

GUIDELINES FOR THE MANAGEMENT OF COMMON MEDICAL EMERGENCIES AND FOR THE USE OF ANTIMICROBIAL DRUGS

St George's Hospital

August 2022

75th edition

	Page
Cardio-vascular emergencies	
Cardiac arrest	3
Hypertension	11
Acute coronary syndromes	16
Management of STEMI	18
Management of NSTEMI-ACS	20
Acute heart failure	22
Disorders of cardiac rhythm	24
Acute deep vein thrombosis (DVT)	27
Acute pulmonary embolism	30
Respiratory emergencies	
Respiratory arrest	34
Oxygen therapy in acute illness	36
Asthma	37
Spontaneous pneumothorax	39
Gastro-intestinal emergencies	
Acute gastro-intestinal bleeding	42
Bleeding oesophageal varices	44
Acute bloody diarrhoea	46
Diabetic & endocrine emergencies	
Diabetic ketoacidosis (DKA)	48
Hyperosmolar hyperglycaemic state (HHS)	51
Hypoglycaemia	54
Addisonian crisis	55
Neurology emergencies	
Acute stroke	57
Status epilepticus (convulsive SE)	61
Acute management of Parkinson's Disease	65
Anaphylaxis	66
Acute pain	67
Suggestions for the use of antimicrobial drugs in adults	
Sepsis	74
Infective endocarditis	76
Diabetic foot infection	78
Infectious diarrhoea	78
Surgical wound infection	78
Malaria	78

Drug overdosage/acute poisoning	79	
Decompensated liver disease	91	
Acute painful joints	94	
Suspected giant cell arteritis (GCA)	96	
Renal emergencies & Electrolyte disturbances		
Acute kidney injury (AKI)	99	
Electrolyte disturbances:		
Hypokalaemia, Hyperkalaemia	101	
Hypocalcaemia, Hypercalcaemia	103	
Hyponatraemia	105	
Hypernatraemia	106	
Sickle cell crises	108	
Acute psychiatric emergencies		
Managing acutely agitated patients	111	
Management and prevention of delirium	116	
Alcohol use disorders & Wernicke's Encephalopathy	119	
Appendix 1	Cardiac markers in acute coronary syndromes	127
Appendix 2	Cardio-pulmonary resuscitation	128
Appendix 3	Perioperative management of diabetes mellitus	129
Appendix 4a	Enteral/parenteral feeding	131
Appendix 4b	Severely ill patients with Eating Disorders	133
Appendix 4c	Prevention and management of refeeding in adults	135
Appendix 5	Assessing the anion gap	141
Appendix 6	Indications for lumbar puncture	143
Appendix 7	Protecting anticoagulated patients from bleeding	145
Appendix 8	HIV post-exposure prophylaxis <i>and</i> Working with AIDS patients	149
Appendix 9	First steps in a major incident	153
Appendix 10	Monitoring selected therapeutic drug levels	154
	Antifungal monitoring	155
	Amikacin, Vancomycin guidelines	155
Appendix 11	Urgent removal of tunnelled lines	155

GENERAL POINTS

In editing the *Grey Book* every attempt is made to ensure that statements are fully compatible with the advice given by the British National Formulary, the Drug and Therapeutics Bulletin, the various professional bodies (such as the British Thoracic Society), the Royal Colleges (particularly the Royal College of Physicians; RCP), National Service Frameworks and NICE. The Intranet version can be found at the St George's NHS Trust Intranet website: <https://stginet.unily.com/sites/grey-book/SitePageModern/48789/grey-book-home>

If you have any comments or questions please send them to the Link Consultant named at the beginning of the section concerned.

Editor takes no responsibility for the content of Intranet links referenced in the *Grey Book*.

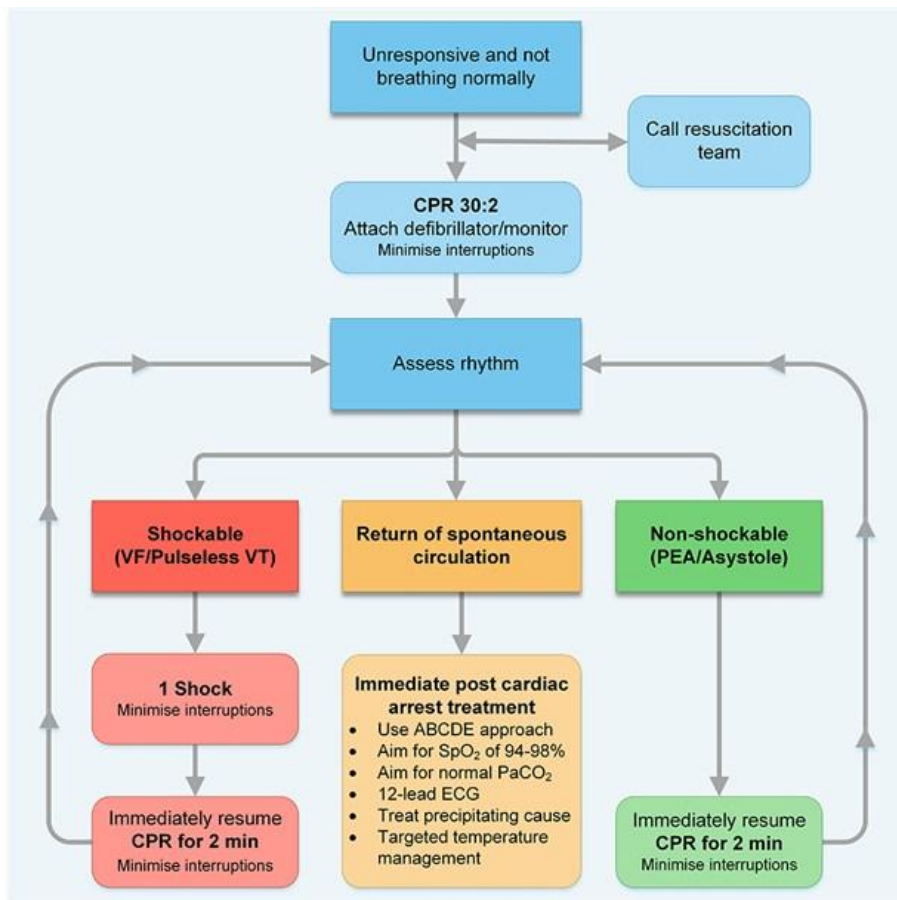
- **The doses given** are for adults unless otherwise stated. If the patient is pregnant, discuss management with the duty obstetric registrar as soon as possible.

- **When medical problems arise** seek advice as follows. During the working day, or when on in-take, refer upwards through your own medical firm. If on "cover" at night and you need advice about a patient on another firm and there is no policy written in the notes, first turn to the in-taking registrar and then to the patient's own consultant. If the patient's consultant cannot be contacted, refer next to the registrar/senior registrar and finally to the in-taking consultant.

Editorial: Teck Khong (editor: tkhong@sgul.ac.uk), Vivien Perkins

Pharmacy liaison: Audrey Khoo

(Link: Jeannie Walls – Lead Resuscitation Officer – Ext 2489)



During CPR

- Ensure high quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced airway in place
- Vascular access (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

Treat Reversible Causes

- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia
- Thrombosis - coronary or pulmonary
- Tension pneumothorax
- Tamponade – cardiac
- Toxins

Consider

- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

Heart rhythms associated with cardiac arrest are divided into two groups:

Shockable Rhythms: Ventricular Fibrillation/pulseless Ventricular Tachycardia (VF/pVT)

Non-shockable Rhythms: Asystole and pulseless electrical activity (PEA).

The main difference in the treatment of these two groups is the need for attempted defibrillation in patients with VF/pVT.

Other actions, including chest compression, airway management and ventilation, vascular access, administration of adrenaline, and the identification and correction of reversible factors, are common to both groups. The ALS algorithm provides a standardised approach to the management of adult patients in cardiac arrest.

Drugs and advanced airways are still included among ALS interventions, but are of secondary importance to early defibrillation and high quality, uninterrupted chest compressions.

Treatment of shockable rhythms (VF/VT)

1. Confirm cardiac arrest – Check for signs of life and normal breathing, and if trained to do so check for breathing and a pulse simultaneously.
2. Call resuscitation team.
3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads – one below the right clavicle and the other in the V6 position in the midaxillary line.
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.
5. Stop chest compressions; confirm VF/pVT from the ECG. This pause in chest compressions should be brief and no longer than 5 seconds.
6. Resume chest compressions immediately; warn all rescuers other than the individual performing the chest compressions to “stand clear” and remove any oxygen delivery device as appropriate.
7. The designated person selects the appropriate energy on the defibrillator and presses the charge button. Choose an energy setting of 150 Joules for the first shock (Manufacturer’s guidance is non-escalating for subsequent shocks.)
8. Ensure that the rescuer giving the compressions is the only person touching the patient.
9. Once the defibrillator is charged and the safety check is complete, tell the rescuer doing the chest compressions to “stand clear”; when clear, deliver the shock.
10. After shock delivery immediately restart CPR using a ratio of 30:2, starting with chest compressions. Do not pause to reassess the rhythm or feel for a pulse. The total pause in chest compressions should be brief and no longer than 5 seconds. If the patient has a definitive airway i.e. ETT or LMA/iGel (if no leak present) Compressions should be continuous with a ventilation rate of 10 breaths per minute.
11. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.
12. Pause briefly to check the monitor.
13. If VF/pVT, repeat steps 6–12 above and deliver a second shock.
14. If VF/pVT persists, repeat steps 6–8 above and deliver a third shock. Resume chest compressions immediately. Give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR. Withhold adrenaline if there are signs of return of spontaneous circulation (ROSC) during CPR.
15. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).
16. Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).

17. If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC (check for signs of life, a central pulse and end-tidal CO₂ if available).
 - If there is ROSC, start post-resuscitation care.
 - If there are no signs of ROSC, continue CPR and switch to the non-shockable algorithm.
18. If asystole is seen, continue CPR and switch to the non-shockable algorithm.

The interval between stopping compressions and delivering a shock must be minimised. Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation. Chest compressions are resumed immediately after delivering a shock (without checking the rhythm or a pulse) because even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation.

The use of waveform capnography can enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved.

Regardless of the arrest rhythm, after the initial adrenaline dose has been given, give further doses of adrenaline 1 mg every 3–5 min until ROSC is achieved; in practice, this will be about once every two cycles of the algorithm. If signs of life return during CPR (e.g. purposeful movement, normal breathing or coughing), or there is an increase in end-tidal CO₂, check the monitor; if an organised rhythm is present, check for a pulse. If a pulse is palpable, start post-resuscitation care. If no pulse is present, continue CPR.

Give amiodarone 300 mg IV after three defibrillation attempts irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest.

Consider a further dose of amiodarone 150 mg IV after a total of five defibrillation attempts.

Lidocaine 1 mg kg⁻¹ may be used as an alternative if amiodarone is not available but do not give lidocaine if amiodarone has been given already.

Witnessed, monitored VF/pVT

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate, ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator – these circumstances are rare. Although there are no data supporting a three-shock strategy in any of these circumstances, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase, immediately after onset of VF/pVT.

If this initial three-shock strategy is unsuccessful for a monitored VF/pVT cardiac arrest, the ALS algorithm should be followed and these three-shocks treated as if only the first single shock has been given.

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm. Its routine use is therefore not recommended. Consider a precordial thump only when it can be used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse. These patients often have some mechanical myocardial contractions, but these are too weak to produce a detectable pulse or blood pressure – this is sometimes described as ‘pseudo-PEA’ (see below). PEA can be caused by reversible conditions that can be treated if they are identified and corrected. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively

Treatment of PEA and asystole

- 1. Start CPR 30:2**
- 2. Give adrenaline 1 mg IV** as soon as intravascular access is achieved (IV/IO)
 - **Continue CPR 30:2** until the airway is secured – then continue chest compressions without pausing during ventilation
 - **When airway is secured** - Ventilate at a rate of 10 breaths per minute
- 3. Recheck the rhythm after 2 min:**
 - If electrical activity compatible with a pulse is seen, check for a pulse and/or signs of life
 - If a pulse and/or signs of life are present, start post resuscitation care
 - If no pulse and/or no signs of life are present (PEA OR asystole)
 - Continue CPR
- 4. Recheck the rhythm after 2 min** and proceed accordingly
- 5. Give further adrenaline 1 mg IV** every 3–5 min (during alternate 2-min loops of CPR)
 - If VF/pVT at rhythm check, change to shockable side of algorithm.

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because the patient may respond to cardiac pacing when there is ventricular standstill with continuing P waves. There is no value in attempting to pace true asystole.

Treat Reversible Causes

Potential causes or aggravating factors for which specific treatment exists must be considered during all cardiac arrests. For ease of memory, these are divided into two groups of four, based upon their initial letter: **either H or T**:

- **Hypoxia**
- **Hypovolaemia**
- **Hyperkalaemia, hypokalaemia**, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders
- **Hypothermia**
- **Thrombosis** (coronary or pulmonary)
- **Tension pneumothorax**
- **Tamponade** – cardiac
- **Toxins**

The four ‘Hs’

Minimise the risk of hypoxia by ensuring that the patient’s lungs are ventilated adequately with the maximal possible inspired oxygen during CPR. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described below, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by hypovolaemia is due usually to severe haemorrhage. This may be precipitated by trauma, gastrointestinal bleeding or rupture of an aortic aneurysm. Stop the haemorrhage and restore intravascular volume with fluid and blood products.

Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient’s medical history (e.g. renal failure). Give IV calcium chloride in the presence of hyperkalaemia, hypocalcaemia and calcium channel-blocker overdose.

Hypothermia should be suspected based on the history such as cardiac arrest associated with drowning.

The four ‘Ts’

Coronary thrombosis associated with an acute coronary syndrome or ischaemic heart disease is the most common cause of sudden cardiac arrest. An acute coronary syndrome is usually diagnosed and treated after ROSC is achieved. If an acute coronary syndrome is suspected, and ROSC has not been achieved, consider urgent coronary angiography when feasible and, if required, percutaneous coronary intervention. Mechanical chest compression devices and extracorporeal CPR can help facilitate this (see below).

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause of cardiac arrest consider giving a fibrinolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported, even in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts. In some settings extracorporeal CPR, and/or surgical or mechanical thrombectomy can also be used to treat pulmonary embolism.

A tension pneumothorax can be the primary cause of PEA and may be associated with trauma. The diagnosis is made clinically or by ultrasound. Decompress rapidly by thoracostomy or needle thoracocentesis, and then insert a chest drain.

Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy. The use of ultrasound will make the diagnosis of cardiac tamponade much more reliable. In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or toxic substances may be revealed only by laboratory investigations. Where available, the appropriate antidotes should be used, but most often treatment is supportive and standard ALS protocols should be followed.

Use of ultrasound imaging during advanced life support

When available for use by trained clinicians, focused echocardiography/ultrasound may be of use in assisting with diagnosis and treatment of potentially reversible causes of cardiac arrest. The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised. A sub-xiphoid probe position has been recommended. Placement of the probe just before chest compressions are paused for a planned rhythm assessment enables a well-trained operator to obtain views within 10 seconds.

Several studies have examined the use of ultrasound during cardiac arrest to detect potentially reversible causes. Although no studies have shown that use of this imaging modality improves outcome, there is no doubt that echocardiography has the potential to detect reversible causes of cardiac arrest. Specific protocols for ultrasound evaluation during CPR may help to identify potentially reversible causes (e.g. cardiac tamponade, pulmonary embolism, hypovolaemia, pneumothorax). Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest is highly predictive of death although sensitivity and specificity has not been reported.

During CPR

High quality chest compressions with minimal interruption

During the treatment of persistent VF/pVT or PEA/asystole, there should be an emphasis on giving high quality chest compression between defibrillation attempts or rhythm checks, whilst recognising and treating reversible causes (4 Hs and 4 Ts), and whilst obtaining a secure airway and intravascular access. Aim for a chest compression pause of less than 5 seconds for rhythm checks, defibrillation attempts, and tracheal intubation. To achieve this rescuers must plan their actions before pausing compressions.

Monitoring during advanced life support

The following methods can be used to monitor the patient during CPR and help guide ALS interventions:

- Clinical signs such as breathing efforts, movements and eye opening can occur during CPR. These can indicate ROSC and require verification by a rhythm and pulse check, but can also occur because CPR can generate a sufficient circulation to restore signs of life including consciousness.
- Pulse checks when there is an ECG rhythm compatible with an output can be used to identify ROSC, but may not detect pulses in those with low cardiac output states and a low blood pressure. The value of attempting to feel arterial pulses during chest compressions to assess the effectiveness of chest compressions is unclear. A pulse that is felt in the femoral triangle may indicate venous rather than arterial blood flow. There are no valves in the inferior vena cava and retrograde blood flow into the venous system can produce femoral vein pulsations. Carotid pulsation during CPR does not necessarily indicate adequate myocardial or cerebral perfusion.
- Monitoring heart rhythm through pads, paddles or ECG electrodes is a standard part of ALS. Motion artefacts prevent reliable heart rhythm assessment during chest compressions forcing rescuers to stop chest compressions to assess the rhythm, and preventing early recognition of recurrent VF/pVT. We suggest that artefact-filtering algorithms are not used for analysis of ECG rhythm during CPR unless as part of a research programme.
- End-tidal CO₂ with waveform capnography. The use of waveform capnography during CPR has a greater emphasis in Guidelines 2015 and is addressed in more detail below.

- The use of CPR feedback or prompt devices during CPR should be considered only as part of a broader system of care that should include comprehensive CPR quality improvement initiatives rather than an isolated intervention.
- Blood sampling and analysis during CPR can be used to identify potentially reversible causes of cardiac arrest. Avoid finger prick samples in critical illness because they may not be reliable; instead, use samples from veins or arteries.
- Blood gas values are difficult to interpret during CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state. Analysis of central venous blood may provide a better estimation of tissue pH.
- Invasive cardiovascular monitoring in critical care settings (e.g. continuous arterial blood pressure and central venous pressure monitoring). Invasive arterial pressure monitoring will enable the detection of low blood pressure values when ROSC is achieved.
- Ultrasound assessment is addressed above to identify and treat reversible causes of cardiac arrest, and identify low cardiac output states ('pseudo-PEA').

Waveform capnography during advanced life support

Use waveform capnography whenever tracheal intubation is undertaken. Although the prevention of unrecognised oesophageal intubation is clearly beneficial, there is currently no evidence that use of waveform capnography during CPR results in improved patient outcomes. The role of waveform capnography during CPR includes:

- Ensuring tracheal tube placement in the trachea (although it will not distinguish between bronchial and tracheal placement).
- Monitoring ventilation rate during CPR and avoiding hyperventilation.
- Monitoring the quality of chest compressions during CPR. End-tidal CO₂ values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value. Whether this can be used to guide care and improve outcome requires further study.⁴¹
- Identifying ROSC during CPR. An increase in end-tidal CO₂ during CPR can indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- Prognostication during CPR. Precise values of end-tidal CO₂ depend on several factors including the cause of cardiac arrest, bystander CPR, chest compression quality, ventilation rate and volume, time from cardiac arrest and the use of adrenaline. Values are higher after an initial asphyxial arrest, with bystander CPR, and decline over time after cardiac arrest. Low end-tidal CO₂ values during CPR have been associated with lower ROSC rates and increased mortality, and high values with better ROSC and survival. The inter-individual differences and influence of cause of cardiac arrest, the problem with self-fulfilling prophecy in studies, our lack of confidence in the accuracy of measurement during CPR, and the need for an advanced airway to measure end-tidal CO₂ reliably limits our confidence in its use for prognostication. The Resuscitation Council (UK) recommends that a specific end-tidal CO₂ value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO₂ values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

HELPFUL INFORMATION FOR USE DURING CARDIAC ARREST
For Difficult IV access – please consider use of Intra Osseous needle.

EZ-IO drills can be found in the following areas:

Lanesborough Wing	St James Wing	Atkinson Morley Wing
PICU <i>1st Floor</i>	ED- Resus area <i>Ground Floor</i>	Cardiac Theatre Recovery <i>1st Floor</i>
Champney's Ward <i>4th Floor</i>	St James' Recovery <i>1st Floor</i>	William Drummond Ward <i>2nd Floor</i>
Paediatric Step Down Unit <i>5th Floor</i>	Paul Calvert Theatre <i>1st Floor</i>	

For Thrombolysis: The Cardiac Arrest dose of Alteplase is 50mg IV Bolus

Consider a further 50mg IV bolus 30 minutes later in prolonged CPR attempt

Nearest Stock is

The critical Care & Outreach Team will be able to advise on how to access Alteplase. If the

Lanesborough Wing	St James Wing	Atkinson Morley Wing
Champney's Ward – 4 th Floor <i>CD Cupboard</i> Ext: 0062	ED Resus <i>CD Cupboard</i> Ext: 1290	William Drummond Ward <i>Ward Stock</i> Ext: 5825

Lucas device is required for transfer, they will organise retrieval and attach it.

CARDIO-PULMONARY RESUSCITATION IN COVID 19 PATIENTS

The Resuscitation Council UK and World Health Organisation have identified Chest Compressions as an aerosol generating procedure. (AGP)

Therefore, during the Covid 19 pandemic, all inpatients must be considered as potentially positive, even if within a 'green' inpatient area.

Please refer to the RCUK guidance on Cardiac Arrest during COVID reproduced on Trust Intranet

<http://stginet/Publications/Clinical%20Publications/The%20Grey%20Book.aspx>

Please note the changes to the algorithm including donning of Level 3 AG – PPE.

SEVERE HYPERTENSION

Link Consultant: Dr Tarek Antonios

Healthcare professionals should first determine the presence or absence of the following:

1. Neurological symptoms:
 - i. Generalized: visual disturbance, agitation, delirium, seizures, coma (Hypertensive encephalopathy)
 - ii. Focal: stroke (thrombotic, haemorrhagic)
2. Fundus: haemorrhage, exudates, papilloedema (malignant hypertension)
3. Pain: chest, abdomen, back (aortic dissection, acute coronary syndrome, MI)
4. Breathlessness: Pulmonary oedema (acute left ventricular failure)
5. Nausea & vomiting: Increased intracranial pressure (Hypertensive encephalopathy)
6. Pregnancy: (Preeclampsia, eclampsia)
7. Drugs: (cocaine, amphetamine, phencyclidine, MAO inhibitors, clonidine withdrawal)

HYPERTENSIVE EMERGENCIES:

This is severe hypertension with Acute & Life-threatening organ damage.

Condition	Contra-indicated drugs	Indicated drugs
Aortic dissection	Vasodilators (on their own)	Metoprolol or Labetalol (\pm SNP), CCB
Hypertensive Encephalopathy	GTN, SNP, Centrally acting drugs	Labetalol
CVAs	GTN, Centrally acting drugs	Labetalol
ACS, MI	Short acting nifedipine	GTN, Metoprolol
Acute LVF	Caution with BB, labetalol, hydralazine	GTN, Diuretics, SNP
AKI	Caution with Diuretics	Labetalol
Malignant HTN	Caution with Diuretics	ACEi, BB, CCB
Cocaine	BB	Diltiazem

- All patients should be admitted to an intensive care unit or a high dependency ward.
- The selection of the anti-hypertensive agent(s) and BP target of treatment depends upon the specific hypertensive emergency.
- In most patients, the aim is to reduce BP in a rapid (within 2-4 hours) controlled way but not overzealous way, to safe (not normal) levels ~160mmHg systolic and ~100mmHg diastolic with the maximum initial fall in BP not exceeding 25% of the presenting value. A too rapid reduction in BP may precipitate cerebral, myocardial or renal infarction.
- The exception is acute aortic dissection where systolic BP should be rapidly lowered to 100-120mmHg within 20 minutes.
- Use oral Nifedipine modified release (MR) or slow release (SR) 10mgs, if appropriate, as per the above table. This dose can be repeated after two hours (with maintenance doses of up to 20mgs TDS).
- Use intravenous anti-hypertensives if the patient is unable to swallow (see recommended drugs in table above).

Management of Hypertensive Emergencies: The key to a successful outcome is the prompt recognition and prompt initiation of treatment. Full medical history and physical examination including palpation of all peripheral pulses and a fundoscopic examination is mandatory. Initial investigations should include FBC, electrolytes, urea, creatinine, urine dipstick, chest x-ray and ECG. These tests should be performed simultaneously with the initiation of antihypertensive therapy.

MALIGNANT (ACCELERATED) HYPERTENSION

Malignant (accelerated) hypertension is a syndrome characterized by severely elevated BP accompanied by retinopathy (retinal haemorrhages, exudates or papilloedema), visual impairment, nephropathy (acute kidney injury), encephalopathy (confusion, lethargy, or coma) and microangiopathic haemolytic anaemia. The resulting vasoconstriction induces severe elevation in BP and widespread endothelial damage. The resulting renal ischemia prompts massive release of renin and angiotensin II triggering a vicious cycle. The rapid increase in BP enhances pressure natriuresis, which further stimulates the RAAS system resulting in secondary hyperaldosteronism, hypokalaemia and metabolic alkalosis.

Malignant hypertension is a very serious condition with very poor prognosis if not properly diagnosed and treated with a mortality rate exceeding 90% within 1 year but with proper treatment 5 years survival is 60-75%. It rarely occurs de novo but is usually a consequence of untreated essential or secondary hypertension, including non-concordance with antihypertensive medications. Most patients who present with malignant hypertension have volume depletion secondary to pressure naturesis. Therefore, further diuresis may exacerbate the hypertension and may cause further deterioration in kidney function.

HYPERTENSIVE ENCEPHALOPATHY:

Hypertensive encephalopathy is believed to be due to cerebral oedema secondary to failure of the cerebral blood flow autoregulation and rapid elevation of cerebral perfusion. Symptoms and signs include headache, nausea, vomiting, visual disturbances, altered level of consciousness, confusion, disorientation, focal or generalized seizures and retinopathy including papilloedema. Diagnosis may be difficult as it is one of exclusion requiring that stroke, encephalitis, vasculitis, subarachnoid haemorrhage and mass lesions need to be excluded. The definite criterion to confirm the diagnosis is a prompt improvement in the patient's clinical condition with the response to antihypertensive treatment. The goal of treatment is to reduce BP by approximately 25% within the first hour or to a level of 160/100 mmHg, whichever value is higher. It must be emphasized that cerebral hypoperfusion and neurological deterioration may result if more reductions in BP are achieved quickly. In this case BP should be allowed to increase and further reductions should be attempted more slowly.

STROKE:

Appropriate treatment of hypertension in the setting of acute stroke remains contentious. There is little scientific evidence and no clinically established benefit for rapid lowering of BP among persons with acute ischemic stroke. Aggressive lowering of BP may cause neurological worsening. However, it is generally agreed that severe hypertension (BP > 180/110 mmHg) may be an indication for treatment as higher BP levels is a contraindication to IV thrombolysis. If thrombolysis is not considered, then emergency administration of antihypertensive drugs should be withheld unless the systolic BP is >220 mm Hg and/or diastolic BP is >120 mm Hg. Treatment could be started with IV labetalol. Previously hypertensive patients with mild to moderate strokes who are not at high risk for increased intracranial pressure may have their usual pre-stroke antihypertensive medications restarted 24 hours after their stroke. GTN is not usually recommended.

AORTIC DISSECTION:

Aortic dissection must be excluded in any patient presenting with severe hypertension and chest pain, back pain or abdominal pain. It is life-threatening with very poor prognosis if not treated. Aortic dissection is classified as type A if it involves the ascending aorta or type B if it does not. Type B responds more favourably to medical treatment. Severe refractory hypertension is nearly omnipresent especially in the acute phase even in patients without history of hypertension.

The immediate reduction of BP and sheer stress is of paramount importance to prevent the extension, haemorrhage and rupture of the dissection. BP should be reduced quickly (within 15-20 minutes) to the lowest tolerated level that preserves adequate organ perfusion. The initial treatment of choice is a combination of IV β -blocker (e.g. metoprolol) or a combined α - β blocker (e.g. labetalol) and a vasodilator (e.g. sodium nitroprusside or dihydropyridine CCB) to decrease systolic blood pressure below 120 mmHg if tolerated. The combination of a dihydropyridine CCB and an ACE inhibitor is effective in reducing central aortic pressure.

ACUTE PULMONARY OEDEMA:

Intravenous GTN is the drug of choice in the initial treatment together with an IV loop diuretic (e.g. frusemide) and diamorphine. GTN reduces both preload and afterload while improving coronary blood flow. There is very little clinical experience with the use of ACE inhibitors in patients with acute LV failure but a short-acting ACE inhibitor (e.g. captopril) may be added if necessary.

It is important to stress here that patients with malignant hypertension who present with acute (flash) pulmonary oedema may not have volume overload. In fact, they may have volume depletion secondary to pressure naturesis. Therefore, IV diuresis may exacerbate the hypertension and cause further clinical deterioration. The use of diuretics should be reserved for patients who are clinically fluid overloaded and should not be prescribed routinely.

STEMI & ACS:

Hypertension is very common in patients presenting with ACS. Intravenous GTN is the drug of choice for ACS & STEMI as it reduces PVR while improving coronary perfusion. β -Blockers attenuate the activity of the adrenergic and the RAAS systems and improve survival in post MI patients. When β -blockers are contraindicated, a non-dihydropyridine CCB (diltiazem or verapamil) can be used if the patient does not have severe LV dysfunction. Short-acting dihydropyridine CCB should not be used in the treatment of hypertensive crisis when associated with ACS or acute STEMI. ACE inhibitors could be added if hypertension persists as they significantly improve survival during STEMI. Sodium nitroprusside, unlike GTN, increases heart rate and provokes ST segment elevation and should not be used alone.

COCAINE OVERDOSE:

Cocaine overdose is often associated with uncontrolled severe hypertension and coronary artery vasoconstriction leading to angina, MI and sudden death. These effects are mediated through α -adrenergic receptors and therefore β -blockers alone (i.e. without α -blockers) may exacerbate the hypertension and the clinical condition and are therefore contraindicated. A non-dihydropyridine CCB (e.g. diltiazem or verapamil) or a combined α - β blocker e.g. Labetalol may be used.

SEVERE PREECLAMPSIA AND ECLAMPSIA:

Preeclampsia is defined as hypertension (BP \geq 140/90 mmHg) in the second half of pregnancy associated with proteinuria and oedema. For preeclamptic patients with severe hypertension IV Labetalol could be given. Sodium nitroprusside can cause profound reflex paradoxical bradycardia and hypotension and should be avoided. ACE inhibitors and ARBs are contraindicated in pregnancy because of the increase in foetal and neonatal morbidity and mortality.

DRUGS FOR THE TREATMENT OF HYPERTENSIVE EMERGENCIES:

Intravenous agents:

- Hypotensive agents should be administered intravenously when organ damage is potentially life-threatening. **All patients should be admitted to an HDU or ITU bed**, for continuous BP monitoring. The choice of drug will frequently depend on the underlying cause or the organ most compromised.

▪ **Sodium nitroprusside (SNP):**

Sodium nitroprusside is the parenteral drug of choice for most hypertensive emergencies. SNP should only be given in HDU or ITU with continuous intra-arterial BP monitoring. SNP is an arteriolar and a venous dilator and has an immediate onset and short duration of action, $t_{1/2}$ 2-3 min. SNP is administered by intravenous infusion starting at 0.3 microgram/kg/min, increasing by 0.5 microgram/kg/min every 5 minutes, to a maximum of 8 micrograms/kg/min. The use of SNP is associated with cyanide toxicity, which is manifested by clinical deterioration, altered mental status, and lactic acidosis. The risk of toxicity is reduced by protecting the drug from light (so minimising degradation), and by not exceeding the equivalent of 2 micrograms/kg/min (over a maximum of 48hrs). The risk of cyanide toxicity is increased in the presence of renal failure, when the dose should be reduced. SNP should be used with caution in preeclampsia.

▪ **Labetalol:**

Labetalol is a combined α - and β -adrenergic blocker which can be used in most hypertensive emergencies and urgencies. It is a logical option for patients with ischaemic heart disease, aortic dissection, stroke and in pregnancy. Labetalol is given either by slow intravenous injection: 20 mg over 1 minute initially, followed by 20-80 mg every 10 minutes to a total dose of 200 mg; or by infusion at a rate of 0.5 to 2 mg/min. Labetalol can cause severe postural hypotension and is contraindicated in patients with acute LV failure, heart block and COPD.

▪ **Glyceryl trinitrate (GTN):**

GTN is a venodilator and to a lesser degree and arteriolar dilator. Its onset of action is 1-3 mins and tolerance quickly develops. It is the drug of choice in acute left ventricular failure, acute pulmonary oedema, and acute coronary syndromes. The initial dose of GTN is 5 micrograms/min to be increased by 10 micrograms/min every 3-5 minutes if needed. However, BP response with GTN is not as predictable as with Na nitroprusside, and higher doses may be required. GTN should not be considered as first-line therapy in other hypertensive emergencies.

• **Hydralazine:**

Hydralazine is an arteriolar dilator which is used particularly in hypertensive emergencies in pregnancy, but labetalol is preferable. A bolus dose of 5 mg can be given by slow intravenous injection, followed by 5 to 10 mg boluses as necessary every 30 minutes. Alternatively, it can be given as an infusion starting at 200-300 micrograms/min; this usually requires a maintenance dose of 50-150 micrograms/min. Hydralazine is contraindicated in ischemic heart disease and aortic dissection.

• **Phentolamine:**

Phentolamine is a short-acting α -blocker, can be used in the first instance when a phaeochromocytoma is known or strongly suspected. It is given by slow intravenous injection, in doses of 2-5 mg over 1 minute, repeated as necessary every 5-15 minutes.

HYPERTENSIVE URGENCIES

(Severe Hypertension with No Life-Threatening Organ Damage):

The situation becomes a Hypertensive Urgency rather than an emergency when there is no evidence of target organ damage. *Always seek advice from the Blood Pressure Unit.* Ideally patients should be admitted to a medical bed and blood pressure reduced slowly; the systolic pressure should be lowered to about 160-180mmHg and diastolic pressure to about 100-110mmHg over 24-48 hours. For known hypertensive patients who are not compliant with their medication, prior therapy should be restarted. For patients taking their medication regularly, therapy should be increased (either by increasing the dose(s) of drugs or adding new drugs). For patients on no treatment, hypertension therapy should be started with oral agents and a follow-up appointment arranged urgently with the hypertension clinic.

Oral agents.

In most patients, oral therapy is adequate, safe and preferred. Again, patients may be hypovolaemic, which often becomes manifest once antihypertensive treatment is given, particularly if the drug used is an ACE inhibitor, angiotensin receptor blocker or direct renin inhibitor. Blood pressure should be measured at regular intervals in the sitting and standing positions. A postural drop of >20mmHg suggests hypovolaemia, which needs correcting.

- Start with nifedipine (SR/MR) 10mg tablets, swallowed whole. The same dose can be repeated at 2 hours if required, with maintenance doses of up to 20mg three times a day.
- Do NOT use nifedipine capsules, long-acting (LA) nifedipine preparations, or amlodipine at this stage.
- Add a β -blocker (e.g. bisoprolol 5mg) as a second line therapy where necessary, particularly when there is co-existing ischaemic heart disease or a resting tachycardia in response to nifedipine.
- ACE inhibitors can be given, but with caution (a rapid fall in blood pressure that occurs in some patients can be treated with intravenous saline).
- Diuretics should be used with caution, unless there is clear evidence of volume overload.

Follow-up management. Renal function should be monitored daily, as the initial BP reduction, to a diastolic pressure of 100-110mmHg, is often associated with deterioration in renal function. This is usually transient and antihypertensive therapy should not be withheld unless there has been an excessive reduction in BP. Once the BP is controlled to this level, then the diastolic pressure can be gradually reduced to 80-90mmHg over the next few weeks.

Before discharge, patients treated for severe hypertension should be referred to the Blood Pressure Unit for investigation of secondary causes of hypertension (e.g. renal artery stenosis, primary hyperaldosteronism, phaeochromocytoma, other adrenal pathology or underlying renal disease).

MANAGEMENT OF ACUTE CORONARY SYNDROMES (ACS)

Link Consultant: Dr Simon Wilson

All patients arriving at hospital with chest pain suggestive of myocardial ischaemia (central or retrosternal pressure, tightness, heaviness, radiating to neck, shoulder or jaw, associated with breathlessness, nausea or vomiting) require an immediate 12-lead ECG and medical assessment. The clinical history is critically important. Management depends upon whether the patient has features of ST-segment Elevation Myocardial Infarction (STEMI) or Non ST-segment Elevation Acute Coronary Syndromes (NSTEMI-ACS). NSTEMI-ACS includes both Non ST-segment Elevation Myocardial Infarction (NSTEMI) and unstable angina.

INITIAL DIAGNOSTIC MEASURES FOR ALL PATIENTS.

The patient should be attached to a cardiac monitor immediately in order to **detect cardiac arrhythmias**. **The 12-lead ECG will be critical in excluding a STEMI.**

Be aware that ST depression in the anterior chest leads may actually represent a posterior STEMI relating to an occluded Circumflex vessel.

If in doubt immediately bleep the Cardiology SpR (bleep 6002) for a review.

➤ **The ECG changes diagnostic of STEMI are:**

ST elevation in two contiguous leads: $\geq 0.25\text{mV}$ in men below the age of 40 years, $\geq 0.2\text{mV}$ in men over the age of 40 years or $\geq 0.15\text{mV}$ in women in leads V2-V3 and/or $\geq 0.1\text{mV}$ in other leads. Left bundle branch block that is new or presumably new, in the context of a convincing history of cardiac sounding chest pain.

In the presence of LBBB, the ECG diagnosis of acute MI is difficult, but often possible if marked ST abnormalities are present. Most LBBB patients evaluated in the emergency department do not have an acute coronary occlusion, nor do they require primary PCI. Blood sampling for serum biomarkers is routinely done in the acute phase but one should not wait for the results to initiate treatment.

➤ **The ECG changes diagnostic of NSTEMI-ACS are:**

Dynamic ST segment depression, T wave inversion, flat T-waves or pseudo-normalisation of T waves. *Caution:* The ECG may be normal in patients presenting with NSTEMI-ACS.

Always perform serial ECGs if there is a clinical suspicion of an ACS (i.e. every 20 minutes during the initial presentation and if ongoing pain).

Atypical ECG presentations that deserve prompt management in patients with signs and symptoms of ongoing myocardial ischaemia include:

Posterior myocardial infarction

Ventricular paced rhythm

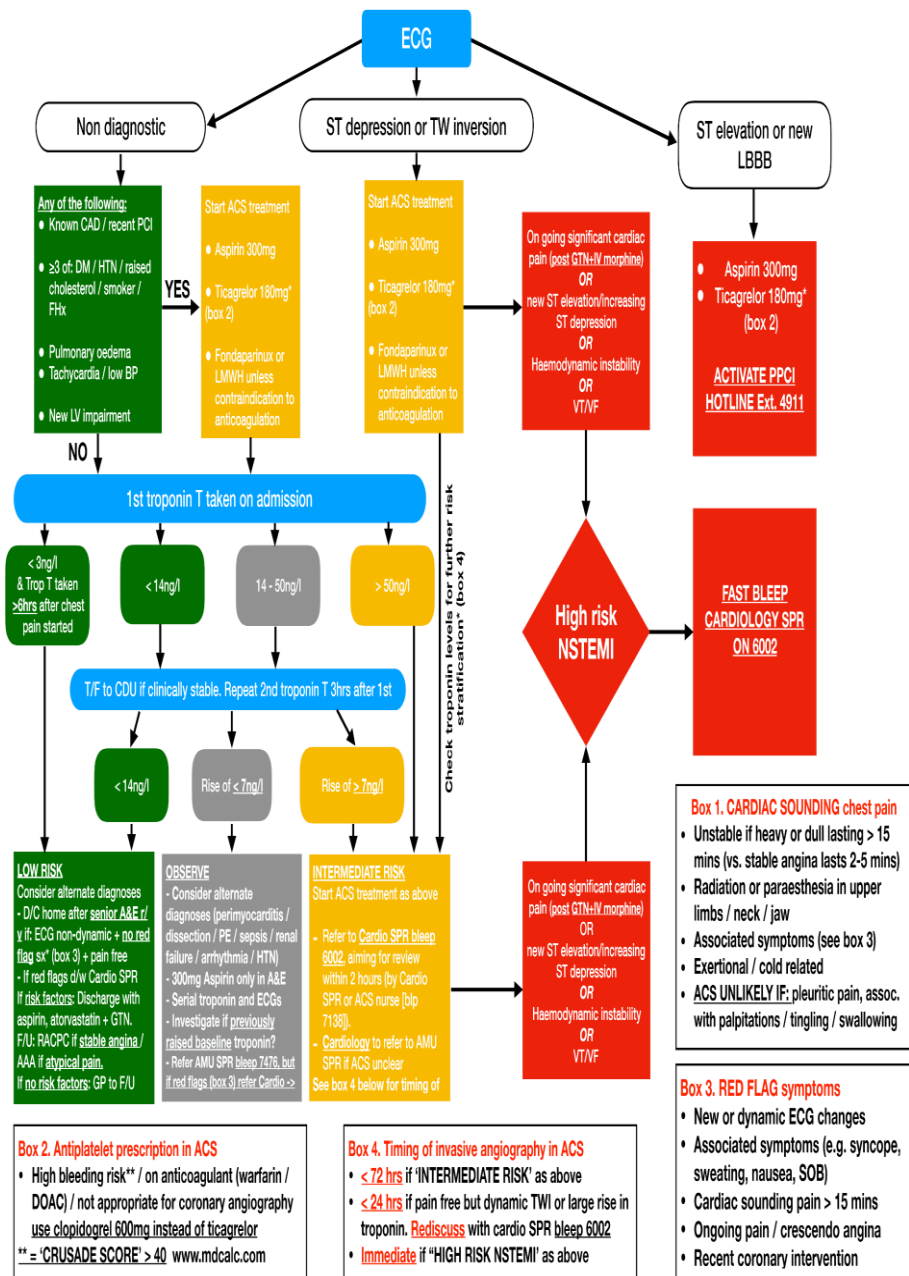
Patients without ECG changes but with persistent ischaemic sounding chest pain

ST elevation in lead aVR (*Caution:* left main stem disease)

If a STEMI is suspected but not definite, *discuss urgently with the duty A&E consultant and then the on-call Cardiology registrar (Bleep 6002).*

Please note that **'High Risk NSTEMIs'** now also require *urgent discussion with the on-call Cardiology registrar (Bleep 6002)*, with a view to immediate primary percutaneous coronary intervention (PPCI) to reperfuse the occluded coronary artery. High risk NSTEMIs are defined by: ongoing significant chest pain despite GTN and IV morphine / new ST depression / haemodynamic instability / ventricular tachycardia/fibrillation.

