia, heart/liver/renal failure. Commonly implicated drugs: SSRIs, PPIs, steroids, diuretics
- Assess: fluid balance status for hypo/eu/hypervolaemia. Measure: paired serum / urine osmolarities and urinary Na
- Management: symptomatic hyponatraemia or Na <126mmol/L or hyponatraemia resistant to treatment - seek guidance from Endocrine SpR. Precise management is dependent on cause, but includes: stopping non-essential culprit drugs, fluid restriction (if hypervolaemic), or IV 0.9% or hypertonic saline if eu/hypovolaemic
- When hyponatraemia has been present for >48hours replacement must be slow to reduce risk of osmotic demyelination syndrome. Target increase is 8-10 mmol/L in 24 hours

Hyperkalaemia

- The risk of hyperkalaemia (>6.5 mmol/L) is myocardial hyperexcitability → VF → cardiac arrest
- Causes: oliguric renal failure, K^+ sparing diuretics, rhabdomyolysis, metabolic acidosis, Addison's disease
- Signs & symptoms: tachycardia, palpitations, chest pain, weakness, faintness. ECG: small P waves, wide QRS, tented T waves
- Non-urgent treatment: treat underlying cause, give po calcium resonium 15g TDS

- Urgent treatment: IV calcium chloride (10ml of 10% calcium chloride via big vein over 10 mins), plus IV insulin (10 units soluble insulin in 50ml of 50% glucose over 30-60 mins). Monitor for hypoglycaemia

GASTROENTEROLOGY

Abdominal pain

- Causes of acute abdomen according to site: Right upper quadrant – cholecystitis, duodenal ulcer, hepatitis, pneumonia, pyelonephritis. Left upper – ruptured spleen, gastric ulcer, pyelonephritis, pneumonia. Right lower: appendicitis, salpingitis, ectopic pregnancy, renal tract stone, mesenteric adenitis, colitis, hernia. Left lower: salpingitis, ectopic pregnancy, renal tract stone, diverticulitis, colitis, hernia
- Abdo pain + diarrhoea without blood**:** IBS (pain with alternating constipation/diarrhoea, bloating, mucus PR); diverticulitis (altered bowel habit with L-sided colicky pain +/- fever); *Norovirus* (48 hrs of watery diarrhoea, vomiting, pain); Enterotoxigenic *E coli* (3 days of watery diarrhoea + cramps)
- Abdo pain + diarrhoea with blood: ulcerative colitis & Crohn's colitis (diarrhoea with blood & mucus, crampy pain, abdominal tenderness); *Campylobacter* (bloody diarrhoea, pain, fever, headache); *Yersinia* (bloody diarrhoea, pain, vomiting, fever, erythema nodosum)
- Acute pancreatitis: epigastric or central abdo pain → back + severe vomiting. Investigations: raised amylase & lipase. Management: nil by mouth, IVI, opiate analgesia. Close monitoring of O_2 sats, bloods (incl glucose) & vital signs
- Other causes of abdo pain: MI; mesenteric ischaemia; leaking AAA; perforated viscus; peritonitis

Diarrhoea: causes and investigation

- Common causes of acute diarrhoea: GI infections, medications, overflow. More rarely: appendicitis, diverticulitis, intestinal ischaemia
- Causes of chronic diarrhoea: IBS, inflammatory colonic disease (diverticulitis, IBD), colorectal cancer, small bowel disease (coeliac, Crohn's, bile acid disorder), pancreatic insufficiency, endocrine causes, chronic infection, alcohol abuse
- Drug causes: antibiotics, metformin, oral magnesium or magnesium-containing antacids, PPIs, SSRIs, NSAIDs, allopurinol, colchicine
- Investigations include: routine bloods; stool for culture/sensitivity, ova, cysts and parasites; stool for *C.difficile*; tests for malabsorption (iron studies, B12 and folate); Coeliac screen; TFTs; faecal elastase; AXR to look for faecal loading. A PR exam should be considered: ?rectal mass, ?faecal loading