*****For management of patients presenting with clinical suspicion of an ACS, please see and follow the revised ACS pathway (August 2019)*****



MANAGEMENT OF STEMI

Link Consultant: Dr Simon Wilson

Refer the patient immediately to Cardiology for Primary Percutaneous Coronary Intervention (PPCI): either fast bleed Cardiology SPR on 6002 or ring PPCI hotline on ext. 4911. The target 'door' - 'reperfusion' time is within 60mins therefore avoid all unnecessary delays. Establish an IV line. Take blood samples for full blood count, U&Es, glucose, high sensitivity troponin T and creatine kinase and lipids. A chest x-ray should be requested but should not delay therapy.

To access the prescribing bundle on iClip use search terms of ACS or acute coronary syndrome.

Aspirin As soon as possible give soluble Aspirin 300 mg to be chewed. This is be followed by Aspirin 75mg od long term. If the patient is allergic to Aspirin seek advice.

Ticagrelor STEMI patients should receive a loading dose of Ticagrelor 180mg stat on arrival. However, in patients with active bleeding, a history of intracranial haemorrhage, or in patients established on formal anticoagulation – i.e those on warfarin or direct oral anticoagulants this should be discussed with the cardiologist first. Ticagrelor should thereafter be prescribed at a dose of 90 mg twice daily for 12 months.

Analgesia Give Morphine 2.5-5mg by slow IV injection (1mg/min) followed by a further 2.5-5.0mg IV if pain persists (and then every 4 hrs as required). To reduce the likelihood of vomiting, give either Metoclopramide (10mg IV over 2 minutes) or Cyclizine 50mg IV immediately afterwards.

Nitrates Give IV Glycerol Trinitrate infusion at a dose of 1-10mg per hour for continuing chest pain or pulmonary oedema if the systolic blood pressure is > 90mmHg and the patient hasn't received a phosphodiesterase inhibitor (eg. Sildenafil) within 24 hours. Please inform the duty cardiology SpR in order that the patient is discussed with the responsible / duty interventional consultant.

Oxygen: Now only recommended in patients with oxygen saturation (SpO_2) $\leq 90\%$: as per European Society of Cardiology 2018 guidelines (previously $<95\%$), following the AVOID and DETO2X randomised controlled trials (RCT). Hyperoxia may be harmful in patients with uncomplicated MI, presumably due to increased myocardial injury. Target SpO_2 94-98%. In patients with chronic obstructive pulmonary disease and who are at risk of hypercapnic respiratory failure the target SpO_2 is 88–92%.

Anticoagulation after PPCI

Assess and prescribe VTE prophylaxis as per hospital guidelines. The strategy regarding longer term anticoagulation with warfarin/direct oral anticoagulants will be stipulated by the responsible interventional cardiologist and should be stated in the angiogram/ PCI report.

Blood glucose management. Manage hyperglycaemia by keeping blood glucose levels below 11.0 mmol/L whilst avoiding hypoglycaemia. In the first instance, consider a dose-adjusted insulin infusion with regular blood glucose monitoring.

Refer newly diagnosed diabetic patients to the diabetes nurse specialist (blp 6236).

<http://stginet/Publications/Clinical%20Publications/Grey%20book/Variable%20Rate%20Intravenous%20Insulin%20Infusi.pdf>

For patients with hyperglycaemia after ACS without known diabetes: assess HbA1c levels before discharge and fasting blood glucose levels no earlier than 4 days after ACS onset (should not delay discharge).

ACE inhibitors Following a STEMI, patients should be commenced on an ACE inhibitor, with the exception of those in renal failure or with a systolic blood pressure (BP) < 90mmHg. A reasonable choice is Ramipril started at a dose of 1.25 mg od. Dosage should be slowly titrated upwards to the maintenance dose of 10mg od, taking care to avoid a fall in BP or reduction in renal function. If Ramipril is not tolerated, try Candesartan (4mg od) or Valsartan (80mg bd).

Beta-blockade Beta (β)-blockers are recommended for all patients except those with:

bradycardia <50bpm	• heart failure requiring therapy
second or third degree heart block	• a history of bronchospasm
cardiogenic shock	• allergy/hypersensitivity to β -blockers

Bisoprolol 2.5 mg daily is the drug of choice. Dosage should be slowly titrated upwards to the maintenance dose of 5.0-10 mg od, taking care to avoid a fall in BP or significant bradycardia.

Statins and lipid-lowering agents All patients should have a lipid profile on admission, and then be commenced on Atorvastatin 80 mg, providing there is no contraindication or history of intolerance, regardless of initial lipid profile. This provides an anti-inflammatory therapeutic effect in addition to lipid lowering efficacy. Reduce dose in patients receiving interacting drugs (Clarithromycin, Cyclosporin, Protease inhibitors, Diltiazem, Amiodarone, Verapamil).

Aldosterone receptor antagonists Arrange for an echo-cardiogram to be done within 24 hrs of admission. If there are clinical signs of heart failure and the left ventricular ejection fraction is (LVEF) \leq 40%, consider an aldosterone antagonist such as Eplerenone 25 mg od (contraindicated if eGFR is < 30mL/min/1.73m², or potassium > 5.0 mmol/L at initiation). If LVEF \leq 40%, please also refer the patient to the heart failure nurses on bleep 7376.

Gastroprotection All patients should be prescribed Lansoprazole 30 mg od. Watch for new hyponatraemia and consider switch to ranitidine if this is a suspected drug reaction.

Addendum: dual anti-platelet therapy in high bleeding risk / high ischaemic risk patients

High bleeding risk patients: a) bleeding history; b) CRUSADE score > 40 (www.mdcalc.com), or c) on anti-coagulants. Discuss these patients with the responsible Cardiologist to consider Clopidogrel 75mg od (after a loading dose) instead of Ticagrelor, and / or a shortened duration of dual anti-platelet therapy (especially in those on anticoagulants following the AUGUSTUS RCT).

High ischaemic risk patients: particularly those with a) severe multivessel diffuse coronary artery disease, b) peripheral arterial disease, and c) recurrent myocardial infarctions. Consider, after the 12 months of dual anti-platelet therapy, Ticagrelor 60mg bd for a further 3 years (following the PEGASUS-TIMI 54 RCT).

Please DO NOT change anti-platelet regimens without discussion with the responsible consultant interventional cardiologist for that patient.

MANAGEMENT OF NSTEMI-ACS Link Consultant: Dr Simon Wilson

Non ST-segment elevation acute coronary syndromes (NSTEMI-ACS) is an umbrella term which includes patients presenting with unstable angina (UA) and non ST-segment elevation myocardial infarction (NSTEMI). Patients with NSTEMI-ACS may complain of rapidly worsening, prolonged (lasting >20 minutes) and increasingly frequent episodes of cardiac sounding chest pain, of cardiac chest pain occurring at rest, or of cardiac sounding chest pain of recent onset (<1 month). Unstable angina refers to patients presenting with ischaemic sounding chest pain in the absence of a Troponin rise. Given that an elevation in the Troponin may not be detectable within the first few hours of presentation, UA and NSTEMI are frequently indistinguishable at the time of the initial evaluation.

DIAGNOSIS

The **clinical history is critical** to the diagnosis. *Please see the 'ACS pathway' (Management of ACS section) for patient risk stratification and management.*

Patients presenting with ischaemic sounding chest pain with a diagnostic ECG (new / deeper T wave inversion, new ST depression), should be admitted and treated for NSTEMI-ACS.

These patients should be referred to the Cardiology SpR (bleep 6002) for a review within 2 hours (either by the Cardiology SpR or by an ACS nurse). If there is a clear diagnosis of ACS with a plan for invasive coronary angiography these patients should be admitted directly to a Cardiology bed. If no cardiology bed is available, the Cardiology SpR will refer to the duty Medical SpR with a management plan.

Otherwise, treat as for ACS and admit under the medical team, or seek the opinion of the ACS nurse practitioner (Mon - Fri 9am – 5pm Bleep 7138).

- Give **Aspirin** 300 mg on admission (unless previously taking Aspirin, or Aspirin contraindicated), and 75 mg daily thereafter. If the patient is intolerant of Aspirin, seek advice. If the patient is already on Aspirin there is no need to re-load. Give their usual 75mg maintenance dose if not already taken on the day of presentation.

- Give **Ticagrelor** 180 mg as a loading dose *unless*

- evidence of active bleeding;

- history of intracranial haemorrhage;

- very high bleeding risk [CRUSADE score >40, www.mdcalc.com];

- or inappropriate for invasive coronary angiography due to frailty.

This should be followed by Ticagrelor 90 mg twice daily.

Patients established on warfarin or direct oral anticoagulants, and those with bleeding risks above, should be discussed with the cardiology team first.

Those not appropriate for Ticagrelor should be considered for Clopidogrel (loading dose 600mg, daily subsequent dose 75mg od).

- Give **Morphine** 2.5-5.0 mg by slow IV injection and repeat if pain persists.

To reduce the likelihood of vomiting, give either Metoclopramide (10 mg IV over 2 mins) or Cyclizine (50 mg IV over 3 mins).

- **Oxygen now only** recommended in patients with oxygen saturation (SpO₂) <90%: as per European Society of Cardiology 2018 guidelines (previously <95%), following the AVOID and DETO2X randomised controlled trials (RCT). Hyperoxia may be harmful in patients with uncomplicated MI, presumably due to increased myocardial injury. Target SpO₂ 94-98%. In patients with chronic obstructive pulmonary disease and who are at risk of hypercapnic respiratory failure the target SpO₂ is 88-92%.

- Give **Fondaparinux** 2.5 mg SC od for 48-72 hours or until the patient has undergone percutaneous coronary intervention (maximum 8 days), *except* when patient is taking a Direct Oral Anticoagulant or Warfarin with an INR >2. If eGFR < 20 ml/min: prescribe IV unfractionated Heparin for 24-48 hrs, then VTE prophylactic dose SC unfractionated Heparin. Post PCI, patients may receive VTE prophylaxis as per Trust guidelines.

- All patients should have a lipid profile on admission (a fasting sample is not required), and should then commence Atorvastatin 80 mg, if no contraindication or history of intolerance, regardless of initial lipid profile.

Reduce dose in patients receiving interacting drugs (Clarithromycin, Cyclosporin, Protease inhibitors, Diltiazem, Amiodarone, Verapamil).

In common with patients with STEMI (see Management of STEMI section) the following are also recommended in NSTEMI-ACS patients:

- Beta blockers are recommended for all patients (see STEMI-ACS sections for contraindications and suggestions for choice and dose)

- Patients with diabetes mellitus or a blood sugar of >11mmol/L should be started on IV insulin (see STEMI section)

- ACE inhibitors (see STEMI section)

- Gastroprotection (see STEMI section)

- Intravenous GTN can be given for continuous chest pain or pulmonary oedema (see STEMI section).

Timing of coronary angiography +/- percutaneous coronary intervention (see box 4 of 'ACS pathway' in Management of ACS section)

'**High Risk NSTEMIs**' now require *urgent discussion with the on-call Cardiology registrar (Bleep 6002)*, with a view to **immediate primary percutaneous coronary intervention** (PPCI) to perfuse the occluded coronary artery.

- **High risk NSTEMIs** are defined by: ongoing significant chest pain despite GTN and IV morphine / new ST depression / haemodynamic instability / ventricular tachycardia/fibrillation.

- **< 24 hours** if any of the following:

- pain free but dynamic T wave inversion on ECG;

- pain free but large rise in troponin;

- recurrent brief ischaemic chest pains at rest (prolonged ischaemic chest pain is defined as a high risk NSTEMI as above).

- These patients require *re-discussion with Cardiology SpR (bleep 6002)*.

- **< 72 hours** if intermediate risk of ACS (see ACS pathway as above)

Further Risk Assessment

- Cardiac biomarkers (high sensitivity Troponin T – see Appendix 1) should be taken on admission and 3 hours from admission.

***** Addendum A: patient management depending on troponin concentrations. Please see ACS pathway in Management of ACS section for details. *****

****** Addendum B: dual anti-platelet therapy in high bleeding risk / high ischaemic risk patients. Please see Management of STEMI section for details. ******

MANAGEMENT OF ACUTE HEART FAILURE

Link Consultants: Dr Robin Ray & Dr Lisa Anderson*

Acute decompensated heart failure carries a 30-day mortality of 15% in those with NT-proBNP > 5000ng/L and 5% in those with NT-proBNP < 5000ng/L¹.

Please contact heart failure nurse specialists (Bleep 7376/ Ext. 4404) as soon as patients are admitted, for specialist in-patient review and for long-term management planning.

DIAGNOSIS

Heart failure should be in the differential of all elderly patients presenting with breathlessness. If heart failure is suspected, request NT-proBNP on admission bloods.

Age (yrs)	<50	50-75	>75
Acute Heart Failure likely if NT-proBNP (ng/L) is	>450	>900	>1800

If the NT-proBNP is normal (< 300ng/L), search for an alternative diagnosis. If the NT-proBNP is higher than the thresholds above, acute heart failure is likely and should be confirmed by echocardiography if not already performed. If the NT-proBNP is intermediate (above 300 ng/L but below acute heart failure levels), reconsider the diagnosis. If after full reassessment, heart failure is still considered likely, request an echocardiogram.

NICE Acute Heart Failure Quality Standards:

- Patients admitted with a new diagnosis of heart failure should have an in-patient echocardiogram prior to discharge and performed within 48 hours of admission.
- Patients admitted with heart failure should have input from the specialist HF team. ESC 2021 Guidelines³
- Patients should be fully evaluated for persistent signs of congestion and guideline directed medical therapy should be optimised before discharge.

Heart failure echo requests

1. NT-proBNP level must be documented on the request form.
2. Repeat echo is not necessary if there is an echo within the last 6 months unless there has been a change in clinical condition or a new lesion (eg. new murmur) is suspected.

MANAGEMENT OF ACUTE HEART FAILURE

Pulmonary oedema reverses rapidly with diuretics, but will recur rapidly after discharge if the underlying cause is not addressed. Audit shows that 50% of patients admitted to CCU with pulmonary oedema require an interventional cardiac procedure (cardiac surgery/TAVI/revascularisation/CRT or ablation).

Acute pulmonary oedema:

- Call the cardiology SpR (Bleep 6002) to arrange admission to CCU and to assess for CHAMPIT causes (ACS/HT emergency/Arrhythmia/Mechanical or valvular/PE/Infection/Tamponade)
- O₂ to maintain SaO₂ (95-98%)
- IV furosemide 40-80mg bolus followed by an infusion at 5-20 mg/hr if required
- Maintain systolic BP > 100mmHg and monitor in a level 2 area
- CPAP (with mechanical ventilation for respiratory failure, physical exhaustion and if appropriate for the patient) Consider IV GTN infusion (10-200 micrograms/min) for patients with concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease.

General measures

- Monitor: pulse, check oximetry and blood pressure every 5-10 mins with continuous ECG. If cardiogenic shock develops, contact cardiology SpR immediately.
- Request chest X-ray; FBC, U&E's, creatinine, NT-proBNP TFTs, LFTs, troponin, glucose, lipids, HbA1c and iron studies.
- Check arterial blood gases if low oxygen saturation/requiring oxygen.
- Review medication: stop nondihydropyridine Ca²⁺ channel blockers (verapamil and diltiazem) and NSAIDs..
- In unstable patients with diabetes, switch to insulin sliding scale.
- Continue ACE and Beta-Blockers: even if the first dose(s) need to be omitted due to hypotension. Withdrawal of beta-blockers in acute heart failure increases mortality risk.
- If patient presents in fast atrial fibrillation and pulmonary oedema, commence digoxin initially until beta-blockers can be initiated. If the patient is unstable, arrange a DC cardioversion.

Management of Chronic Heart Failure with Left Ventricular Systolic Dysfunction (LVEF<40%)³

To reduce mortality, the following 4 classes of drugs should be used in all patients.

1. ACE inhibitors/ARNI (eg sacubitril valsartan – HF team to initiate). Start ACE- (*e.g.* ramipril) and titrate until reviewed by HF team.
2. Beta-blockers. Start a betablocker *e.g.* bisoprolol and titrate upwards
3. Mineralocorticoid Receptor Antagonist.
Start Spironolactone or Eplerenone 12.5–25mg od.
4. SGLT2-. Contact the HF team to commence SGLT2-.

Diuretics are used for the relief of congestive symptoms and fluid retention only.

Intravenous iron should be considered in stable patients prior to discharge if LVEF <50%, ferritin < 100ng/ml or 100-299ng/ml with TSAT <20% and Hb <145.

Additional Treatment for selected patients (HF team review)

CRT +/- Defibrillator.

Ivabradine

Angiotensin receptor blocker if intolerant of ACE-/ARNI.

Revascularisation/valve intervention

Management of Heart Failure with Preserved LV Function (HFpEF)

Fluid balance management

Blood pressure management

DISCHARGE AND FOLLOW-UP

All patients with acute heart failure need community heart failure nurse follow-up. Please arrange this via the in-patient heart failure nurses (ext. 4404, Bleep 7376). Follow-up arrangements should be clearly documented.

- If ACE inhibitors, beta-blocker or spironolactone doses have been reduced or discontinued during the admission, state the reason (*e.g.* hypotension, renal impairment, hypo/hyperkalaemia) in the discharge summary so that re-initiation can be considered in the community.
- Record patient's weight on discharge and presence of any residual oedema at this weight.

¹ Chronic heart failure in adults: Diagnosis and management. NICE Clinical Guideline 109, September 2018

² NICE Acute Heart Failure: diagnosis and management in adults. Clinical Guideline 187 Quality Standards (NICE 2015)

³ ESC Heart Failure Guidelines 2021. EHJ 2021. 42, 3599-3726

DISORDERS OF CARDIAC RHYTHM

Link Consultant: Dr Anthony Li

SINUS BRADYCARDIA

This requires no treatment unless it is causing symptoms. If treatment is deemed necessary, give atropine 600-1200micrograms IV in the first instance. Persistent symptomatic bradycardia requires pacing (temporary or permanent). If temporary pacing is required, transvenous pacing under X-ray control is optimal. For advice, contact the Cardiology registrar on call.

ATRIOVENTRICULAR BLOCK

First and second-degree block found incidentally do not usually need emergency treatment but further investigation is often necessary. After acute MI patients with second degree block will need temporary pacing if the block is impairing cardiac function. Complete (3rd degree) AV block requires careful evaluation and should be discussed with the cardiology registrar on call immediately. Overnight admissions must be discussed with the on-call consultant by the Cardiology registrar before a decision not to insert a temporary pacemaker is taken.

<i>Indications for Temporary Pacing – Emergency/Acute</i>
Acute MI with: <ul style="list-style-type: none"> ▪ Asystole ▪ Symptomatic bradycardia not responsive to atropine ▪ Bilateral bundle branch block (alternating BBB or RBBB with alternating LAHB/LPHB) ▪ New or indeterminate age bifascicular block with 1st degree AV block ▪ 2nd or 3rd degree AV block after an acute anterior MI
Bradycardia not associated with acute MI: <ul style="list-style-type: none"> ▪ Asystole ▪ Any symptomatic bradycardia resistant to medication ▪ 2nd or 3rd degree AV block with haemodynamic compromise or syncope at rest ▪ Asymptomatic AV block with severe bradychardia (<30bpm) +/- QRS>120ms+/- QTc prolongation >500ms
VT secondary to bradycardia eg Torsades de Pointes
Suppression of drug-resistant VT or SVT
Drug overdose, eg. digoxin, beta blockers, verapamil

<i>Indications for Temporary Pacing – Elective</i>
<ul style="list-style-type: none"> ▪ Support for procedures that may promote bradycardia ▪ General anaesthesia with: 2nd or 3rd degree AV block; intermittent AV block ▪ Cardiac surgery when epicardial pacing has failed ▪ Rarely considered for coronary angioplasty

Patients with symptomatic block usually require immediate pacing even if symptoms have resolved upon arrival. Asymptomatic patients with marked bradycardia (heart rate <40bpm), a broad complex escape rhythm (QRS >120ms) and/or significant QT prolongation (QTc >500ms) are at high risk of arrest and will require urgent pacing. This is preferably achieved by prompt implantation of a permanent pacemaker but a temporary one may suffice for overnight/over-weekend management.

Complete AV block associated with inferior myocardial ischaemia is usually transient but will require temporary pacing if the patient is compromised or permanent pacing if

the heart rate remains slow. When associated with anterior infarction temporary pacing is always indicated regardless of presence or absence of symptoms. Patients with acute bifascicular block following acute myocardial infarction should be considered for temporary pacing particularly if the PR interval is increased or increasing. If required temporary transcutaneous pacing can be achieved rapidly but is rarely needed; contact the cardiology registrar for advice immediately.

SUPRAVENTRICULAR TACHYCARDIAS.

The commonest types are:

- a) atrial fibrillation, atrial flutter and atrial tachycardia
- b) junctional re-entry tachycardia (AV nodal and atrioventricular)

A 12-lead ECG must be obtained in all cases. It is important to diagnose the disturbance accurately, as therapy will depend on the particular rhythm. All types can be paroxysmal or persistent and treatment should be tailored accordingly. Paroxysms should be terminated and preventive treatment started. Chronic arrhythmias which cannot be terminated should be slowed.

Chronic AF, flutter and atrial tachycardia can be treated with AV nodal blocking drugs (diltiazem, beta-blockers, digoxin). AF of recent onset (≤ 48 hours) is best terminated by IV flecainide (1-2mg/kg over 10 min; maximum dose 150mg). In the presence of heart failure or acute ischaemia, amiodarone should be used (300mg bolus via large bore cannula in a large vein or centrally, then a 900mg 24-hour infusion). For more urgent and effective treatment DC cardioversion may also be considered. Unless otherwise contraindicated, patients in AF for more than a day should be anticoagulated as they are at risk of developing cardiogenic embolism. In some patients acute cardioversion is appropriate and Trans-Oesophageal Echocardiography will be required to exclude thrombus; seek advice from the on-call cardiology registrar.

Junctional re-entry tachycardias are most effectively terminated with IV adenosine. Give an initial 6mg dose over 2 secs. If no effect is seen within 1 min give a second injection of 12mg. Further doses are not recommended. Remember, adenosine should not be given to patients with asthma or severe obstructive airways disease. Intravenous verapamil (5mg) can be used as an alternative. If the patient is refractory to drugs seek advice. All supraventricular arrhythmias may be treated by ablation. Patients who have syncope due to Wolff Parkinson White (WPW) syndrome or atrial flutter with 1:1 conduction, should be referred immediately to the cardiology registrar on call and considered for urgent in-patient ablation. Any other patient who has an episode of atrial flutter or junctional re-entry tachycardia should be referred to an interventional electrophysiologist as an outpatient so that therapy by ablation can be discussed. Patients with recurrent and highly symptomatic AF should also be referred.

NB DO NOT GIVE AV NODAL BLOCKING DRUGS TO WPW PATIENTS WITH PRE-EXCITED ATRIAL ARRHYTHMIAS.

VENTRICULAR TACHYCARDIA (MONOMORPHIC)

This is very common and may present with a wide range of symptoms from moderate discomfort (haemodynamically stable tachycardia) to profound collapse or arrest (haemodynamically unstable tachycardia). Do not be misled into thinking that stability excludes a diagnosis of VT.

The commonest causes include acute infarction/ischaemia and chronic left ventricular scarring after infarction. First get the diagnosis correct by examining the 12 lead ECG. If this cannot be obtained because of collapse, urgent DC shock is required – otherwise record the ECG. Most instances of VT can be correctly diagnosed but if in doubt treat broad complex tachycardia as VT. Features of VT include:

- wide QRS complexes (more than 0.14 sec or 3.5 small squares).
- AV dissociation sometimes with capture and fusion beats;
- a leftward axis shift compared to sinus rhythm;
- any previous history of IHD (MI, PTCA, CABG)

Therapy depends on the clinical situation and whether the patient has an implantable cardiac defibrillator (ICD). If the patient is hypotensive, in cardiac failure or has ischaemia, cardioversion should be undertaken. If stable then initial treatment should be with lidocaine 1.5mg/kg IV. If this terminates tachycardia continue as an infusion at 2mg/min for up to 24 hours. If tachycardia continues an additional lidocaine bolus of 0.5-0.75mg/kg should be considered. Otherwise consider giving amiodarone (300mg bolus via large bore cannula in a large vein or centrally, then a 900mg 24-hour infusion). *Do not give more than one additional drug* – polypharmacy can be dangerous. If drug therapy fails, or the patient has poor cardiac function, direct current cardioversion (150J-200J Biphasic) under sedation is the best therapy (if help needed contact the cardiac registrar for advice). Whatever method is used, full facilities for resuscitation should be to hand. Further cardiological assessment is mandatory in all cases not associated with acute ischaemia or infarction. Remember to check electrolyte levels. The administration of magnesium, initial dose 8mmol (4mL of 50%) may help when the arrhythmia is refractory.

NB: DO NOT TREAT A POSSIBLE VT WITH VERAPAMIL

Some patients presenting with ventricular arrhythmias who have an ICD implant may have received shocks from the device. The presence of an ICD does not prevent the use of emergency defibrillation or cardioversion in the event of a cardiac arrest or compromising VT that has not responded to ICD therapy. As appropriate, follow relevant ALS protocols or arrhythmia management strategies as described above, but attempt to defibrillate away from the device itself (usually left infraclavicular site). Haemodynamically stable VT or successfully treated patients may benefit from immediate ICD reprogramming. Contact the ICD clinic (ext.1372) 9-5pm, Monday-Friday for assistance, as well as the Cardiology registrar. Out of hours the cardiology registrar should be called and the on-call ICD technician contacted if required. If patients present with multiple shocks from their ICD and there is cause to believe these may be inappropriate (i.e. shocked whilst on a monitor with no ventricular arrhythmia noted), a magnet should be placed over the device and the patient placed on continuous cardiac monitoring. All such patients should be admitted to cardiology.

POLYMORPHIC VT

This is less common and usually causes presyncope, syncope or cardiac arrest depending on the duration of arrhythmia. It may be associated with QT prolongation (Torsade de Pointes) when temporary pacing, betablockers and potassium and magnesium replacement may treat the arrhythmia successfully but precipitants such as certain drugs or hypokalaemia must be removed. ***Beware of subarachnoid haemorrhage as a cause.*** Other causes include ischaemia when QT prolongation may not be present. Betablockers and urgent assessment for cardiac catheterisation will be necessary. Involve the cardiology registrar on call early in these cases.

VENTRICULAR FIBRILLATION (see Cardiac Arrest).

VENTRICULAR ECTOPIC BEATS.

These are ubiquitous and do not require treatment unless they are causing symptoms such as palpitations or dizziness, when the patient should be referred for investigation and management. The urgency of this or the need for in-patient investigation will depend on the severity of symptoms. Eg. syncope requires in-patient assessment. Frequent ectopy, whether symptomatic or not, may indicate underlying structural heart disease and referral for non-urgent investigation as a minimum requirement is appropriate.

ASYSTOLE (see Cardiac Arrest)

Patients with acute MI who develop CARDIAC FAILURE or CARDIOGENIC SHOCK, should be referred to the on-call cardiology registrar as soon as possible.

ACUTE DEEP VEIN THROMBOSIS (DVT)

Link Consultant: Dr Pamala Kanagasabapathy

DVT is common, particularly in hospital. Above knee thromboses can extend proximally and embolise to the lungs. Treatment aims to reduce the risk of embolism and restore vein patency so avoiding the long-term problems of venous obstruction. If the DVT occurs during pregnancy, involve the obstetricians before proceeding.

Arrangements for diagnosis

Diagnosis of acute DVT should be confirmed as soon as possible by compression duplex ultrasound.

If DVT is suspected and there will be a delay of more than four hours before ultrasonography, then therapeutic anticoagulation (see Standard Treatment below) should be commenced unless there is a contraindication (these cases should be discussed with haematology). If DVT is excluded then therapeutic anticoagulation should be discontinued.

- **Inpatients** should have an ultrasound requested on iClip and the Ultrasound (US) Department should be contacted – the scan can then be performed on the next inpatient list.
- **Outpatients** please see the ‘DVT pathway’ on the ED homepage (<http://stginet/Units%20and%20Departments/Emergency%20Department/Medicine%20Guidelines/Medicine%20Guideline.aspx>)
- Patients with confirmed DVT will be assessed by the **Thrombosis clinical nurse specialist** (blp 7380; x2826). They will initiate treatment, counsel the patient and book the patient into the anticoagulation clinic for follow up.
- Patients at enhanced risk of bleeding (eg. those with liver disease, peptic ulcer, alcohol abuse, uncontrolled hypertension, heart failure, recent major trauma, GI, GU or ICH less than 4 weeks ago, or on drugs that interfere with the anticoagulant’s effect), need to be assessed before treatment is started and may require admission to hospital.
- The following patients are not suitable for ambulatory management: pregnant, age under 16, suspicion of PE, co-existent serious medical pathology, bilateral DVT or extension to common iliac vein, severe acute venous obstruction, heparin sensitivity or history of HIT, angina or SOB on minimal exertion, no fixed abode, compliance issues, unable to understand instructions, CrCl <15ml/min.
- *If there is clinical suspicion of active major bleeding, anticoagulation should be withheld while urgent confirmatory tests are performed. Evaluation of the relative risk of bleeding vs thromboembolism is required.*

Treatment

1. For positive compression ultrasound

Standard Treatment

If a compression ultrasound done within working hours confirms a DVT, and provided anticoagulants are not contraindicated immediately, start rivaroxaban (provided that creatinine clearance is ≥ 15 ml/min). The dose is 15mg twice daily for 21 days post-acute thrombosis and then 20mg once daily for maintenance from

day 22 onwards (if creatinine clearance ≤ 50 ml/min and the risk of bleeding outweighs the risk of recurrent thrombosis then use 15 mg once daily for maintenance).

Details on the use of rivaroxaban for VTE treatment are available on the Trust Intranet:

<http://stginet/Units%20and%20Departments/Haematology/ANTICOAGULATION/VTE%20Treatment%20Algorithm%20DVT.pdf>

For cancer-associated thrombosis, pregnancy-related thrombosis or patients who are breast feeding, therapeutic dose dalteparin should be given (rather than rivaroxaban). Dalteparin should not be used in patients with a CrCl <30ml/min. IV unfractionated heparin is a short-term option for inpatients as per Trust heparin policy. For long-term management options in patients with chronic renal impairment please contact haematology for advice.

For renal transplant patients taking immunosuppressive therapy please contact a renal specialist for advice (regardless of the patient's renal function).

Second Line Treatment

If the patient is intolerant/contraindicated to rivaroxaban or has developed further thrombosis on rivaroxaban, please contact haematology to discuss an alternative Direct Oral Anticoagulant (DOAC).

If there is a contraindication to DOAC therapy then therapeutic dose dalteparin (or unfractionated heparin) should be used, followed by warfarin

i) For patients initiated on rivaroxaban

For patients initiated on rivaroxaban, a 'Screening and notification of initiation of treatment' form needs to be completed and faxed to [the](#) patient's GP; available via: <file:///\\stg1nas01\formulary\Rivaroxaban%20for%20VTE%20-%20Notification%20of%20Initiation%20of%20Treatment.pdf>

The patient should be referred to the anticoagulation team using a referral on iClip, as they will require a follow up appointment at the St George's thrombosis clinic to assess tolerance and obtain further supplies of rivaroxaban. Patients must be provided with a relevant patient information pack – '[Rivaroxaban: A patient's guide to deep vein thrombosis treatment](#)', an [anticoagulant alert card](#) and verbally counselled about the use of rivaroxaban.

These are available from pharmacy or the anticoagulation clinic, and are also kept as stock in ED and on Richmond Ward.

ii) For patients initiated on warfarin.

The INR should be checked in the anticoagulation clinic within 3-4 days and the dose adjusted as necessary. Warfarin dosing clinic runs on Tuesdays and Fridays in St James Wing. The dose of dalteparin, which should be given subcutaneously whilst the patient's INR is subtherapeutic, is dependent on the patient's weight according to the schedule below:

Body Weight	Daily Dalteparin Dose
Under 46kg	7,500 units od
46-56kg	10,000 units od
57-68kg	12,500 units od
69-82kg	15,000 units od
83-110kg	18,000 units od
over 110kg	10,000 units bd

NB. These doses differ from the BNF dosing for patients over 110kg. This is due to inhouse experience and expert consensus. Please use the above Trust doses and not the BNF doses.

The dose of dalteparin should be continued until the INR is within range **on two consecutive days**, 24 hours apart. If dalteparin is given for more than five days, assess renal function and **if creatinine clearance is ≤ 30 ml/min** use unfractionated heparin. For patients needing ambulatory management of DVT, dalteparin injection can be selfadministered. However, patients should be referred to the district nurse team if they are unable to self-administer the injections or if they are discharged on unfractionated heparin. Patients discharged with supplies of dalteparin should be provided with a sharps bin. Information on disposal of sharps bins can be found on the Anticoagulation and Thrombosis homepage.

The duration of anticoagulation therapy varies and should **be decided in the thrombosis clinic**.

For in-patients **being discharged on warfarin**, an INR is required on the day of discharge to allow dose forecasting. The anticoagulation team will issue a Yellow Anticoagulant booklet and provide information on dose forecasting. For patients within the St George's catchment area for warfarin monitoring (SW11, SW12, SW16-19, CR4), the anticoagulation team will arrange a follow up appointment at the St George's Anticoagulation clinic. For patients who fall outside of the St George's catchment area for warfarin monitoring, the ward team will need to make an appointment on the next available clinic date for the patient at their nearest warfarin clinic.

Details of the discharge procedure are available on the anticoagulant web page <http://stginet/Units%20and%20Departments/Haematology/ANTICOAGULATION/Anticoagulation%20and%20Thrombosis.aspx>

Draw the patient's attention to the counselling points in the yellow booklet.

For INR ranges for different indications see the Anticoagulation Clinic Guideline

2. For negative compression ultrasound

If the initial compression ultrasound is negative, discharge the patient from DVT care and consider other possible diagnoses. If there is a high clinical suspicion for DVT in the absence of a positive ultrasound use clinical judgement. This could include repeating the US Doppler within 5–7 days and continuing anticoagulation if the clinical situation suggests a high risk of DVT. A scheme for this assessment, together with advice on the additional diagnostic tests that might be needed in this situation, is available from the **thrombosis clinical nurse specialist** (Bleep 7380; Ext. 2826). Out-of-hours advice can be given by the on-call haematology registrar via switchboard.

ACUTE PULMONARY EMBOLISM

Link consultant: Dr Adrian Draper

Pulmonary embolism (PE) should be considered in anyone presenting with:
Breathlessness, chest pain, cough/haemoptysis and syncope.

Various risk factors increase the likelihood of the patient having a PE but arguably the most important are recent lower limb surgery/immobilisation, pregnancy/immediate post partum period and active malignancy.

In suspected PE use a clinical prediction score to assist diagnosis such as the revised Geneva clinical prediction tool:

Items	Original	Simplified
Previously objectively diagnosed DVT or PE	3	1
<i>Heart rate</i>		
75-94 bpm	3	1
≥/≤ 95 bpm	5	2
Surgery or fracture in last month	2	1
Haemoptysis	2	1
Active cancer	2	1
Unilateral lower limb pain	3	1
Pain on lower limb deep venous palpation & unilateral oedema	4	1
Age >65 years	1	1
Clinical probability		
<i>Three-level score</i>		
Low	0-3	0-1
Intermediate	4-10	2-4
High	≥11	≥5
<i>Two-level score</i>		
PE-unlikely	0-5	0-2
PE-likely	≥6	≥3

If PE is clinically suspected arrange CTPA and commence anticoagulation.

Once PE is confirmed use PESI score to assess risk of mortality and classify risk as below:

Risk factor	Score	Class	Mortality
Age	Age in years	Class I: ≤ 65 points	Very low: 0-1.6%
Male Sex	+10 points		
Cancer	+30 points	Class II: 66-85 points	Low: 1.7-3.5%
Chronic heart failure	+10 points		
Chronic pulmonary disease	+10 points		
Heart rate >110 beats/min	+20 points	Class III: 86-105 points	Moderate: 3.2-7.1%
Systolic BP < 100mm Hg	+30 points		
Respiratory rate > 30/min	+20 points		
Temperature < 36°C	+20 points	Class IV: 106-125 points	High: 4.0-11.4%
Altered mental status	+60 points		
Arterial saturation < 90%	+20 points	Class V: > 125 points	Very high: 10.0-24.5%

Classification of PE severity and early in-hospital mortality risk:

Early mortality risk	Haemodynamic instability	PE Severity PESI class III or IV	RV dysfunction (Echo or CTPA)	Elevated cardiac troponin
High	+	(+)	+	(+)
Intermediate-high	-	+	+	+
Intermediate-low	-	+	One (or none) positive	
Low	-	-	-	-

Patients who are class I-II can be considered for early discharge on Rivaroxaban. Rivaroxaban should be avoided if severe renal impairment or antiphospholipid syndrome (use heparin followed by warfarin).

Patients who are class III-V should have further testing with troponin T and ECHO. Patients with positive troponin and RV dysfunction should be monitored and if they deteriorate haemodynamically should receive thrombolysis. Patients with one of positive troponin T or RV dysfunction (intermediate-low risk) require hospital monitoring whilst initiating Rivaroxaban for a few days with clinical judgement regarding discharge.

High risk patients should be thrombolysed with Alteplase. Provided there are no contraindications, give Alteplase as an infusion of 100mg over 2 hours or if cardiac arrest is imminent 0.6mg/kg over 15 minutes (max 50mg), followed by IV unfractionated heparin for 48 hours.

Where thrombolysis is contraindicated consider catheter directed therapy or surgical embolectomy.

Intermediate-high risk patients (who could deteriorate and require thrombolysis) should be commenced on IV unfractionated heparin with a weight adjusted bolus dose.

*** Definitions of Haemodynamic instability**

1 Cardiac arrest	Needs CPR
2 Obstructive shock	Systolic BP <90 mmHg or vasopressors needed to maintain BP \geq 90mmHg despite adequate filling <i>and</i> End-organ hypoperfusion (altered mental state; cold, clammy skin; oliguria/anuria; increased lactate)
3 Persistent hypotension	Systolic BP < 90mmHg or systolic BP drop \geq 40 mmHg, lasting longer than 15 min and not caused by new onset arrhythmia, hypovolaemia or sepsis

Subsequent management of confirmed PE

Patients will require oxygen therapy if hypoxic, and analgesia if in pain (paracetamol is often sufficient). Rivaroxaban should now be considered first line anticoagulant for the treatment of PE unless the patient has high risk of death PE (use IV heparin), or is pregnant or breastfeeding (use LMW heparin) or has GI cancer. While awaiting confirmation of PE, the patient should receive their first ‘loading dose’ of rivaroxaban at 15mg twice daily, *unless there is clinical suspicion of active major bleeding, anticoagulation should be withheld while urgent confirmatory tests are performed. Evaluation of the relative risk of bleeding versus thromboembolism is required.*

If PE is confirmed, please refer patient to **thrombosis team** ext.1332 or b/p 8409 and assess pulmonary embolism severity score (PESI). Rivaroxaban should continue at a dose of 15mg bd for 3 weeks followed by a maintenance dose of 20mg od (the maintenance dose should be reduced to 15mg od in patients with mild-moderate renal impairment CrCl 15-49ml/min if risk of bleeding outweighs risk of recurrent thrombosis). Unfractionated heparin should be used in renal failure CrCl< 15ml/min. An alternative DOAC can be used on the advice of a haematologist if rivaroxaban is contraindicated or not tolerated. If DOACs are contraindicated or not tolerated use warfarin.

After discharge it is essential patients are referred for follow up by both chest and thrombosis clinics where decisions regarding duration of therapy will be made.

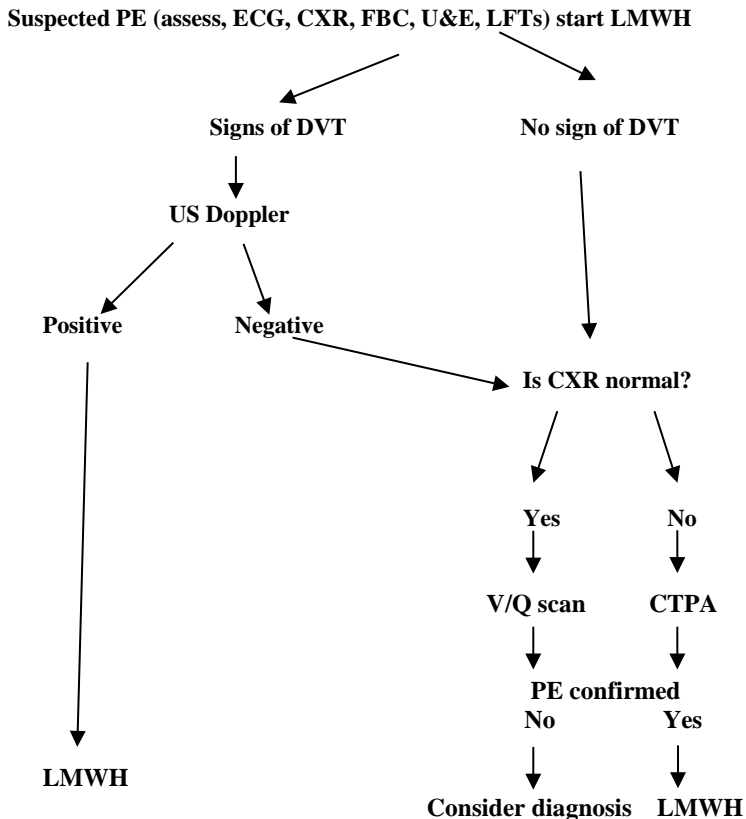
Pregnancy and PE

Pregnancy and the immediate post-partum period are important risk factors for venous thromboembolism. During pregnancy there are often medical and maternal

concerns about imaging involving radiation. The major issues relate to radiation dose to the foetus (perfusion scanning) versus radiation dose to mother (CTPA radiation to breasts).