- Keep stool chart and fluid balance chart. Complications of diarrhoea: dehydration, electrolyte disturbance, AKI

Antibiotic-associated diarrhoea (AAD)

- AAD occurs due to disruption of the gut microbiome and direct effects on GI mucosa
- ~30% of AAD is due to infection by toxin-producing strain of *Clostridium difficile* caused by antibiotic therapy. Diagnosis: send stool sample to microbiology for immunoassay and toxin detection
- Antibiotics commonly associated with *C.difficile* infection: broad-spectrum penicillins, cephalosporins, clindamycin, fluoroquinolones. Additional risk factors: increased age, acid suppressants (eg PPIs), underlying morbidity
- *C.difficile* diarrhoea produces spores which can be passed onto others. Confirmed cases of *C.difficile* infection should isolated in a side-room & barrier nursed
- Management of AAD: stop or rationalise antibiotics; specific management: mild - po metronidazole, severe - po vancomycin or fidaxomicin. Complications: pseudomembranous colitis, toxic megacolon

Upper gastro-intestinal bleeding

- Typical causes of haematemesis & melaena are peptic ulcer, Mallory-Weiss tear, oesophageal varices, drugs, upper GI cancer
- Investigations: FBC, U&E, LFT, coag, x-match, CXR, ABGs, ECG
- Insert IV line and urinary catheter (+/- central line). Give IV fluids or blood (O Rh neg, if needs be)
- Correct clotting abnormalities (vit K, FFP, platelets). If varices, consider IV terlipressin (1-2mg/6 hrs)
- Arrange urgent endoscopy

Decompensated cirrhosis

- Acute decompensation of chronic liver disease carries a high mortality. It is associated with sepsis, AKI, variceal bleeding, alcohol / drug abuse, hepatocellular carcinoma, cardiorespiratory failure
- At presentation look for precipitating cause (eg sepsis). Investigations: FBC, U&Es, LFTs, coag, blood lactate and ammonia, serum AFP, US liver, urine & blood culture. Patients should be assessed for suitability of liver transplant.
- First manifestation of liver decompensation is often ascites. Perform paracentesis: cell count, culture and protein level. Start low sodium diet - if this fails, start spironolactone 100 mg OD +/- furosemide 20 mg OD

- Encephalopathy manifests as a variety of neuro-psychiatric abnormalities ranging from behaviour changes to asterixis and coma. Primary management is oral lactulose → >2x loose bowel movements / day. Involve ITU early in deeply encephalopathic patients due to poor airway reflexes and risk of aspiration. These patients require daily phosphate enemas.
- Spontaneous bacterial peritonitis (SBP) presents with fever, reduced consciousness and abdominal pain. Risk factors: Child-Pugh Grade C cirrhosis, ascitic protein <10, upper GI bleed. Diagnosis: ascitic neutrophil count >250, or mono-microbial ascites culture. Treatment: broad spectrum antibiotics, eg piperacillin/tazobactam.

Constipation

- Constipation: <2 bowel motions/week with straining, pain or sense of incomplete evacuation (tenesmus). Exclude serious pathology:
- Constipation + rectal bleeding +weight loss: ?colorectal cancer; Constipation + lack of flatus: ?GI obstruction; Constipation + menorrhagia: ?hypothyroidism
- In the elderly, check stool chart daily, constipation is a common cause of delirium. If there is loss of weight, PR bleed, & microcytic anaemia: PR exam is essential
- Give pre-emptive dietary advice if prescribing drugs that constipate (opioids, iron, furosemide, Ca^{2+} channel blockers, aluminium antacids, tricyclics)
- Investigations: in elderly - FBC, ESR Ca^{2+}, TFT. In young - nil. AXR will show faecal loading

Laxatives & management of constipation

- Increase oral fluid intake when treating constipation (especially important in elderly)
- First line: *Bulking agents* - eg ispaghula husk (Fybogel) 1 sachet in water BD after meals. Not in elderly, faecal impaction or opioid constipation
- Second line: *Osmotic laxatives* - eg lactulose 15 ml BD, or macrogol (Movicol) 1-3 sachets daily. Can cause bloating
- Third line: *Stimulants* - eg senna 2 (to 4) tablets at night for 3 days, or bisacodyl 5- 10 mg ON, or docusate sodium 100 mg TDS. Good for opioid constipation; can cause low K^{+}
- Additional: *Rectal stimulants* - eg glycerin suppositories; s*tool softeners* - eg arachis oil enema; m*icro-enemas* - eg docusate sodium or sodium citrate

INFECTIONS

Urinary tract infection

- Dysuria, frequency, nocturia, fever, suprapubic pain, haematuria, anuria. Catheterised patients at particular risk. UTI is common cause of new-onset confusion or delirium, esp in older patient
- Investigations: urine dipstick, MSU or catheter bag specimen. If systemically septic (NEWS >3) take blood cultures and other sepsis investigations
- If NEWS ≥3, resuscitate the patient, start empiric antibiotic therapy in keeping with Trust antimicrobial guidance
- If NEWS ≤2, assess patient for dysuria, new nocturia, cloudy urine, malodorous urine:
- If 2 or 3 of these symptoms are present, start empiric antibiotic therapy. If 1 or none of these symptoms present, await MSU analysis

Infective endocarditis

- Fever + new murmur = endocarditis. Also: rigors, night sweats, weight loss, malaise. Often asymptomatic. Consider in pts with prosthetic valve, previous endocarditis, or PUO
- Other signs: splenomegaly, splinter haemorrhages, Janeway lesions, Osler's nodes, peripheral skin infarcts & other signs of emboli
- Investigations: 3 sets of blood cultures taken at 30-minute intervals. Highlight investigating endocarditis → prolonged incubation. Urinalysis: microscopic haematuria
- Common organisms: *Strep viridans, Staph aureus, Strep bovis,* enterococci, *Coxiella burnetii.* Send serum for: Coxiella, Mycoplasma, Brucella, Legionella
- Contact microbiology urgently to guide antibiotic prescribing

Sepsis

- Consider sepsis if: NEWS ≥3; new confusion/agitation; increased oxygen requirement; concern raised by staff
- Clinical features to help identify source: new respiratory symptoms; new urinary symptoms; soft tissue infection; hot joint
- Investigations 1: FBC, U&E, LFT, pro-calcitonin, bone profile, coag, CRP, VBG for lactate
- Investigations 2: blood culture, urine culture, CXR, skin/mucosal swab, joint aspirate
- Management: Give oxygen (O_2 sats 94-98%), IV antibiotics within 1 hour (local antimicrobial policies), IV fluid if shocked (systolic BP>100), monitor urine output

Use of major antibiotic classes

- Penicillins (eg benzylpenicillin): gram + infection, *Strep*, cellulitis, endocarditis. Piperacillin + tazobactam (Tazocin): gram + & gram - infections, *Pseudomonas*, neutropenic sepsis
- Carbapenems (eg meropenem): gram + & gram - infections, aerobes, anaerobes
- 3rd generation cephalosporins (eg ceftriaxone): gram + & - infection, meningococcus, not *Pseudomonas*
- Aminoglycosides (eg gentamicin): gram - infection
- Lipopeptides (eg vancomycin): gram + infection, MRSA, given po for *C. difficile*

NEUROLOGY

Headache

- The life-threatening disorders associated with a headache include:
- Sub-arachnoid haemorrhage**:** sudden onset headache, often occipital, 'worst ever', neck stiffness, meningism, decreased consciousness
- Venous sinus thrombosis: headache of sub-acute onset, cough-initiated pain, worse in morning & bending forward, papilloedema
- Meningitis: severe headache & photophobia, neck stiffness, meningism, fever, purpura, decreased consciousness
- Encephalitis: infectious prodrome + headache, fever, meningism, focal neurology, odd behaviour, decreased consciousness