The RCOG recommends performing imaging as required and there is no justification for avoidance of imaging (the total foetal radiation dose of a CXR, perfusion scan and CTPA is still less than background radiation for the duration of gestation).

Please follow the algorithm below.



References

2019 ESC Guidelines on diagnosis and management of acute PE

<https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Acute-PulmonaryEmbolism-Diagnosis-and-Management-of>

RESPIRATORY ARREST

Link Consultant: Dr Clare Shoults

Respiratory arrest must be reversed rapidly if the patient is to survive. The cause should be determined as soon as possible; the common causes in hospital include:

- Acute respiratory disorder, eg asthma, severe pneumonia.
- Acute or chronic respiratory failure.
- Overdose of respiratory depressant drugs, eg morphine, barbiturates.
- Obstruction, eg foreign body. Laryngeal impaction quite often leads rapidly to cardiac arrest. The heart will probably re-start with a few chest compressions and before intubation has been attempted. The possibility of obstruction should always be kept in mind. Arrest can also occur in patients who are already intubated if the tube is suddenly obstructed.
- Neuromuscular failure, eg Guillain-Barre syndrome, myasthenia gravis. In these conditions there is usually a warning period of decreasing vital capacity and tidal volume. This should be looked for as dyspnoea may be absent until the failure is well advanced.
- Secondary to cardiac arrest.
- Plugging of a tracheostomy.

Airway management

Once obstruction by a foreign body has been excluded or removed the initial management involves either mouth-to-mouth breathing, or insertion of an airway and breathing by means of mouth-to-mask or bag and mask techniques. If cardiac output has ceased, as judged by the pulse, external cardiac massage must be undertaken. In most patients, subsequent treatment will consist of endotracheal intubation followed by hand ventilation with 100% oxygen. Intubation should be attempted by the first person arriving with the necessary experience; in difficult cases this will need the help of an anaesthetist. Continued bag and mask ventilation is the best option if intubation skills not available.

Treatment of the cause

The underlying cause of the arrest should be treated as appropriate. Non-specific respiratory stimulants are of little value. However, when the arrest has been caused by an opiate, Naloxone should be given. The initial dose is 0.4mg IV and if the patient fails to respond, the dose should be repeated every 2-3 mins until depression is reversed. If IV access is not available, Naloxone can be given IM or subcutaneously. The drug is not effective in buprenorphine overdose but will occasionally work in patients with alcohol overdose. If arrest is secondary to benzodiazepine overdose try flumazenil IV (200micrograms over 15 sec followed by 100micrograms every 60 sec if required, up to 1mg total dose). Use with caution, if other psychotropic drugs (especially tricyclic anti-depressants) may have been ingested as their toxic effects may be potentiated; if the patient is known to be benzodiazepine dependent; or if the patient is epileptic and has been taking a benzodiazepine for a prolonged period. Flumazenil has a short duration of action, the patient should remain under close observation until all possible central benzodiazepine effects have subsided.

Tracheostomy problems

If the patient has a plugged tracheostomy, clear the secretions by suction, re-inflate the cuff and seek advice from an ENT, anaesthetic or respiratory registrar urgently. Guidelines for the care of patients with tracheostomies generally are on each ward.

Ongoing management

Most patients who survive a respiratory arrest will require intermittent positive pressure ventilation. This should be carried out on the Intensive Therapy Unit under the

strictest supervision. Even if the patient is deemed not to require intermittent positive pressure ventilation, any patient who has had a respiratory arrest should be closely watched for the next 24 hours and their management discussed with a member of the respiratory, or ITU, team.

Respiratory failure

In some situations the occurrence of respiratory arrest is preventable. Patients with type one respiratory failure who are tiring should be moved urgently to the high dependency unit as they may need invasive ventilation.

The indications for non-invasive ventilation (NIV) are:

- acute hypercapnic respiratory failure in the acute, or acute-on-chronic, patient who does not yet require tracheal intubation and who has

- a $p\text{CO}_2 \geq 7$

- a $\text{pH} < 7.35$

- an increased respiratory rate despite optimisation with oxygen therapy

- acute hypercapnic respiratory failure with chest wall deformity, neuromuscular disorder or decompensated obstructive sleep apnoea

- cardiogenic pulmonary failure refractory to CPAP

- patients who might otherwise receive tracheal intubation, but in whom this is better avoided or not appropriate

- patients being weaned from mechanical ventilation

Patients requiring NIV should be discussed with the respiratory registrar or, if out-of-hours, with the respiratory consultant on call. Full NIV guidelines are on the

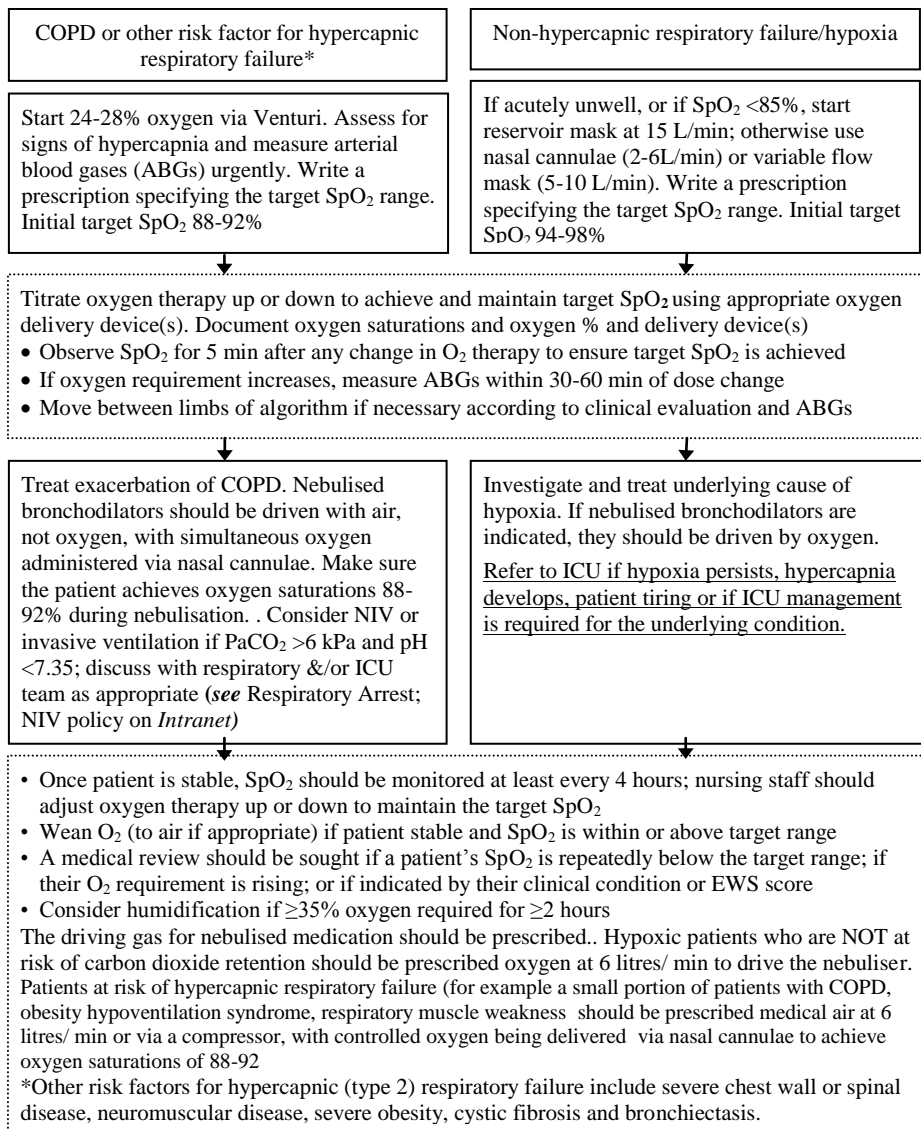
Intranet http://stginet/Policies/Clin 5-PatientMment/Clin_5_25.pdf

OXYGEN THERAPY IN ACUTE ILLNESS

Link: Dervilia Griffin, Lead Respiratory Nurse Specialist

Appropriate oxygen therapy is a vital component in the management of acute illness; it must be administered urgently in critical illness and in patients with severe hypoxia. However, excessive oxygenation generally provides no extra benefit, and may be harmful, particularly in patients with chronic respiratory failure. Therefore oxygen therapy must be titrated to maintain a target oxygen saturation (SpO₂) range, guided by a written prescription. In emergencies, it should *initially* be administered without prescription. Refer to BTS Guideline for oxygen use in adults in healthcare and emergency settings (2017) www.brit-thoracic.org.uk

Algorithm for Oxygen Therapy in Acute Illness



ASTHMA

Link Consultants: Dr Jane Evans & Dr Sachelle Ruickbie

In the UK approximately 1500 people die each year from acute asthma. Failure to recognise and appropriately manage acute severe asthma are contributory factors. Patients presenting with any of the following features should be considered unstable and may warrant admission:

- nocturnal symptoms interrupting sleep (usually cough and dyspnoea)
- worsening cough
- increased use of β_2 -agonists (less effective and relief shorter lasting)
- decreased efficacy of rescue medication (such as corticosteroids)

Remember that a previous admission to hospital, particularly if it required treatment in ITU, should be taken to indicate that the patient is prone to life-threatening episodes.

The features of severe asthma include:

- peak flow < 50% predicted or best achievable by patient
- tachypnoea (> 25 breaths/min)
- tachycardia (> 110 beats/min)
- unable to complete full sentences

The features of potentially fatal asthma include:

- peak flow < 33% predicted or best achieved by patient
- cyanosis/hypoxia
- silent chest on auscultation
- bradycardia/hypotension

MANAGEMENT – Monitoring

Measure arterial blood gases on admission and repeat as necessary to assess progress. A PCO_2 greater than 6kPa suggests the patient is at imminent risk of respiratory failure and so in need of mechanical ventilation. Use pulse oximetry to monitor the patient's oxygen saturation and assist in assessing response to treatment if the patient has either deteriorated rapidly over a few hours or has previously been in ITU with an attack of asthma. Record peak flow on initial assessment, before and after bronchodilator treatment, and again after at least one to two hours.

MANAGEMENT – Treatment

Oxygen: Patients with acute severe asthma are hypoxaemic and this should be corrected urgently with controlled supplementary oxygen adjusted to keep SAO_2 94-98%.

Bronchodilators: A bronchodilator, such as Salbutamol (2.5-5 mg) should be started as soon as possible via an oxygen-driven nebuliser (drive at a flow rate of at least 6 L/min). This dose can be repeated at 15-30 min intervals if no improvement is seen. Nebulised Ipratropium bromide (500 micrograms) helps in about 30% of patients with acute asthma and may be given every 6 hours. Parenteral/IV beta-2 agonists may have a role in ventilated patients or those in extremis but there is limited evidence to support this.

Corticosteroids: Patients should be given Hydrocortisone 100 mg IV 6-hourly or Prednisolone 40-50 mg od by mouth as soon as the initial assessment is made. No material benefit can be expected for several hours but it is essential not to delay administration. Whichever steroid is given initially, corticosteroids should be continued for a minimum of 5 days or until recovery. The Prednisolone dose does

not need to be tapered off, unless the patient is on a maintenance dose or steroids are required for more than 3 weeks. Inhaled steroids should be started as soon as possible.

Hydration: Some patients require intravenous hydration. Monitor electrolytes, particularly potassium, as hypokalaemia may develop.

Magnesium: In patients with severe asthma who respond poorly to initial treatment, or with life-threatening asthma, after discussion with senior medical staff, consider giving a single dose of intravenous Magnesium at a dose of 2 g (8 mmol) in 250 mL of NaCl 0.9% over 20 minutes.

Aminophylline: Intravenous Aminophylline should only rarely be given in acute asthma because it is difficult to use and has limited efficacy. Its administration should be limited to patients in whom all other treatments have failed, the patient continues to deteriorate and intubation is imminent. Cardiac monitoring is essential during administration and levels should be monitored.

Inpatient Management: A progressive improvement in morning peak flow should be seen before discharge. Patients should normally be transferred from nebulised to inhaler therapy when peak flow approaches normal limits. Prior to discharge, it is essential to check that the patient has a good inhaler technique, that if the technique is poor the patient is re-taught, and that the correct device is prescribed for their needs.

Discharge: Patients should be discharged on inhaled and/or oral steroids (as appropriate to their previous history and current severity) and an asthma action plan. They should be reviewed by their GP in 2 days and by an asthma specialist within 4 weeks. Peak flow monitoring should be undertaken by patients who have difficulty telling if their asthma is deteriorating. The Respiratory Nurses (Bleep 7697) can provide advice on asthma management (patient 'self-management plan') and on follow-up arrangements. For specific advice first contact the on call respiratory SpR (Bleep 6614) or consultant.

SPONTANEOUS PNEUMOTHORAX

Link Consultant: Dr Helen Meredith

The sudden entry of air into a pleural space and the subsequent collapse of the underlying lung presents with pain or shortness of breath (or both) or very rarely with cardiorespiratory arrest (as occurs in a tension pneumothorax). In most instances the air enters through a spontaneous leak in the pleura and no precipitating factor is found; alternatively air entry may follow trauma or surgery.

MANAGEMENT

For most patients there is no immediate threat. Once a pneumothorax is suspected, x-ray the chest to confirm the diagnosis, to assess the degree of any collapse. The need for intervention is primarily based on symptoms. We also use the size of the pneumothorax on chest x-ray to guide the decision to intervene, although this is less important compared to the degree of symptoms. A large pneumothorax is defined as a visible rim of air > 2cm between the lung margin and the chest wall (at the level of the hilum).

Symptoms in primary spontaneous pneumothorax (age <50 and no underlying lung disease) may be minimal and frequently no intervention is needed.

Secondary spontaneous pneumothoraces (known underlying lung disease; assume if age > 50) often have a higher morbidity.

Tension pneumothorax

Patients with a tension pneumothorax will require immediate aspiration of the entrapped air followed by intercostal tube drain placement. This is a clinical diagnosis and an emergency; a chest X-ray should not be taken until after the chest drain is inserted. Cardiac arrest can occur, so be prepared to start CPR immediately. Signs of severe breathlessness and respiratory distress indicate a potential tension pneumothorax.

Primary Spontaneous Pneumothorax

This is often a benign condition. Use symptoms to guide management decision.

See diagram below (BTS Pleural guidelines 2010)

If patient requires no intervention, discharge with written advice to re-attend the ED if worsening breathlessness. Refer to Pleural clinic 1-2 weeks via ED respiratory referral form or e-mail h.meredith@nhs.net.

Secondary Spontaneous Pneumothorax

All patients with underlying lung disease should be admitted to hospital and will require at least overnight observation and may require chest drain placement. CT guided drain placement may need to be considered if the patient has decompensated but only has a small pneumothorax.

See **Management flow chart below** (BTS Pleural guidelines 2010).

Pleural procedures for pneumothorax:

The chest drain or aspiration cannula should be inserted in the safe triangle bounded by the apex of the axilla, the nipple (ie 4th intercostal space in the mid clavicular line) and the base of the scapula or anteriorly in the 2nd intercostal space.

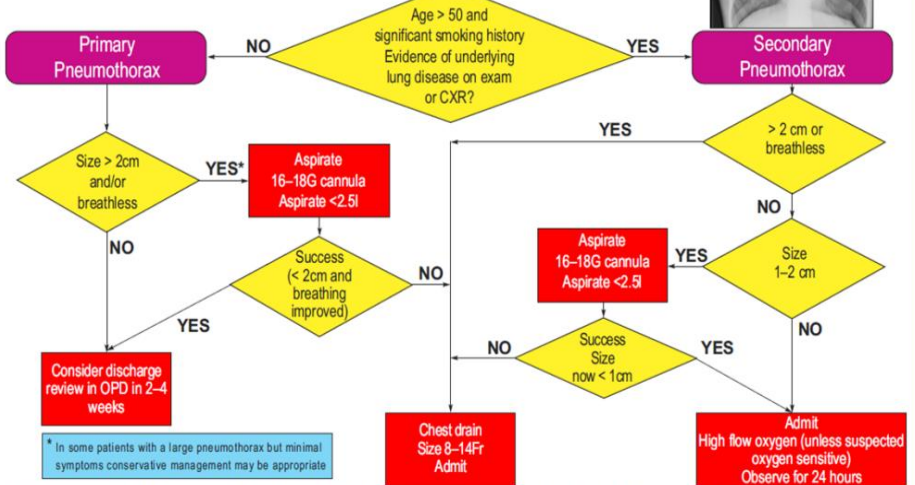
See footnote 1 for equipment required.



BTS Pleural Disease Guideline 2010
MANAGEMENT OF SPONTANEOUS PNEUMOTHORAX

Spontaneous Pneumothorax
 If Bilateral/Haemodynamically unstable
 proceed to chest drain

Measure the interpleural distance at the level of the hilum



The BTS Pleural Disease Guideline is endorsed by: Royal College of Physicians, London; Royal College of Surgeons in England; Royal College of Physicians of Edinburgh; Royal College of Surgeons in Edinburgh; Royal College of Physicians and Surgeons of Glasgow; Royal College of Radiologists; Royal College of Anaesthetists; Royal College of Pathologists; College of Emergency Medicine; Society for Acute Medicine; Association for Clinical Biochemistry; British Society of Clinical Cytology.
 BTS Guideline for Pleural Disease 2010 is published in Thorax Vol 65 Supplement 2 and is available online at: <http://www.brit-thoracic.org.uk/clinical-information/pleural-disease.aspx>

Seek advice from a respiratory specialist registrar or consultant if:
 Seek advice from a respiratory specialist registrar or consultant if:

- the lung fails to expand
 - the patient develops surgical emphysema
 - the drain continues bubbling for more than 24 hours
- Advise smoking cessation to reduce risk of further event.

On discharge refer the patient for an appointment for the pleural or a general respiratory clinic in 1-2 weeks. The patient should be told to report back to hospital immediately if symptoms deteriorate, and advised not to travel by air until the pneumothorax has been confirmed to have been resolved on chest x-ray.

Further information: **please see BTS pleural guidelines:**
<https://www.brit-thoracic.org.uk/document-library/clinical-information/pleural-disease/pleural-disease-guidelines-2010/pleural-disease-guideline/>

Written consent should be obtained for all procedures other than an emergency and the modified WHO safety checklist should be used (see footnote 2).

Pleural procedures - Equipment

All pleural procedures

- Consider pre-medication with analgesia +/- sedation
- Skin cleaning- Chlor prep Drape x2 Surgical gown Sterile gloves
- Needles (1 drawing up, 2 green, 1 orange) 10ml syringe 10-15mls
- 1% lidocaine
- Sterile gauze
- Consent form
- Pleural procedure safety checklist
- Inco pad- for patient +/- on floor

Therapeutic aspiration

Rocket thoracocentesis set- 6F

Tegaderm- cannula dressing 2.0 Stitch if leaving in situ

Rocket wide bore adaptor if connecting to an underwater seal drain- bottle as below

Chest drain- Seldinger

Portex 12F seldinger drain

2.0 Stitch Large tegaderm- 1628

Rocket wide bore adaptor

Drain tubing, underwater seal drain, sterile water

Chest drain- large bore/ blunt dissection

Rocket 28F blunt dissection chest drain insertion pack 2.0 Stitch

Drain tubing, underwater seal drain, sterile water

**1. PLEURAL PROCEDURE CHECKLIST
(PLEURAL ASPIRATION AND CHEST DRAIN INSERTION)**

<p>SIGN IN (read out loud) Before giving local anaesthetic</p>		<p>SIGN OUT (read out loud) Before any team member leaves</p>
<p><i>Have all team members introduced themselves by name and role?</i> Yes <i>All team members verbally confirm:</i> <input type="checkbox"/> What is the patient's name? <input type="checkbox"/> What procedure, site and position are planned? <i>Has the patient been consented?</i> <input type="checkbox"/> Yes <input type="checkbox"/> Written consent [] <i>Allergies?</i> <input type="checkbox"/> No <input type="checkbox"/> Yes <i>Please state.....</i> <i>Has essential imaging been reviewed?</i> <input type="checkbox"/> Pneumothorax – confirm side <i>Effusion – US used or</i> <i>Effusion - surgical blunt dissection- confirm side & mark</i> <i>Do you have all the equipment you need?</i> <input type="checkbox"/> Yes <input type="checkbox"/> No <i>Have risk factors for bleeding been checked?</i> <input type="checkbox"/> Yes INR..... Platelets..... <input type="checkbox"/> Clopidogrel/anticoagulants adequately stopped <i>Are there any critical or unexpected steps you want the team to know about?</i> <input type="checkbox"/> Yes <input type="checkbox"/> N/A</p>	<p>Ward.... Date... Surname... First name... DOB Hosp no..... Performed by.....</p> <p>Infection [] Pneumothorax [] Bleeding [] Pain [] Drain dislodgement [] Drain blockage [] Visceral injury [] Failure</p> <p>Equipment required for Insertion See Equipment List</p> <p>Local Anaesthetic : Lignocaine 1% [] 2% []mls (3mg/kg maximum = 20ml of 1% for 70kg person)</p>	<p><i>Has the procedure been documented in the notes?</i> <input type="checkbox"/> Name [] Site [] Side [] <input type="checkbox"/> Seldinger [] Wide bore [] Size..... <input type="checkbox"/> Sutured atcm Complications Yes [] No [] (Further details in medical notes) Is there a post-procedural care plan in place? CXR booked [] reviewed [] Drainage plan in medical notes [] Chest drain chart in nursing notes [] Are the specimens labelled? Equipment <i>Has all invasive equipment used been accounted for? []</i> <i>Are there any problems that need to be addressed? []</i> Send samples for: Biochemistry- 2-5ml plain container for protein & LDH (+ send serum LDH & protein) MC&S- 5ml in plain container+ 5ml in blood culture bottle if infection suspected. Cytology – at least 20ml Consider <i>pH if infection suspected (run as ABG if non-purulent)</i> <i>Mycobacteria culture, glucose, adenosine deaminase</i></p>

ACUTE UPPER GASTROINTESTINAL BLEEDING

Link Consultant: Dr Chris Groves

Immediate Assessment

Once the diagnosis of a bleed has been made, take blood for haemoglobin, blood grouping/cross match, and coagulation studies. Enquire about drug usage (especially NSAIDs) and alcohol, retching (Mallory Weiss tear) and previous dysphagia. Examine for signs of chronic liver disease and portal hypertension (palpable spleen, abdominal veins), and check for melaena by rectal examination. If endoscopy is to be undertaken, adequate resuscitation should be ensured prior to the procedure.

Immediate Management

This should be based on the severity of the bleed and the predicted risk to the patient. It is convenient to divide patients into two main groups - 'low risk' and 'high risk'. The 'high risk' patients can be further divided according to the severity of the bleed and the urgency for endoscopy and possible surgical intervention (*see flow diagram below*).

Consider activating the CODE RED protocol if there is suspected or confirmed massive haemorrhage by telephone to 6789.

Initial Management

- Patients at '**low risk**' include those with no sign of haemodynamic compromise; Hb > 10g/dl; aged < 60 years, and previously fit. In low-risk patients allow oral fluids, observe for signs of continued or re-bleeding and arrange an OGD for the next routine list. Referral for endoscopy should be made on an endoscopy request form. It is important to complete all sections of the form to allow appropriate prioritisation of the patient. Inform the Endoscopy Unit of the need for endoscopy by 9am. Start patient on oral Omeprazole 40mg BD.
- Patients at '**high risk**' include those with haematemesis or fresh melaena; systolic hypotension (<100mmHg); tachycardia (pulse >100 beats per min); postural drop in diastolic BP; Hb<10g/dL; severe concomitant disease (liver/cardiovascular/respiratory); age >60 years.

In high-risk patients restore blood volume with blood/blood substitutes, admit to high dependency ward, monitor closely (pulse rate, blood pressure, CVP), inform GI bleed registrar and discuss/arrange emergency endoscopy.

High risk patients or those with haematemesis who are vomiting, where endoscopy is planned but not imminent, can be given IV Omeprazole 40mg BD until ready for an OGD. The endoscopist should enter the OGD findings in the Endoscopy Unit computer. If the endoscopist sees a bleeding ulcer, the patient should be given omeprazole (80mg) as a stat injection IV, followed by an infusion at 8mg/h for 72 hours.

Subsequent Management

The next stage of management depends on the state of the patient, his or her 'risk assessment' and the findings on endoscopy, (see 'Second phase' diagram above).

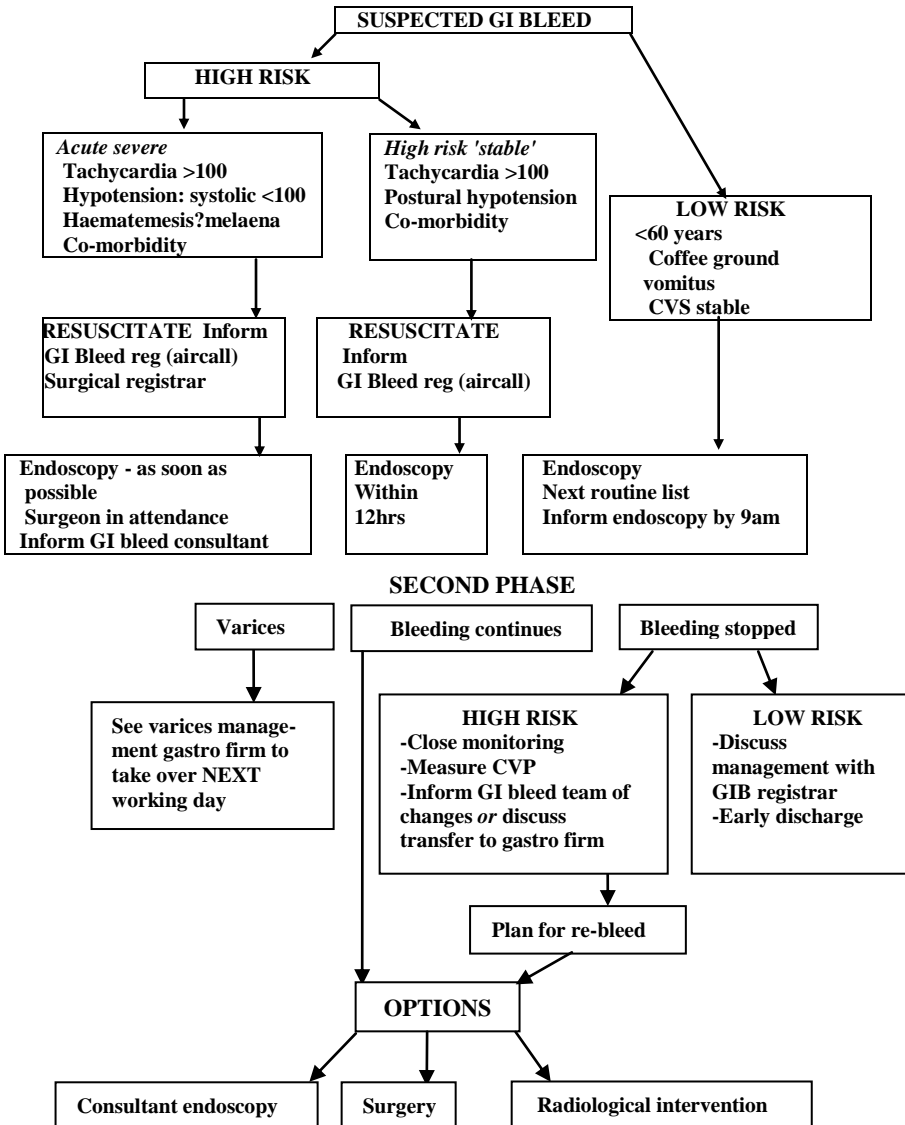
A patient with a gastric or duodenal ulcer who has had endoscopic treatment of a visible vessel should have high dose PPI. Omeprazole 8mg/ hr via a syringe driver for 72 hrs is recommended. Eradication therapy for H.Pylori should be given either now or at discharge.

A patient with a visible vessel or endoscopic evidence of recent or active bleeding is at high risk of rebleeding. Observe for continued bleeding or rebleeding as indicated by a fall in systolic BP, rise in pulse rate, fall in CVP or overt evidence of bleeding.

Surgery

Surgery should be considered if bleeding continues or recurs after hospital admission despite endoscopic therapy, since this is associated with a tenfold increase in mortality. A high transfusion requirement (>4 units if patient older than 60

years; >8 units if younger) should also alert the team to the possible need for surgery. A consultant surgeon should be involved in the decision on whether to operate.



General Measures

The patient may be allowed to drink water and start a light diet as soon as the initial endoscopy has been performed and surgery is not contemplated. Gastric ulcers require endoscopic follow up at 8 weeks to ensure healing. There is no need to rescope duodenal ulcers unless symptoms recur in which case an H.Pylori breath test is indicated.

BLEEDING OESOPHAGEAL VARICES

Link Consultant: Prof Daniel Forton

Each episode of acute variceal bleeding is associated with a 30% mortality at time of admission, if current guidance is not followed. Survivors of an episode of active bleeding have a 70% risk of recurrent haemorrhage within one year. Prompt resuscitation, control of bleeding and supportive care are essential to maximise any chance of survival.

1. RESUSCITATION

- Insert two 16 gauge peripheral venous cannulae.
- Take blood for FBC, coagulation screen, U&Es, LFTs, group and save; cross-match if overt bleeding.
- Intubate to protect the airway if the patient:
 - has severe encephalopathy (very sleepy or confused);
 - has severe uncontrolled haematemesis;
 - has aspiration pneumonia;
 - is unable to maintain SpO₂ above 90%.
- Correct blood volume cautiously and carefully:
 - Use plasma expanders to maintain haemodynamic stability. A systolic blood pressure of 100mmHg is generally adequate and overuse of plasma expanders can perpetuate bleeding. Monitor urine output, aiming for hourly urine output (as measured by urinary catheter) of 0.5mL/kg/hr.
 - Use packed red cells to maintain the haemoglobin at approximately 80-100g/L.

Correct clotting:

- Maintain platelet count $>50 \times 10^9/L$, in patients who are actively bleeding, with platelet transfusion
- Give vitamin K (phytomenadione) 10mg IV slowly.
- Give fresh frozen plasma (12mL/kg) if clotting is abnormal (INR > 1.5 or Fibrinogen $<1g/L$).
- Involve critical care early especially if there is haemodynamic instability, drowsiness and/or confusion and a risk of aspiration. This is especially valuable if the patient has renal, pulmonary or cardiac dysfunction.

2. TREATMENT

- Correct clotting problems as above.
- Vasoconstrictor drugs

Give terlipressin 2mg IV followed by 1 or 2mg every 4-6 hrs. Start *before* diagnostic endoscopy if you strongly suspect variceal bleed, continue for 2-5 days after endoscopy. Reduce dose in patients with ischaemic heart disease or peripheral vascular disease.

- Antibiotic prophylaxis

Blood and an MSU should be sent for microscopy, culture etc.

Antibiotic prophylaxis is essential and should be started from admission, following antimicrobial guidance e.g. co-amoxiclav 1.2g IV TDS or ciprofloxacin 400mg IV bd, *only* if Penicillin allergic.

- Endoscopy - please arrange urgently. It is often performed under general anaesthetic in theatres. Liaise early with theatres and plan ICU bed.

For general advice and to arrange endoscopy, contact endoscopy unit/GI SpR (blp 7464, normal hours) or on-call GI bleed registrar (via switchboard after hours).

Band ligation is the treatment of choice. Start sucralfate liquid 1g qds for 7 days after banding and omeprazole 40mg od for up to 7 days. Patients should not remain on a PPI indefinitely.

Repeat endoscopy after one week unless earlier intervention is needed because of further bleeding.

- Prevent encephalopathy

Encephalopathy may be precipitated in any patient with hepatic dysfunction who bleeds. Give oral lactulose 20mL tds. Avoid benzodiazepines. Opiates can be used cautiously but unwanted side effects may need to be reversed by naloxone. Check blood glucose if drowsy.

Discuss all cases with the Hepatology Team or On-Call Endoscopist

3. FAILURE TO CONTROL ACTIVE BLEEDING

- ET Tube

When necessary, introduce an endotracheal tube and arrange transfer to ITU.

Tamponade with a Sengstaken-Blakemore tube or an endoscopically placed Danis stent should be considered and positioned by the on call Endoscopist. .

- Transjugular intrahepatic portosystemic stent shunt (TIPPS).

4. SECONDARY PROPHYLAXIS OF VARICEAL HAEMORRHAGE

Hepatology team (Prof Forton/Drs Clark/Hughes/Singanayagam/Barker/Yalcin) should take over care the next working day. Request early ultrasound of abdomen and hepatic and portal dopplers, and full liver screen if aetiology unknown.

Do not initiate beta blockers until discussed with Consultant Hepatologist.

BLOODY DIARRHOEA AND ACUTE SEVERE ULCERATIVE COLITIS

Link Consultant: Dr Kamal Patel

Management of patients with severe bloody diarrhoea, (passing 6 or more bowel motions per 24 hrs) will depend on the underlying condition. In patients presenting with bloody diarrhoea for the first time, the diagnosis usually lies between ulcerative colitis (UC) and infective colitis – ulcerative colitis should always be suspected until proved otherwise. Other causes, and their frequency of presentation are as follows:

<i>Common</i>	<i>Less common</i>	<i>Rare</i>
Ulcerative colitis	Pseudomembranous colitis/ <i>Clostridium difficile</i> associated diarrhoea*	Enterohaemorrhagic <i>E.coli</i> associated with Haemolytic-uraemic syndrome
Crohn’s disease	Ischaemic colitis	Yersiniosis
Bacterial dysentery (eg camylobacter, salmonella, shigella, etc.)	Amoebic dysentery	TB enteritis
	Colorectal cancer	Schistosomiasis
	Diverticular disease	HIV-related opportunistic Infection, eg. CMV, HSV, etc.

*normally non-bloody

Ulcerative colitis

In a patient with an established diagnosis of ulcerative colitis, the features of an acute severe exacerbation are: passing 6 or more bloody bowel motions in 24 hrs plus at least one of the following:

- fever >37.5°C
- tachycardia >90bpm
- ESR >30 or CPR >45
- haemoglobin <10g/L
- albumin <30g/L

This is a potentially life-threatening condition and all patients fulfilling these criteria will usually require admission and should be discussed with the Gastro team as soon as possible either via the Gastro Registrar (blp 7464) or the IBD clinical nurse specialist (blp 7994) available via the switchboard.

Immediate investigation

Blood + stool

- full blood count/ESR
- U & E (K+), LFTs (albumin), CRP
- stool microscopy culture and sensitivity x 2
- *C. difficile* toxin

Endoscopy

- Sigmoidoscopy (rigid or flexible) and biopsy

Radiology

- daily plain abdominal X-ray (toxic megacolon is indicated by a transverse colon diameter ≥ 6cm)

- a labelled white cell scan may also be of value in assessing the extent and severity of the disease or alternatively CT-abdomen if concerns of perforation-but please discuss with Gastro team

Management - on admission:

- Start hydrocortisone 100mg qds IV immediately BUT in a mild to moderate attack, BO < 6 day, topical treatment with 5-aminosalicylic acid (mesalazine) suppositories, foam enemas or liquid enemas for proctitis, distal or Left sided colitis respectively. These preparation are greatly preferable to steroids
- Start appropriate fluid replacement with normal saline and potassium supplement
- Request early surgical review particularly if concerns of perforation (ideally from a colorectal surgeon)
- Perform daily abdominal x-rays; dilatation of the transverse colon >6cm indicates toxic megacolon and usually requires urgent colectomy: evidence of mucosal islands is also a very poor prognostic feature
- Start low molecular weight Heparin (Dalteparin 2500-5000units s/c every 24 hrs) since these patients are at increased risk of thromboembolism
- Start stool chart documenting frequency, consistency and blood and review daily
- Check temperature, pulse and blood pressure every 6 hours
- Check full blood count; perform U&E daily and LFT; albumin and CRP daily
- Rescue therapy of steroid refractory UC with ciclosporin or infliximab should be initiated after 72hrs of steroid treatment and be administered by a Gastroenterologist. For this reason it is imperative the patient is referred to the Gastro team as soon as possible after admission
- Remember that patients should not usually be kept nil by mouth unless surgery is imminently scheduled.

DIABETIC KETOACIDOSIS (DKA)

Link Consultant: Dr Kenneth Earle

It is better to contact the diabetes team earlier rather than later via Diabetic Unit, ext 1429, during working hours or by paging the consultant SG295. Key features of DKA compared to other hyperglycaemic emergencies are summarised below. Patients with hyperosmolar hyperglycaemic syndrome (HHS) should be referred directly to HDU/ICU.

During COVID, please refer to modified guidance:

<http://stg1wordpress01/trustnews/wordpress/wp-content/uploads/2020/04/COVID-DKA-GREY-BOOK-April-2020.pdf>

Causes of hyperglycaemic emergencies and their differentiation

	Blood glucose	Urinary Ketones	Dehydration	pH	Serum Osmolality (osmol/kg)
Severe DKA	11 to 33 mmol/L	≥ +++	+++	<7.35	Variable
Normoglyc ketoacidosis	<11mmol/L	+++	+++	<7.35	Normal
HHS	>33mmol/L	Negative	++++	≥7.35	>320
HHS/DKA mixed	>33mmol/L	++ to +++	++++	<7.35	>320
Lactic acidosis	Variable	0 to 1+	0 to 1+	<7.35	

Use the Trust DKA chart for all insulin and fluid prescriptions and to record all relevant observations for the duration of the DKA episode

IMMEDIATE MANAGEMENT (0 to 60 minutes)

STEP 1: Initial investigations

- Insert two iv cannula of sufficient bore size to allow fluid resuscitation
- Take blood for full blood count, renal profile, blood glucose (BG), lactate, venous blood gas analysis, and culture
- Ensure urinalysis, ECG, CXR and culture of any other potential infective source are done

STEP 2: Initial intravenous fluid therapy

- Give sodium chloride 0.9% (without potassium) 1 litre over 10 to 15 minutes
- Then start sodium chloride 0.9% (without potassium) 1 litre over 1 hour

STEP 3: Intravenous insulin infusion

- Prescribe IV insulin infusion as Actrapid 50 units in 0.9% sodium chloride to a total volume of 50ml
 - Infuse iv insulin initially at a fixed rate of 6units/hr via infusion pump
 - If BG at presentation is <14mmol/litre then infuse 10% glucose at 100ml/hr in addition to 0.9% sodium chloride, and reduce IV insulin rate to 3 units/hr
 - (Only give a stat dose of Actrapid (SC) insulin if a delay of ≥ 1 hour is anticipated in setting up an insulin infusion)
- Continue long-acting subcutaneous (SC) insulin – glargine (Lantus®) or detemir (Levemir®) – if the patient was taking this before admission; withhold short and intermediate-acting insulins

OTHER ASPECTS OF MANAGEMENT

<u>Discuss with HDU/ ICU if:</u> <ul style="list-style-type: none">• BG > 33 mmol/L• Hypokalaemia ($K^+ < 3.5$mmol/l)• GCS < 15• Pulse > 100bpm or < 60bpm• SBP < 90 mmHg	<u>Essential monitoring</u> <ul style="list-style-type: none">• Hourly EWS & BG• Hourly fluid balance• Electrolytes (esp K^+) every 2 to 4 hours (venous blood gas analysis)
---	--

- Consider central line, continuous cardiac monitoring, nasogastric tube, urinary catheter
- Prescribe VTE prophylaxis unless contraindicated

ON-GOING MANAGEMENT (60 minutes to 5 hours)

STEP 1: Fluid therapy and clinical monitoring

- Fluid therapy: After the first 2 litres of fluid (Immediate Management Step 2), switch to 0.9% sodium chloride with 40mmol potassium chloride/litre. Administer 2 litres over the next 4 hours at 500ml/hr.
- Important notes re potassium: Do not give potassium if anuric or if serum potassium > 5.5 mmol/L; If potassium remains < 3.5mmol/L continue 0.9% sodium chloride with potassium chloride 40mmol/L and call HDU/ICU; The maximum rate of potassium infusion is 20 mmol/hr; Use ready-mixed infusion bags
- Clinical monitoring: Monitor vital signs using EWS. Alert senior decision maker if patient triggers a response. Be especially vigilant of conscious level – call HDU/ICU if any impairment of consciousness
- Glucose monitoring: Perform capillary blood glucose measurements every hour (if HI send laboratory sample for accurate result)
- Biochemical monitoring: Perform venous blood gas for pH, bicarbonate & potassium at the end of hrs 1, 2 & 4
- Other considerations: Exercise caution in the elderly, pregnant, adolescent, heart or kidney failure, other serious co-morbidities. Catheterise if oliguric (urine output < 0.5mL/kg/hr).

STEP 2: Adjustment of insulin infusion rate

- If BG is not falling by ≥ 3 mmol/hr: Increase infusion rate by 1 unit/hr (check infusion pump is working and connected)
- When BG is < 14 mmol/L: Add 10% glucose infusion at 100ml/hr (continuing 0.9% sodium chloride infusion) and reduce insulin infusion rate to 3 units/hr or to a rate that maintains BG in the range 9 to 14 mmol/L – **do NOT stop insulin**
- Otherwise continue insulin at 6 units/hr

SUBSEQUENT MANAGEMENT (beyond 5 hours)

Fluid and insulin therapy

- Glucose management: Maintain BG in the range 9 to 14 mmol/L by adjusting the insulin infusion rate as described in box 6. Continue 10% glucose infusion at 100 ml/hr (do not alter the rate of glucose infusion)
 - Fluid therapy: Continue 0.9% sodium chloride with potassium 40mmol/L, adjusting the rate of administration as appropriate to maintain euvoelaemia and to keep the serum potassium within the reference range
 - Oral intake: Allow if nausea and vomiting resolved and bowel sounds present
 - Conversion to subcutaneous (SC) insulin: Convert to SC insulin when the patient is eating and drinking and the serum bicarbonate concentration is > 15 mmol/L
 - Stop IV fluids and IV insulin 1 hour AFTER the first dose of subcutaneous insulin.
- **Refer to DKA chart for full details of how to transfer to SC insulin**

DISCHARGE PLANNING

Assessing suitability for discharge and arranging follow-up

- Refer to Specialist Diabetes Team before discharge
- Patient should not be discharged until:
 - Biochemically normal
 - Normal diet and established on usual SC insulin
 - Patient/carer able to administer SC insulin

HYPEROSMOLAR HYPERGLYCAEMIC STATE (HHS)

Link Consultant: Dr Kenneth Earle

During COVID, please refer to modified guidance:

<http://stg1.wordpress01/trustnews/wordpress/wp-content/uploads/2020/04/COVID-HHSGREY-BOOK-April-2020.pdf>

It is better to contact the diabetes team earlier rather than later via Diabetic Unit, ext 1429, during working hours or by paging the Diabetes SpR bleep 7762.

Admission investigations for the diagnosis of HHS and mixed HHS/DKA

Diagnostic criteria	HHS	Mixed HHS/DKA
Serum pH	≥ 7.3	< 7.3
Serum bicarbonate (mmol/L)	> 15	≤ 15
Lab blood glucose (mmol/L)	> 33	> 33
Urine ketones	$< 2+$	$\geq 2+$
Capillary ketones (mmol/L)	< 1.0	$1.0 - 3.0$
Osmolality* (mOsm/L)	> 320	> 320

* Calculate Osmolality = $(2 \times \text{Na}^+) + \text{urea} + \text{glucose}$

If glucose < 33 and pH < 7.3 , use DKA protocol instead

Prescribe HHS treatment on ePMA using the treatment plan: HHS – Hyperosmolar Hyperglycaemic State

IMMEDIATE MANAGEMENT (0 to 60 minutes)

STEP 1: IV access and initial investigations

- Obtain IV access – most patients with HHS will require at least two peripheral cannulae or central venous access
- Take blood for full blood count, renal profile, liver function, blood glucose (BG), lactate, CRP, Troponin, HbA1c, bone profile, magnesium, venous blood gas and blood culture
- Ensure urinalysis, ECG, CXR and culture of any other potential infective source are done

STEP 2: Initial intravenous fluid therapy

- Give sodium chloride 0.9%, 500ml to 1 litre over the first hour
- If serum potassium concentration ≤ 5.5 mmol/L, infuse 0.9% sodium chloride with potassium chloride 20 mmol/L
- Do **NOT** attempt to correct abnormal serum sodium concentration

STEP 3: Mixed HHS/DKA

- **IF mixed HHS/DKA with significant ketonaemia (capillary ketones ≥ 1 mmol/L) or ketonuria (urine ketones $\geq 2+$)**
- Commence IV insulin at a fixed rate of **0.05 units/kg/hr** using a syringe driver, aiming to decrease glucose level by < 5 mmol/L/hr
- Do **NOT** give an IV/SC insulin bolus injection
- **Continue any usual doses of long-acting insulin**

STEP 4: Refer all patients to ADU

Essential monitoring for the first 6 hours

- Hourly NEWS, fluid balance, GCS and VBG
- Continuous cardiac monitoring
- Arterial line and regular biochemistry
- Urinary catheter
- Document glucose and blood gas electrolytes

Consider:

- Antibiotics
- VTE prophylaxis
- Nasogastric tube
- Central venous access

Discuss with ICU if any of the following apply:

- GCS < 12
- Creatinine > 200 umol/L
- pH < 7.1
- Osmolality > 350 mOsm/L
- Na > 160 mmol/L
- K < 3.5 mmol/L
- HR > 100 /min
- SBP < 90 mmHg
- Lactate > 5 mmol/L

ON-GOING MANAGEMENT (1 to 6 hours)

STEP 5: Monitoring and assessment

- Clinical monitoring
 - Monitor vital signs/NEWS, fluid balance and conscious level hourly
 - Perform hourly blood gases for potassium, sodium, glucose and pH
 - Aim for glucose to fall by < 5 mmol/L/hr
 - Send bloods for U&Es and lab glucose at 6 hours, calculate and document osmolality
- If **conscious level** deteriorates, **this is an emergency** and senior help must be sought to assess for cerebral oedema
- Ensure VTE assessment completed and prophylaxis given if not contraindicated
- Ensure feet checked for ulceration and protect heels if non-mobile
 - Refer to diabetes team (bleep7762)

STEP 6: Fluid and insulin guidance

• Fluid replacement:

- Infuse 0.9% sodium chloride with 40 mmol potassium chloride/L at 500ml/hr for hour 2, then 100 to 200ml/hr as determined by clinical fluid status -
- Aim for 2 to 3 litre positive fluid balance after 6 hours

• Sodium:

- **Sodium will rise by 2.4 mmol/L for every 5.5 mmol/L fall in glucose**
- Do **not** actively correct hypernatraemia in the first 48 hours
- Ensure osmolality falling by average 5 mOsm/L/hr

• Potassium:

- Infuse 0.9% sodium chloride with potassium chloride 40 mmol/L unless anuric or potassium > 5.5 mmol/L
- If potassium < 3.5 mmol/L consider need for ICU and replacement via central line

• Glucose/Insulin:

- Start IV insulin infusion only once glucose no longer falling by 5 mmol/L/hr **AND** fluid replete. (N.B. insulin may not be needed)
- If needed, commence IV insulin at a fixed rate of 0.05 units/kg/hr
- Adjust insulin rate by 1 unit/hr as needed to ensure glucose falling at < 5 mmol/L/hr
- Once blood glucose is < 14 mmol/L, to prevent hypoglycaemia, introduce 5% glucose at 125 ml/hr alongside the sodium chloride 0.9% with potassium chloride 40 mmol/L infusion

SUBSEQUENT MANAGEMENT (6 to 24 hours)

STEP 7: Treatment targets

- Aim to keep glucose level 10 to 15 mmol/L. Check blood glucose every hour
- Aim to reduce osmolality by no more than average 5mOsm/L/hr. If too fast, reduce rate of fluid (+/-insulin)
- Aim to reduce sodium by no more than 10 mmol/24 hours
- Expect 3 to 6 litres positive fluid balance after 12 hours, with the remainder replaced after 24 to 72 hours in total

ONGOING MANAGEMENT (24 hours+)

STEP 8: Stepping down treatment

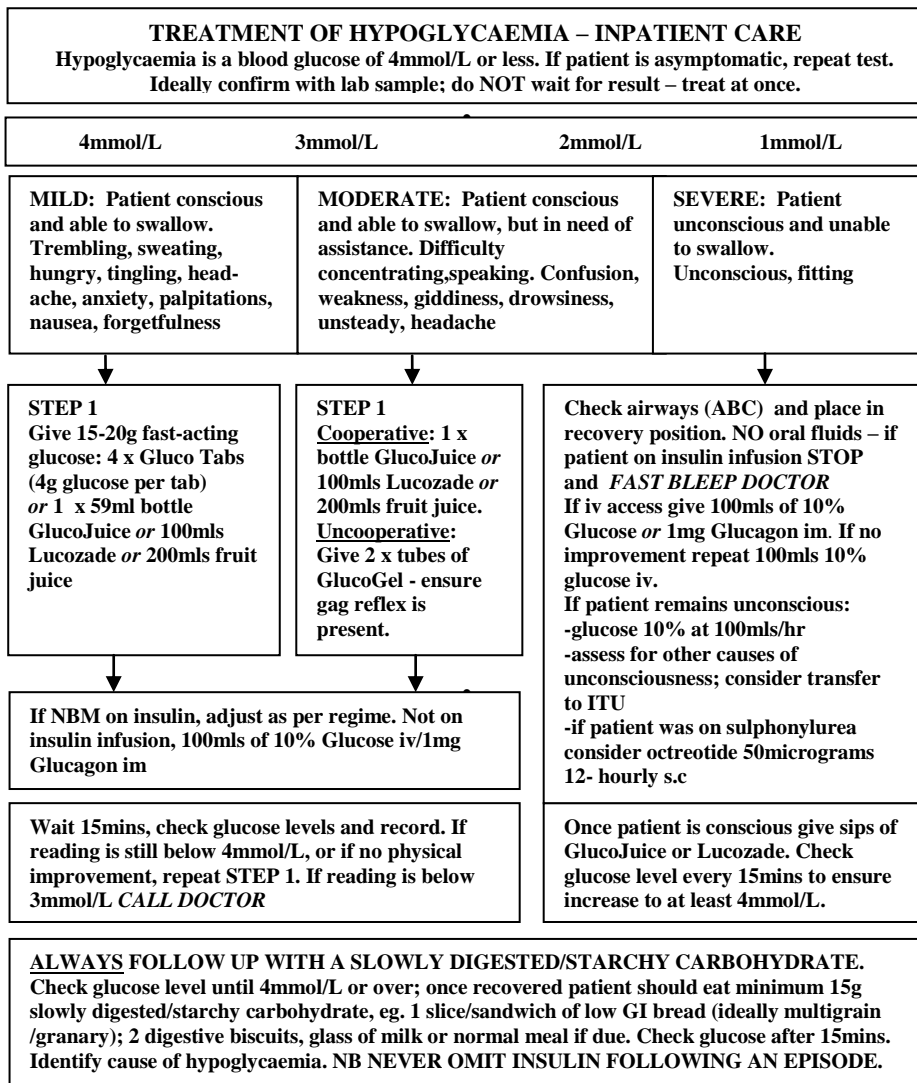
- Once metabolic state normalised and eating and drinking as normal, consider switch to SC insulin or oral diabetes therapy and discontinue IV fluids
- If patient new to insulin refer to diabetes team (bleep 7762)
- Examine feet daily
- Consider need to continue VTE prophylaxis beyond discharge if high risk

Converting to subcutaneous (SC) insulin therapy

- Ensure patient is able to eat and drink before discontinuing the IV insulin infusion
- Choose a meal time to switch over
- IV insulin has a very short half life (3 to 5 mins) therefore ensure SC insulin is given 1 hour before the infusion is discontinued
- If it is necessary to stop the IV insulin infusion overnight, seek advice from the medical team. Check blood glucose hourly

HYPOGLYCAEMIA

Link Consultant: Dr Kenneth Earle



Hypoglycaemia is unusual except in patients with diabetes who commonly suffer from excessive effects of their hypoglycaemic drugs. Occasionally it is induced by these drugs used in suicide bids by patients who are not diabetic. Other drugs (eg alcohol and Aspirin) may cause hypoglycaemia. It can also arise as part of an underlying disease such as insulinoma, carcinoid or sepsis (particularly in children and neonates). If you suspect that hypoglycaemia is iatrogenic, send blood/urine for screening (eg. sulphonylurea screen, estimate of insulin concentration).

ADDISONIAN CRISIS

Link Consultant: Dr Gul Bano

An Addisonian crisis occurs when the adrenal gland fails to secrete sufficient cortisol for the body's needs. This can lead to rapid deterioration, is potentially life-threatening and may cause severe morbidity and mortality if undiagnosed or not treated appropriately. It is a **MEDICAL EMERGENCY** and **REQUIRES IMMEDIATE TREATMENT**.

Signs and symptoms of an Addisonian Crisis

Clinical features of an Addisonian crisis include:

- Weakness
- Dizziness
- Drowsiness
- Mental agitation/confusion
- Nausea/Vomiting
- Abdominal tenderness
- Hypotension especially postural hypotension
- Shock
- Hyponatremia
- Hyperkalemia
- Hypoglycemia

These are non-specific and may go unnoticed until a patient becomes haemodynamically compromised. Therefore, **always treat a patient with suspected adrenal insufficiency before waiting for confirmation of diagnosis**.

Patients and situations in which acute adrenal crisis can occur:

All patients taking steroid therapy, particularly for longer than 3 weeks and regardless of the underlying indication, are at risk of an Addisonian crisis.

Such patients include:

- Patients who have primary (Addison's disease) or secondary (hypothalamic or pituitary) adrenal failure
- Patients on adrenal suppressive doses of prednisolone or other steroids, including dexamethasone in a dose equivalence >5 mg once daily for longer than one month
- Patients with congenital adrenal hyperplasia
- Patients on long term inhaled steroids
- Patients on high doses of topical steroids

Remember that cortisol requirements increase when individuals become acutely unwell or experience significant stress. Therefore, an Addisonian crisis may be precipitated in patients on steroid therapy particularly during:

- An acute illness; or,
- An infection, major surgery, vomiting with inadequate steroid absorption or major stress

Therefore

- i) Always consider a potential diagnosis of an Addisonian crisis and treat immediately when features are present in such patients and especially in situations listed above.
- ii) Increase steroid therapy during the above periods

Immediate Management ^[1,2]

1. Insert cannula and take blood for urea, electrolytes, glucose and cortisol
2. Commence an IV infusion of 0.9% saline (this helps reverse fluid and sodium deficiency)
3. Correct hypoglycaemia (Please see Management of hypoglycaemia)
4. Give 100mg IV Hydrocortisone bolus immediately followed by 100mgs of IV Hydrocortisone six hourly for 24 - 48 hours or until oral therapy can commence. Hydrocortisone Sodium Phosphate or Hydrocortisone Sodium Succinate by slow IV bolus (to avoid vascular damage) or IM should be used.
(NB. DO NOT use Hydrocortisone Acetate due to its slow-release microcrystalline formulation)
5. Consult on-call Endocrine registrar BIp 7778 for further on-going management.

References

1. Krasner AS. Glucocorticoid Induced Adrenal Insufficiency. JAMA 1999; 282: 671-6.
2. Turner H and Wass J. Oxford Handbook of Endocrinology and Diabetes. Oxford University Press 2003

ACUTE STROKE

Link Consultant: Dr Brian Clarke

All patients with suspected acute stroke less than 4.5 hours from onset should be considered for thrombolysis. This is a medical emergency – page the stroke team through switch. Potential stroke patients in SW London should, if possible, be admitted to St. George's, and then repatriated to their local SU within 72 hours, or local medical wards within 24 hours if they are a stroke mimic.

Stroke is an acute focal cerebral deficit lasting for 24 hours or resulting in death that occurs secondary to cerebrovascular disease ie. cerebral infarction, haemorrhage, subarachnoid haemorrhage and cerebral venous thrombosis. To direct management it is essential to know the underlying pathology (haemorrhage or infarction), the site (e.g. carotid or vertebrobasilar territory), underlying aetiology (e.g. carotid stenosis or cardiac embolism) and residual disability.

Transient ischaemic attack (TIA) is an acute focal cerebral deficit lasting less than 24 hours (most commonly within 1 hour). They can be seen in the TIA clinic – please use the proforma on SGH website (discuss with stroke SpR). However, TIAs may be admitted depending on the clinical view at the time. If the patient is an in-patient, we can often arrange the tests before they go home.

Admission

Good management of patients with stroke reduces mortality by 25% and the risk of recurrence by up to 75%. It reduces complications and residual disability. All patients who develop features of stroke or a TIA should, if possible, be admitted directly to the Hyper Acute Stroke Unit (HASU, William Drummond Ward, 3rd Floor AMW). The exception are those in whom the episode is not the major current condition e.g. ST elevation MI.

Many patients are referred to the stroke team if they are FAST positive when assessed by the LAS. If not, they should have the ROSIER performed by A&E staff. If the ROSIER score is negative, stroke is unlikely. FAST/ROSIER positive patients are referred directly to the stroke SpR. If stroke is suspected even if the patient is FAST/ROSIER negative, the admitting A&E SpR should assess the patient and then contact Bleep 7317 (24 hours/day). In-patients should be referred immediately for stroke assessment. If the patient cannot be admitted directly to the HASU, care should be started in a general ward but every effort made to transfer the patient to the HASU as soon as possible. If the HASU is full, a patient (usually the one who has been there the longest) will be moved to make way for the new admission. The 'moved' patient will either be transferred to a stroke unit bed, or if unavailable, a general medical bed. If the transfer occurs out of hours, the stroke team will hand over to the receiving team on the next working day with clear details of diagnosis, secondary prevention and ongoing management plan, and a discharge letter.

History & Examination

The history should be recorded in the stroke proforma (available from HASU or the ED), include time and mode of onset (sudden/gradual), progression since onset and vascular risk factors. The neurological examination should assess the patient's conscious level (use the Glasgow coma scale), gait, cognitive function (orientation, language, memory, visuospatial skills, AMTS), visual fields, speech, swallowing, limb weakness, cerebellar signs, reflexes, plantar responses and presence/absence of incontinence, and check for neck stiffness and Kernig's sign if subarachnoid haemorrhage is suspected. The NIHSS should be completed in all admissions. The general examination must include vital signs (especially BP), cardiac or respiratory signs, peripheral pulses and assessment of presence/absence of carotid bruits and cardiac murmurs.

Investigations

All patients should have a CT scan within 1 hour of A&E admission. MRI scanning is the optimal imaging modality, although its use is limited by availability. Abnormalities are detected earlier than with CT and it is particularly indicated in patients with small regions of infarction which may not be well seen on CT (lacunar stroke and posterior circulation stroke). An MRI scan is also indicated in patients suspected of having carotid dissection and cerebral venous thrombosis (*see below*).

The scan should be performed immediately in A&E in all patients. Urgent scanning is also required in patients with coma, deteriorating consciousness, brain stem or cerebellar signs or progression, acute stroke symptoms whilst on anticoagulants, or suspected subarachnoid haemorrhage. A scan is needed to confirm diagnosis, distinguish infarction from haemorrhage and exclude non-vascular causes in order to determine treatment. Remember an early scan may be normal in some patients with cerebral infarction. If the diagnosis is in doubt a repeat CT or MRI scan may help (advice can be obtained from the stroke team). The scan, if normal, confirms the safety of lumbar puncture where the history and findings on examination suggest subarachnoid haemorrhage. It is essential to look for xanthochromia in the CSF if subarachnoid haemorrhage is suspected and the CT scan has not shown sub-arachnoid blood. Red cells alone in the CSF can occur with a traumatic lumbar puncture and can confuse diagnosis if the supernatant fluid is not examined.

All patients should have a stroke order set from iClip. This includes FBC, ESR, Coag screen, U&Es, glucose and cholesterol levels, ECG and chest X-ray. Patients with an ischaemic stroke should have a Doppler study (carotid and vertebral) to check for a stenosis. In some patients an MR or CT angiogram may also be necessary. Patients with haemorrhagic stroke should have a clotting screen, and patients with ischaemic stroke under the age of 60 may need a thrombophilia screen (protein C, protein S, antithrombin III, APC resistance, lupus anticoagulant), auto-antibody and anticardiolipin antibody screen. An echocardiogram should be considered in those under the age of 65 or suspected of having a significant cardiac abnormality (either from the history, examination or ECG, or in whom the pattern of infarction is consistent with embolism i.e. in multiple cerebral vascular territories). Urgent echo and blood cultures should be performed in patients with suspected endocarditis (fever, murmur, peripheral emboli, raised inflammatory markers).

In those under the age of 50 or with recurrent unexplained stroke, transoesophageal echocardiogram should be considered. Cerebral angiography may also be required in subarachnoid haemorrhage, intracranial haemorrhage, carotid stenosis, brain stem or cerebellar strokes or in patients under the age of 50 and is performed in the AMW neuroradiology department after discussion.

Acute Medical Management

1. Thrombolysis

Thrombolysis given *within 4.5 hours* of ischaemic stroke improves outcome. All patients admitted *within 4 hours* of stroke or with in-hospital stroke, should be referred immediately to the stroke SpR (Bleep 7317) before arranging investigations. They will organise brain imaging and start tPA if appropriate. Check blood glucose, insert two IV lines and perform an ECG after contacting the stroke SpR. Intra-arterial thrombolysis or thrombectomy is now available for selected patients from 9am to 5pm. If patients have high initial Blood Pressure (> 185/110 mmHg) either GTN patch (5mg initially) or bolus dosing with labetalol (10-20mg according to BP) can be used to lower BP. Following boluses, labetalol infusion may sometime be required to ensure BP remains within target in the first 24 hrs following lysis.

2. Anti-platelet therapy and anti-coagulation.

All patients with ischaemic stroke and in whom imaging has excluded a haemorrhage should be loaded with a single anti-platelet agent followed by daily maintenance dose.

- Patients should usually be loaded with Aspirin 300 mg (given orally or rectally), followed by a daily dose in the range 75-300 mg.

- However, if the patient is already on Clopidogrel, then Re-load with Clopidogrel 300 mg in ED.

Further antiplatelet drug changes or use of dual agents will be decided by the attending Stroke or Neurology Consultant on the HASU ward round. .

- Full heparinisation should be reserved for patients with cerebral venous thrombosis, or where the risk of a cardioembolic source is high.
- Delay anti-coagulation for 2 weeks in patients with atrial fibrillation or other cardioembolic source, if the stroke is large. If the stroke is small, anticoagulation can be started sooner. If in doubt, seek advice from the stroke SpR.

3. Anti-hypertensive therapy

- Patients already on antihypertensive medication should continue their usual treatment unless their blood pressure (BP) is low.
- *Acutely elevated BP is common following ischemic stroke and should not be treated aggressively.* In patients with a systolic BP > 220mmHg, or a diastolic BP > 110 mmHg, blood pressure should be reduced gradually (see *Severe hypertension*).
- In patients with *acute intracerebral haemorrhage, blood pressure should be treated intensively with the aim of achieving a target systolic BP of < 140 mmHg.*

4. Other treatments

Much of the mortality and morbidity following stroke is from secondary complications. To minimise these:

i) **Complete the VTE form.** In patients at high risk of DVT and pulmonary embolism, prescribe intermittent pneumatic compression stockings for 30 days. Low molecular weight heparin is no longer routinely used.

ii) Only if swallowing is inadequate, give fluid replacement via nasogastric tube or, if this is not possible, via an IV line. If in doubt about swallowing capacity, check with stroke team or speech therapist. Patients who cannot swallow or eat adequately will need feeding supplementation.

iii) If the blood glucose remains > 10mmol/L, consider giving Insulin, as high blood glucose can worsen the ischaemic damage.

iv) Refer patients to physiotherapy, occupational therapy and dieticians on the working day after admission. If the patient has difficulty swallowing or communicating, refer for speech therapy.

v) Treat fever (persistent temperature over 37.5°C) with Paracetamol (1g 6-hourly), and identify and treat the site of infection.

vi) Give oxygen (24%) to patients with oxygen saturations persistently below 95%.

vii) Look out for mood disturbance, especially depression, as this is common after acute stroke. The need for treatment should be assessed by a multi-disciplinary team.

Specific Stroke Syndromes

- **Carotid Dissection:** Clues to diagnosis include young age, history of neck trauma, and Horner's syndrome on the side of dissection. If suspected, the imaging of choice is an MRI scan with cross-sectional views through the carotid artery in the neck (ask the radiologist specifically for these) as well as carotid MRA. Refer patients with dissection to the neurology SpR for advice.

- **Cerebral Venous Sinus Thrombosis:** This may present with headache, seizures, reduced consciousness and focal neurological signs. Brain imaging may show infarction and also haemorrhagic infarction. Its incidence is increased in those with a prothrombotic state. Investigations of choice are MRI brain and magnetic resonance venography. Refer suspected patients to the stroke SpR. Most patients should be anticoagulated with heparin and then warfarin even if some evidence of haemorrhagic infarction (*seek advice*).

Cerebellar Haemorrhage: Patients with cerebellar haemorrhage should be referred for urgent neurosurgical opinion. The haemorrhage can lead to obstruction of CSF flow and secondary hydrocephalous.

• **Malignant Middle Cerebral Artery infarction:** Patients under 60 with large MCA infarction may come in the first few days after stroke. They should be under observation on the HASU.

• **Subarachnoid Haemorrhage:** SAH is most commonly due to a berry aneurysm, and carries a high risk of a further bleed. Clues to diagnosis include sudden onset (thunderclap) headache, neck stiffness, photophobia, vomiting at onset, and reduced consciousness levels. The investigation of choice is CT imaging which may show free blood. If this is negative and the index of suspicion is high, lumbar puncture should be performed. Xanthochromia should be specifically sought. If the diagnosis is made or is likely, refer the patient urgently to the neurosurgeons on AMW.

• **Intracerebral Haemorrhage:** The most common causes are hypertension, amyloid angiopathy in the elderly, or an underlying arteriovenous malformation, aneurysm or tumour. Frequently the underlying cause is obscured by blood. MRI imaging is usually done after 1 to 2 months to exclude an underlying lesion. In young patients cerebral angiography should be considered.

Intracerebral bleeding whilst on anticoagulants generally requires urgent reversal of anticoagulation (Beriplex, a prothrombin complex concentrate [PCC] and IV vitamin K as per haematology advice - see Appendix 7) to prevent haematoma expansion. Discuss with neurology team urgently.

Prevention/reduction of risk of recurrence

- Hypertension should be investigated and treated after the acute stage (see above).
- Patients with carotid stenosis demonstrated should be referred urgently to the stroke SpR
- Consider anti-coagulation in patients with atrial fibrillation (age alone IS NOT a contraindication).
- Treat other risk factors: eg. diabetes, smoking, cholesterol.
- Patients with ischaemic stroke who are not anticoagulated should be treated with anti-platelet therapy. First-line treatment is Clopidogrel (75mg per day), Aspirin and Dipyridamole is the alternative option. The choice of antiplatelet is made after stroke team review.
- All patients admitted with TIA/stroke should be followed up in the stroke follow up clinic (fax discharge letter to Ext. 4591)

STATUS EPILEPTICUS AND CLUSTER SEIZURES IN ADULTS

(Convulsive Seizures in patients aged > 16 years old)

Link consultant: Prof Hannah Cock

Status epilepticus (SE) is defined as continuous seizure activity which has failed to self-terminate leading to a risk of neurological damage. The risks are highest with tonic-clonic (convulsive) seizures. Convulsive SE may present as either a run of discreet tonic-clonic seizures without full recovery in between (ie without regaining consciousness), or continuous tonic-clonic seizure activity. Most convulsive seizures terminate spontaneously within 3 minutes, and do NOT need emergency treatment. Convulsive seizures lasting longer than 5 minutes or recurring without recovery should be managed as Convulsive SE, unless the patient is known to habitually have longer seizures with self-termination (eg information from relatives, friends, or the patient's epilepsy card or diary). The mortality and morbidity of convulsive status epilepticus is high, and it is important to control fits as soon as possible, to use adequate doses of 1st and 2nd line agents, but not to over-treat patients in whom seizures have terminated but are slow to recover.

Repeated or Cluster seizures (typically 3 or more self-terminating within 24 hours), if atypical for that patient, should also be managed as a medical emergency (NICE 2022).

In both settings, if the patient has an individualised emergency care plan available, follow that. Where this is not immediately available:

CLUSTER SEIZURES

- Consider giving a benzodiazepine (Clobazam 10mg, or Midazolam 10mg) immediately. This can be repeated once within 24 hour period if further seizures occur.
- As soon as possible, request Urgent Specialist Review (to be seen within 24hours of presentation). Depending on timing of presentation, this may be using the Refer To Epilepsy form on iClip (for review Mon-Friday in working hours), or via on-call neurology (Bleep 7277 for review at weekends or bank holidays). This must include consultant review to consider risk of repeated clusters and whether an individualised emergency management plan should be provided prior to discharge (NICE 2022)

CONVULSIVE STATUS EPILEPTICUS:

0-5 minutes: Stabilization phase

- padded bed rails. Do not restrain.
- Time seizure from onset, Institute regular monitoring (temperature, cardiac, respiration, BP) including ECG monitoring.
- Administer oxygen. During an inter-ictal period insert an airway. Do not attempt to insert anything in the patient's mouth during a seizure, even if the tongue is injured.
- Place the patient in a semi-prone position with the head down to prevent aspiration.
- If there is any suggestion of alcohol abuse or impaired nutrition, give thiamine as high potency intravenous Pabrinex BEFORE GLUCOSE.
- Estimate blood glucose rapidly using a blood test. If the patient is hypoglycaemic, give 100ml of 10% glucose rapidly, and if still fitting unconscious, repeat and then start 10% glucose at 100ml/hr. (Refer to management of Hypoglycaemia).
- Consider possibility of non-epileptic status.

Attempt IV access & Emergency investigations: venous blood count, clotting, glucose, urea, sodium, potassium, calcium, liver function and anti-convulsant drug levels (irrespective of known history at this stage). Save 5ml blood and 50ml urine for toxicology. Treat acidosis if severe.

5-20 minutes Initial therapy & investigate cause phase.

- Check if any pre-hospital benzodiazepines have been given. If two adequate doses (an adequate dose in adults is 4mg Lorazepam, or 10mg of diazepam/Midazolam) of any benzodiazepine have been administered and seizures have recurred within a 24-hour period, move straight to 2nd line/established status treatment.
- **Initial Benzodiazepines:** The drug of first choice is lorazepam given as an IV bolus injected at 2mg/min, (4mg first dose for adults. If Lorazepam is unavailable, give 10mg Diazepam iv or 10mg Buccal Midazolam. If no iv access, give Buccal Midazolam. A 2nd dose of a benzodiazepine may be repeated once within 10-20minutes. Give usual antiepileptics if already on treatment. NB: Benzodiazepines must be written up on the 'stat dose' rather than the 'prn' part of the drug chart. Write up a maximum of two stat doses with clear instructions on when to give, eg. 'for convulsions > 5mins' (not just 'if fits'). **The biggest risk to respiratory function in status epilepticus is ongoing seizures, NOT respiratory depression from benzodiazepines. These are evidence-based guidelines; repeated smaller doses are not recommended.**
- **Establish aetiology:** *Gain information* (Is there evidence of previous epilepsy, any anticonvulsant drugs, diary or wallet card or bracelet?). Consider need for urgent CT (no previous epilepsy history, new focal neurology, any refractory case). Alert anaesthetist and ICU. Identify and treat medical complications. Consider pressor therapy if needed.

20-40minutes, 2nd line therapy (established status)

- Alert the on-call anaesthetist in case of later need.
- Start an iv infusion of a 2nd line antiepileptic agent. Either Valproate or Levetiracetam are suitable in terms of efficacy and safety for this indication at stated doses and have practical advantages over the licensed agent Phenytoin. Guidance for each is in the table:

Dose, indications and contraindications for second line antiepileptic agents in the treatment of status epilepticus

Drug	Dose; Rate (Maximum)	May be preferable	Contraindicated
Valproate	40mg/kg; 10mg/kg/min (3000mg)	Already taking Valproate, suspected poor adherence** Generalized epilepsy* Comorbid migraine, mood disorder Alternatives contraindicated or previously ineffective	Women of childbearing age** Pre-existing liver disease or pancreatitis Known metabolic disorder predisposing to hepatotoxicity
Levetiracetam	60mg/kg; 6mg/kg/min (4500mg)	Already taking Levetiracetam, suspected poor adherence Need for minimal drug interactions Alternatives contraindicated or previously ineffective	May not be best choice in •acute or prior brain injury •known mood./behaviour disorder (may exacerbate) •Dose adjustments as per BNF in renal impairment (GFR <80mL/minute/1.73m ²).

*known or suspected idiopathic (genetic) generalised epilepsy, eg. history of myoclonus or typical absence seizures.

**Valproate should not be used as first line for SE in women of childbearing age, other than for a woman known to be prescribed VPA already presenting in Convulsive Status, in whom poor adherence is the most likely cause, or in whom levetiracetam is known to have failed. In this situation Valproate is the most appropriate option for mother and any potential pregnancy in the emergency situation. If none of the above are available/ suitable, Phenytoin 20mg/kg (50mg/min infusion) can be used. Contraindications to Phenytoin including significant hypotension, bradycardia, heart block, porphyria and overdose of recreational drugs or antidepressants.

iv Phenobarbital 15mg/kg is also an option.

40-60minutes, third therapy phase (refractory status)

If ongoing seizures there is no clear evidence to guide treatment. Unless there is a clear clinical reason to avoid ICU if possible, most will require anaesthetic doses of one of the following, titrated to effect, with EEG monitoring if possible. Anaesthesia should be continued for 12-24 hrs after last clinical or electrographic seizure, then dose tapered.

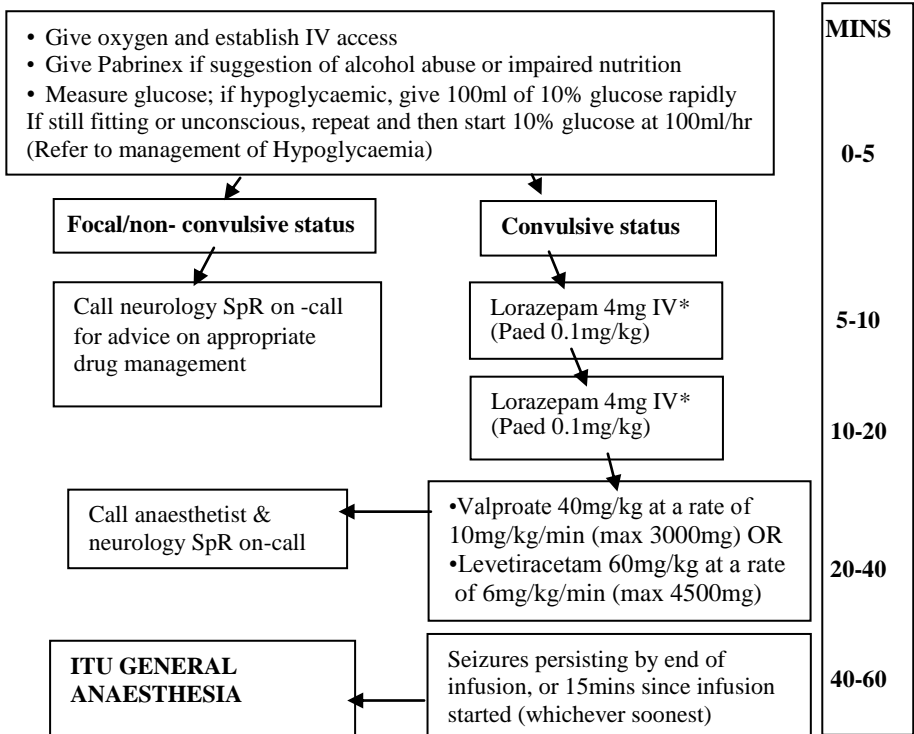
- Propofol (1-2mg/kg bolus then 2-10mg/kg/hr). Risk of infusion syndrome increases with duration of therapy
- Midazolam (0.1-0.2mg/kg bolus, then 0.05-0.5mg/kg/hr)
- Thiopental sodium (3-5mg/kg bolus then 3-5mg/kg/hour). After 2-3 days infusion rate needs reduction as fat stores are saturated.

Administering an alternative 2nd line agent is also an option if clinical rationale to avoid ICU. Neurology advice should always be sought for on-going management in adults. Send blood for Pyridoxine (vitamin B6) level and give Pyridoxine 50mg iv (as Pabrinex) if not already given. At least intermittent EEG monitoring is necessary for refractory status. The recommended primary endpoint is suppression of epileptic activity on EEG, with a secondary end point of burst-suppression pattern.

ALL:

- CXR when clinically stable to assess for possible aspiration
- Further assessments to investigate the cause
- Consider maintenance anti-epileptic medication if no identifiable fully reversible cause.
- For all adult (> 16years:) patients with convulsive status epilepticus, as soon as possible, request Urgent Specialist Review (to be seen within 24hours of presentation). Depending on timing of presentation, this may be using the Refer To Epilepsy form on iClip (for review Mon-Friday in working hours), or via on-call neurology (Bleep 7277 for review at weekends or bank holidays).This must include consultant review to consider risk of repeated clusters and whether an individualised emergency management plan should be provided prior to discharge (NICE 2022)

Treatment of Convulsive Status Epilepticus in adults (also valid for children >2yrs)

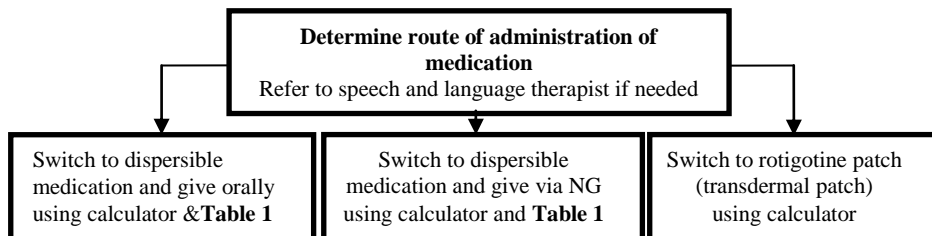


*If Lorazepam is unavailable, give 10mg Diazepam iv (paed 300-400 micrograms/kg - max.10 mg) or 10mg Buccal Midazolam (paed 300 micrograms/kg). If no iv access, give Buccal Midazolam.

ACUTE MANAGEMENT OF PARKINSON'S DISEASE (PD)

Link consultant: Dr Dominic Paviour

- Do Not** Suddenly stop anti-parkinsonian medication including Apomorphine (this is NOT an opiate); this can be life threatening, causing a neuroleptic malignant type syndrome.
- Do** Treat/investigate infection, dehydration, constipation which are frequent causes of admission.
- Do** Make sure patient's with PD receive their medication regularly and on time.
- Do** Contact the Parkinson's Nurse and / or treating consultant early in admission.



The following calculator can be used to guide switching the patient's regular oral PD medication to a 'levodopa equivalent dose' (LED): <http://parkinsonscalculator.com/>

REMEMBER TO CONVERT BACK TO ORAL PD MEDICATION WHEN ABLE TO SWALLOW SAFELY

Table 1: Administration if swallowing difficulties or nasogastric tube (NG)

Entacapone	Disperse tablets in water (do not crush)
Selegiline, Rasagiline	Crush tablet and disperse in water
Amantadine	Open capsules and disperse contents in water
Stalevo® (co-careldopa and entacapone)	Switch to single entities as non-formulary (co-careldopa and entacapone disperse in water)
Opicapone	Not suitable to be given via NG – hold in acute phase
Pramipexole, Pramipexole MR, Ropinirole, Ropinirole MR	Convert modified release to equivalent immediate release formulation and split dose three times a day. Crush tablet and mix with water

Medications that can be safely omitted if no NG in situ:

Entacapone, Opicapone	Refer to PD team if long term omission
Selegiline, Rasagiline	
Amantadine	

Non-oral treatment (continue existing treatment if possible):

<u>Apomorphine</u> Subcutaneous infusion DO NOT change dose or stop pump until discussed with PD CNS/neurology registrar APO-go (apomorphine) 24 hour helpline: 08448801327	<u>Duodopa®</u> Intestinal gel (levodopa 20mg/carbidopa 5mg per ml) If unable to continue treatment, calculate LED by adding continuous rate + morning bolus dose Duodopa® 24 hour helpline: 0800 4584410
---	--

Nausea and vomiting:

<u>AVOID</u> -Metoclopramide -Prochlorperazine -Promethazine -Haloperidol WORSEN PARKINSONIAN SYMPTOMS	<u>USE</u> -Domperidone -Ondansetron -Cyclizine SHORT TERM ONLY (cardiac side effects)
--	--

**USEFUL NUMBERS: Neurology Registrar bleep:7277. PD CNS bleep: 8487 extn: 2141
 DO NOT STOP PARKINSON'S DISEASE MEDICATIONS: CONTACT THE WARD PHARMACIST OR ON-CALL PHARMACIST (bleep: 6267) OUT OF HOURS**

ANAPHYLAXIS

Link consultant: Dr Yee Ean Ong

Anaphylaxis is life threatening but rapidly reversible if treated properly. The symptoms, which include bronchospasm, hypotension, laryngeal and facial oedema and urticaria, can develop within minutes of challenge. Common precipitants include food (eg shellfish, peanut); wasp/bee sting; drugs such as penicillins, contrast media, vaccines; antigens given for “desensitisation”, or allergy to latex. Treatment principles are similar for adults and children but drug doses differ; the doses quoted below are for adults. **See resuscitation UK guidelines for further information**

Management

- Remove allergen (eg stop drug infusion)
- Give high-flow oxygen and preserve airway
- Give adrenaline (epinephrine), 0.5mL of a 1:1000 solution (ie 0.5mg) IM into antero lateral aspect of middle third of thigh. Repeat after 5 mins if there is no improvement. Several doses may be needed, especially if improvement is transient or the patient deteriorates.

Giving adrenaline IV is potentially hazardous and should be reserved for patients with immediately life-threatening profound shock by an experienced specialist (seek early critical care support).

- Intravenous crystalloid eg Hartmann’s fluid challenge 500-1000 mL immediately and more as needed if hypotensive.
- If no improvement after 2 does of im adrenaline, termed refractory anaphylaxis, give im adrenaline every 5 mins until iv adrenaline started
- Nebulised salbutamol and ipratropium are a useful adjunct if bronchospasm is a major feature which has not responded rapidly to other treatment.
- Routine use of corticosteroids is not advised, consider after initial resuscitation for refractory reactions or ongoing asthma/shock.
- Antihistamines are not recommended as part of the initial emergency treatment for anaphylaxis, consider oral cetirizine for skin symptoms but their use must not delay treatment with adrenaline and iv fluids.
- **NB Beware** the possibility of early or late recurrence of symptoms and consider observation for a minimum of 6-12 hrs
- *Write the name of the agent that caused the reaction – prominently in the patient’s notes and drug chart.*
- After a suspected anaphylactic reaction, mast cell tryptase should be taken as soon possible after emergency treatment and 1-2 hours (but not later than 4 hours) later.
- Patients should be discharged with appropriate information and an adrenaline auto-injector if needed.