Acute haemorrhagic stroke

- Suspect in sudden onset focal neurological deficit which is ongoing or >24 hrs. FAST and ROSIER = CVA diagnostic tools. Exclude hypoglycaemia, perform urgent non-contrast CT head
- If suspected / confirmed haemorrhagic CVA, contact stroke team & consider neurosurgical intervention. Previously fit pt should be considered for surgery following intracerebral haemorrhage if they have hydrocephalus
- The following should receive medical (not surgical) treatment: small deep haemorrhages; lobar haemorrhage without either hydrocephalus or rapid neurological deterioration; large haemorrhage + significant pre-CVA comorbidities; GCS < 8 (unless dues to hydrocephalus); posterior fossa haemorrhage
- Patients taking anticoagulants should have this treatment stopped and reversed. If on warfarin return clotting to normal using IV vitamin K
- Offer rapid BP control if a) <6 hours onset and systolic BP 150-220mmHg, or b) >6 hours onset or systolic BP >220mmHg. Aim for a systolic target of 130 to 140 mmHg within 1 hour of starting treatment and maintain this blood pressure for at least 7

days. Avoid BP control if: underlying structural cause; GCS < 6; early neurosurgery to evacuate haematoma; massive haematoma + poor prognosis

Acute ischaemic stroke

- Suspect in sudden onset focal neurological deficit which is ongoing or >24hrs. FAST and ROSIER = CVA diagnostic tools
- Exclude hypoglycaemia and perform urgent non-contrast CT head. Once haemorrhagic stroke excluded, administer 300mg aspirin PO/PR +/- PPI. Contact stroke team
- Thrombolysis with alteplase if stroke <4.5hrs onset and no contraindications (administered by staff experienced in thrombolytics). Referral for mechanical thrombectomy if meets time of onset criteria & radiological criteria, and no contraindications
- Do not lower BP in acute phase unless hypertensive complications (or having thrombolysis where BP<=185/110). Maintain glucose, O_2, temp, and hydration
- Ischaemic stroke + AF: give aspirin for 2 weeks before consideration of anticoagulant. Do not give warfarin in acute phase. If already on anticoagulation, switch to aspirin for 7 days to reduce risk of haemorrhagic transformation

Status epilepticus: principles of management

- Status epilepticus = seizures lasting >30mins, or repeated seizures without recovery periods. Risk of permanent brain damage and death increases with length of attack
- Secure the airway. Use suction. Give 100% oxygen
- Urgent bloods: FBC, U&E, glu, Ca, toxicology, anticonvulsant levels (when appropriate)
- IV lorazepam 4mg as slow bolus. If no response, give 2nd dose after 15mins
- If fits continue, give phenytoin infusion at 50mg/min (total dose for 60kg pt = 1g; dose for 80kg pt = 1.5g; max dose = 2g)

HAEMATOLOGY

Investigation of anaemia

- Acute anaemia: examine for signs of blood loss / haemodynamic instability. Resuscitate. Investigations: FBC, U&E, LFT, coagulation screen, group and save/crossmatch and fibrinogen (in massive haemorrhage). Endoscopy or interventional radiology may be required to identify / control bleeding source
- Chronic anaemia: classify as microcytic (MCV < 80 fL), normocytic (MCV 80-100 fL) or macrocytic (MCV > 100 fL)

- Causes of microcytic anaemia: iron deficiency, anaemia of chronic disease, thalassemia. Investigations: serum iron, total iron binding capacity, transferrin saturations, ferritin, Hb electrophoresis (thalassaemia)
- Causes of normocytic anaemia: blood loss, anaemia of chronic disease, chronic kidney disease, haemolytic anaemia. If blood loss and anaemia of chronic disease excluded undertake haemolysis screen: blood film, reticulocytes, haptoglobin, bilirubin, lactate dehydrogenase, direct antiglobulin test
- Causes of macrocytic anaemia: vitamin B12 or folate deficiency, hypothyroidism, pregnancy, drugs (methotrexate, azathioprine) and myelodysplastic syndromes

Deep vein thrombosis (DVT)