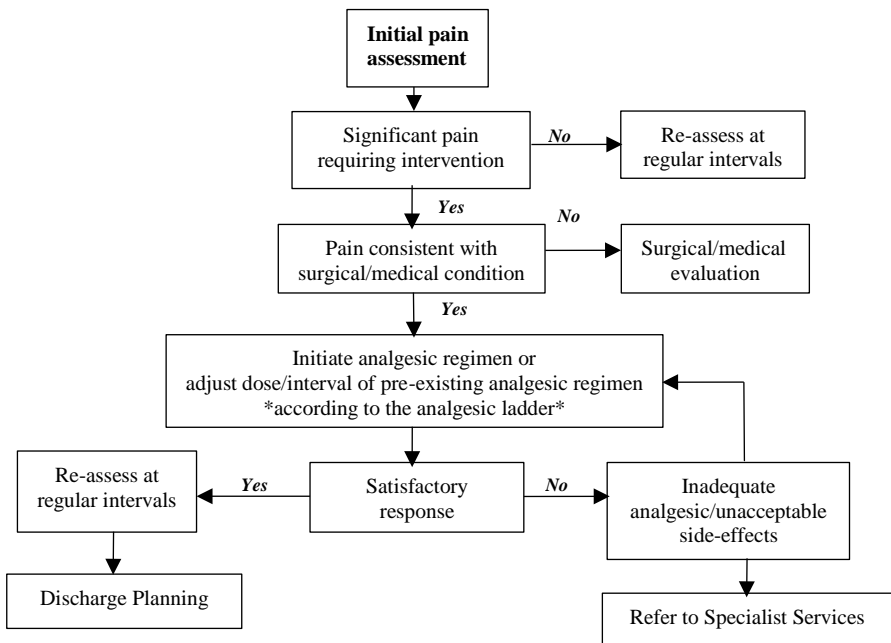
ACUTE PAIN

Link Consultant: Dr Liana Geary

Note that for some conditions, such as acute coronary syndromes, acute painful joints, and sickle cell crises, analgesic approaches differ.

Comprehensive guidelines are available on the Inpatient Pain Team's intranet page <https://stginet.unily.com/sites/inpatient-pain-service>

Acute pain, whether due to a medical or surgical condition, should be relieved as soon as possible. Simultaneously investigate and treat the underlying cause – it is rare for analgesia to mask a diagnosis. Pain may be classified as mild, moderate, severe and treated accordingly. In general it is more realistic to *strive for comfort* rather than complete abolition of pain.



THE “ANALGESIC LADDER”

Step 1

Mild pain

Regular, simple analgesics such as paracetamol and/or a non-steroidal anti-inflammatory drug (NSAID) if not contraindicated e.g. Ibuprofen.

Step 2

Moderate pain

Add weak opioid to Step 1 analgesia. If prescribing combination analgesic indicate the strength of the components.

Step 3

Severe pain

Change weak opioid to strong oral opioid + continue Step 1 analgesia (consider IV Paracetamol)

TREATMENT DETAILS

Simple Analgesics

- **Paracetamol:** 1g PO/Enteral tube/IV 4-6 hourly

(Maximum 4g/day **if over 50kg** and **no risk** of hepatotoxicity)

*Note: For **adult patients <50kg and/ or malnourished or at risk of hepatotoxicity, the maximum dose should be 15mg/kg 4-6 hourly (Maximum 60mg/kg in 24 hours)***

- **Non-Steroidal Anti Inflammatory Drugs (NSAIDs)**

Ibuprofen: 200-400mg PO 4-6 hourly (Maximum 2.4g/day)

Naproxen: 500mg BD OR 250mg 6-8 hourly PO (Max 1.25g/day)

The lowest possible dose should be used for the shortest period of time to control symptoms. Patients with moderate or high risk of gastrointestinal adverse events or those requiring extended courses should be on gastro-prophylaxis while on NSAIDs. Caution should be exercised in the over-65 years of age.

Contraindications:

Bleeding diathesis, peptic ulceration, renal dysfunction, allergy to NSAIDs (higher risk in asthmatics), coexisting severe cardiac disease, hepatic failure, 3rd trimester pregnancy

- **Combination Analgesic**

Co-dydramol 10/500 (Dihydrocodeine 10mg + Paracetamol 500mg per tablet)

1-2 tablets PO 4-6 hourly (Maximum 8 tablets/day).

Opioids

Be cautious when prescribing in renal impairment, elderly or opioid naive patients. The lowest effective dose should be used. Whenever prescribing opioids consider adding antiemetics and regular laxatives.

- **Weak Opioids**

Dihydrocodeine: 30mg PO 4-6 hourly (maximum 180mg/day)

Codeine Phosphate: 30mg PO 4-6 hourly (maximum 240mg/day)

Tramadol: 50-100mg PO 4-6 hourly (maximum 400mg/day)

- **Strong Opioids**

Morphine: recommended starting dose 5-10mgs PO 4-6 hourly
(Half the dose in elderly)

Oxycodone: recommended starting dose 2.5-5mg PO 4-6 hourly
(Half the dose in elderly and patients with moderate to severe renal impairment)

Morphine is the preferred opioid. Oxycodone should be used first line instead of morphine in patients with renal impairment or those unable to tolerate morphine. Oral route should be the first choice.

Morphine and oxycodone may also be given on the wards as an IM, SC injection (in appropriate situations for rescue analgesia) or a IV-Patient-Controlled Analgesia (PCA).

Note: if a patient is on a PCA, the dose of a step-2 opioid should be reduced as appropriate. Refer to PCA policy.

Severe acute pain often requires morphine to be given by injection to give adequate control. **IV morphine as a bolus should only be given in A&E, ICUs and Theatres with the patient under continuous observation. If a patient is to have IV morphine outside these areas, it needs to be under the supervision of a suitably qualified practitioner.**

For Morphine injections use the dosage regimens given in the following table:

Bolus IV Morphine <i>In A&E, ICUs and Theatres ONLY</i>		
Age (yrs)	Dose	
	Pain severe	Less severe
< 70	2mg	1mg
> 70	1mg	0.5mg

IM/ SC Morphine	
Age (yrs)	Dose
20-39	10mg
40-59	5-10mg
60-69	2.5-5 mg
70-85	2.5mg
>85	1mg

Assess the patient 10 minutes after IM, and every 5 minutes after IV injection for at least 30 minutes after administration. Assuming there is no evidence of opioid overdose (see section below for diagnosis and treatment), then if:

- *Pain relieved*, convert the patient to oral opioids and continue regular pain assessments.
- *Pain persists*, contact the [Inpatient Pain Team](#) on extension 2080/ bleep 6477 or complete a referral on iCLIP to the Inpatient Acute Pain Service to seek further advice.

OPIOID OVERDOSE

a. In the Acute Pain setting

If a patient who has been given an opioid shows signs or symptoms of an overdose such as drowsiness or respiratory depression (respiratory rate of less than 8 per minute) then:

1. Stop the opioid.
2. Assess Airway, Breathing, and Circulation. Administer oxygen by face mask. Support vital functions as necessary.
3. Give Naloxone IV injection 100-200mcg every 2-3 minutes until patient is rousable and respiratory drive returns. Expect to see an improvement after 1-2 doses. Call for senior help if patient does not make expected recovery.
4. Be aware that the antagonistic effect of naloxone may precipitate a pain crisis.
5. Inform the Inpatient Pain Team so the patient can be followed up as appropriate.

b. In the Palliative Care Setting

In the palliative care setting the use of inappropriate doses of naloxone can cause a rapid reversal of the physiological effects of long-term opioids used for pain control. This can cause intense pain and distress, and acute withdrawal syndrome. This can lead to cardiovascular instability, pulmonary oedema and even cardiac arrest.

If respiratory rate \geq 8/minute and the patient easily rousable and not cyanosed, adopt a policy of 'wait and see'; consider reducing or omitting the next regular dose of opioid.

If respiratory rate $<$ 8/minute, patient barely rousable/unconscious and/or cyanosed:

1. Stop the opioid
2. Administer oxygen by face mask
3. Dilute a 1mL ampoule of naloxone 400mcg to 4mL with sodium chloride 0.9%
4. Administer 1mL (100mcg) IV (from dilution above) every 2 minutes until the patient's respiratory status is satisfactory. Even lower doses may be used: 0.2mL (20mcg) IV every 2 minutes until respirations are satisfactory. If IV access is not present / appropriate then naloxone can be given IM or SC. Call for senior help if the patient does not make the expected recovery.
5. Close observation is needed to ensure the patient is breathing satisfactorily and pain control is maintained.
6. If repeated doses of naloxone are needed then consider a naloxone infusion.
7. Ensure the palliative care team is informed so the patient can be followed up as appropriate (see contact details below)

Naloxone is shorter-acting than opioids and therefore the patient needs to be kept under observation to ensure that the signs of an overdose do not recur. A Naloxone infusion may be needed to prevent it. Discuss with ICU.

If a patient has been on a transdermal opioid patch, be aware that it may take up to 30 hours for the levels of opioid to decrease to 50% and the patient will require continuous monitoring.

Communications:

Inpatient Pain Team (bleep 6477);

On-Call Anaesthetist (bleep 6111/7647);

Palliative Care Team (bleep 6508).

Out of Hours Palliative Care Advice (Royal Trinity Hospice 0207 787 1000 and ask for on call doctor)

Email: acute.painservice@stgeorges.nhs.uk

SUGGESTIONS FOR THE USE OF ANTIMICROBIAL DRUGS IN ADULTS
Links: Marina Basarab and Amber Arnold

THE FOLLOWING ADVICE RELATES DIRECTLY TO TRUST POLICY AND GUIDELINES

All clinicians should ideally within one hour of (or as soon as possible after) identifying a possible bacterial infection, undertake the following:

A) START SMART

- **DO NOT START ANTIBIOTICS** in the absence of clinical evidence of bacterial infection (does not apply to febrile neutropenic patients)
- If there is evidence of bacterial infection, use the trust empiric antibiotic guidelines to initiate prompt effective antibiotic treatment. Take appropriate cultures prior to initiation of antibiotic therapy if possible.
- In patients with sepsis initiate antibiotics within one hour of diagnosis.

For Trust policy for specific conditions including surgical prophylaxis see the Microguide website: <http://microguide.horizonsp.co.uk/viewer/sguh/adult> or use the Microguide smartphone app (or follow quick link at bottom of Intranet homepage)

- If the patient is colonised with resistant organisms, such as MRSA or those producing ESBLs (extended-spectrum beta-lactamases) empiric treatment should be modified accordingly. Discuss with microbiology.
- Penicillin allergy – check and document nature of reaction: mild = rash; moderate to severe = angioedema, swollen tongue, anaphylaxis
- Document on drug chart and in medical notes: clinical indication, duration or review date, route and dose.

Antibiotics in hospitals are often continued unnecessarily because clinicians caring for the patient do not have information indicating why the antibiotics were initially commenced and how long they were planned to be continued. This problem is compounded where primary responsibility for patient care is frequently transferred from one clinician to another. Ensuring that all antibiotic prescriptions are always accompanied by an indication and a clear duration or review date will help clinicians change or stop therapy when appropriate.

• **Obtain Cultures First**

Knowing the susceptibility of an infecting organism can lead to narrowing of broad-spectrum therapy, changing therapy to effectively treat resistant pathogens and stopping antibiotics when cultures suggest an infection is unlikely. Use strict asepsis when taking blood cultures – contaminated samples lead to clinical confusion and inappropriate antibiotics.

• **Prescribe single dose antibiotics for surgical prophylaxis where antibiotics have been shown to be effective.**

Critical to this advice is that the single dose is administered within the 60 minutes *prior* to surgical incision or tourniquet inflation to enable peak blood levels to be present at the start of the surgical procedure. A repeat dose of antibiotic prophylaxis is required when the operation for prolonged procedures and where there is significant blood loss. A treatment course of antibiotics may also need to be given (in addition to appropriate prophylaxis) in cases of dirty surgery or infected wounds.

THEN FOCUS

REVIEW THE CLINICAL DIAGNOSIS and the continuing need for antibiotics by 48 hours and make a clear plan of action - the “Antimicrobial Prescribing Decision”.

Antibiotics are generally started before a patient's full clinical picture is known. By 48 hours, when additional information is available, including microbiology, radiographic and clinical information, it is important for clinicians to re-evaluate why the therapy was initiated in the first place and to gather evidence on whether there should be changes to the therapy. Review the need for antibiotics on every ward round.

The five Antimicrobial Prescribing Decision options are:

Stop, Switch, Change, Continue and OPAT:

1. **Stop** antibiotics if there is no evidence of infection
2. **Switch** antibiotics from intravenous to oral when:
 - temperature has been $< 38^{\circ}\text{C}$ for 48 hours or more;
 - oral foods/fluids are tolerated;
 - there is no unexplained tachycardia (HR < 90 bpm for 48 hours); *and provided that:*
 - there is no evidence of impaired absorption;
 - it is not a condition such as endocarditis or meningitis, for example, in which extra high tissue antibiotic concentrations are essential;
 - a suitable oral formulation is available.
3. **Change** antibiotics – ideally to a narrower spectrum – or broader if required
4. **Continue** and review again at 72 hours
5. Outpatient Parenteral Antibiotic Therapy (**OPAT**).

It is essential that the review and subsequent decision is clearly documented in the medical notes. (*Source: ARHAI Antimicrobial Stewardship Guidance 18.11.11*).

- Remember the potential harm caused by antibiotics, in terms of side effects and selection of resistant organisms such as MRSA and *Clostridium difficile*. Cephalosporins and ciprofloxacin have particular risk of selecting *C.difficile* or MRSA.
- Treat the clinical condition and not the microbiology result – a positive culture may represent colonisation, normal flora or contamination, as well as infection. This is especially true of catheter urines samples and sputum samples.
- Non-antibiotic measures may be equally important in treating some infections – eg: drainage/debridement of deep wound infections or abscesses, removal of foreign bodies such as IV lines or urinary catheters, hygienic measures for infected skin ulcers or superficial wound infections, physiotherapy in the management of pneumonia.
- Always consider the implications for cross-infection. Infection Control advice can be found on the Trust Intranet, or obtained from the Infection Control Team (x5675).
- Certain antibiotics (principally Cephalosporins, Ciprofloxacin, Meropenem, Ertapenem, Tazocin) are designated ‘Protected’ – their use is restricted to certain departments and/or specific clinical indications as listed in Trust guidelines; Microbiology approval is required for all other indications.
- IV antibiotics should only be used if the patient is seriously ill or unable to take medication orally. IV ciprofloxacin, sodium fusidate or clindamycin are rarely necessary because oral preparations have very good bioavailability.
- Doses are for adult patients with normal renal and hepatic function – seek advice in patients with impaired clearance.

The infection care group at St Georges is an integrated care group consisting of medical microbiology, adult infectious diseases, OPAT and infection control.

Seek further advice within working hours as appropriate from:

- Infection (Microbiology) Registrar (including A&E): Bleep 6480
- Infection (Microbiology) Registrar covering Intensive Care and Paediatric Specialities Registrar (covers GICU, CITU, NICU, PICU, NNU, Paediatrics, Paediatric Surgery): Bleep 7118
- McEntee Ward Registrar (Infection (Adult Infectious Diseases) Registrar: Bleep 7568
- Antimicrobial Pharmacist: Bleep 7508
- Ward Pharmacists, or Infection Control Nurses

For out-of-hours advice contact the respective department via switchboard.

Infections in inpatients such as TB, meningitis or infections in returned travellers should generally be managed by the McEntee Ward registrar (bleep 7568). The same specialists, plus GU Medicine should be contacted for advice on management of patients with HIV (*Appendix 8*).

Outpatient Parenteral Antibiotic Treatment (OPAT) Service

Some patients who are in hospital *only* because of the need for intravenous antibiotics (e.g. patients with cellulitis or osteomyelitis) may be suitable for inclusion in the OPAT service. To refer or for advice contact the OPAT registrar (bleep OPAT nurse (bleep 8170) Please refer to OPAT webpage under ‘Units & Departments’ on the Trust Intranet site for further details and referral form.

<http://stginet/Units%20and%20Departments/OPAT%20Service/OPAT%20Service.aspx>

INDICATIONS	CONTRA-INDICATIONS
Medically stable, fit for discharge	Oral antibiotics a feasible option
Stable home environment	Drug abuse / alcoholism
Patient understands OPAT and willing to avail of the service	Homeless or chaotic lifestyle, or dangerous home environment
Need IV antibiotics – no oral option	Active relevant psychiatric conditions (eg suicidal ideation, psychosis)
Once daily intravenous antibiotic available (unless patient is able to self-administer)	Unstable medical or surgical condition.
	Severe cognitive impairment eg senile dementia

ANTIBIOTIC MANAGEMENT OF COMMON INFECTIONS IN ADULTS

**PLEASE REFER TO TRUST INTRANET
GUIDELINES
USING THIS LINK:**

<http://microguide.horizonsp.co.uk/viewer/sguh/adult>

**MANAGEMENT OF SEPSIS:
PATIENTS WITH AN EARLY WARNING SCORE (EWS) GREATER THAN 4
Link Consultant: Dr Catherine Cosgrove**

Sepsis has a high mortality and morbidity. About 50% of patients with an Early Warning Score (EWS) >4 will have sepsis. Prompt action can save lives.

Patients with a high EWS score should be reviewed for signs of infection. The immunosuppressed are particularly vulnerable and neutropenic sepsis guidelines should be reviewed:

<http://stginet/Units%20and%20Departments/Acute%20Oncology%20Service/AOS%20Homepage.aspx>

On recognising a patient who is septic the practitioner should start the 'Sepsis 6' (see diagram below) or document why it is not appropriate, such as they have an alternative diagnosis or end of life plan.

The choice of IV antibiotics should be guided by likely source of infection and by the hospital antibiotic guidelines:

<http://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Antimicrobial%20Prescribing%20Info%20for%20staff.aspx>

Refer to the microguide: <http://microguide.horizonsp.co.uk/viewer/sguh/adult>

**Is your patient scoring on EWS?
Do they have, or are they likely to have, an infection?**

High Risk Criteria

Responds only to voice or pain/ unresponsive
Systolic B.P \leq 90 mmHg (or drop >40 from normal)
Heart rate $>$ 130 per minute
Respiratory rate \geq 25 per minute
Needs oxygen to keep SpO₂ \geq 92%
Non-blanching rash, mottled/ ashen/ cyanotic
Not passed urine in last 18 hours
Urine output less than 0.5 ml/kg/hr
Lactate \geq 2 mmol/l
Recent chemotherapy in past 6 weeks



- **Initiation of the Sepsis Six to be completed as soon as possible, but always within 60 minutes - take blood, urine and sputum cultures if indicated.**
- **IV antibiotics- write a review date for 48 hours**
- **Review by competent decision-maker, such as a doctor grade ST3 or above, immediately.**
- **Telephone conversation with consultant immediately following initial review.**
- **Referral to critical care or document why not**

Moderate Risk Criteria

Relatives or Nursing concerns re mental state
Acute deterioration in functional ability
Trauma/ surgery/ procedure in last 6 weeks
Systolic B.P 91- 100 mmHg (or drop >40 from normal)
Heart rate 111- 130 per minute or new arrhythmia
Respiratory rate 21-24 per minute or breathing hard
Not passed urine in last 12- 18 hours
Clinical signs of wound, device or skin infection



- Sepsis Six to be completed as soon as possible, but always within 60 minutes - remember to take blood, urine and sputum cultures if indicated.
- Review by competent decision-maker, such as a doctor grade ST3 or above, within 60 minutes.
- IV antibiotics- write a review date for 48 hours
- Review by consultant within 14 hours
- Observations every 30 minutes until EWS score is below 4 for 4 successive hours.
- Repeat lactate measurement within 2 hours.
- Repeat laboratory blood tests within 14 hours, unless observations indicate earlier need (eg reducing urine output, jaundice, bleeding).
- Escalate immediately if septic shock (including cryptic shock) develops or if organ dysfunction requires reconsideration of need for critical care (eg acute kidney injury).

The Sepsis Six

- 1 High-flow oxygen to maintain oxygen saturation >95%
- 2 Blood cultures and consider source control
- 3 Intravenous antibiotics
- 4 Intravenous fluid resuscitation
- 5 Check haemoglobin and serial lactates
- 6 Hourly urine output measurement

If after delivering the Sepsis Six, patient still has:

- systolic BP <90 mmHg
- reduced level of consciousness despite resuscitation
- respiratory rate over 25 breaths per minute
- lactate not reducing

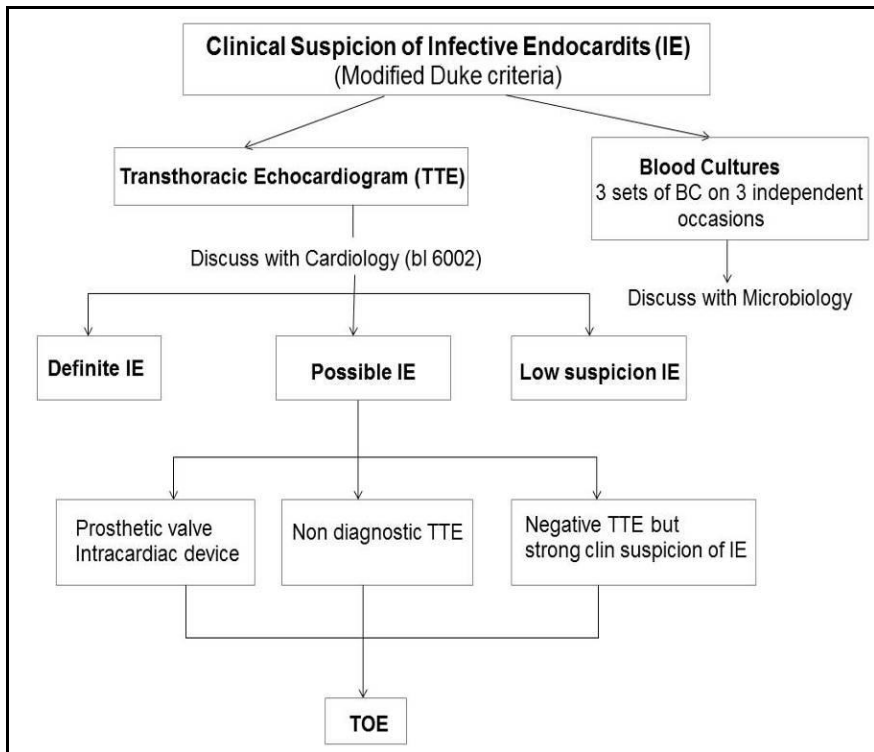
or if patient is clearly critically ill at any time

Refer to Critical Care Outreach

INFECTIVE ENDOCARDITIS

Suspected endocarditis: See flow chart below. Take blood cultures (3 sets at 3 different times, plus a serum sample), request an *urgent* ECHO & seek an *urgent* review by a *senior* cardiologist. The decision as to when to start treatment depends on the severity of illness – in general terms, clinical sepsis should not go untreated. Discuss all cases of endocarditis with the Infection (Microbiology) team, blp 6480 ext 5676/1970 or via switchboard if out-of-hours.

Patients with endocarditis should be under the care of a named Consultant Cardiologist and managed by Cardiology or jointly by Cardiology & Infection. There is a joint Cardiology – Infection MDT that reviews endocarditis patients on a weekly basis, usually on a Wednesday morning.



Empiric treatment of endocarditis is based on:

- whether a patient has a native valve or prosthetic valve
- time of presentation after surgery for a prosthetic valve.

Native valve or late prosthetic valve (>1 yr post surgery) infections are usually treated empirically with beta lactams antibiotics such as Flucloxacillin combined with Amoxicillin to cover *S. aureus*, *Streptococcus spp* and *Enterococci spp*. Gentamicin is added to provided synergy for *Streptococcus* species.

Prosthetic valve infections that are early (<1 year post surgery) need to be initially treated with antibiotics cover for other organisms especially coagulase negative staphylococcus. The combination usually includes vancomycin, gentamicin and rifampicin.

Please refer to latest Trust guidelines on the Microguide App or web page for empiric treatment: <http://microguide.horizonsp.co.uk/viewer/sguh/adult>

Speciality Guidelines>Endocarditis Guidelines – Treatment

If or once the organism is known, discuss with Infection (Microbiology) for definitive treatment.

EMPIRICAL TREATMENT OF DIABETIC FOOT INFECTION

Please refer to Trust Intranet Guidelines:

<http://microguide.horizonsp.co.uk/viewer/sguh/adult>

INFECTIOUS DIARRHOEA

The following advice relates directly to trust policy and guidelines:

Patients with suspected infectious diarrhoea should be managed according to the ST GEORGE'S NHS TRUST ADULT INPATIENT DIARRHOEA PROTOCOL

<http://stginet/Units%20and%20Departments/Infection%20Control/diarrhoea%20protocol%202012%20v3.doc>

SURGICAL WOUND INFECTION

Antimicrobial treatment is indicated only if the wound shows signs of spreading inflammation or if the patient is systemically ill. Blood cultures as well as pus from the wound should be sent to the laboratory. Take advice on initial treatment. The diagnosis of a wound infection is a clinical diagnosis not a microbiological one. The growth of organisms in specimens does not necessarily mean an infection and microbiology specimens should be taken to guide treatment, not make the diagnosis of an infected wound.

MALARIA OR FEVER IN RETURNING TRAVELLERS

Link Consultant: Professor Derek Macallan

Malaria should be considered in any ill or febrile patient who has travelled in a malaria-endemic area.

For country information, see <http://nathnac.org/pro/index.htm>. Note that the incubation can be very prolonged.

Typhoid fever is an important differential diagnosis. Request an FBC, an urgent malaria blood film, blood cultures (for typhoid) and a serum sample. For recent returnees (≤ 21 days) from high-risk areas, also consider the remote possibility of viral haemorrhagic fever (*see* Trust protocol).

All febrile adult travellers returning from the tropics should be referred to Infectious Diseases: CIU registrar, Bleep 7568 during daytime, or via switch after hours.

If malaria is diagnosed in an adult, enquire if there are children in the family who are currently unwell.

Children suspected of having malaria should be evaluated in paediatric A&E the same day. Inform the Paediatric Registrar on duty, Bleep 7474.

DRUG OVERDOSAGE/ACUTE POISONING

Link Consultant: Dr Mark Haden

This section describes the general measures that should be taken to support patients in the first 24 hours after poisoning. It also offers advice on the treatment of some of the more common causes of poisoning. The guidelines are far from exhaustive and so for more detailed information, or for advice on the treatment of less common situations, contact Toxbase (the National Poisons Information internet site) at <http://www.spib.axl.co.uk> or the National Poisons Information Service on 0844 892 0111.

PRIMARY ASSESSMENT

- Is airway protected?

If not, crash bleed the anaesthetic registrar and intubate patient with cuffed endotracheal tube. If these procedures are delayed lay the patient in the recovery position.

- Is ventilation adequate?

Check clinical indices; respiratory rate, depth and drive, oxygen saturation + arterial blood gases. If ventilation inadequate, consider giving naloxone (up to 2mg) to reverse opiates, and providing ventilatory support. Give O₂ to all patients until it is clearly not required.

- Is circulation adequate?

If hypotensive give IV fluid – initially sodium chloride 0.9%. Introduce a central venous line if help is needed for monitoring fluid replacement. Attach cardiac monitor to check for dysrhythmias and treat as appropriate. Avoid vasoconstrictors.

- Assess conscious level and pupil size and reactivity.
- Check body temperature – those with hypothermia may well need warming.
- Check capillary blood glucose at the bedside.
- Is the patient pregnant? If yes, seek advice from the on-call obstetric SpR or the NPIS. If unsure, consider doing a pregnancy test
- Check U&Es, renal and liver function, blood glucose and acid base balance as appropriate.
- Do an ECG if appropriate and a CXR if aspiration a possibility.
- Establish means to monitor vital signs.

IDENTIFY THE POISON

Take history from patient or relatives (or phone GP) to find out what medications the patient had available, and to assess amount taken and when

- Retain tablets or containers found with patient
- Check paracetamol and salicylate blood levels (4hrs after ingestion if timing possible)
- Consider sending blood, urine, gastric fluid for toxicology
- If information on definitive treatment of specific poisons is needed this can be sought as follows:
 - a) Use Toxbase (see above for website)
 - b) If adequate information cannot be obtained by these means, or for further advice on cases that are clinically or toxicologically complex, ring NPIS (0870 600 6266).

PREVENT ABSORPTION OF DRUG/POISON

Removal of drug from the GI tract is controversial. The potential benefits of reducing drug absorption may be outweighed by the hazards of the methods used, eg aspiration of stomach contents, paradoxical increase in drug absorption. Syrup of ipecac should **not** be used to induce vomiting. Gastric lavage and activated charcoal have a place but they should only be used according to strict criteria:

A. Gastric lavage

Indications

Lavage should be undertaken if presentation is within 1 hour of ingestion, if the patient has taken a potentially life threatening drug overdose, and if the procedure is agreed by a senior member of Emergency Department staff.

Contraindications to lavage

Lavage should not be undertaken if:

- the patient has depressed conscious level, unless airway is protected by cuffed ET tube
- the substance ingested is a hydrocarbon or corrosive
- the patient is at risk of GI haemorrhage or perforation

B. Activated charcoal (50-100g) as a single dose to reduce drug absorption

Indications

Presentation within 1 hour of ingestion of a potentially toxic amount of a drug known to be adsorbed to charcoal (check with NPIS or Toxbase if drug is not on the list).

Adsorbable drugs include:

- antiepileptics (phenytoin, phenobarbital, carbamazepine, valproate)
- analgesics (paracetamol, salicylates, dextropropoxyphene, piroxicam)
- cardiac drugs (disopyramide, amiodarone, digoxin, Ca channel blockers)
- antidepressants (SSRIs, tricyclics)
- miscellaneous (theophylline, quinine, dapsone)

Presentation 1-2 hours after ingestion of a potentially toxic amount of drug adsorbed to charcoal and known to delay gastric emptying. Such drugs include: salicylates, opioids, tricyclic antidepressants, sympathomimetics, theophylline.

Contraindications

- Drugs not adsorbed by activated charcoal (metals, alcohols, acids, alkalis)
- Depressed conscious level, unless airway is protected by cuffed ET tube

Complications

- The administration of activated charcoal is associated with aspiration and GI obstruction

SECONDARY ASSESSMENT

Continue to monitor and treat problems that arise in ED and on the ward.

Airway and Breathing – monitor respiration and oxygen saturation. Protect airway with cuffed endotracheal tube and support breathing with ventilation as appropriate.

Circulation – pulse, blood pressure. IV fluids for hypotension. Avoid vasoconstrictors.

Cardiac monitor for dysrhythmias if appropriate.

Conscious level – neurological observations and pupils.

Body temperature - check.

Urine output – IV fluids if urine output falls to <400mL/24 hour. Check bladder. If distended, attempt to empty it with fundal pressure before considering catheterisation.
Other active medical problems? History from patient and/or relatives plus physical examination to assess intercurrent medical problems which may precipitate or complicate overdose.

If there is currently, or potentially, a need for High Dependency or Intensive Care, discuss with ITU registrar early (contact through ITU x3295 or x3296).

ENHANCE GI ELIMINATION OF DRUG/POISON A. Multiple-dose activated charcoal

Indications

Consider multiple-dose activated charcoal to increase drug elimination if the patient has taken a life-threatening dose of carbamazepine, theophylline, phenobarbital, quinine or dapsone, or a tricyclic antidepressant. It should also be used for salicylate poisoning when the blood concentrations are still rising.

Contraindications:

- Unprotected airway; Intestinal obstruction
- Drugs not adsorbed by activated charcoal (metals, alcohols, acids, alkalis)

Protocol

- Give an initial 50g dose of activated charcoal
- Activated charcoal to be drunk by patient, or if this is not possible it can be given via an NG tube. Consider giving an antiemetic intravenously if charcoal poorly tolerated.
- Repeat charcoal administration at a dose of 50g every 4 hours and review the clinical status of the patient after 4 doses (Total 200g).
- Continue charcoal until patient's clinical and laboratory parameters, including plasma drug concentrations, are improving
- Give a laxative to prevent constipation

B. Whole bowel irrigation

Indications

- Life-threatening overdose of a sustained-release or enteric coated drug, or drug not absorbed by activated charcoal (e.g, iron, lithium)
- After ingestion, or insertion (into lower GI tract), of packets of illicit drugs

Contraindications

- **Bowel obstruction, perforation, ileus, GI haemorrhage**
- Haemodynamic instability
- Compromised, unprotected airway
- Patients with debility or a condition that irrigation may exacerbate

Protocol

- Give irrigation solution by mouth or NG tube using reconstituted polyethylene glycol (4 sachets of Klean-Prep oral powder dissolved in 4 litres of water) at 1500 – 2000mL/hr (for adults)
- Patient should be seated or at least at 45°
- Continue whole bowel irrigation until rectal effluent is clear

SPECIFIC MEASURES FOR COMMON DRUG OVERDOSES PARACETAMOL

Paracetamol overdose, even in small amounts, can cause fatal liver damage. To prevent this:

- Paracetamol should be suspected as a component of all overdoses.
- Plasma concentrations should be measured and compared against a paracetamol treatment graph (see below)

Patients with plasma paracetamol concentrations above the treatment line are at risk of liver damage and require antidote treatment.

- N-acetylcysteine, which acts as an antidote and prevents paracetamol-induced liver damage, should be used as described below.
- The treatment of patients who have taken a paracetamol overdose depends on the timing of presentation after overdose, as well as the way in which the overdose was taken.
- Where the time of ingestion is unknown, patients should be managed as per the staggered paracetamol overdose.
- If there is uncertainty about whether the presentation was due to therapeutic excess, the patient should be managed as a staggered paracetamol overdose.

Within 4 hours of ingestion

- <1 hour, give activated charcoal if more than 150mg/kg of paracetamol is ingested.
- Measure plasma concentrations at 4 hours post ingestion. If levels are above treatment line on treatment graph, give N-acetylcysteine intravenously using the following regimen:
 - First infusion: 150mg/Kg (maximum 16.5g) in 200mL 5% glucose as IV infusion over 60 minutes
 - Second infusion: 50mg/Kg (maximum 5.5g) in 500mL 5% glucose as IV infusion over next 4 hours
 - Third infusion: 100mg/Kg (maximum 11g) in 1L 5% glucose as IV infusion over next 16 hours

Within 4-8 hours of ingestion

- Measure plasma paracetamol concentrations at presentation
- Compare concentrations with treatment graph to determine whether N-acetylcysteine is indicated.

Within 8-15 hours of ingestion

- Take blood for urgent measurement of plasma paracetamol concentrations (preferentially prior to administration of N-acetylcysteine).
- Start N-acetylcysteine infusion immediately if more than 150mg/kg of paracetamol is ingested.
- Stop treatment if level is below the treatment line on the treatment graph.

Within 15-24 hours of ingestion

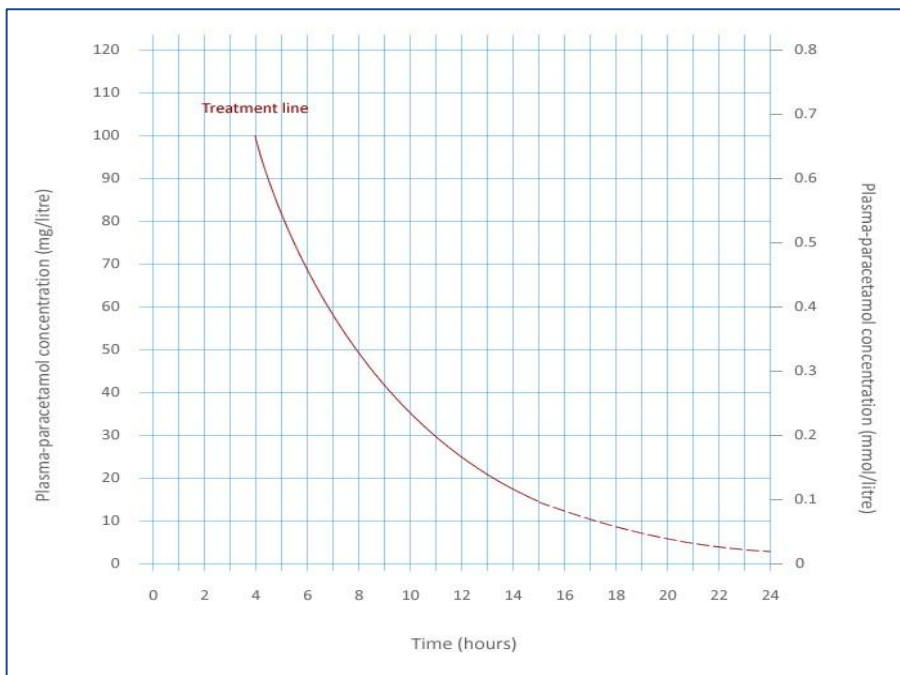
- Take blood for urgent measurement of plasma paracetamol concentrations (preferentially prior to administration of N-acetylcysteine).
- Start N-acetylcysteine infusion immediately

In patients presenting more than 12-15 hours after overdose, the concentration of paracetamol requiring treatment with acetylcysteine may be close to the limit of detection of the paracetamol assay.

If at 24 hours the patient is asymptomatic, INR, blood, gases and plasma creatinine are normal and plasma paracetamol concentration $<10\text{mg/L}$, then the N-acetylcysteine infusion can be stopped. If any of these are abnormal then continue N-acetylcysteine at 150mg/Kg over 24 hours.

Take blood for plasma paracetamol concentrations and if the patient is asymptomatic and the INR, LFTs, venous bicarbonate and plasma creatinine figures are all 'normal', the patient can be seen as medically fit and told to return if abdominal pain or vomiting develop.

- Treat with N-acetylcysteine if the patient is clearly jaundiced or has hepatic tenderness. Otherwise wait for the results of blood tests before commencing treatment.
- Patients with a chronically elevated ALT (e.g. chronic liver disease), may not require acetylcysteine treatment if the ALT and INR have not significantly changed from previously documented values. These cases should be discussed with the NPIS.
- Treat with acetylcysteine if;
 - A. ALT is above the upper limit of normal,
 - B. INR is greater than 1.3 (in the absence of another cause, e.g. warfarin)
 - C. Paracetamol concentration is detectable



Situations where N-acetylcysteine should be given without guidance of the treatment graph and managed as per staggered overdose:

- Where timing of overdose is unknown
- Where overdose was staggered (tablets taken at 2 or more times)

- All patients presenting with evidence of severe toxicity or fulminant hepatic failure regardless of the time post overdose

Staggered overdose (non-therapeutic ingestions of excessive paracetamol over a period of more than one hour)

MHRA advice is that treatment with acetylcysteine should be commenced without delay in ALL patients who have ingested a staggered overdose.

Check blood tests **at least 4 hours** after the last paracetamol ingestion.

Clinically significant hepatotoxicity is unlikely if at least 4 hours or more after the most recent paracetamol ingestion:

- the paracetamol concentration is less than 10 mg/L, **AND**
- the ALT is within the normal range, **AND**
- the INR is 1.3 or less, **AND**
- the patient has no symptoms suggesting liver damage.

Acetylcysteine can be discontinued *in patients not considered to be at risk of clinically significant liver damage*. If the patient also has a normal serum creatinine, they can be discharged.

Therapeutic excess (ingestions of excessive paracetamol with intent to treat pain or fever and without self-harm intent)

Patients with clinical features of hepatic injury such as jaundice or hepatic tenderness should be treated urgently with acetylcysteine. In other patients, management is determined by the maximum dose of paracetamol ingested in any 24-hour period.

A. Maximum dose more than 75 mg/kg within any 24-hour period:

- Check paracetamol concentration, LFTs, INR, U&Es, creatinine, bicarbonate and FBC at least **4 hours** after the last paracetamol ingestion.
- Acetylcysteine should be commenced if the patient is symptomatic or blood tests indicated a risk of hepatotoxicity.

B. Maximum dose more than the licensed 24-hour dose for that patient (e.g. 4g in an adult) but less than 75mg/kg/24 hours over the preceding 2 or more days:

- Blood tests to be considered particularly if there is doubt about the doses used, **OR** other factors are present that may increase risk of hepatotoxicity.
- Acetylcysteine should be commenced if the patient is symptomatic or blood tests indicated a risk of hepatotoxicity.

C. Maximum dose consistently less than the licensed 24-hour dose for that patient (e.g. 4g in an adult) AND consistently less than 75mg/kg for that patient over the preceding 24-hour period:

- Further assessment is not needed, provided a reliable history has been obtained and the patient is well.

Acute kidney injury may occur as part of acute hepatic injury (hepatorenal syndrome) or, rarely, in the absence of hepatic injury. The latter is a well-recognised complication. Even a small rise may be clinically significant. Acetylcysteine has not been tested as an antidote for this complication and there is uncertainty about its efficacy as a treatment for isolated renal impairment. If creatinine has risen significantly then recheck in 8-12 hours.

Treat renal failure conventionally. Maximal abnormalities usually occur 3-7 days after exposure; recovery of renal function is the norm.

Post treatment

Monitor urine output and plasma glucose. Take blood for urea, creatinine and electrolytes, INR, liver function tests, and blood gases. Use to determine whether patient is fit for discharge, in-patient care should be prolonged or advice sought from specialist liver centre.

Contact specialist liver centre if:

- INR post-ingestion >2 at 24 hours, >4 at 48 hours, >6 at 72 hours
- There are other indices of severe hepatotoxicity i.e. any of elevated creatinine, acidosis, renal failure, hypotension (mean arterial pressure <60mmHg), encephalopathy

ASPIRIN (SALICYLATE)

In overdose salicylate stimulates the respiratory centre, resulting in hyperventilation and a respiratory alkalosis. There is a compensatory increase in renal excretion of bicarbonate, sodium, potassium and water, resulting in metabolic acidosis with dehydration and electrolyte imbalance. Acidosis increases the amount of salicylate that can cross into the CNS and causes CNS effects such as coma and convulsions. If the patient has tinnitus it is likely that the plasma salicylate concentration is greater than 400mg/l.