- Suspect DVT when unilateral, painful, swollen, red leg. Risk factors: immobility, trauma, hormone treatment, pregnancy, cancer
- Undertake two-level DVT Wells score for DVT likelihood: >2 likely DVT; <2 unlikely DVT
- If **likely:** ultrasound doppler of leg within 4 hrs. if unable to perform immediate US, then check D-dimer, anticoagulate (check Trust guidelines), arrange US within 24 hrs. If US negative, stop anticoagulation and repeat US in one week
- If **unlikely**: check D-dimer first. If positive, arrange US. If unable to perform US within 4 hrs then anticoagulate and arrange US within 24 hours. If D-dimer +/- US negative then consider alternative diagnosis
- Management: anticoagulate for 3 months in provoked DVT (clear trigger); anticoag for 6 months in unprovoked DVT; anticoag lifelong for recurrent DVT

VTE risk assessment & prophylaxis

- Assessment of venous thromboembolism (VTE) risk against the risk of bleeding from anti-coagulation should be undertaken in all patients admitted to hospital
- Assess as soon as possible after admission and start pharmacological prophylaxis within 14 hours of admission
- Mechanical prophylaxis using anti-embolism stockings unless there is arterial disease, peripheral neuropathy or severe leg oedema. Intermittent pneumatic compression is first line for acute stroke and trauma patients
- Pharmacological VTE prophylaxis with low molecular weight heparin for acutely ill medical patients and surgical patients. Dose adjustment or use of unfractionated heparin if there is renal impairment
- VTE prophylaxis for medical patients usually stops on discharge. Check hospital guidelines for continued prophylaxis in post-operative patients

DERMATOLOGY

Red Legs

- Swelling and redness of the lower legs & feet is common in the elderly, immobile and obese. Sleeping in a chair contributes to the pathogenesis. Exacerbated by heart failure
- The disorder, caused by failure of both lymphatic and venous vasculature, is termed lymphovenous insufficiency or the dependency syndrome
- Involved skin is sore and feels hot. The redness and swelling may be complicated by venous eczema, serous exudate ('weeping') and oedema blisters
- Lymphovenous insufficiency is often mistaken for bilateral cellulitis. Lesional skin can become secondarily infected, but true cellulitis of both legs is rare
- Having excluded arterial disease (ABPI) management is compression bandaging / hosiery. Also: topical skin therapy, heart failure treatment, sleeping in bed, leg elevation

Pruritus (itching)

- Itch is a common symptom in hospital in-patients. Although often multifactorial, itch can be divided into skin causes and systemic causes (including drugs)
- Skin causes: dermatitis (atopic, allergic contact, irritant contact, venous), psoriasis, urticaria (can be drug-induced), scabies, senescence (old age)
- Systemic causes: chronic kidney disease (worse in haemodialysis than peritoneal dialysis); hepatobiliary disease (generalised itch or localised to hands/feet); Hodgkin's lymphoma; polycythaemia vera (aquagenic itch); myelodysplasia; hyperthyroidism
- Drug-induced itch: opiates, imatinib. Urticaria can be caused by NSAIDs, codeine, morphine, iodinated radio contrast. Skin dryness & itch can be caused by diuretics
- Management: Treat underlying condition / stop causative drug. Treat skin inflammation with emollient & topical steroid. Treat urticaria with regular antihistamine. Dermatitis is commonly infected with *Staph aureus* (or MRSA); scabies is highly contagious

Leg ulcers

- Most leg ulcers are venous in origin, some are arterial, some are mixed arterio-venous. Leg ulcer complications: pain, immobility, infection, discharge, smell
- Venous ulcer: medial lower leg, above malleolus, surrounding venous eczema & lipodermatosclerosis

- Venous ulcers are best treated with 4-layer compression bandaging (once arterial disease excluded by ABPI): Layer 1 - padding bandage; layer 2 - crepe bandage; layer 3 - compression bandage; layer 4 - cohesive bandage
- Arterial ulcer: lateral lower leg or dorsal foot, painful (especially at night), well-defined borders, 'punched out'. Often requires vascular surgery input
- Other causes of a leg ulcer: pyoderma gangrenosum (rapid expansion, dusky margins, pain++), basal cell carcinoma (rolled, pearly margins), squamous cell carcinoma (heaped, keratotic margins), vasculitis (surrounding purpura, stellate outline), calciphylaxis (chronic renal failure, stellate outline, pain++)