Monitoring

- U & Es, CVP (for moderate to large overdoses) – correct dehydration and electrolyte abnormalities with IV fluids, may need large volumes
- pH and arterial blood gases
- Blood sugar
- Prothrombin time

Treatment

- The benefit of gastric decontamination is uncertain.
- If <1hour since ingestion of 125mg/kg or more give activated charcoal (dose: 50g adults; 1g/kg child).
- A second dose of charcoal may be warranted in patients whose plasma salicylate concentration continues to rise.
- Consider gastric lavage in adults within 1 hour of a potentially life-threatening overdose (suggested dose 500mg/kg salicylate or more), providing the airway can be protected.
- Measure plasma salicylate level at least 2 hours (symptomatic patients) or 4 hours (asymptomatic patients) post ingestion and every 2 hours until plasma salicylate level starts to fall.
- Check acid-base status, U&Es, INR/PTR, FBC and blood glucose.
- Asymptomatic patients with normal acid-base status can be considered for discharge after observation for 6 hours following the overdose, provided their plasma salicylate concentration is below 300 mg/L (2.2 mmol/L).
- Treat hypokalaemia urgently; this will reduce the risk of severe hypokalaemia with bicarbonate therapy if this becomes necessary later.

Urinary alkalinisation

This enhances elimination of salicylates and reduces CNS effects, and is indicated if the salicylate level is greater than 500mg/L in adults or 350mg/L in children or the elderly.

Aim for:

- Correction of hypokalaemia (hypokalaemia prevents urinary excretion of alkali)
- Urine pH 7.5 to 8.5, but plasma pH <7.6

Indications for haemodialysis

- Renal failure
- Congestive heart failure or non-cardiogenic pulmonary oedema
- Hypoxia
- Coma, convulsions, CNS effects not resolved by correction of acidosis
- Acid-base or electrolyte imbalance resistant to correction
- Persistently high salicylate concentrations unresponsive to urinary alkalinisation
- If the salicylate concentration is greater than 700mg/L.

OPIOID OVERDOSE

Features

Severe opioid toxicity produces depression of the respiratory and central nervous systems. If untreated the depression of the level of consciousness can lead to deep coma, convulsions and respiratory arrest.

Milder opioid toxicity may produce nausea, vomiting, nightmares, anxiety, agitation, euphoria, dysphoria, depression, paranoia and hallucinations. While pin-point pupils are often present, this is not a reliable clinical sign and their absence does not exclude opioid toxicity. Sedation may be associated with hypotension, bradycardia and hypothermia. The route of opioid administration may produce important clinical features such as soft tissue infections and abscesses at the sites of intravenous heroin injection. The presence of infections distant to the injection site should also be specifically determined (for example, endocarditis, lung abscesses). Inadvertent intra-arterial injection can cause severe limb ischaemia.

The time to peak concentration depends on the formulation and route of administration (see below). The mean plasma elimination half-life for morphine is about 2 hours but this may be disproportionately prolonged in overdose.

Oral solution – peak for free morphine at 15 minutes

Capsule - 2 to 4 hours

MR tablet - 1-5 hours

IV injection - 15 minutes

Management

1. Manage the patient using an ABC approach. Give 100% oxygen via facemask.
2. Stop any ongoing opioid administration and check for and remove any transdermal patches.
3. Naloxone is a competitive opioid receptor antagonist. The mean half-life is around 30mins which is shorter than that of most opioids. The dose of naloxone depends on the clinical circumstances:
 - For opioid-induced respiratory depression following **acute overdose**, rapid titration of naloxone is necessary to reverse potentially life-threatening effects.

- For patients who have been on long term opioids (either prescribed or illicit) 1) the use of inappropriate doses of naloxone can cause a rapid reversal of the physiological effects of long-term opioids.. This can cause an acute withdrawal syndrome which can lead to cardiovascular instability, pulmonary oedema and cardiac arrest.. If such patients have a respiratory rate ≥ 8 , are easily rousable and not cyanosed/normal pCO₂ on ABG, consider omitting any further opioid doses and monitoring the patient closely. If required, use smaller doses of naloxone titrated to effect – dilute one ampoule of naloxone (400micrograms) to 10ml in 0.9% sodium chloride (40 micrograms/ml) and give 0.5-1.0ml every 2-3mins titrated to effect.
 - If, following naloxone administration, the patient subsequently develops signs of opioid toxicity again, consider starting the patient on a naloxone infusion. The starting rate of the infusion per hour should be set at 60% of the initial bolus dose of naloxone which the patient required.
For example if the patient required 2mg of naloxone to achieve the desired level of consciousness, the infusion should be started at 1.2mg/hr.
4. Patients should be monitored closely for any of the above signs of severe opioid toxicity. Patients who have received multiple doses of naloxone or who are on a naloxone infusion are likely to require admission to an HDU environment.
 5. Monitor BP, pulse, respiratory rate and conscious level every 15 minutes initially. Monitor oxygen saturation continuously in patients with a reduced level of consciousness. Consider arterial blood gas analysis to test for hypercapnia or hypoxia in patients with reduced respiratory rate. Check U&Es and CK in patients with a history of prolonged unconsciousness.
 6. The length of observation required prior to discharge will be dependent on the type of opioid preparation taken:
 - Standard Preparation – 6hrs
 - Prolonged Release – 12hrs
 - Methadone – 24hrs
 7. Prior to discharge, ensure the patient has been symptom-free for at least 6 hours after the last dose of naloxone.
 8. Patients at risk of recreational opiate overdose can be prescribed TTO Prenoxad. These are pre-filled syringes containing 2mg naloxone which a patient’s carer or relative can use to treat opiate overdose before the arrival of an ambulance. Please contact the Drug and Alcohol Liaison Team or the Homeless Inclusion Team who can prescribe and dispense this.

OPIOID OVERDOSE IN THE MANAGEMENT OF ACUTE PAIN AND IN PALLIATIVE CARE

Please refer to *Acute Pain* for the management of patients with opioid overdose in these settings.

BENZODIAZEPINES

- Supportive measures, particularly airway maintenance and ventilatory support if required.

- Activated charcoal may be given to patients who have taken more than 1mg/kg within 1 hour, *providing* they are not too drowsy.
- The use of flumazenil is contraindicated in benzodiazepine overdose, and should not be given as a diagnostic test or in a mixed overdose.

TRICYCLIC ANTIDEPRESSANTS

- Correct hypoxia; if hypercarbic, assist ventilation.
- Give activated charcoal (50g) if it is estimated that the patient has taken more than 5mg/kg within the last hour (the dose is similar for the tricyclics generally). A second dose of charcoal (50g) should be considered after 2 hours in patients with central features of toxicity.
- If hypotensive, raise foot of bed and, if necessary, expand intravascular volume.
- Monitor ECG until heart rate < 100 bpm, QRS normal and no conduction defect. Check K⁺. Treat arrhythmias by correcting hypoxia and acidosis
- Treat convulsions with IV diazepam (10-20mg in adults or lorazepam 4mg), and delirium with oral diazepam (may require 20-30mg every 2 hours).
- Indications for NaHCO₃: pH<7.1, QRS>0.6 seconds, or patient has developed arrhythmias, hypotension or seizures. Give 1-2mmol/kg as a bolus then infuse as required.

The target pH is 7.45-7.55.

- If cardiotoxicity is unresponsive to the above consider the use of a lipid emulsion.
- In adults & children: 1.5 mL/kg of 20% Intralipid as an intravenous bolus followed by 0.25- 0.5 mL/kg/min for 30-60 mins to an initial maximum of 500 mL.
- The bolus could be repeated 1-2 times for persistent cardiovascular collapse or asystole.
- The infusion rate should be titrated against clinical response.

CARBON MONOXIDE - Diagnosis

- Sources: inadequately ventilated gas/propane/butane heater/boiler; car exhaust fumes; rarely inhalation of fumes from paint stripper (methylene chloride).
- Early features: headache, nausea, irritability, weakness and tachypnoea, then dizziness, ataxia, agitation, impaired conscious level, respiratory failure, cerebral oedema, metabolic acidosis. Also skin blisters, rhabdomyolysis, acute renal failure, pulmonary oedema, myocardial infarction, retinal haemorrhage, cortical blindness, choreoathetosis, mutism.
- Late features: neuropsychiatric features (including impaired memory, disorientation, apathy, mutism, irritability, impaired concentration, personality change, Parkinsonism, parietal lobe lesions, incontinence, gait disturbance).
- Features of chronic poisoning: headache, nausea, flu-like symptoms.
- Suspect diagnosis if more than one member of household affected.
- Measure carboxyhaemoglobin (heparinised sample) although correlation between blood levels and clinical features is poor; and arterial blood gases (for metabolic acidosis). NB pulse oximetry is unreliable.

Treatment

- Give oxygen at maximum concentration +/- IPPV (via a tight-fitting mask). Treat metabolic acidosis with O₂, avoid IV sodium bicarbonate. Monitor ECG.

- Anticipate cerebral oedema; if necessary give mannitol 1g/kg (as 20% solution over 20 minutes).
- Discuss hyperbaric oxygen treatment with NPIS (tel. 0870 600 6266) if:
 - Unconscious at any time since exposure
 - Carboxyhaemoglobin > 20%
 - Any neuro/psychiatric symptoms (particularly check for cerebellar signs.)
 - CVS complications (including ischaemic ECG)
 - Pregnancy

WHAT TO DO IF THE PATIENT REFUSES TREATMENT

Under common law, treatment can generally only be given where the patient gives consent. Consent can be signalled by word, gesture or in writing.

1) Questions when the patient refuses treatment:

a. Does the patient have the capacity to consent?

- assess patient's capacity to consent and mental illness state
- document assessment in the notes

- ensure these processes are witnessed by a third party e.g. senior nurse
- consider independent second medical opinion and/or psychiatric opinion

In order to give or refuse consent a patient must have the *capacity* to reach such a decision, defined as being able to:

- comprehend and retain treatment information
- believe such information
- use the information and weigh it up to arrive at a choice

Capacity may be affected by:

- state of mind that led to overdose
- drug/poison taken by patient and consequent hypoxia, hypotension
- hypoglycaemia
- stress, fatigue or pain
- psychiatric illness

b. Does the patient have a psychiatric illness?

If in doubt obtain *early* psychiatric opinion

- daytime - liaison psychiatry (Bleep 6501)
- out-of-hours - contact duty psychiatrist via Springfield Switchboard

The treatment options

a. When the patient is judged to lack capacity to consent

- if lack of capacity is judged transient then only give treatment essential to save life
- if lack of capacity is judged permanent then treatment can be given if it is considered to be in the patient's best interest

If either of these situations arise it is important to continue to try to get consent without coercion and to discuss the situation with patient's relatives as appropriate.

b. When the patient has psychiatric illness

The patient may be detainable under the Mental Health Act. If the overdose is considered to be a consequence of a mental disorder, then the patient can be treated medically for the overdose under the Mental Health Act – but only under the direction of the patient’s responsible medical officer – i.e. the psychiatrist taking care of the patient.

c. When the patient is unconscious or medically unwell

If the patient is unconscious or medically unwell, the doctor should treat the patient according to clinical judgement of the patient’s best interest. It is good clinical practice to consult and involve relatives in decision-making, but relative’s consent has no legal standing.

MANAGEMENT OF DECOMPENSATED CHRONIC LIVER DISEASE

Link Consultant: Prof Daniel Forton

Patients with chronic impaired liver function can remain stable (compensated) for many months but can also decompensate rapidly. The commonest causes of acute (rapid) decompensation are hypovolaemia (sometimes secondary to a GI bleed), alcohol, sepsis, drugs and renal impairment. Rapid 'decompensation' may also occur with the development of hepatocellular carcinoma (HCC).

The Decompensated Cirrhosis Care Bundle should be completed within the first 6 hours of admission.

Investigations

Blood Tests

1. FBC
2. Clotting screen
3. Urea, electrolytes, creatinine, glucose
4. Liver function tests, γ GT, albumin
5. α feto-protein (HCC marker)
6. Arterial blood gases if patient has encephalopathy, renal impairment or sepsis
7. Viral screen/autoantibodies/transferrin saturation/copper studies as appropriate to help establish aetiology
8. Septic screen – blood cultures, urine cultures, sputum cultures and ascitic tap

Radiology

1. CXR
2. Early abdominal ultrasound: define the texture of the liver; visualise any liver tumours; define the biliary tree; establish spleen size; look for ascites; and establish the patency of the portal and hepatic veins and hepatic artery.

Management

Ascites

(Remember, treatment may not be needed if the patient is asymptomatic, and if there is renal impairment, accept the presence of ascites).

- Do diagnostic paracentesis (ask for urgent cell count to check for spontaneous bacterial peritonitis (SBP) defined as >250 neutrophils/mm³ or >500 leucocytes/mm³. Send samples for WBC count, culture in blood culture bottles, albumin and cytology as appropriate.
- *Moderate ascites*, if plasma Na⁺ >130 mmol/L and renal function is normal, give spironolactone 100mg plus furosemide 40mg daily.
- Measure weights daily, target weight loss ~ 500 g/day (up to 1kg/day if peripheral oedema)
- Increase dose of both diuretics simultaneously every 3–4 days to achieve target weight loss; maintain a 100:40 ratio up to a maximum of 400mg spironolactone:160mg furosemide. Maximal effect of dose adjustment will be seen in 7 days.
- Do daily U&E. Caution: rapid changes can lead to encephalopathy.
 - If hyponatraemic, (<120 - 125 mmol/L) stop diuretics and restrict Na⁺ to 88mmol (2000mg)/day and fluid to 1-1.5litres/day (arrange with dietician).
 - If hypokalaemic (<3 mmol/L) stop furosemide; if hyperkalaemic (>6 mmol/L) stop spironolactone.
- *Massive ascites* –
 - Seek advice from hepatology team about total paracentesis from Hepatology team (Prof Forton/Drs Clark/Hughes/Barker/Singanayagam/Yalcin).
 - Note:** paracentesis is not usually performed if the patient has SBP.

Infection

If patient's temperature >37.5°C it is important to exclude infection, do:

1. Blood cultures
2. MSU
3. Sputum culture
4. Ascitic tap –send ascites inoculated in culture-medium bottles to increase diagnostic yield

SBP is defined as >250 neutrophils/mm³ or >300-500 lymphocytes/mm³

While awaiting culture results start IV antibiotics as per Microguide

If SBP, add albumin replacement 1.5g/kg of HAS on day 1 and 1g/kg on day 3 to prevent hepato-renal syndrome (20g albumin in 100ml of 20% human albumin).

Jaundice

1. Exclude haemolysis, do conjugated bilirubin and blood film
2. Exclude biliary obstruction
3. If there is a possibility of alcoholic hepatitis, discuss urgently with the Hepatology team with a view to a transjugular liver biopsy and specific therapies.

Coagulopathy

1. Give vitamin K (menadiol sodium phosphate) 10mg IV daily for 3 days. If severe coagulopathy, vitamin K (phytomenadione) can be given IV 10mg *slowly* and, if response is inadequate, repeated every 3 hours, up to a total dose of 40mg in 24 hours.
2. Do not give clotting products unless patient is bleeding
3. Note that moderate coagulopathy is not itself a contraindication to central line insertion or ascitic tap.

Venous Thromboembolism

1. Consider all patients to be at risk of VTE and assess as per '*Guideline for the prophylaxis of venous thromboembolism and peri-procedural management of coagulation status in patients with advanced liver disease*' on the anticoagulation and Hepatology pages on the Intranet.
2. Remember, coagulopathy is not reflective of overall thrombotic risk due to altered levels of anticoagulant factors in cirrhosis

Encephalopathy

1. Give lactulose 20mL tds (titrate dose to achieve at least 2 loose stools/day), via nasogastric tube if necessary
2. Give phosphate enemas bd/tds – especially if not taking oral medication
3. Stop diuretics if plasma Na⁺ <130mmol/L as this increases the risk of encephalopathy
4. Avoid sedatives
5. Consider IV broad spectrum antibiotics:) if no other trigger for encephalopathy is identified. Antibiotics as per trust guidelines for *Sepsis of Unknown Origin*. Avoid nephrotoxic agents eg. aminoglycosides as these may potentiate hepato-renal syndrome
6. If grade 3 or 4 encephalopathy, consider intubating to protect the airway
7. Other causes of reduced Glasgow Coma Scale, eg. sepsis, Wernicke's (give Pabrinex), intracranial bleed (consider CT head)
8. Rifaximin can only be initiated by Hepatology consultant as per trust [Rifaximin Protocol](#)

Renal Impairment

In the context of liver failure, this has a very poor prognosis if not corrected quickly. Hepatology team should be contacted early.

1. Stop diuretics and all nephrotoxic agents including beta blockers
2. Stop NSAIDs; contraindicated in liver failure
3. Catheterise bladder and monitor urine output
4. Check urine sodium

5. Fluid resuscitate if appropriate with 5% HAS or sodium chloride 0.9%. Consider early discussion with critical care for difficult fluid balance. (Aim for MAP >80mmHg to achieve 0.5ml/kg/hr based on dry weight)

6. If adequately fluid resuscitated and still oliguric, start terlipressin 1mg qds: reduce dose in patients with ischaemic heart disease or peripheral vascular disease. If creatinine does not decrease by at least 25% after day 3, dose should be increased stepwise up to maximum 2mg every 4 hours. Terlipressin should be used in combination with 100mL 20% HAS BD, once euvoaemic.

Portal hypertension (defined by the presence of moderate to large varices on endoscopy)

Consult with Consultant Hepatologist prior to initiation of beta-blocker.

See **Grey Book** entry [Bleeding Oesophageal Varices](#) for management of this condition.

Fluid replacement

In decompensated cirrhosis there is generally total body sodium excess, therefore avoid sodium chloride or sodium-containing colloids if possible, unless the patient requires urgent fluid resuscitation, as this will worsen ascites or oedema. If the patient is hyponatraemic ($\text{Na}^+ < 125\text{mmol/l}$) seek specialist advice.

Nutrition

Patients are invariably protein and/or energy malnourished. Feeding should be given enterally, if necessary with a nasogastric tube provided the airway can be protected.

With dietician's advice give:

1. High protein diet (protein restriction is very rarely required unless deep HE)
2. High calorie diet
3. No added salt diet
4. Thiamine replacement (Pabrinex[®] ONE pair IV over 10 mins for 3 doses, then thiamine 100mg po bd for 2 weeks. If signs of Wernicke's Encephalopathy or acute alcohol withdrawal, give Pabrinex[®] TWO pairs TDS for 5-7 days)

Analgesia

Pain is not usually a feature of liver failure. If analgesia needed:

1. Paracetamol is safe in the conventional doses for short term use, reduce dose to 1g TDS (if >50kg) for long-term use. (NB NSAIDS are contra-indicated)
2. Opioids may be used, but may precipitate encephalopathy (less likely with morphine or dihydrocodeine than codeine phosphate). Remember that opioids may accumulate even when given at traditional doses, monitor for constipation and hepatic encephalopathy

Referral to Hepatology team

All patients with decompensated liver disease should be referred to the hepatology team as soon as possible with early escalation to critical care unless there is an end of life plan in place.

They should also be referred if they have:

1. Organ failure in addition to liver disease
2. Hepatocellular carcinoma
3. Variceal haemorrhage
4. Massive ascites, likely to need total paracentesis
5. Hepatic encephalopathy (<12 weeks of onset of jaundice)
6. Incipient renal failure
7. Alcoholic hepatitis
8. Sepsis
9. Viral hepatitis

ACUTE PAINFUL SWOLLEN JOINT(S)

Link Consultant: Professor Nidhi Sofat

A patient with a painful, swollen and (often) stiff joint needs prompt treatment both to relieve discomfort and to prevent permanent damage. Management principally turns on whether symptoms are due to bacteria (septic arthritis), trauma, crystal deposition (gout), blood (haemarthrosis), or are part of a more generalised process such as rheumatoid arthritis. By the end of a careful history and examination it should be possible to make a “working” diagnosis although this will still need confirmation by appropriate investigations.

HISTORY AND EXAMINATION

Ask about time course of symptoms (gout can develop fully over hours, rheumatoid over weeks), assess whether more than one joint is involved (in gout, septic arthritis or haemorrhage the involvement of one joint only is the rule, in a rheumatoid process oligo- or poly-arthritis is more likely), take drug history (thiazides may precipitate gout, arthritis is a recognised part of some drug allergies), ask about recent trauma, check for possible infective source, and look for extra-articular clues such as –

- urethritis (eg in sexually acquired reactive arthritis)
- rash (eg in psoriatic arthritis)
- nodules (eg in RA)
- pyrexia (eg in sepsis)
- pallor (eg in anaemia of chronic disease)
- hepatosplenomegaly (eg in autoimmune rheumatic disease)
- pericarditis/pleurisy (eg in SLE)
- bruising (local trauma, clotting defect)
- diarrhoea (eg in inflammatory bowel disease)

INVESTIGATIONS

Immediate. If an effusion is present aspirate the joint where possible and send sample for *urgent* analysis. Macroscopic appearance coupled with microscopy, gram stain and culture will help confirm (or exclude) infection. Polarised light microscopy should be used to detect crystals of uric acid or pyrophosphate. The exclusion of infection will permit local steroid injection. If aspirate looks infected seek possible bacterial source by taking appropriate culture samples (eg blood, MSU, urethral swab).

Within 24 hours. Take blood for full blood count (to detect increase/decrease in haemoglobin, white cell and platelet numbers), ESR (this may be elevated in an acute phase response, eg inflammation in autoimmune rheumatic disease), and uric acid (this is usually elevated in gout). If a viral cause is suspected screen for viral antibodies (include parvovirus).

Later. Screen for anti-nuclear antibody and rheumatoid factor if you suspect an autoimmune rheumatic disease.

TREATMENT

The joint(s) should be immobilised when inflamed; start rehabilitation as soon as symptoms have resolved. If diagnosis unclear or if septic arthritis is diagnosed, seek advice from the rheumatology team.

Analgesia

Paracetamol	0.5-1g/4-6 hourly
Codeine phosphate	60mg/4 hourly

(Codeine is especially useful where infection is suspected as it does not affect temperature and so allows the response to an antibiotic to be assessed).

Non-Steroidal anti-inflammatory drugs

Ibuprofen	400mg 6-8 hourly.
Naproxen	500 mg bd (12 hourly)

Alternatively, for gout, give

Colchicine 500micrograms/2 hourly (maximum 8 daily)

especially useful where an NSAID is not tolerated or does not work.

(Note: Allopurinol and probenecid should not be started during an acute attack of gout, but should not be stopped if already being taken following a previous attack).

Antibiotics

Current Trust guidelines suggest flucloxacillin as empirical treatment or intravenous vancomycin if patient is Penicillin allergic (this should cover *S. aureus* and other gram +ve cocci). In children below 3 years give amoxicillin or a cephalosporin such as cefotaxime or ceftriaxone (to cover *H. influenzae*). Switch to specific treatment once synovial fluid culture results are known. Do not start an antibiotic until bacterial culture samples have been taken. Do not give the antibiotic by injection into the joint.

Corticosteroids Intra-articular corticosteroids are indicated for significant non-infectious joint inflammation that has not responded to a NSAID within 24 hours. The following drugs can be used: hydrocortisone acetate (25mg); methylprednisolone acetate (40-80mg). Lignocaine (1%) can be added for additional pain relief.

MANAGEMENT OF GIANT CELL ARTERITIS (GCA)

Link Consultant: Dr Arvind Kaul

Section A: Background

Giant Cell Arteritis is part of the spectrum of Large Vessel Vasculitides (LVV) which occurs in older people >50 years. While commonly associated with medium size arteries such as the Temporal arteries (headache) and Ciliary Arteries (blindness), inflammation can extend to large arteries including the cranial vessels, aorta and its thoracic branches.

The complications of GCA include blindness which usually occurs before diagnosis or during the first week after treatment. Other complications include stroke, arterial stenosis, aneurysm, dissection or rupture. GCA is a medical emergency so early diagnosis based on history, examination, investigations and immediate treatment with steroids is essential.

Section B: Making the diagnosis

History

Cranial Symptoms

- Headache, unilateral or bilateral, temporal
- Scalp tenderness/hyperaesthesia
- Jaw claudication (pain on chewing food, relieved by rest, dental causes excluded)
- Temporal Artery tenderness, nodularity or reduced pulsation;
- Indications of tongue or scalp ischemia that may precede necrosis
- Audio-vestibular symptoms (hearing loss, tinnitus)

Visual Symptoms

- Visual manifestations including diplopia, blurring, amaurosis fugax, temporary loss of vision or changes to colour vision;
- **Patients presenting with a history of new visual loss (transient or permanent) or diplopia must be evaluated urgently by an ophthalmologist. Contact Moorfields on emergency extension 6115 or via switchboard to Ophthalmology on-call out of hours.**

Extra-cranial symptoms

- Limb claudication (i.e. pain on exertion, resolves on rest, associated pallor of limbs or reduced perfusion, absent pulses, bruits)
- Polymyalgia Rheumatica symptoms (pain, early morning stiffness of the shoulder and hip girdles)
- Weight loss
- Chest pain
- Constitutional symptoms: fatigue, fever, sweats

Examination:

- **Cardiovascular examination:** Assess bruits, Right and Left arm BP difference, carotidynia, peripheral pulses, examine temporal artery for nodularity or reduced pulsation
- **Respiratory Examination:** signs of infection
- **Abdominal:** assess for aortic aneurysm
- **Neurological:** assess for any neurological deficits and CNS involvement
- **ENT:** look for signs of oral infection, otitis externa, shingles rash
- **Ophthalmological** examination if visual disturbance is reported

Initial Investigations:

- Bloods: FBC, U +E, LFT, CRP, ESR, INR, random glucose
- Urine dip: excludes infection
- CXR: exclude infection, assess for aortic root dilatation
- ECG: if chest pain is reported
- CT Aorta or PET scan: to view distribution of vasculitis if history and signs warrant

Differential Diagnoses

- Neurological headache: Migraines
- Space occupying lesions: tuberculoma, abscess, metastases, intracranial haemorrhage
- Infections: sinus, dental, soft tissue infections, shingles
- ENT conditions including TMJ dysfunction
- Chronic pain conditions e.g. Fibromyalgia

Section C: the GCA referral pathway

If GCA is felt likely, follow the 5-step pathway below -

1. Refer

- **Referrals from inpatient ward teams:**
Refer directly to Rheumatology on-call service bleep 7787 (Mon-Friday 9-5 pm)
Follow pathway below
- **Referrals from GP/A&E/AMU between Mon-Friday 9-5 pm:** Refer to Acute Medicine Team for assessment and investigations Follow pathway below

2. Investigate

Both tests below must be organised and will increase the chance of an accurate diagnosis

- **Temporal artery biopsy**
Email Vascular Surgeons co-ordinator with history summary including relevant medications:
Andrea Browne, andrea.browne@st.georges.nhs.uk
Christine Brown, christine.brown@stgeorges.nhs.uk
Call 4984 or 4312 on Mon-Fri 9-5 pm if needed

- **Temporal artery ultrasound to include Axillary artery if possible** Order on iClip under ‘VL US Temporal Artery Both’.
Do NOT request under general ultrasound.
If Mon-Fri 9-5 pm the Vascular lab can be contacted directly on 2151 to discuss if needed.

3. Treat

Start therapy:

- Assess severity

Standard Presentation

Prednisolone 40-60mg PO daily

Severe presentation

IV Methylprednisolone 250-500mg OD, for 3 days consecutive days if severe presentation

(blindness, transient visual loss, stroke, stenosis, aneurysm)

Assess history of comorbidities and medications that could predispose to glucocorticoidrelated adverse effects, including:

Active infection

Hypertension

Diabetes

Osteoporosis

Psychiatric history

- Co-Prescribe Omeprazole 20mg once daily or alternative
- Start Adcal D3 TT once daily
- Counsel to return to ED immediately if visual symptoms occur or become clinically unwell

4. Educate

- Patient information: Versus Arthritis Leaflet on GCA
<https://www.versusarthritis.org/about-arthritis/conditions/giant-cell-arteritis-gca/>
- For doctors: British Society for Rheumatology
Guideline for the Management of Giant Cell Arteritis,
Mackie et al, Rheumatology 59: 487-494 (2020)

5. Follow up

- Discuss with Rheumatology Consultant/SpR on-call (Bleep 7787) Mon-Friday 9-5 pm to organise early OP follow in weekly Hot Clinic (Friday PM)

ACUTE KIDNEY INJURY (AKI) **Link Consultant: Dr Debasish Banerjee**

Acute kidney injury (AKI), characterised by a sudden rise in blood creatinine and due to fall in glomerular filtration rate (GFR), is common (15-20%) in hospital. Causes include hypovolaemia (surgery, haemorrhage, burns), sepsis or nephrotoxic insult (eg drugs, iv contrast media, myoglobinaemia or haemo-globinaemia). Other less common causes of AKI are obstruction, acute interstitial nephritis (due to drugs or infection), and glomerulonephritis, primary or secondary (such as due to lupus)

Stages of AKI: There are 3 stages of AKI with increasing severity:

Stage 1: Creatinine rise 1.5-2 fold from baseline or $\geq 26.4 \mu\text{mol/L}$ and/or urine $\leq 0.5 \text{ml/kg/hr} > 6 \text{h}$

Stage 2: Creatinine rise 2-3 fold from baseline and/or urine $\leq 0.5 \text{ml/kg/hr} > 12 \text{h}$

Stage 3: Creatinine rise > 3 fold from baseline or $\geq 354 \mu\text{mol/L}$ and/or urine $\leq 0.3 \text{ml/kg/hr} > 24 \text{h}$

Management : see figure below

1. Fluid management: Assess fluid status with monitoring of pulse, BP, JVP and urine output. CVP monitoring is useful only in ITU and HDU. Correct hypovolaemia using 0.9% saline, ideally in boluses of 250 ml and continuous monitoring of fluid status. If urine output remains low after 2 litres of fluid seek expert advice. If patient remains hypotensive (SBP <100 mm Hg) after fluid therapy seek ITU advice. Once patient is adequately volume resuscitated maintain fluid intake at a rate of urine output + 30 ml/hr.
2. Treat sepsis with appropriate antibiotics.
3. Stop all nephrotoxic drugs like ACE inhibitor (ACEI), Angiotensin Receptor Blocker (ARB), NSAIDs, aminoglycosides.

Figure 1: Steps in diagnosis and management of AKI

DIAGNOSIS: Creatinine rise ($26 \mu\text{mol/L}$) or > 1.5 times from baseline ($> \times 3$ - SEVERE AKI)
Check Pulse, BP, Temperature, Respiratory rate, Urine output (U/O)
Maintain airway, breathing and circulation, administer oxygen and contact ITU if necessary

REHYDRATE if volume depleted using Normal Saline (0.9%) 250 ml boluses upto 2 L
Monitor Pulse/BP/Temperature/Respiration and hourly urine (urine catheter if necessary)

INVESTIGATE: *Urine* for protein, blood, leucocyte, microscopy&culture, electrolytes
Blood: U&E, FBC, LFT, Arterial Blood Gas, Calcium, Immune and Myeloma screen
Radiology: Ultrasound scan of Kidneys, Ureters and Bladder

STOP Nephrotoxic medications: NSAIDs, ACEI, ARB. **ADJUST** drug doses
SEPSIS: If sepsis: Start antibiotics, avoid gentamicin

REFER Renal registrar on bleep 6415 or ext 1080; if uncontrolled hyperkalaemia (> 6.0), acidosis (pH < 7.2), fluid overload +/- anuria, significant haematuria proteinuria, low Hb, or AKI 3

4. Treat hyperkalaemia (K^+ greater than 6mmol/L) - *see* management of [Hyperkalaemia](#).
5. Order urgent ultrasound scan (if no other obvious cause is found) and relieve obstruction if present (using catheter and urology advice). Order ANA, ANCA, anti-GBM antibodies, complements, serum electrophoresis, urine Bence Jones Protein – if haematuria and proteinuria present; LDH, bilirubin, retics, CK if necessary.
6. Diuretics can help to reduce fluid overload.

If dehydrated and urine sodium is <10mmol/L or fractional excretion of sodium <1% think of prerenal failure and administer adequate fluids.

Renal biopsy should be considered if there are atypical clinical features, haemo-proteinuria or features suggesting multisystem disease.

Indications for dialysis in patients with AKI are:

- Life-threatening or intractable pulmonary oedema
- Uncontrollably rising K^+
- Severe ($pH < 7.2$) or worsening acidosis

Prevention: AKI can often be prevented. So, for example, take special care to avoid volume depletion in high-risk patients (eg. those with diabetes, myeloma, or established renal failure), and those subjected to overnight fast, surgery or investigations involving iv contrast. Hypovolaemia due to blood or fluid loss should be avoidable or rapidly reversible. Be very cautious when using drugs such as aminoglycosides, vancomycin and NSAIDs that might cause AKI.

For patients due to receive radiocontrast, the following should be implemented to reduce the risk of AKI due to contrast nephropathy (see figure 2):

Prevention of AKI due to radiocontrast nephropathy:

Identify high risk patients with CKD, Diabetes, Age>65, Heart failure and use IV Fluids 0.9% saline 1ml/kg/hr, 3-12 hours before and 6-12 hours after procedure

(to maintain urine output 150ml/hr) Sodium bicarbonate can also be used
Stop potential nephrotoxic agents eg NSAIDs, ACEi, ARB and Diuretics

Guidelines for management are available at <http://www.londonaki.net>, <http://publications.nice.org.uk> or St Georges intranet – the renal home page

ELECTROLYTE DISTURBANCES

Link Consultant: Dr Joyce Popoola

HYPOKALAEMIA

Low serum potassium can cause muscle weakness (leading to paralysis), cardiac arrhythmias, and in susceptible patients hepatic encephalopathy. It can also potentiate the unwanted cardiac effects of digoxin and of drugs that prolong the QT interval.

Indication for treatment. In general, potassium supplements should be given to any patients with a serum potassium < 3 mmol/L, or < 3.5 mmol/L if they are taking a drug that has arrhythmic side effects enhanced by low potassium or who have cardiac disease. An exception should be made for patients with renal failure. Hypokalaemia commonly occurs immediately after haemodialysis and is usually transient and self-correcting. Hypokalaemia in those with end-stage renal failure or after dialysis, is complex and supplements should not be given without first discussing with the renal team.

Causes. Low K^+ is commonly secondary to increased losses (vomiting, diarrhoea, thiazides, loop diuretics, corticosteroids). It can also be due to alkalosis (including loss of hydrogen ions through persistent vomiting), beta-blockers, xanthines and insulin, all of which cause potassium to enter cells rather than cause overall deficit.

Treatment. Remember, a plasma K^+ of 3 mmol/L secondary to potassium loss represents a total deficit of around 300 mmol (2 mmol/L– 600 mmol). If possible, and if there is time, first treat the cause. Replacement can be by mouth or by intravenous infusion.

- Oral replacement is preferable – it is certainly safest. Sando-K (12 mmol/tablet) is the first choice; Slow K (8 mmol/tablet) should be reserved for those unable to tolerate Sando-K. The usual dose is 40-120 mmol/day. The maximum daily dose is 300 mmol.
- Intravenous replacement should be reserved for those:
 - i. with symptoms (paralysis, arrhythmia, hepatic encephalopathy).
 - ii. in whom the K^+ is below 2.5 mmol/L.
 - iii. intolerant of oral K^+ .

Infuse potassium into a large vein at up to 20 mmol K^+ /hr (not more than 200 mmol/day). If plasma $K^+ < 2$ mmol/L with arrhythmia, 40 mmol K^+ may be given over 1h. Bags for IV potassium infusion are available through Pharmacy.

NB. The risk of thrombophlebitis from infusion of solutions via peripheral veins should be weighed against concern that central K^+ infusion might worsen cardiac arrhythmia. Remember that the risks of iatrogenic hyperkalaemia are potentially more serious than those of hypokalaemia.

Monitoring. Measure serum potassium at frequent intervals. Continuous trace of cardiac rhythm. Check serum creatinine (expect more rapid rate of rise of K^+ in patients with renal failure).

HYPERKALAEMIA

Serious clinical manifestations associated with raised serum potassium (K^+) include cardiac arrhythmias, asystole, muscle weakness and paralysis.

Treatment Indications: Attempts should be made to lower if serum $K^+ > 6$ mmol/L.

Thresholds maybe higher in dialysis or chronic kidney disease (CKD), but always contact the renal unit as specialist input is needed. Exclude causes of pseudo-hyperkalaemia *e.g.* haemolysis, delayed transit to laboratory. If unexpected, repeat sample but do not delay treatment.

Causes of increased Potassium concentrations:

a) **Reduced renal excretion** e.g. renal failure, Potassium-sparing diuretics, ACE inhibitors/Angiotensin-II receptor blockers, NSAIDS, Addison's disease.

b) **Potassium leaving cells** e.g. acidosis, diabetic hyperglycaemia, cell damage (trauma, rhabdomyolysis, burns, haemolysis). Remember with K^+ movement between body compartments, total body K^+ may be low or normal, measuring arterial pH, pCO_2 and pO_2 help differentiate.

Acute Management of Hyperkalaemia

- If the ECG is abnormal, give 10 mL of 10% calcium gluconate intravenously (IV) slowly (maximum rate 2 mL/min), repeat dose if necessary until ECG normalises (maximum dose 40 mL), ideally with cardiac monitoring.
- To move potassium into cells give glucose 25g in 50mls (50% glucose) with 10 units of soluble human Insulin (Dextrose/Insulin infusion), over 15 mins.
- ***In addition, give 10% glucose by infusion @50mls/hour for 5 hours for patients with pre-treatment blood glucose <7.0mmol/l to avoid risk of hypoglycaemia.
- Check blood glucose every 30 minutes for 2 hours then hourly. Blood glucose monitoring is required up to 12 hours after glucose insulin infusion.
- If hyperkalaemia persists, repeat infusion and involve renal registrar.
- The renal team should be involved in hyperkalaemic cardiac arrests.

- **Measure Bicarbonate & Arterial pH**

Measurement of bicarbonate & arterial pH is **essential**. to guide treatment:

a) **Mild to moderate acidosis (pH 7.1-7.3)**. If patient is not fluid overloaded, use 500 mL 1.4% $NaHCO_3$ over 2-3 hours then recheck bicarbonate.

b) **Severe acidosis (arterial pH <7.1)**. Use 500mL 1.4% $NaHCO_3$ in 1 hour; (volume overload or cardiac arrest, use 50mL 8.4% $NaHCO_3$ slowly *via* a large vein).

- Sodium bicarbonate should not be given in Type 2 respiratory failure or hypocalcaemia (*adjusted* calcium < 2.0mmol/L).
- Consider use of 10-20 mg nebulised salbutamol. This is not mono-therapy (no response in 40% of patients). Caution in patients with tachycardia or ischaemic heart disease.
- An oral potassium binder, Sodium Zirconium Cyclosilicate (*Lokelma*) 10g three times a day for up to 72 hours may be useful. Discuss with renal team.
- Once acute episode managed check serum K^+ concentration at least twice daily, then once daily once K^+ is < 6.0 mmol/L.
- Remember to stop all potassium-retaining/containing drugs if possible, ensure dietary review of dietary potassium. Chronic use of potassium-binders requires specialist input.

Renal Registrar bleep number 6415

References include: Renal Association Guidelines on Treatment of Acute Hyperkalaemia in Adults, Hyperkalaemia Cardiac Arrest (update in process 2019/2020

HYPOCALCAEMIA

The most prominent feature of low plasma concentrations of calcium is increased neuromuscular activity with paraesthesia, then leading to muscle cramps, carpo-pedal spasm, laryngeal stridor and convulsions. These effects are determined by the concentration of ionised calcium and are influenced by plasma pH (available calcium concentration falls the more alkaline the plasma).

Indications for treatment. Attempts to raise the available calcium should be made if the plasma ‘adjusted’ calcium is below 1.8 mmol/L or the patient has unequivocal signs of hypocalcaemia with a low calcium, *ie* tetany, positive Chvostek or Trousseau’s sign, or seizures.

Calcium levels should always be corrected against albumin. To calculate ‘adjusted’ calcium: adjusted calcium (mmol/L) = unadjusted calcium (mmol/L) + 0.02 x (40 – serum albumin (g/L)).

Causes. While alkalosis increases the likelihood of symptoms and signs, and occasionally (*eg* prolonged hyperventilation) is the sole cause of the clinical picture, other causes include primary hypoparathyroidism, renal failure, vitamin D deficiency and malabsorption. A low plasma Mg^{2+} can also cause hypocalcaemia without any change in total body calcium. Measure magnesium if in doubt – hypomagnesaemic hypocalcaemia should be treated with intravenous magnesium alone. Seek specialist advice.

Treatment. Supplements can be given either by mouth or intravenously.

- Oral route. Give 12.5g of $CaCO_3$ (5g of elemental Ca) over 24h not with food. One CalciChew tablet contains 0.5g of elemental Ca. Alfacalcidol should be given in a dose of 1-5micrograms daily.
- Intravenous infusion. Give 10mL of 10% calcium gluconate ($2.2\text{mmol } Ca^{2+}$), no faster than 2mL/min. The effect is short-lasting so the infusion should be followed by iv calcium gluconate 10%, 40mL (in 500mL 0.9% NaCl or 5% dextrose) over 24h; this will provide 8.8 mmol of Ca^{2+} . Measure Ca^{2+} concentration 3-4 times daily until serum Ca^{2+} is within the normal range, adjusting the infusion rate as appropriate.

HYPERCALCAEMIA

An elevated serum calcium concentration may be asymptomatic or cause symptoms such as thirst, polyuria, nausea, vomiting, constipation, abdominal pain, confusion or coma. If malignant hypercalcaemia is suspected specialist advice should be sought from the Acute Oncology or Palliative Care Teams.

Indications for treatment.

Attempt to lower the serum calcium in anyone with an ‘adjusted’ serum calcium of > 3 mmol/L unless the value is stable and the patient is completely asymptomatic. In patients with malignancy treat calcium > 3 mmol/L even if minimal symptoms and consider treatment in hypercalcaemia $< 3\text{mmol/L}$ if symptomatic. (For calculation of adjusted calcium see section on *Hypocalcaemia*). Patients with hypercalcaemia are usually volume deplete, and this should be corrected.

Causes.

Hypercalcaemia can occur as a result of reduced excretion, increased absorption or a shift of calcium between body compartment

- Common causes are: malignant disease (can occur without bone metastases), primary hyperparathyroidism, thiazide diuretics, vitamin D intoxication, calcium-containing drugs

- Less common causes include: sarcoidosis, tuberculosis, thyrotoxicosis, cortisol deficiency, Pheochromocytomas, VIPomas, immobilisation, recovery phase of AKI, post renal transplant, Lithium, Vitamin A.

Treatment.

- First record the patient’s weight.
- Stop drugs known to cause hypercalcaemia.
- Give 0.9% NaCl to render the patient euvolaemic, aiming to increase urine volume to 200 mL/hr. May require >4 litre in 24 hours. Cautious rehydration in patients at risk of CCF
- Only give Furosemide (40-80 mg orally or IV) once hypovolaemia is corrected to increase urine flow and calciuresis it is essential that the patient is not rendered hypovolaemic.

Diuretics should not be given routinely in cases of malignant hypercalcaemia.

If the serum calcium is still raised after 24 hours a bisphosphonate can be given following consultation with a specialist as the choice and dosing of the bisphosphonate will vary according to the aetiology.

- Renal failure patients particularly those on renal replacement therapy may have hypercalcaemia associated with tertiary hyperparathyroidism. Specialist advice should always be sought prior to intervening as bisphosphonates may not be appropriate in this situation.
- In particular, patients with hypercalcaemia and known metastatic disease should be given IV Zoledronic as in (a) below:

In patients with hypercalcaemia and known malignant disease

a) Give IV Zoledronic acid over 15 minutes. **Recheck serum calcium Day 5 after treatment. If persistent hypercalcaemia, seek specialist advice regarding repeat dosing of zoledronic acid.**

Maximum frequency of maintenance IV Zoledronic dose is 3-4 weekly.

NB. Doses should be adjusted if eGFR is reduced, as below:

eGFR (mL/min/1.73m ²)	>60	50-59	40-49	30-39	<30
Dose of IV Zoledronic acid	4mg	3.5mg	3.3mg	3.0mg	Do not use*

*** If eGFR < 30 mL/min, then Denosumab 120mg SC can be given following specialist advice.**

Or, (in absence of known malignant disease)

b) Give IV Pamidronate over 2-3 hours in a dose of 15-90 mg dissolved in 500 mL 0.9% NaCl. IV Pamidronate should not be used for patients with renal impairment and eGFR < 30 mL/min* but may be used in patients with an eGFR between 30-59 mL/min at rate of infusion not exceeding 20 mg/hr. The serum calcium should fall within 24-48 hours with the maximum response taking 4-5 days. Further doses of Pamidronate should not be given within this period. If the plasma calcium remains elevated, seek help.

Dose should be adjusted for serum calcium as below:

i) Doses of IV Pamidronate in patients with eGFR ≥ 60 mL/min

Serum calcium	Up to 3 mmol/L	3-3.4 mmol/L	3.5-3.9 mmol/L	≥ 4 mmol/L
Dose of IV Pamidronate over 2-3 hrs	15-30 mg	30-60 mg	60-90 mg	Mg

ii) Doses of IV Pamidronate in patients with renal impairment and eGFR < 60 mL/min

	In patients with eGFR between 30-59 mL/min		In patient with eGFR < 30 mL/min*	
Serum calcium (mmo/l)	< 4 mmol/L	≥ 4 mmol/L	< 4 mmol/L	≥ 4 mmol/L
Dose of IV Pamidronate	40 mg	60 mg	Do not give	
Rate of infusion	< 20 mg/hr		n/a	n/a

HYPONATRAEMIA

Hyponatraemia ($\text{Na}^+ < 135 \text{ mmol/L}$) results from H_2O retention, Na^+ loss or a combination of the two. Although the definition of hyponatraemia is $\text{Na}^+ < 135 \text{ mmol/L}$, it is only clinically significant if the sodium concentration is $< 125 \text{ mmol/L}$, or has fallen rapidly ($> 20 \text{ mmol/L}$ in 24 hours). Hyponatraemia can lead to shift of H_2O into cells, with cell swelling ie. cerebral oedema. The concentration of plasma sodium does not give any indication of volume status, ie. hyponatraemic patients can be fluid-overloaded, euvoalaemic or volume deplete.

Hyponatraemia is usually asymptomatic. The causes include:

- renal loss of Na^+ (caused by, for example, diuretics, tubular disorder)
- gain of H_2O due to
 - Vasopressin (ADH) release in response to intravascular hypovolaemia, nausea or pain.
 - Syndrome of inappropriate ADH secretion (SIADH).
 - Excessive water intake (as with, for example, Dextrose 5% infusion, water irrigation after trans-urethral prostatectomy (TURP), compulsive drinking).

Hyponatraemia is usually associated with hypo-osmolality (plasma osmolality $< 275 \text{ mmol/kg}$). The combination of hyponatraemia and normal or elevated plasma osmolality indicates the presence of an additional, osmotically active, substance (eg. glucose, mannitol.).

Clinical assessment

- Confirm plasma sodium below 135 mmol/L
- Measure urinary sodium concentration
- Measure plasma osmolality and assess volume status;
 - If osmolality greater than 275 mmol/kg , assume the problem is hyper-glycaemia or renal failure and treat as such
 - If osmolality less than 275 mosmol/kg , then treatment will depend on whether the patient is:
 - hypovolaemic (*causes*: diuretics, vomiting, diarrhoea, cortisol deficiency)
 - euvoalaemic (*causes*: diuretics, hypothyroidism, primary polydipsia, cortisol deficiency, SIADH or irrigation with glycine or sorbitol during TURP, pregnancy, drugs such as ecstasy, chlorpropamide, exercise)
 - hypervolaemic (*causes*: congestive cardiac failure, renal failure, conditions associated with hypoalbuminaemia)

Calculation of serum osmolality (s. Osm.):

s. Osm. (mmol/kg) = $2 \times s. [Na] + \text{glucose} + \text{urea}$. (All units are mmol/L)

Note calculating the serum osmolality has limitations so ideally a sample should be sent to the laboratory for precise assessment.

Therapy

Principles:

- i) Treatment of underlying disease where possible
- ii) Initial therapy to raise the serum sodium
- iii) Prolonged therapy in patients with persistent SIADHiv) Treatment should raise serum Na^+ by no more than 8mmol/L in 24hrs and be within 24hrs.

Clinical management depends on type of hyponatraemia:

a) Hypovolaemic hyponatraemia:

- Give IV 0.9% NaCl
- Stop diuretics
- Give anti-emetics if necessary

The amount of Na^+ required in hypovolaemic hyponatraemia is determined as follows:

Na^+ requirement (mmol) = $0.6 \times \text{body weight in kg} \times (\text{desired } Na^+ - \text{actual } Na^+)$

Calculate volume of 0.9% saline (150 mmol/L) to be given over 24 hr from this formula.

b) Euvolaemic hyponatraemia:

- Restrict fluid to 1L/day
- Stop diuretics
- Give liothyronine or L-thyroxine if hypothyroid
- Replace corticosteroid if deficient
- Consider oral sodium
- Consider demeclocycline 300 mg – 600 mg bd if no response to fluid restriction

IV Conivaptan can be considered following discussion with the renal team; appropriate adjustments for liver or renal impairment must be made.

c) Hypervolaemic hyponatraemia:

Restrict fluid to 1L/day

- Restrict sodium intake
- Give diuretic as necessary
- Replace K^+ loss
- Treat underlying disease

Hypertonic saline should be reserved for patients with seizures or other life-threatening neurological complications of hyponatraemia. In such cases contact the ICU staff and discuss further management.

HYPERNATRAEMIA

Hypernatraemia is defined as serum sodium concentration $> 145\text{mmol/L}$, but is usually only clinically significant if the concentration is $> 155\text{mmol/L}$, or there has been a rapid rise ($>20\text{mmol/L}$ in 24hrs). The symptoms of hypernatraemia range from mild confusion to coma, and can occasionally be associated with intracerebral or subarachnoid haemorrhage.

Hypernatraemia is almost always due to H₂O loss (deficiency) rather than to Na⁺ gain (excess). It is important to determine whether acute or chronic loss when assessing cause(s).

Causes include:

- H₂O loss without adequate H₂O intake
- Diuretics/Laxative abuse
- Osmotic diuresis (e.g. hyperglycaemia: beware pseudohyponatremia)
- Na⁺ gain (ingestion of sea water, infusion of large volumes of intravenous NaHCO₃ 8.4%)
- Central Diabetes insipidus (DI) e.g. brain/pituitary surgery or brain injuries
- Nephrogenic DI - inherited or acquired e.g Lithium, Demeclocycline, Vaptans

Management

1. Stop H₂O loss. Depending on the cause this may involve giving an anti-emetic for vomiting, stopping diuretics or treating diarrhoea.

2. Calculate the H₂O deficit, where

$$H_2O \text{ deficit(L)} = \text{body weight in kg} \times 0.6 \times \frac{(\text{actual Na}^+ (\text{mmol/L}) - 140)}{140}$$

3. Encourage enteral replacement orally or via NG tube if difficulty with swallow. Aim to replace 1/3 of the water deficit in addition to usual fluid maintenance in the first 24 hours.

4. If IV hydration is required in patients with chronic hypernatraemia, give IV 5% Dextrose at a rate of 1.35 mL/hour x patient's weight in kg, up to a maximum rate of 150 mL/hr.

This is approximately: 70 mL/hr for a 50kg patient
 100 mL/hr for a 70kg patient

Note 5% dextrose/0.18% sodium chloride contains 30 mmol/L of Na⁺, while 0.9% sodium chloride contains 150 mmol/L.

5. Check serum Na⁺ 12 hourly in first 24 hrs then daily; it should not fall by > 8-10 mmol/L in 24 hrs to avoid cerebral oedema.

NB If hypernatraemic is due to acute water loss, senior help should be sought immediately. Acute water loss should be managed in the **High Dependency Unit**. Correction should be with 5% Dextrose at a rate 3-6 mL/kg/hr with monitoring of plasma sodium every 1-2 hours. Aim to correct sodium in 24-48 hours. Hypernatraemia in neonates and young children also requires early senior input.

If hypernatraemia is due to central or nephrogenic DI, specialist input should be sought early.

SICKLE CELL CRISES

Link Consultant: Dr Julia Sikorska

At least 500 patients with sickle cell diseases (HbSS, HbSC, HbSBthal) live in the St George's catchment area, with St George's having around 250 admissions a year due to sickle cell. In-taking teams can expect to see >100 crises/year. Many patients have a personal management protocol which is kept in a file in their name in A&E Majors. Copies of patient protocols are also on EPR under electronic documents and should be consulted for advice on prompt initial treatment, since it may differ in important details from the generic advice given below.

PAIN CRISIS

The most common type of crisis presents as agonising and relentless pain. The pain may be localised to a single long bone, present symmetrically in several limbs, or involve the axial skeleton (lumbar spine, ribs or pelvis). Pain can lead to behavioural changes including becoming non-communicative or occasionally panicked and aggressive. If pain is bad enough to bring the patient to hospital, the patient usually warrants admission. Patients will often have tried a variety of analgesics at home including some form of opiate.

In the Accident and Emergency Department there are ED guidelines available:

<https://www.stgeorges.nhs.uk/wp-content/uploads/2020/03/ED-Guideline-Management-of-Acute-Sickle-Cell-Crisis.pdf>

Achieving fast and adequate pain control is the priority.

- Patients with sickle cell disease should be triaged as urgent.
- Nurse assessment with vital sign observations
- If pain crisis: administer analgesia as per protocol if one is available, or as per ED guidelines
- ***Analgesia must be given within 30 mins***
- Assess pain every 30 mins until adequate pain relief has been achieved and give a second dose at 30 mins if needed
- For parenteral analgesia the subcutaneous route is preferred to intramuscular (to preserve muscles)
- If no protocol and requiring parenteral opiate then 0.1mg/kg morphine sc is an appropriate starting dose. This can be repeated at 20min intervals until pain control achieved
- Pethidine is not used at St George's Hospital any longer for sickle crises - it is a cerebral irritant which can cause seizures and has poor bioavailability
- If patients from elsewhere request pethidine please discuss with haematology SpR
- Morphine alternatives include oxycodone and hydromorphone
- Entonox should not be used after leaving the ambulance due to risk of irreversible neuropathy
- Adjuvant analgesics include paracetamol and NSAIDs (as long as no evidence of nephropathy)
- Please ensure laxatives, antiemetics and antipruritics are co-prescribed. Ensure regular review of SpO₂s and respiratory rate in patients needing opioid analgesia.

Supplementary management

- Oxygen, keeping oxygen saturations above 94%
- IV fluids if not orally maintaining adequate hydration
- Aim for 1 – 1.5 x maintenance volume once volume depletion corrected
- Broad spectrum antibiotics if signs of infection (Co-amoxiclav with Clarithromycin or Levofloxacin alone if Penicillin allergic. (People with sickle cell disease are effectively asplenic and therefore susceptible to infection with encapsulated organisms such as Streptococcus pneumoniae and Haemophilus influenzae B). Antibiotics can be given orally unless clinical concern warrants intravenous administration.

- Liaise with haematology SpR and, if in hours, the Sickle Cell Clinical Nurse Specialist for admissions.
- Medical assessment for complications requiring specific/urgent intervention and treatment (*see below for details of life threatening crises*).
- Full history and examination (focussing on chest, abdomen and CNS).
- Regular assessment of vital signs:
Hourly observations to monitor pain, sedation, vital signs, respiratory rate and oxygen saturation for the first 6 hours and 4-hourly thereafter until they leave hospital or the episode has ended. Staff should be alert to potential risk of opiate toxicity and act on any concerning observations.
- Blood samples for FBC, reticulocyte count, renal and liver profile, group and save.
- Blood cultures if suspicion of infection.
- CXR if chest signs / symptoms.
- There is usually no need to X-ray painful bones in a simple pain crisis
- Blood transfusions are usually not indicated and should only be considered after discussion with haematology SpR

Admission

If the patient is to be admitted (most cases) immediately contact the Bed Manager and advise the Haematology team. No patient admitted with sickle cell crisis should be placed on a ward outside the Medical Service Centre - there are specific cohorted sickle beds. After admission to the ward continue 2 hourly SC Morphine or analgesia regimen prescribed. Give at the dosage indicated on the patient's personal protocol if available.

The patient should wait no more than 4 hours in A&E. During this wait and if delayed longer please ensure that:

- i). the analgesia regimen is followed.
- ii). the patient has fluid input maintained.
- iii). the patient has antibiotic regimen maintained.
- iv). the patient is observed regularly to ensure all vital signs are maintained and pain levels assessed.

If a patient is discharged from, or leaves A&E, then:

- Contact the haemoglobinopathy specialist nurse (SGH blp 7520; or via Balham Health Centre on 0208 700 0615 if community-based) and give details of the admission and assessment.
- Give the patient sufficient analgesia to ensure effective pain management until the patient may see their GP or a specialist nurse counsellor.

LIFE-THREATENING CRISIS

Patients can present with a variety of other acute manifestations which may be rapidly fatal if not recognised and treated quickly.

Infection: Patients prone to sickling have reduced splenic function and are at risk of overwhelming septicaemia (pneumococcus, meningococcus, rarely haemophilus) even if taking Penicillin prophylaxis. Peak risk is in childhood. The patient may present with fever, shock, seizures, coma, meningitis (often with delayed CSF pleocytosis) or even profuse diarrhoea. Early IV antibiotics to cover pneumococcus and staphylococcus (Co-amoxiclav and clarithromycin, or if Penicillin allergic then levofloxacin alone) and volume support are vital. If osteomyelitis suspected, discuss with Microbiology.

Splenic or Liver Sequestration: During infection children may suffer a rapid fall in haemoglobin and growth of the spleen – changes often noted by the mother. Death can result from hypovolaemia and anaemia. Early transfusion is vital. In adults, liver sequestration is more common and can present similarly with profound anaemia and hepatomegaly. Transfusion is often required in these patients as well.

Chest crisis: Severe shunting & hypoxia caused by intra-pulmonary sickling and mimicking pulmonary embolus/pneumonia, may start in one lobe and then spread to others. It sometimes begins as a pain crisis affecting ribs or shoulders. Treat with fluids and oxygen; observe arterial O₂ tensions – a falling PaO₂ will require exchange transfusion and needs expert advice. Encourage patients with chest pain to attempt one maximal inhalation every 5-10 mins ('incentive spirometry') to aerate basal lung segments; this reduces the risk of progressive sickle chest syndrome. Non invasive respiratory support may well be required, as well as urgent exchange transfusion. Discuss with haematology urgently.

Girdle syndrome: If sickling occurs in the splanchnic bed, abdominal pain with rigidity, loss of bowel sounds and increasing icterus may develop. IV fluids are vital. A surgeon should be consulted to exclude other abdominal events, but surgery should be withheld unless unavoidable, and then only after exchange transfusion and discussion with haematologists.

Cerebral sickling: Patients can present with strokes, fits, coma, bizarre behaviour or psychosis, and sickling should be excluded in any susceptible patient with such signs. IV fluids are vital and early exchange transfusion a possibility. Patients are at risk of both haemorrhagic and ischaemic strokes.

Priapism: Priapism typically affects only the corpora cavernosa. Major or prolonged attacks post puberty can result in permanent loss of erectile function. Urgent referral to Urology is essential as early decompression can be achieved by aspiration +/- intracavernosal Phenylephrine.

Blood transfusion: In a patient with Sickle Cell Disease blood transfusion can be dangerous. Never give a simple transfusion for anaemia (except in those sequestering), without reducing HbS level by exchange. If this precaution is not taken the blood viscosity will increase and make the patient worse. Consider if Hb < 6 g/dl or if there has been a 2 g/dl fall from steady state. Get haematological advice and ensure that the blood transfusion department knows that the patient due to receive blood has sickle cell, so that appropriately phenotyped blood can be provided.

Surgery: Do not plan or carry out surgery without first assessing the patient with the Haematology Team. Special pre- and post-operative care, often including blood exchange, is essential to optimise outcome.

Acute renal failure: In sickle cell disease this is most commonly multifactorial with causes including dehydration, sepsis, nephrotoxic drugs (especially NSAIDs) as well as acute papillary necrosis. Urine dipstick for haematuria is important, as is a MSU to exclude infection and these patients should be discussed with the nephrology team as well as haematology. Intravenous fluid replacement is important (minimum of 3 L/24 hours) and ensure nephrotoxic drugs are withheld.

MANAGING ACUTELY AGITATED PATIENTS

Link Consultant: Runa Patel-Kumar & Dr Marcus Hughes*

STEP 1: Non-drug approaches to de-escalate the situation

A. Identify and treat causes of agitation

The following should be considered:

- Underlying diagnosis: delirium, dementia, acute mental illness, acutely distressed state or learning disability
 - Pathophysiological factors: infection, medication, electrolyte disturbance, alcohol or drug withdrawal, hypoxia, CNS disease, all causes of encephalopathy, hypoglycaemia, epilepsy, head injury and poisoning
 - Situational factors - establish reasons for acute agitation from patient or relative if possible
- An agitated patient must have a senior medical review, preferably by a consultant physician or medical registrar within one hour**

B. Optimise the patient's environment

- Ensure patient resides in a calm, quiet, appropriately lit environment. Aim for continuity of care, minimising 'new faces'
- Encourage presence of family or friends and enquire if there are any known triggers that cause distress or actions that can give reassurance
- Consider one-to-one nursing or Registered Mental Health Nurse special if the patient is very distressed
- Use frequent reorientation to place and reason for admission if confused
- Contact relevant specialist hospital team depending on initial diagnostic assessment – Liaison Psychiatry/ Learning Disability/ Drug and Alcohol Liaison teams

C. Observation

- Monitor patient using Early Warning Score (EWS) by a registered nurse. Inform nurse in charge, seek medical review and increase frequency of observations if EWS score increases.
- When patients are acutely agitated it may be very difficult to record vital signs such as BP. In these situations, the patient's physiological status must be assessed using A-E clinical assessment (Airway, Breathing, Circulation, Disability, Exposure), recording as many vital signs on the EWS as possible and documenting the A-E clinical assessment. If the nurse or other clinician is concerned about the patient's physiological status, whether the patient requires treatment with medication, or is to be physically restrained the patient's care must be escalated to a senior doctor and nurse. Monday-Friday daytime this should be the nurse in charge of the ward. Day time out of hours this would be the site manager on Bleep 7626/6007 (17.00-20.00 hrs daytime and 08.00-20.00 hrs weekends) and at night the Hospital at Night team on Bleep 7740.

D. Assessing capacity

- Establish what motives the patient has and compare this with what the clinician wants or is trying to achieve. Is there a conflict between the two?
- If there is conflict, does the patient have capacity? Make sure the patient is given information necessary to make a decision
- Be clear if you are treating a consenting patient, or giving the treatment in best interests under the Mental Capacity Act
- Consider the need for restraint if a patient who does not have capacity is displaying behaviour that is putting themselves or others at risk of harm:
http://www.rcn.org.uk/development/communities/rcn_forum_communities/mental_health/resources/a-z_of_resources/restraint
- Regardless of the patient's capacity, staff should always explain what they are doing and seek patient's understanding and agreement
- For further guidance see Mental Capacity Act Code of Practice:
<http://www.justice.gov.uk/protecting-the-vulnerable/mental-capacity-act> and Trust Safeguarding Adults policy

If above measures remain inadequate, proceed to Steps 2-5 (a) for patients aged >65 yrs or (b) if aged 18-65yrs. Should medication be required, ensure the patient is monitored according to protocol below.

(a) Steps 2-5 (>65yrs): Drug Management for Acutely Agitated Patients aged >65 years

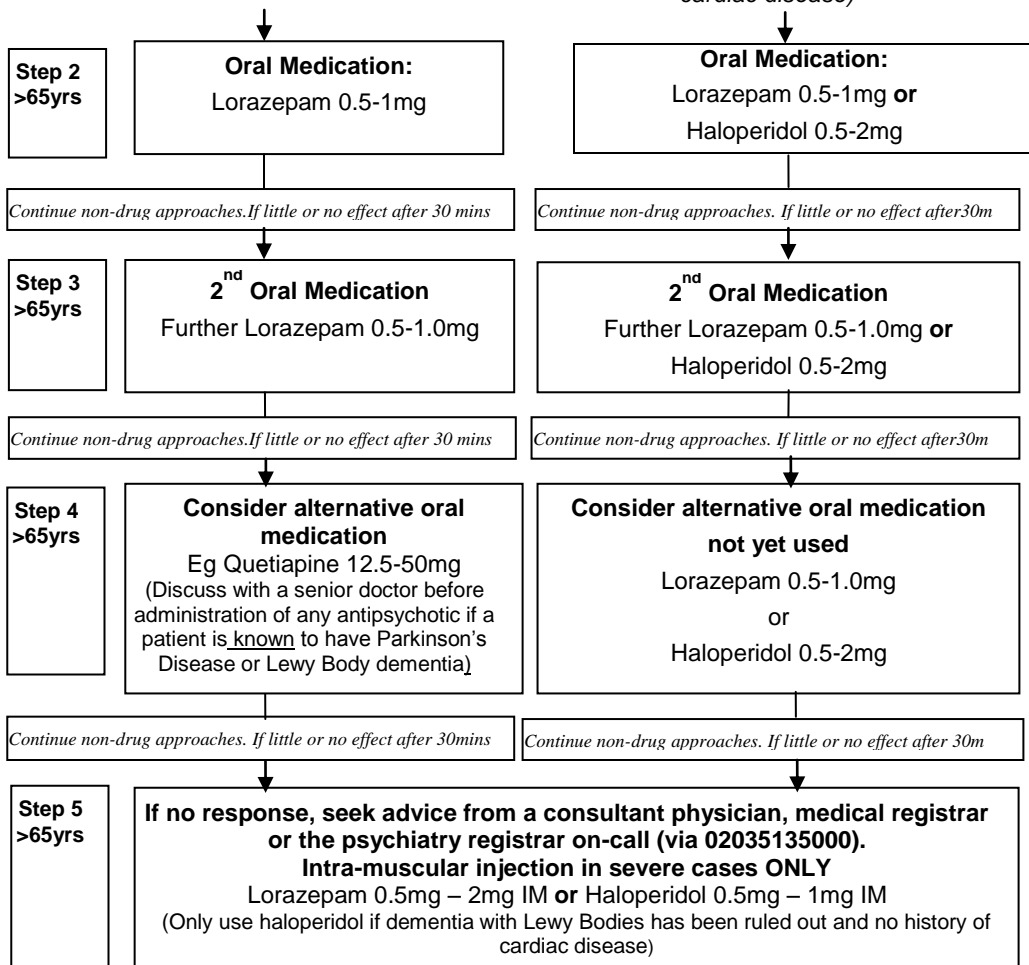
The following pharmacological interventions may be appropriate for patients aged >65 years who have not responded to Step 1 above. These steps may be considered for a consenting patient, or as 'best interests' treatment for a patient who lacks capacity. All treatment must be a proportionate and reasonable response to the risk posed to/by the patient.

Usually start at step 2, but in extreme cases move to step 4 below, all drugs must be prescribed, administered and their effect monitored by a senior doctor and nurse. Each step needs to be under the direction of a consultant physician or medical registrar.

Dementia with Lewy Bodies (either a confirmed diagnosis or cannot be excluded), Parkinson's Disease, Cardiac disease.

Contraindications* to antipsychotics have been ruled-out

(* including: dementia with Lewy bodies, Parkinson's, cardiac disease)



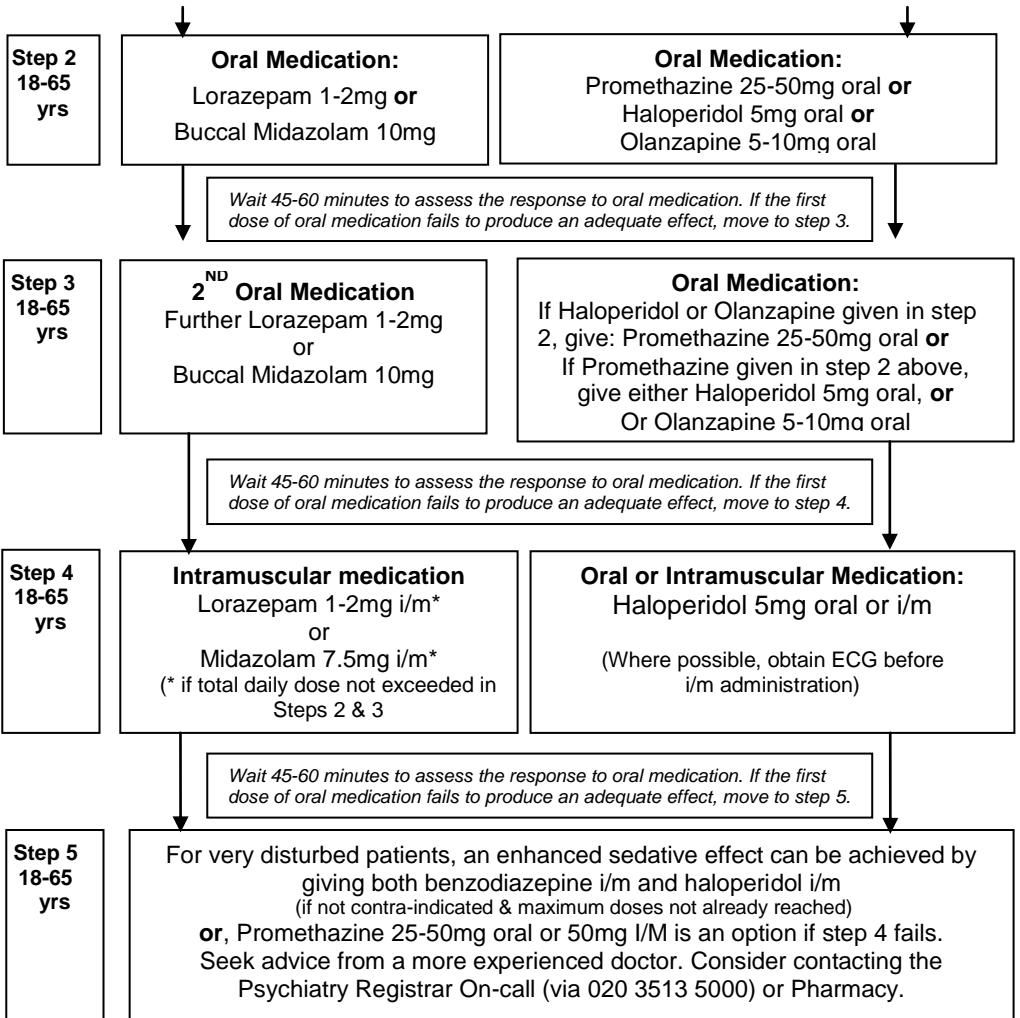
b) Steps 2-5 (18-65yrs): Drug Management for Acutely Agitated Patients aged 18-65 yrs

The following pharmacological interventions may be appropriate for patients aged >65 years who have not responded to Step 1 above. These steps may be considered for a consenting patient, or as ‘best interests’ treatment for a patient who lacks capacity. All treatment must be a proportionate and reasonable response to the risk posed to/by the patient.

Usually start at step 2, but in extreme cases move to step 4 below, all drugs must be prescribed, administered and their effect monitored by a senior doctor and nurse. Each step needs to be under the direction of a consultant physician or medical registrar.

For agitated patients with no contra-indications to benzodiazepines and patients with cardiac disease:

For patients in whom benzodiazepines are contra-indicated: (Impaired respiratory function, paradoxical aggression can be caused by benzodiazepines.)
NB. HALOPERIDOL IS NOT SUITABLE FOR PATIENTS WITH CARDIAC DISEASE



Monitoring of patients following the use of medicines for controlling agitation

After medication had been given, the following must be monitored & recorded by a qualified nurse continuously where possible or at least every 15 minutes:

(If the patient is undergoing control & restraint, or has his/her posture otherwise affected, vital signs will need more frequent monitoring)

- Blood pressure, pulse, temperature, respiratory rate, oxygen saturation, oxygen usage and conscious level using the Early Warning Score
- Extra-pyramidal movement side effects - muscle spasm, tremor, stiffness and restlessness
- Hydration status - is the patient dehydrated, eating and drinking. Check capillary blood glucose
- Where possible, obtain an ECG before giving haloperidol I/M. Check QTc interval.

Use of flumazenil to reverse benzodiazepine toxicity

- Flumazenil must be given if the respiration rate falls to < 10/minute after a benzodiazepine has been used
- Give flumazenil 200micrograms intravenously over 15 seconds. If the desired level of consciousness is not obtained within 60 seconds, a further 100micrograms can be injected and repeated at 60-second intervals to a maximum total dose of 1mg (1000micrograms) per course (initial dose plus 8 further doses). Monitor respiration rate continuously until it returns to the baseline level.

N.B. the effect of flumazenil may wear-off and respiratory depression can return – monitoring must therefore continue beyond the initial recovery of respiratory function.

Use of anticholinergics

- An anticholinergic medicine may be given to counteract an acute dystonic or parkinsonian reaction. It may be administered orally, IM or IV depending on severity of symptoms and patient's ability to swallow.
- Response to IV administration will be seen within 5 minutes and IM in about 20 minutes. e.g. procyclidine 5-10mg by IM, IV or oral (tablet or liquid). Maximum dose is 30mg/24hrs

Maximum recommended doses (see table below)

- Other medicines prescribed for the patient must also be considered (especially regular prescriptions for antipsychotics or benzodiazepines)
- These maximum doses must not be considered a license to use more than the minimum effective dose, they are provided purely for guidance and do not replace the need for careful clinical judgement by the staff caring for the patient. A decision to exceed these doses must be carefully recorded.
- The maximum daily dose should include both regular medication as well as “PRN” doses given in response to disturbed behaviour.

DRUG	(a) For patients aged >65 years old		
	Maximum oral dose in 24hrs	Maximum injectable dose in 24 hrs	Maximum dose (oral and i/m) in one 24hr period (including prn and regular doses)
Lorazepam	2mg	2mg i/m	2mg
Quetiapine	50mg	n/a	50mg
Haloperidol	10mg	5mg i/m	5mg oral + 5mg i/m
Midazolam	n/a	n/a	n/a
Olanzapine	n/a	n/a	n/a
Promethazine	n/a	n/a	n/a

DRUG	(b) For patients aged 18 - 65 years old		
	Maximum oral dose in 24hrs	Maximum injectable dose in 24 hrs	Maximum dose (oral and i/m) in one 24hr period (including prn and regular doses)
Lorazepam	4mg	4mg i/m	4mg
Haloperidol	20mg	12mg i/m	10mg oral + 5mg i/m
Midazolam	20mg	15mg i/m**	7.5 i/m + 10mg buccal
Olanzapine	20mg	n/a	20mg
Promethazine	100mg	100mg i/m	100mg

** High strength midazolam 10mg/2ml injection is restricted to certain areas in accordance with the NPSA Rapid Response Alert. If a dose is needed during normal working hours the ward pharmacist must sanction the supply and the patients details recorded in the CD order book. During 'out of hours', the resident on-call pharmacist must be contacted on Bleep 6267 to sanction supply and the patient details recorded in the CD order book.

MANAGEMENT AND PREVENTION OF DELIRIUM

Link Consultants: Dr Marcus Hughes & Dr Helen Jones

What is Delirium?

A syndrome of disturbed consciousness (drowsy or agitated), with acute onset and fluctuating course caused by medical illness or medication/alcohol/toxins or their withdrawal. Usually it is multifactorial.

It should be considered as the most likely cause of new-onset confusion, paranoia, or hallucinations in a hospital inpatient.

Patients at particular risk are those aged 65 or older, acutely unwell (EWS 5 or more), admitted with a hip fracture or with CNS disease, especially any form of cognitive impairment.

Prevention and Recognition of Delirium

Delirium is very common in hospital inpatients and causes increased length of stay and risks complications such as pressure sores, malnutrition and hospital acquired infections. Although some patients present with delirium on admission, in others it develops during the admission and in some is preventable with good medical and nursing care.

Recognising that a patient has a delirium enables us to determine the causes, optimize the environment and reassure staff and families. All adult patients admitted overnight to hospital should be assessed for their risk of developing delirium, screened for current delirium and have their modifiable risks addressed.

In St George's we have an iClip 'Delirium Assessment' that is ordered automatically when an adult patient is admitted to a ward. This must be filled out within 24 hours of admission by going to 'Tasks' and then 'Medical' tab and clicking on 'Delirium Assessment'. If the patient has any of the major risks for delirium (age 65 or older, acutely unwell, hip fracture, cognitive impairment), the assessment will prompt you to: look for behavioural indicators of delirium, complete a 4AT screen, decide if you can make a diagnosis of delirium, and ensure all precipitating factors are addressed. If the patient has no major risk factors then the assessment is complete and no further assessment is required.

Nursing staff assess patients for signs of delirium daily using the RADAR screen, if they pick up new signs of a possible delirium they will ask you to review – order a new delirium assessment on iClip under 'orders', 'delirium assessment'.

Behavioural indicators of Delirium

What aspects of a patient's behaviour might lead you to consider delirium?

- Cognitive Function: poor concentration, slow responses, obvious disorientation, confusion.
- Perception: visual or auditory hallucinations.
- Physical Function: reduced movement or mobility, restlessness, agitation, sleep disturbance.
- Social Behaviour: lack of cooperation with reasonable requests, withdrawal, change in mood, attitude, communication.

4AT Screen

The 4AT is a screening tool to look for Delirium and moderate to severe cognitive impairment, validated for use in hospital inpatients. It does not replace clinical judgement or give a definitive diagnosis of delirium. Although higher scores are associated with

higher mortality during the inpatient stay, it is not a severity score and is not designed to be used on a frequent basis on the same patient to monitor delirium progression.

Alertness -Normal -0, mild sleepiness on waking only -0, clearly abnormal -4

AMT4 (DOB, age, place, current year) -No mistakes-0, 1 mistake-1, 2 or more or untestable-2

Attention (months of the year backwards) -Greater than 7-0, <7 or refuses-1, untestable-2

Acute change or fluctuating course -No-0, Yes-4

Scores: 0= delirium and significant cognitive impairment unlikely; 1-3= possible cognitive impairment; 4 or above=delirium likely

Management of Delirium

First make a diagnosis. Take as full a collateral history as possible of the confusion and behavioural changes, especially the symptoms listed in the behavioural indicators above. Always ask specifically about previous psychiatric history and any previous episodes of confusion, agitation and memory problems. Use the 4AT as a screening tool or use CAM (confusion assessment method) if you are confident in its use. Think about whether you can diagnose delirium based on the DSM-V criteria: disturbance in consciousness and a change in cognition that develops over a short period of time and fluctuates.

The key to managing delirium is to identify and treat the causes of the delirium.

Ensure as full a physical examination as possible. Remember to use the no touch observation guidelines and if unable to fully assess due to agitation, proceed as per the agitated adult guidelines.

Severe agitation is a medical emergency.

Possible causes to consider: urinary retention; constipation; pain; metabolic disturbances (especially sodium, calcium, renal, liver, thyroid, glucose); infection; cardiac or respiratory problems (especially those causing hypoxia or CO retention); medications (especially opiates, tricyclics, Parkinson's medications, steroids, anticholinergic drugs such as oxybutynin); alcohol, nicotine or drug withdrawal (including legally prescribed drugs such as benzodiazepines); CNS disease: head injury such as concussion and other TBI, epilepsy with post-ictal confusion, meningitis or encephalitis, Wernicke's encephalopathy.

Remember delirium is usually multifactorial. Look for and treat the medical causes, and also ensure that you optimise the patient's environment, to reduce the nursing and environmental factors that can worsen delirium.

Optimising environmental factors that may worsen delirium

Avoid catheterisation if at all possible and review catheters and other invasive lines daily. Minimise polypharmacy and review medication every 24 hours. Encourage the presence of family or carers – especially if patient is agitated, they can be allowed to visit and often help to calm and reassure. If the patient does not speak good English, ensure a translator is available. Aim for continuity of nursing care, minimising 'new faces', avoid ward and bed transfers, especially at night. Nurse in a well-lit bay close to the nursing station, use frequent reorientation to place and reason for admission. Distressed or agitated patients may require enhanced care with a 1 to 1 HCA, or in extreme cases a RMN. Encourage early mobilisation, aim to sit out of bed at least as soon as possible – proactively ask nursing staff to do this. Encourage adequate hydration and good nutrition, avoid constipation, make sure fluid, food and bowel charts are completed and reviewed

and patient is given assistance with eating and drinking. Patients' glasses should be clean and used, ensure hearing aids working and ears clean – can use a communicator device from audiology if deaf but no hearing aids available.

Explain the diagnosis to patient and family and provide our delirium information leaflet to relatives and/or patient. Document the diagnosis of delirium in patient's notes – if iClip Delirium Assessment is completed this will automatically be added as a problem and appear on the discharge summary.

Further Advice

Refer to the Dementia and Delirium team on iClip ('orders' 'Refer to delirium') for advice on difficult diagnosis, management if delirium not resolving or advice on behaviour that challenges. The team are available Mon-Fri 9am to 5pm, bleep 7001, ext 6851. Consult the intranet for the Delirium Pathway, patient information leaflet on delirium and further resources, search 'Dementia and Delirium Team'. Out of hours, help is available through the psychiatry liaison team, the Senior Health consultant on call can also give telephone advice.

ALCOHOL USE DISORDERS
Link Consultant: Dr Mark Haden

MANAGEMENT OF ALCOHOL WITHDRAWAL

Advice on managing alcohol withdrawal is available from SGH Drug & Alcohol Liaison Team on Ext. 0595 or Bleep 6915, Monday to Friday 9am to 5pm. For non-urgent referrals or referrals out-of-hours please refer on iClip: search under orders for “Refer to DALT”. All patients with suspected alcohol problems should be referred to this team, as early as possible once this is identified. Please see the details on the Alcohol Liaison Team pages on St George’s intranet for details on how to refer out of hours, or patients being discharged from the ED.

[Drug and Alcohol Liaison Team - Drug and Alcohol Liaison Team \(unily.com\)](#)

Definition

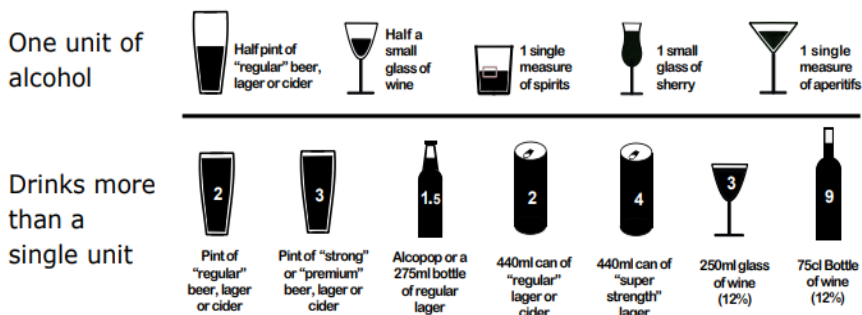
Acute alcohol withdrawal may be defined as: The physical and psychological symptoms that people can experience when they suddenly reduce the amount of alcohol they drink if they have previously been drinking excessively for prolonged periods of time. Symptoms are typically seen within 6-8 hours of the last drink and may develop before the blood alcohol level has fallen to zero. Symptoms outlined below may vary in severity, commonly peaking at 10-30 hours and usually subsiding by 40-50 hours.