RHEUMATOLOGY

Painful joint

- Causes of a monoarthritis: septic arthritis, gout, OA. Causes of oligoarthritis (<5 joints): gout, psoriatic arthritis (PsA), reactive arthritis, ank spon. Causes of polyarthritis (> 5 joints): RA, OA, reactive arthritis, PsA
- Septic arthritis: acutely inflamed joint + fever. Risk factors: RA, DM, immunosuppression, chronic kidney disease, prosthetic joint. Organisms: *Staph aureus*, streps, *N gonococcus*, Gram negatives. Management: IV fluclox OR IV vancomycin + 3rd generation cephalosporin
- Gout: >50% affects MTP joint of big toe. Investigation: raised plasma urate, crystals on joint aspiration. Precipitants: surgery, starvation, infection, diuretics. Management: high-dose NSAID OR colchicine OR prednisolone
- Osteoarthritis: can be localised, usually hip or knee. Examination: joint tenderness, mild synovitis, crepitus, pain. Management: paracetamol + topical or oral NSAID
- Rheumatoid arthritis: swollen, painful, small joints of hands and feet; can rarely present with monoarthritis. Accompanied by extra-articular features: nodules, ILD, pericarditis, eye signs. Management: early use of DMARDs & biologics can prevent joint destruction

Back pain

- Back pain caused by malignant or infective disorders should be considered if: acute in onset, pt on immunosuppression, in presence of fever, night sweats & weight loss
- Signs of sinister back pain are progressive pain, nocturnal pain, thoracic pain, bilat or alternating pain, sphincter disturbance, exercise-related leg weakness (=spinal stenosis). The 2 most important syndromes are:
- Acute cord compression: bilateral pain, sphincter disturbance, LMN signs at level of compression, UMN and sensory loss below level of compression

- Acute cauda equina compression: nerve root pain in legs, saddle anaesthesia, bladder & bowel incontinence
- If there is a neurological deficit organise urgent neurosurgical review

COGNITION & CONFUSION

Agitation: causes & management

- Restlessness + mental distress. Underlying medical or psychiatric causes are common, and include:
- Unaccustomed environment, head trauma, drugs, alcohol, sepsis, hypoxia, metabolic disturbance, delirium, autism, antisocial personality disorder
- Treat medical conditions and attempt to de-escalate agitation verbally. Ensure patient and staff are safe. If verbal approach unsuccessful, consider medication
- Oral drugs: lorazepam, promethazine, midazolam, olanzapine, risperidone, quetiapine, haloperidol. IM drugs: lorazepam, promethazine, olanzapine, aripiprazole, haloperidol. IV drug: diazepam
- Patient needs close monitoring, encourage oral fluids, monitor vital signs & O_2 sats

Delirium: causes & management

- Delirium is common in hospital in-patients. It can by hypoactive, hyperactive, or mixed. The '4AT' score can be used to screen for delirium in those at higher risk (> 65 yrs, cognitive impairment/dementia, previous delirium)
- Identifying and managing possible causes is essential. Common causes include dehydration, poor nutrition, constipation, metabolic derangement, pain, sleep deprivation, drugs, and infection
- Mainstay of treatment focuses on reorientation and reassurance whilst underlying causes are treated. Day-night lighting, visible 24-hour clock and calendar, family visits are all helpful. Use of hearing and visual aids, if required
- Specific management depends on the cause. These may include: IV or subcut fluids, regular laxatives, regular analgesia, correction of electrolyte abnormalities, antibiotics for infection. Reduce medical/nursing interventions overnight
- Treatment under DOLS and a 1:1 may be appropriate. If the patient is felt to be a risk to themselves there is a role for drug treatment e.g. low dose haloperidol

Dementia

- Neurodegenerative syndrome with progressive decline in several cognitive areas. Typical presentation: memory loss over months/years. Affects 20% > 80 yrs**.** Sub-types include Alzheimer's disease, vascular dementia, Lewy body dementia, fronto-temporal dementia. Other causes: alcohol/drug abuse, repeated head trauma, HIV, Parkinson's, Huntington's, CJD

- Diagnosis made from history: patient and collateral. Cognitive testing: dementia screen, tests of function/language, mental state examination. Consider risk factors for vascular dementia and Parkinson's. Consider drug-induced cognitive impairment
- Investigations to look for reversible/organic causes: bloods/MSU/MRI head/functional imaging e.g PET and EEG.
- Management: refer to integrated memory services for further assessment/management. Avoid drugs that impair cognition (eg neuroleptics, sedatives). Treat depression with SSRI or mirtazapine. Tackle social withdrawal with CBT. Non-pharmacological interventions include music, multi-sensory stimulation
- Dementia has significant knock-on stresses for the carer/family. A care coordinator (via Social Services) can co-ordinate: OT/district nurse/CPN/respite care/laundry. Capacity assessment is needed to delineate patient's ability to make decisions re: medical/financial affairs. Consider appointment of a Lasting Power of Attorney

Mini mental state examination

- The MMSE tests orientation, registration (immediate memory), attention, short-term memory, and language functioning. It is used for screening cognitive function, typically dementia. The domains tested are:
- **Orientation** (date, place); **Registration** (naming objects)
- **Attention and calculation** (spelling, simple mathematical sequencing); **Recall** (asking for names of the objects learned earlier); **Language** (naming 2 objects, repeat a difficult sentence, follow a 3-stage command, follow a written instruction, write a sentence)
- Scores: 25-30 normal; 21-24 mild cognitive impairment (ci); 10-20 moderate ci; < 10 severe ci
- The MMSE will not detect subtle memory loss nor problems with long-term memory. It may not be appropriate if patient has learning or communication disabilities

MISCELLANEOUS

Management of alcohol withdrawal

- Alcohol withdrawal is a clinical diagnosis: symptoms range from mild (tremor, sweating, anxiety, cravings) to severe (hallucinations, seizures, delirium tremens). Involve Alcohol Care Team and specialist nurses
- Patients should be assessed regularly. Withdrawal symptoms are graded and recorded with assessment tool, eg 'CIWA' or 'AWS'
- Moderate-to-severe alcohol withdrawal: treat with benzodiazepines - doses and delivery depend on clinical symptoms, scoring systems and local guidelines. Lorazepam, diazepam and chlordiazepoxide are all used
- As patients are frequently malnourished supportive treatment is essential: IV fluids, IV pabrinex (to reduce the risk of Wernicke's encephalopathy), replacement of electrolytes, nutritional support

- Delirium tremens is separate from alcoholic hallucinations and requires early identification and aggressive management. Patients need close observation: ITU admission may be appropriate.

Obese in-patients

- As well as numerous health problems, obesity produces challenges with clinical assessments, practical procedures, imaging, hospital equipment and medication
- Obesity-associated health problems: type 2 DM, IHD, hypertension, steatohepatitis, OA hips/knees/spine, cancer (breast, bowel), sleep-apnoea syndrome
- Assessment problems: pulse readings, BP readings, JVP assessment, abdo exam. Procedure problems: venesection & cannulation. Interpretation problems: ultrasound & radiography
- Specialist medical equipment: bariatric bed (4.5 ft wide); bariatric wheelchair (34.5 inches wide – an ordinary door frame is 30-32 inches in width); bariatric BP cuff (12.5 inches wide)
- Obesity & drug problems: large reservoir for lipophilic drugs (increased half-life); fatty liver disease causes decreased hepatic clearance; enhanced renal blood flow causes increased drug clearance. Orlistat side effects: steatorrhoea, flatulence, abdo pain, headache

'Sectioning' & the Mental Health Act

- The Mental Health Act (1983) covers assessment, treatment, and rights of people with a mental health disorder. People can be detained under the MHA for assessment/treatment when they are at risk of harm to themselves or to others
- Admission under Section 2 for *assessment* is undertaken by an Approved Mental Health Professional + 2 doctors. The patient can be detained for up to 28 days under Section 2.
- Admission under Section 3 for *treatment* is undertaken by an Approved Mental Health Professional + 2 doctors. The patient can be detained for up to 6 months under Section 3.
- Patients admitted under section 2 or 3 can be given medication without their consent (although consent should always be sought)
- The mental health professional in charge of a patient detained under the MHA is the Responsible Clinician. The RC can reverse a section at any time if it is no longer needed

Capacity

- In order to have capacity a person must be able to:
- Understand the information about a decision presented to them
- Retain this information long enough to make a decision
- Arrive at a decision having considered the information, and be able to communicate this decision
- If any of the above is absent, they are deemed to lack capacity

Deprivation of liberty safeguards (DOLS)

- DOLS are a legal set of safeguards to protect a patient who has had his/her liberty deprived (because of a lack of capacity) as part of ongoing care & treatment
- The DOLS are designed to ensure the patient has 1) an independent representative, 2) regular review of the DOL, 3) a legal basis on which to challenge the DOL (through the court of protection)
- The representative is usually a patient's relative or carer. If patient has dementia and there is no relative/carer the representative may be an Independent Mental Capacity Advocate
- If a DOLS is needed, apply for both an emergency DOLS (lasts up to 7 days) and a standard DOLS (lasts up to 12 months)
- The team instituting it should continually monitor the need for a DOLS. The patient or their representative can request a DOLS review

APPENDIX

Acute coronary syndromes

https://www.nice.org.uk/guidance/NG185

Acute heart failure

https://www.nice.org.uk/guidance/cg187

Acute kidney injury

https://www.nice.org.uk/guidance/NG148

Alcohol withdrawal
https://www.nhs.uk/conditions/alcohol-misuse/treatment

Anaemia

https://bestpractice.bmj.com/topics/en-gb/93

Antibiotic-associated diarrhoea

https://cks.nice.org.uk/topics/diarrhoea-antibiotic-associated

Asthma

https://www.nhs.uk/conditions/asthma

Atrial fibrillation

https://bestpractice.bmj.com/topics/en-gb/3

Back pain

https://bestpractice.bmj.com/topics/en-gb/189/diagnosis-approach

Capacity

https://www.nice.org.uk/guidance/NG108

Community-acquired pneumonia

https://www.nice.org.uk/guidance/NG138

COPD

https://www.nhs.uk/conditions/chronic-obstructive-pulmonary-disease-copd

COVID-19

https://www.nhs.uk/conditions/coronavirus-covid-19

Constipation

https://cks.nice.org.uk/topics/constipation/management

Delirium 4AT score

https://www.the4at.com

Dementia

https://www.nice.org.uk/guidance/ng97

Diabetic ketoacidosis

https://bestpractice.bmj.com/topics/en-gb/3000097

DOLS

https://www.dgt.nhs.uk/patients-and-visitors/deprivation-liberty-safeguards-dols

DVT

https://www.nice.org.uk/guidance/NG158

Frailty

https://www.scfn.org.uk/clinical-frailty-scale

Glasgow Coma Scale

https://www.glasgowcomascale.org

Hyperosmolar hyperglycaemic state

https://bestpractice.bmj.com/topics/en-gb/3000124

Infective endocarditis

https://www.nice.org.uk/guidance/cg64/ifp/chapter/infective-endocarditis

Leg ulcer

https://cks.nice.org.uk/topics/leg-ulcer-venous/management

Mental Health Act

https://www.nhs.uk/.../mental-health-and-the-law/mental-health-act

NEWS2

https://www.rcplondon.ac.uk/projects/outputs/national-early-warning-score-news

Sepsis

https://www.nice.org.uk/guidance/NG51

Stroke

https://cks.nice.org.uk/topics/stroke-tia/management

Upper gastro-intestinal bleeding

https://www.nice.org.uk/guidance/cg141

UTI

https://www.nice.org.uk/guidance/qs90