Identification of risk

Treatment for withdrawal in hospital may be required either in individuals attending the hospital with symptoms and signs of unplanned acute alcohol withdrawal, or in those patients admitted for other reasons, who have a history of significant alcohol consumption. Hence all patients should be screened for alcohol misuse, to identify those at risk. This can be simply done using the AUDIT-C screening tool, as below (Table 1). A score of ≥ 5 is a positive screen, and indicates increasing or higher risk drinking. Those patients should be offered the opportunity to speak to one of the Alcohol Liaison Nurses, who will perform a full screen to identify whether there are features of hazardous, harmful or dependent drinking. Patients admitted to hospital who consume > 15 units alcohol daily or with features of alcohol dependence are at high risk of alcohol withdrawal.

Table 1. AUDIT-C screening tool

Questions	Scoring system					Your score
	0	1	2	3	4	
How often do you have a drink containing alcohol?	Never	Monthly or less	2-4 times per month	2-3 times per week	4+ times per week	
How many units of alcohol do you drink on typical day when you are drinking?	1-2	3-4	5-6	7-9	10+	
How often have you had 6 or more units if female, or 8 or more units if male, on a single occasion in the last year?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily	



Interventions and Brief Advice (IBE)

The primary goal of IBE is to reduce alcohol consumption by showing the patient:

- What the consequences of their drinking might be
- What they can do about it
- What help and support can be accessed

Step 1. Feedback on their AUDIT-C score

- Eg. so your scored in that screening questionnaire, that tells me that you're currently not in the low risk drinking category.
- Ask permission to discuss what this means, if they say no offer information to take away (they may still say no that's ok - just document this)

Step 2. Remind or explain the current guidance on low-risk drinking

Ask if they know the current guidance and get them to explain to you

If they don't know then explain to them

- You are safest not to drink more than 14 units per week.
- Spread this drinking over three days or more during the week.
- No more than 2-3 units per day
- Keep the risk lower by having several alcohol-free days each week.

If you are pregnant or planning a pregnancy, the safest approach is not to drink alcohol at all, to keep risks to your baby to a minimum.

See next page for units in common drinks

Step 3. Make it relevant

Remind them of the risks associated with alcohol consumption, particularly the ones related to their presentation (eg. if came with hypertension remind them about increased cardiac disease, if came with injury (even if not drunk at the time) remind them about increased accident and injury)

Step 4. Next steps

- Give them the generic alcohol information leaflet (under patient information leaflets on the ED intranet page or the DALT page) AND
- The borough specific leaflet if they live in: Wandsworth, Merton, Lambeth or Sutton (available on the DALT intranet page)

If they live in another borough / would rather go home and look things up online their local service can also easily be found via:

- Talk To Frank
- Or via their GP

If none of the above is possible then offer DALT referral (under orders on iclip)

ASSESSMENT OF WITHDRAWAL

The following **symptoms and signs** suggest the possibility of acute alcohol withdrawal:

<ul style="list-style-type: none">• Anxiety/agitation/irritability• Nausea/vomiting/anorexia• Tremor of hands, tongue or eyelids• Insomnia• Confusion	<ul style="list-style-type: none">• Hallucinations• Sweating• Fever with or without infection• Tachycardia and mild hypertension
---	---

In addition to full history, an adequate **alcohol history** should be obtained, to include the following:

- Drinking pattern
 - How many days per week alcohol consumed
 - How many units on each occasion
 - What type of alcohol
 - How often > 6 units consumed on a single occasion
- Time of last drink
- Previous withdrawal symptoms, particularly seizures or Delirium Tremens
- Tolerance
- Morning drinking
- Prioritisation of drinking above other factors (physical health, mental health, social or family life, work, financial or legal matters)

Physical examination

In addition to assessment for the signs listed above, patients should be examined for the signs of chronic liver disease, and standard observations should be recorded 2 hourly for the first 24 hours, and until symptoms stabilised:

- Temperature
- Pulse rate
- Blood pressure
- Respiratory rate

Investigations

- Blood tests: FBC (MCV), U&Es, LFTs, GGT, bone profile, magnesium, blood clotting, B12, folate
- Further specific investigations as guided by symptoms and signs

Assessment of need for hospital admission

NICE recommends admission to hospital for medically assisted withdrawal for those in acute alcohol withdrawal, in the following groups:

1. Those with, or who are assessed to be at high risk of developing, alcohol withdrawal seizures or delirium tremens.
2. Young people under 16 years who are in acute alcohol withdrawal.

3. Certain vulnerable people (for example, those who are frail, have cognitive impairment or multiple comorbidities, lack social support, have learning difficulties or are 16 or 17 years of age).

Delirium Tremens (DTs)

DTs occurs in only about 5% of patients undergoing alcohol withdrawal but accounts for the highest morbidity and mortality. The onset of DTs is two to five days (most commonly at 2-3 days) after cessation of alcohol. It represents a medical emergency.

Features of Delirium Tremens include:

- Severe tremor
- Clouding of consciousness
- Delusions
- Confusion and disorientation
- Tachycardia >100/min
- Agitation
- Violent behaviour
- Delirium
- Fever with or without infection: temperature > 101°F/38.3°C
- Severe hallucinations (mainly visual, may be tactile or auditory) often causing extreme fear

ALCOHOL WITHDRAWAL TREATMENT

The treatment of alcohol withdrawal has two broad components; pharmacotherapy to treat symptoms and signs, usually with benzodiazepines, and vitamin supplementation to treat or prevent Wernicke's Encephalopathy (see below).

Considerations

1. For patients admitted to hospital for other reasons, deemed to be at risk of alcohol withdrawal, but with no overt symptoms and signs, benzodiazepine treatment may not be required, but symptoms should be monitored during first 72 hours. PRN doses may be prescribed if there is concern, for example over inaccurate alcohol history.
2. Patients who are being discharged from hospital, in particular the ED, who have a history of alcohol dependence, should be advised to gradually reduce their consumption rather than stopping abruptly. These patients should be offered referral to the Alcohol Liaison Team, who will make contact with the patient and ensure that they are signposted or referred to the appropriate local community alcohol service.

Treatment regimens

Chlordiazepoxide is a long-acting benzodiazepine, which is the first line treatment for alcohol withdrawal. Table 2 shows suggested regimens for treating the symptoms of alcohol withdrawal, according to previous alcohol consumption. There are 2 methods of medicating patients in alcohol withdrawal: a) the symptom-triggered approach or b) fixed dose regimens. NICE recommends following a symptom-triggered approach to dosing medication in hospital inpatients.

This can be through clinical judgement in an experienced individual, or through more objective assessment using the CIWA scale. The CIWA-Ar score (The Clinical Institute Withdrawal Assessment – Alcohol, revised) is a validated 10-item assessment tool that can be used to quantify the severity of the alcohol withdrawal syndrome, and to monitor and

medicate patients throughout withdrawal. CIWA scores can be recorded on iClip under the “Assessment and Obs” tab under “Adult Assessment”.

Symptom-triggered (PRN only) regimens present a particular challenge on our busy acute hospital wards and where possible the appropriate fixed dosing regimens should be used to prevent unwanted escalation of withdrawal symptoms. One of the commonest errors in managing alcohol withdrawal is under-dosing or too-rapid weaning of chlordiazepoxide. Withdrawal seizures and DTs are more likely to occur in this scenario. In order to minimise the risks of this, we advocate following one of the fixed-dose regimens below, prescribed regularly, with clinical judgement or CIWA scoring used to administer additional, “as-required” doses. PRN doses vary according to the fixed dose the patient is taking.

Patients should monitored at least 2 hourly in the first 24 hours, and until symptoms stable. Consider referring the patient to the Critical Care Outreach Team if the total daily dose of chlordiazepoxide (or equivalent) exceeds 240mg.

Table 2. Recommended chlordiazepoxide fixed dose regimens

Severity	MODERATE	SEVERE	VERY SEVERE
Alcohol units/day	15 – 25	30 – 49	50 – 60
Chlordiazepoxide prescribing (mg)			
Day 1 (mg)	20 QDS	30 QDS	40 QDS
Day 2	15 QDS	25 QDS	35 QDS
Day 3	10 QDS	20 QDS	30 QDS
Day 4	5 QDS	15 QDS	25 QDS
Day 5	5 BD	10 QDS	20 QDS
Day 6	5 nocte	5 QDS	15 QDS
Day 7		5 BD	10 QDS
Day 8		5 nocte	5 QDS
Day 9			5 BD
Day 10			5 nocte
Prn	10-20	20-30	30
Max daily dose	240	240	240

Considerations

1. There are a number of factors influencing chlordiazepoxide metabolism, therefore an individualised approach to the treatment regimen may be required in order to achieve symptom control. All patients should be reviewed regularly by the Alcohol Liaison team, who are available for advice on prescribing.
2. If a patient is requiring frequent PRN doses, this should be reflected in an increase in the regular dose, and the reducing regimen will need to be amended.
3. Doses should be reduced if over-sedation occurs.
4. High doses should be avoided in the elderly, or lorazepam considered as a short acting alternative benzodiazepine.
5. Caution in patients with decompensated liver disease – lorazepam should be considered.
6. Chlordiazepoxide should NOT be prescribed as discharge medication. Patients who are motivated and committed to stopping drinking should be given the opportunity to complete

their detox. The Alcohol Liaison team will advise on whether there are opportunities to consider completion of detox through ambulatory pathways with local alcohol services.

Lorazepam may be required in the elderly or those with liver impairment:

Approximate equivalent Benzodiazepine doses

30mg Chlordiazepoxide \approx 10mg Diazepam \approx 1mg Lorazepam

Treatment of Delirium Tremens

- Reorientation and a calm environment
- Pharmacological: benzodiazepines
- Oral Lorazepam is the first-line treatment 1– 2 mg as required, max 8mg per day according to response/CIWA-Ar. Doses should be given in 6 hourly intervals. (Note 8mg Lorazepam \approx 240mg Chlordiazepoxide)
- If symptoms persist or oral medication is declined/not feasible, consider parenteral Benzodiazepines.
- Titrate IM/IV Lorazepam up to 0.5 to 1mgs every thirty minutes according to response, max 8mg per day. Injectable Lorazepam contains propylene glycol, polyethylene glycol and benzyl alcohol which can precipitate hypersensitivity. IM/IV Lorazepam must be diluted 1:1 with sodium chloride 0.9% prior to administration
- Titrate IV Diazepam emulsion 5-10 mgs every 30–60 minutes (should be given at a rate of not more than 5 mgs per minute into a large vein). Try to avoid IM administration due to unpredictable absorption.
- Baseline physical observations prior to IV drug administration to be repeated every 15 mins for the first hour and half hourly for the next 4 hours.
- Antipsychotics may be added for behavioural disturbance, hallucinations and paradoxical effects of benzodiazepines: Haloperidol 1-5mg po/IM or Olanzapine.
- Large doses of benzodiazepines may be required in the treatment of DT's. Consider referring the patient to the Critical Care Outreach Team if the total daily dose of chlordiazepoxide (or equivalent) exceeds 240mg.
- Seizure threshold may be lowered, and adequate treatment with benzodiazepines to manage the underlying withdrawal is the priority.

Treatment of seizures

- Alcohol withdrawal seizures may be self-limiting
 - There is no evidence to support the use of conventional anti-epileptics such as phenytoin to prevent recurrence of seizures
- i). If prolonged seizure occurs treat with lorazepam 1-2mg IV or diazepam 5-10mg PR, repeated 5-10 mins later if needed. Lorazepam is less of a respiratory depressant and can be given up to 8 mg/24 hours.
- ii). Consider excluding organic cause for seizure
- iii). If a seizure occurs during treatment for alcohol withdrawal the dosing regimen should be adjusted, with increase in fixed doses

WERNICKE'S ENCEPHALOPATHY

Wernicke's Encephalopathy (WE) is an acute life-threatening neurological syndrome consisting of confusion, apathy, dullness, delirium, palsies of the ocular muscles, nystagmus, disturbances in equilibrium, and ataxia. Its most common cause in industrialized countries is thiamine deficiency associated with alcoholism. If not treated immediately with thiamine, the patient is likely to progress to an amnesic state, and may

even die. A late neuropsychiatric manifestation is Wernicke-Korsakoff syndrome or psychosis, characterised by memory loss and confabulation.

Thiamine plays a role in metabolizing glucose to produce energy for the brain. An absence of thiamine therefore results in an inadequate supply of energy to the brain, particularly the hypothalamus and mammillary bodies. Heavy alcohol use increases the demand for and interferes with the metabolism of thiamine, so even in cases where patients are eating a balanced diet while drinking heavily, the metabolic problem persists because most of the thiamine is not absorbed.

Signs of Wernicke's Encephalopathy (WE)

The classical text book triad of signs is ataxia, confusion and ophthalmoplegia, however in reality this only occurs in 10% of patients

Signs may be:

- Confusion (82%)
- Ophthalmoplegia or nystagmus (29%)
- Ataxia (23%)
- Hypothermia and hypotension
- Memory disturbances
- Coma or unconsciousness

Treatment of Wernicke's

This is a medical emergency. Patients showing sign of WE require:

- i). IV Pabrinex ® TWO pairs TDS for 5-7 days
 - Pabrinex ® is diluted in 50-100mL of normal saline or Dextrose 5%, and given by IV infusion over 30 minutes
 - The first IV dose should be given in normal saline and should be administered prior to any glucose
- ii). Continue IV Pabrinex ® at dose of One pair OD until there is no further improvement in clinical symptoms
- iii). Oral therapy should follow parenteral therapy, with thiamine 100mg TDS.

Use of Pabrinex ® in other scenarios

1. Acute alcohol withdrawal

- Give IV Pabrinex ® TWO pairs TDS for 3 days
 - a) If low risk for WE treatment can then switch to thiamine 100mg TDS.
 - b) If high risk for WE (eg malnourished, liver disease, very high alcohol consumption) parenteral treatment should continue with IV Pabrinex ® ONE pair OD for the duration of the detox, followed by oral therapy

2. Harmful or dependent drinkers

- NICE recommends offering ONE pair IV Pabrinex ® once daily for at least 3-5 days in harmful or dependent drinkers:
 - if they are malnourished or at risk of malnourishment or
 - if they have decompensated liver disease
- AND**
- they attend an emergency department or
 - are admitted to hospital with an acute illness or injury.

FURTHER RESOURCES

Alcohol liaison team page, St George's Hospital intranet

www.nice.org.uk/guidance/cg100

www.nice.org.uk/guidance/cg115

<https://www.nice.org.uk/guidance/ph24>

CARDIAC MARKERS IN PATIENTS WITH SUSPECTED ACUTE CORONARY SYNDROMES**Link Consultant: Dr Mahtab Sharifi**

In a patient in whom an acute coronary syndrome is suspected, measurements of cardiac markers should be used to confirm or exclude myocardial infarction. The tests currently available provide measurement of creatine kinase (CK) and cardiac troponin T (cTnT). Diagnosis of AMI requires elevation of a cardiac troponin. Measurements can be particularly helpful in providing an accurate diagnosis in patients with musculoskeletal injury causing rises in CK and CK-MB. Moreover, levels remain elevated for at least 7 days following acute myocardial infarction, so can be used in diagnosis when the patient presents late. It should be noted that cTnT also rises in other conditions where there is cardiac damage, such as myocarditis.

Measurement of cTnT is particularly helpful when making decisions about patients:

- presenting more than 12 hours after the onset of symptoms
- whose CK elevation may be of musculoskeletal origin as in trauma or after surgery
- without ST segment elevation but who are being considered for angiography and subsequent intervention.

CK and cTnT should be requested on admission, and 3 hrs from admission in all patients with chest pain with the possible diagnosis of acute MI. If clinical suspicion persists, or the patient is at high risk, a further sample should be taken at 6-10 hrs from admission. In accordance with the new universal definition of MI, an increase in troponin by more than 7 ng/L to above 14 ng/L with appropriate clinical features is required for a definitive diagnosis of MI. An increase from < 14ng/L to more than 200 ng/L is highly suggestive of AMI. Re-infarction may be detected by repeat measurement.

DO NOT ATTEMPT (DNA) CARDIO-PULMONARY RESUSCITATION(CPR)
Link: Jeanette Walls, Resuscitation Officer & DNACPR lead

Where patients are admitted to hospital acutely unwell their resuscitation status should be considered as soon as is reasonably possible if a cardiopulmonary arrest is anticipated. Clinicians need to involve and consult patients (and/or their families where the patient lacks capacity) when decisions are being made about DNACPR decisions. A failure to involve and consult in such cases may be a breach the patient's Article 8 ECHR rights. When no explicit decision has been made about resuscitation before a cardiopulmonary arrest, and the expressed wishes of the patient and family are unknown, it should be presumed that staff would attempt to resuscitate the patient.

All patients will be automatically assumed to be appropriate for CPR in the event of cardiac arrest unless a completed DNA CPR form is visible in the patient's notes. Senior medical and nursing colleagues should support anyone initiating CPR where DNA CPR documentation has not been carried out. A DNA CPR decision only applies to CPR and *not* to other aspects of care (eg analgesia, antibiotics, suction, treatment of choking or anaphylaxis etc. – which are sometimes loosely referred to as resuscitation).

It is also essential to identify those patients who would not want CPR to be attempted in the event of an arrest and who competently refuse this treatment option. Some competent patients may wish to make an Advance Decision about treatment (such as CPR) that they would not wish to receive in some future circumstance. These statements must be respected as long as these decisions are informed, current and made without coercion from others.

If you are in doubt about any aspect of the decision making process seek advice from senior colleagues and the Trust's legal services manager on ext 2901.

The responsibility of a DNA CPR decision is that of the most senior clinician responsible for the patient's care (usually medical consultant in hospital or the GP in community-based facilities). It is wise to reach consensus with the patient, staff and relevant others and to complete documentation in accordance with the **Trust's DNA CPR policy***, ensuring the decision is communicated to all involved in the patient's care. The most senior clinicians are responsible for any future revised decision.

Junior doctors with full GMC licence to practise can sign the DNA CPR form but the decision must be fully discussed and agree with the responsible Senior Clinician who should then sign at the next available opportunity. Doctors without full GMC licence to practise (Foundation Year 1) should NOT make this decision.

*http://stginet/Procedural%29documents/Patient%20related/Patient_Management/Clin_1_1.pdf

For further guidance please refer to:

1. 'Decisions relating to Cardiopulmonary Resuscitation'. A Joint Statement from the BMA, the Resuscitation Council (UK) and the Royal College of Nursing (October 2007, updated October 2014).

2. End of Life Guidance for Doctors – General Medical Council (July 2010)

http://www.gmcuk.org/End_of_life.pdf_32486688.pdf

3. Clinical Ethics Committee: email cec@sghms.ac.uk, helpline x4971, or Legal Services x2901.

*http://stginet/Procedural%29documents/Patient%20related/Patient_Management/Clin_1_1.pdf

PERIOPERATIVE MANAGEMENT OF DIABETES MELLITUS

Link Consultants: Dr Grainne Nicholson* &

Dr Kenneth Earle

All patients with diabetes undergoing procedures or surgery, or who are nil by mouth, should have their glycaemic control managed according to the variable rate intravenous insulin infusion (VRIII) protocol available via intranet through the following link:

<https://stginet.unily.com/sites/grey-book/SitePageModern/48789/grey-book-home>

Patients who do not require insulin infusion should still have their Capillary Blood Glucose (CBG) checked at least hourly, peri-operatively and in recovery.

1) Pre-operative assessment:

- i). Patients with diabetes should ensure that they maintain acceptable glycaemic control ($\text{HbA}_{1\text{C}} < 69$ mmol/mol) in the months and weeks prior to surgery, as this improves the healing process post-operatively.
- ii). Patients who have poor glycaemic control should ideally be referred to the diabetes team for optimisation of diabetes control prior to surgery. However urgent and emergency surgery should not be delayed or cancelled and such a decision should only be made in discussion with the responsible team.
- iii). All diabetic patients who are scheduled for an elective surgery with a period of starvation, should attend a pre-operative assessment clinic as soon as possible, and receive an individualised diabetes management plan with written instructions on the management of their medications pre-operatively according to the guideline available on: <http://stginet/Units%20and%20Departments/Anaesthetics/Protocols.aspx>
- iv). Patients who are undergoing procedures that involve giving intravenous radio-contrast, should ideally discontinue metformin 24 hours prior the procedure and re-start 48 hours after procedure. If this is not possible, then Metformin can be stopped on the day of procedure.

2) On admission (Prior to operation):

- i). Patients with diabetes should be, wherever possible, scheduled first on the operating list, in order to avoid unnecessarily long starvation periods.
- ii). To ensure that blood glucose is controlled within normal limits before surgery (target 4-12 mmol/L), random blood glucose should be obtained soon after the patient is admitted. If it is not in the target range, advice should be sought from the diabetes team (Diabetes Specialist Nurse - Bleep 6236; or, SpR - Bleep 7778), the anaesthetic team or both.
- iii). CBG must be monitored at least hourly peri-operatively and in recovery. More frequent monitoring will be required if the CBGs are not well controlled.
- iv). If VRIII is needed, then an intravenous cannula should be inserted and VRIII should be initiated either by 8 AM for morning operations or 12 pm for afternoon procedures.
- v). glucose is temporarily stopped, eg. en route to theatre, insulin must also be stopped temporarily

vi). Never administer Sodium Chloride 0.9% as the sole intravenous fluid to a patient receiving Insulin. Fluid management should be with 500ml 5% Dextrose plus 0.45% NaCl with 0.15% KCl (premixed bag at 85 ml/ hr.) (If the patient has renal impairment, omit the potassium). See VRIII protocol for guidance.

vii). If on admission a patient has hypoglycaemia (CBG is < 4 mmol/L) this should be treated with 100ml of intravenous Glucose 10% and CBG monitored every 15 minutes. For patients with fluid restriction 50ml of intravenous Glucose 20% can be used. Trust guideline for the treatment is available via intranet through the link below:

<http://stginet/Publications/Clinical%20Publications/Grey%20book/Hypoglycaemia.pdf>.

At this point please discuss with the relevant anaesthetist.

viii). No patient should remain on VRIII for more than 48 hours without discussion with diabetes team.

3) Post-operative management

i). Blood glucose should be monitored hourly until the reading is stable and within normal range. Readings can then be taken two hourly. Serum potassium should be measured on alternate glucose samples.

ii). Patients for whom VRIII has been initiated should have their first meal following surgery while VRIII continues, to check that meal is tolerated.

iii). Usual oral anti-diabetes medications and insulin regimen should be re-started as soon as patient is able to eat and drink according to the VRIII protocol (page 6).

iv). VRIII should not be stopped in patients who are usually administering insulin as part of their diabetes management until one hour post administration of the first dose of subcutaneous insulin. The exception is for patients who have not missed their background insulin.

ENTERAL/PARENTERAL FEEDING**Link Consultant: Dr Penny Neild**

Full guidance on nutritional support is detailed in the Trust policy available on the intranet via the following link:

<http://stg1.wordpress01.wordpress/wp-content/uploads/2016/> Malnutrition – overt or covert – delays recovery and increases the risk of clinical complications. Patients at risk of malnutrition by virtue of disease or complications should be referred to the ward Dietitian via iClip with a Malnutrition Universal Screening Test (MUST) score or iNUT (renal or heart failure patients) complete. Nutritional Care plans as per iClip are to be implemented whilst awaiting Dietitian review.

[06/Nutrition-Support-AdultOral-Enteral-and-Parenteral.pdf](http://stg1.wordpress01.wordpress/wp-content/uploads/2016/06/Nutrition-Support-AdultOral-Enteral-and-Parenteral.pdf)

A decisional flow chart is available for patients refusing oral intake including MCA:

<http://stg1.wordpress01.wordpress/wp-content/uploads/2016/06/Appendix-K-1.pdf>

In addition, Trust guidance and policy on use of naso-gastric tubes can be found via the following link:

<http://stg1.wordpress01.wordpress/wp-content/uploads/2020/04/Nasogastric-TubesInsertion-Policy-2020.pdf>

Indications for Parenteral Feeding

Oral or enteral feeding routes are preferred for nutrition support. Parenteral nutrition (PN) is available if these routes are not accessible, but can often be avoided with forethought. The wide range of specialist enteral feeds available allows successful feeding in virtually all clinical states, and is superior to PN in respect of infection complications and maintenance of gut function. There is no clinical advantage in embarking on IV feeding if the patient is expected to resume oral-enteral feeding within 3-5 days unless there is a history of malnutrition.

Any patient being considered for parenteral feeding must be referred to the ward dietitian for review in the first instance. The ward dietitian will escalate to the Nutrition Team Dietitian if required who will inform the Nutrition Support Team.

The cut-off time for the submission of Parenteral Nutrition referrals for feeding to initiate the same working day is 09:30am. Referrals received outside of this time will commence feeding on the following working day. PN will not be instigated outside weekday working hours or at weekends except within the critical care setting.

Initiation of Parenteral Feeding

Patients must have 'Adult TPN' (order set on iClip) bloods ordered daily. Electrolytes must be corrected if abnormal (particular attention to K, Mg, and PO₄). Refer to the refeeding section of the Nutrition Support Policy for guidance on replacement of electrolytes and provision of thiamine (IV Pabrinex®):

<http://stg1.wordpress01.wordpress/wp-content/uploads/2016/06/Appendix-A-Refeeding-Syndrome-and-Electrolyte-Supplementation-Guidance.pdf>

This is particularly important in those at risk of re-feeding syndrome in patients initiated on nutrition support, especially those with low BMI, significant weight loss or inadequate oral intake for >10 days.

Patients accepted for IV feeding must be referred to Venous Access for CVC placement with a dedicated clean lumen for PN (PICC/Hickman).

Ongoing Management

The managing medical/surgical team must ensure the following monitoring occurs:

- Daily “Adult TPN” blood profile, unless directed otherwise by the NST
- Twice daily blood glucose measurement unless directed otherwise by the NST
- Strict fluid balance documented
- Weekly weight

Once a patient starts PN the NST dietitian, pharmacist and nutrition nurse specialist will review the patient (Mon-Fri) and the NST will review the patient on a weekly Consultant or Registrar led ward round.

Line Care

Full aseptic technique must always be used when accessing the CVC to prevent line infections and complications.

In the event of possible line sepsis differential peripheral and central line cultures should be taken and source clearly labelled.

Refer to the Nutrition Support policy for guidance on management, which includes details of when IV access should be removed.

Admission of complex Home PN (HPN) patients

Patients managed on HPN may be admitted for a variety of complications related to their IV feeding and other unrelated medical conditions. Please inform any member of the Nutrition Support team of these admissions within normal working hours. Advice can be sought from the PN pharmacist (Bleep 7554) for the use of patients HPN bags and for the supply of TPN giving sets and light protective bags.

Complications such as line sepsis/complications, AKI and electrolyte disturbances should be managed as per Trust guidance.

Out of hours, if home PN unavailable, please provide IV fluids and electrolytes to match the patients usual parenteral nutrition prescription. Patients should be encouraged to bring their HPN bags to hospital for administration on that date. The on-call pharmacist (Bleep 6267) can be contacted for advice outside normal working hours (9am-5.30pm), including cold chain storage and the supply of giving sets/light protective bag covers.

Contact details

Any team member may be contacted during standard working hours via:

Dr Penny Neild, Consultant Gastroenterologist (Ext. 3429) Dr John Louis-Auguste, Consultant Gastroenterologist (Ext. 3429); the Gastroenterology SpR (Bleep 6590); Emma Pindard & Jennifer Brown nutrition nurse specialists (Bleep 8050/8498); Alison Green and Evanna Leavy GI Services dietitians (Bp 6171/8894).
Sheung Lee Lead Pharmacist Adult PN (Bleep 8580)/ PN Pharmacist (Bleep 7554);

Out of hours

For questions or concerns regarding existing patients contact the on-call pharmacist via Bleep 6267.

INITIAL MANAGEMENT OF SEVERELY ILL PATIENTS WITH EATING DISORDERS (ED)

Link Consultant: Dr Penny Neild

Patients admitted to St. George's Hospital with severe ED (Anorexia Nervosa [AN] or Bulimia) and particularly those with an acute medical condition are at significant risk of death, as they have minimal reserves to combat any illness. The following should be borne in mind when admitting such patients:

- a) Hypoalbuminaemia does not usually occur as a result of ED. If present, underlying sepsis or an alternative acute event should be strongly suspected and sought. Such patients may not exhibit typical symptoms or signs of infection, e.g. fever, raised inflammatory markers and white cell count.
- b) It is essential to start feeding such patients as soon as possible, but carefully and with close monitoring (see below). Even 24 hours delay can have significant deleterious consequences for such markedly malnourished individuals.
- c) If a patient is a 'voluntary' admission (i.e. not held under a section), but refuses to eat, a psychiatric opinion should be sought as a matter of urgency.
- d) If a patient is detained under Section (2 or 3), this includes the provision for compulsory feeding (usually by means of NG tube). Again, if the patient refuses to eat or be fed by NG tube, a psychiatric opinion should be sought as a matter of urgency.

On admission the two main aims of care are:

1. Manage any acute medical problem which may have precipitated the current admission.
2. Oversee the management of re-feeding syndrome.

For both oral and tube fed patients the following needs to be monitored during the refeeding process:

- i. Order daily U+E's including potassium, phosphate and magnesium.
- ii. Correct any electrolytes as required based on the below criteria.
- iii. Monitor blood glucose levels every 4 hours until stable.
- iv. The patient needs to be prescribed:
 - 1L fluid in form of water PO or NG
 - Thiamine or Pabrinex as described below
 - Additional supplementation with a multivitamin if at high risk of refeeding (see below)

NUTRITIONAL TREATMENT

Within working hours refer the patient to the Dietitian on Bleep 7703

If out-of-hours make the referral the following working day.

Weekend service Dietitians are available on blp 8169, Saturdays and Sundays, 08.30 – 1630The regimen below outlines an out-of-hours nutrition plan. Only to be used until a referral is made on the next working day to the above dietitian.

Oral Re-feeding targets:

A daily meal plan is as follows which provides an increased calorie intake every 2 days:

Day1-2: ½ breakfast, yoghurt, ¼ lunch, yoghurt, ¼ supper = 550-600kcal

Day3-4: ½ breakfast, yoghurt, ½ lunch, yoghurt, ½ dinner = 750-800kcal

Day 5: ½ breakfast and glass of milk, yoghurt, ½ lunch, rice pudding, ½ supper, yoghurt = 1000kcal

Portion size to be checked by 1-1 nurse and not the patient

All food intakes should be recorded in the Food Record Charts by the patient's named nurse

Each meal must contain a source of carbohydrate

Use phosphate rich foods e.g. milk, yoghurt

Allow the patient 3 specific food dislikes (individual foods not food groups e.g: can dislike lamb but not all meat, or can dislike potatoes but not all starchy foods)

If patient is not eating meals the following sip feeds are appropriate:

Fortisip Compact 300 calories, 37 g carbohydrate, 12g protein

Substitute as:

Day 1-2: ½ a sip drink at each meal not eaten

Day 3 onwards: 1 x full sip drink at each meal not eaten

Enteral Re-feeding targets:

If the patient is to be tube fed they should be kept NBM and a continuous feed used. Below is the out-of-hours regimen for a refeeding patient:

NOTE: Do not use the standard out-of-hours inpatient Dietitian feeding protocol (it will overfeed an ED patient)

Day 1: 500ml Nutrison 1.0 as 25mls per hour x 20 hours. Rest 4 hours and repeat on day 2.

Day 3: 800ml Nutrison 1.0 as 40mls per hour x 20 hours. Rest 4 hours and repeat day 4.

Day 5: 1L Nutrison 1.0 as 50mls per hour x 20 hours. Rest 4 hours and repeat until Dietitian review.

Give 4 boluses of 100ml sterile water throughout the day.

If PO fluids are allowed they need to be prescribed on the drug chart and recorded on fluid balance chart.

Continuous pump feeding is most appropriate for out of hours, however if a patient is thought likely to attempt to remove feeding tubes, bolus feeding would be safer and the following out of hours regimen can be used:

Day 1: 1 x 200ml Fortisip bolus at 10:00 and 1 x 200ml Fortisip Bolus at 14:00.

Day 2: Repeat Day 1.

Day 3: 1 x 200ml Fortisip bolus at 09:00, 1 x 200ml Fortisip bolus at 13:00 and 1 x 200ml

Fortisip bolus at 17:00. Repeat until Dietitian review.

Flush before and after each bolus with 50mls sterile water.

Plus additional 4 x 100ml sterile water throughout the day.

If additional fluids are required liaise with the patient's medical team.

APPENDIX 4c

**GUIDELINES FOR THE PREVENTION AND MANAGEMENT OF REFEEDING
IN ADULTS**

Link Consultant: Dr Penny Neild

Definition of refeeding syndrome

Refeeding syndrome is defined by severe electrolyte and fluid shifts and associated with a group of clinical symptoms and signs that occur in starved or malnourished patients on commencement of nutrition support (oral, enteral or parenteral)¹. The physiological consequences of refeeding syndrome can lead to severe haematologic, neuromuscular, respiratory and cardiac symptoms and even death¹(Figure 1²).

Starvation results in reductions in cellular activity and organ function which is coupled with micronutrient and electrolyte depletion. The breakdown of protein and lipid occurs and gluconeogenesis as a result of insulin levels decreasing and an increase in glucagon levels.

Those at risk who have rapid reintroduction of nutrition can have shifts in fluid and electrolytes¹. However, it is common for potassium, magnesium and phosphate to be within normal serum levels prior to feeding due to homeostatic mechanisms¹. The consequence of these shifts can result in many different presentations from fluid retention, cardiac arrhythmias, respiratory insufficiency and possibly death¹.

Clinical signs of starvation include¹:

- Bradycardia (HR <60 beats per minute)
- Hypothermia (<36°C)
- Hypotension (Systolic BP <90mmol HG)
- Hypoglycaemia
- Peripheral and Pulmonary oedema

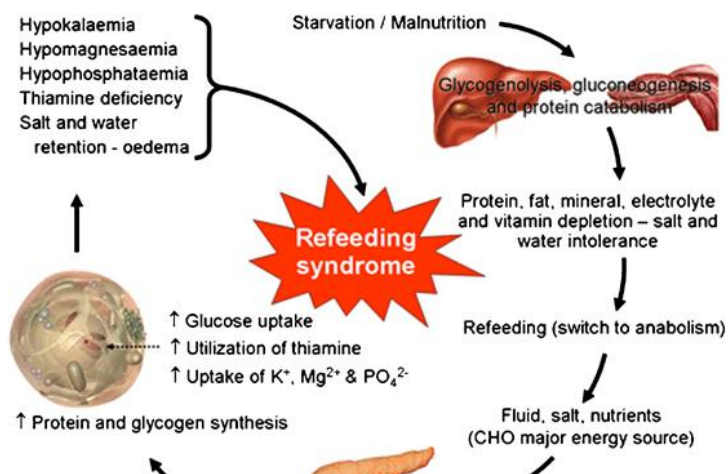


Figure 1: Pathogenesis and features of Refeeding Syndrome²

Identifying at-risk groups¹

- Anorexia nervosa
- Chronic diseases causing under-nutrition (eg cancer and cardiac cachexia, chronic obstructive pulmonary disease, cirrhosis, chronic vomiting)
- Gastrointestinal disorders causing malabsorption (eg Crohn's disease), maldigestion (eg pancreatic insufficiency), prolonged diarrhea or steatorrhea
- Diabetic hyperosmolar states resulting from poorly controlled or undiagnosed diabetes
- Morbid obesity with large weight loss (e.g. after gastric banding)

The following has been based on NICE CG36 guidelines³.

Step 1:

Identifying Refeeding Risk	
At Risk	<ul style="list-style-type: none"> • Very little intake for greater than 5 days
High Risk	<p><i>The Patient has <u>one or more</u> of the following:</i></p> <ul style="list-style-type: none"> • BMI less than 16kg/m² • Unintentional weight loss greater than 15% within the previous 3-6 months • Very little nutritional intake for greater than 10 days • Low levels of potassium, phosphate or magnesium prior to feeding <p><i>Or the patient has <u>two or more</u> of the following:</i></p> <ul style="list-style-type: none"> • BMI less than 18.5kg/m² • Unintentional weight loss greater than 10% within the previous 3-6 months • Those with very little intake for greater than 5 days • A history of alcohol abuse or drugs including insulin, chemotherapy, antacids or diuretics (interpret with caution)
Extremely High Risk	<p><i>The patient has <u>either</u> of the following:</i></p> <ul style="list-style-type: none"> • BMI less than 14kg/m² • Negligible intake for greater than 15 days

Step 2:

Vitamin and Mineral Supplementation	
Oral	
Thiamine	100 mg BD-TDS for 10 days ***
Vitamin B Co Strong	1 or 2 tablets TDS for 10 days
Multivitamin: Valupak Multivitamin	OD for 10 days
NBM	
Pabrinex	Give one pair of ampoules OD for 10 days* ** ***
Multivitamin: Abidec Liquid	1.2 mL OD**
Renal	
Dialysis pts	Renavit OD and Thiamine
*For patients who are extremely high risk please consider giving two pairs OD for 10 days	
** If patients are no longer NBM for the 10 day duration please swap to the oral route after three days for cost effectiveness	
***Please aim to give prior to feeding or increase in nutrition	

Step 3:

Reintroducing Nutrition	
At risk	<ul style="list-style-type: none"> • Nutrition support should be introduced at no more than 50% of requirements for the first two days • If clinical and biochemical monitoring reveals no refeeding symptoms, aim to meet full nutritional requirements on Day 3
High Risk	<ul style="list-style-type: none"> • Start Nutrition support at a maximum of 10 kcal/kg/day • Increase levels slowly to meet or exceed full nutritional requirements within four to seven days
Extremely High Risk	<ul style="list-style-type: none"> • Start Nutrition support at a maximum at 5 kcal/kg/day • Monitor cardiac rhythm continuously • Restore circulatory volume and monitoring fluid balance and overall clinical status closely

Step 4:

Replacing electrolytes: Potassium			
Serum Levels 3.5- 4.7mmol/L	Mild Higher than 3mmol/L	Moderate Asymptomatic 2.5-3 mmol/L	Severe Less than 2.5mmol/L or symptomatic
Patient able to tolerate oral or enteral therapy <i>NB: Use i.v. therapy if patient has diarrhoea or high output stoma.</i>	Potassium effervescent tablets (Sando-K® 12mmol /tablet). Two tablets twice daily (48mmol).	Potassium effervescent tablets (Sando-K® 12mmol/tablet). Two tablets three times daily (72mmol).	Not appropriate. See i.v. therapy
Patient on intravenous therapy only	Infuse potassium into a large vein at up to 20 mmol K+/hr (not more than 200 mmol/day). If plasma K+ < 2 mmol/L with arrhythmia, 40 mmol K+ may be given over 1hr . Bags for IV potassium infusion are available through Pharmacy.		
Renal:	The medical team should seek advice from the renal team for potassium replacements in CKD 4-5. Potassium levels should be monitored daily and dose of any supplementation adjusted as per bloods and renal team advice. Please be aware if the patient is on renal replacement therapy. Use pre-dialysis bloods only to identify need for replacement.		

[References for this section]

1. Crook M, Hally V & Panteli J. The importance of the refeeding syndrome. Nutrition. 2001; 17, 632-7.
2. Stanger Z, Brunner A, Leunenberger M, Grimble R, Shenkin A, Alison S and Lobo D. Nutrition in Clinical practice – the refeeding syndrome: illustrative cases and guidelines for prevention and treatment. 2007; 62: 687-694 . Available from: doi:10.1038/sj.ejcn.1602854 (Accessed 25.5.17)
3. NICE. GC32. Nutrition support for Adults: oral nutrition support, enteral tube feeding and parental nutrition. London: National Institute of Clinical Excellence; 2006

Replacing electrolytes: Phosphate			
Serum Levels 0.75-1.5mmol/L	Moderate (Asymptomatic) 0.4-0.6 mmol/L	Moderate Symptomatic 0.4-0.6 mmol/L	Severe Less than 0.4mmol/L
Patient able to tolerate oral or via enteral tube feeding <i>NB: Use i.v. therapy if patient has diarrhoea or high output stoma.</i>	Phosphate Sandoz [®] Effervescent tablets (16.1mmol/tablet) Two tablets twice daily (64mmol)	Oral therapy not appropriate. Use i.v. therapy – See below	
Patient on intravenous therapy only	Dilute one 20mL vial of Glycophos [®] in 250mL of sodium chloride 0.9% or glucose 5% and give over 6-8 hours (phosphate 20mmol, sodium 40mmol) Check plasma calcium, if high seek pharmacist advice prior to supplementation. Not appropriate for patients with high plasma sodium. Do not recheck phosphate for at least 6 hours post infusion (to allow for distribution). With patients less than 40kg check dose with pharmacist first.		
Intensive Care	INTENSIVE CARE UNIT and acutely ill patients with low phosphate alone may not be due to refeeding syndrome. This can occur with ventilator assisted patients and post anaesthetic. Replace phosphate according to i.v. monograph and only follow the above if refeeding risk is established.		
Renal	The medical team should seek advice from the renal team for phosphate replacements in CKD 4-5. Phosphate levels should be monitored daily and the dose of any supplementation should be adjusted as per bloods and renal team advice. Please consider if the patient is on renal replacement therapy. Use pre-dialysis bloods <u>only</u> to identify need for replacement. In renal impairment check dose of IV prescription with renal pharmacist		

Replacing electrolytes: Magnesium		
Serum Levels 0.75-1.03mmol/L	Mild Greater than 0.5mmol/L	Severe Less than 0.5mmol/L
Patient on intravenous therapy only	10mmol Magnesium sulphate in 50ml sodium chloride 0.9% over 4 hours	20mmol Magnesium sulphate in 50mls sodium chloride 0.9% over 4 hours
Patient able to tolerate oral or via enteral tube feeding <i>NB: Use i.v. therapy if patient has diarrhoea or high output stoma.</i>	Magnesium glycerophosphate 4mmol tablets/solution Two tablets three times a day (24mmol)	Not appropriate – See i.v. above

Replacing electrolytes: Corrected Calcium

Serum Levels 2.15-2.5mmol/L	Monitor corrected calcium when magnesium levels are low. It is important to look at corrected calcium when making any assessment of calcium supplementation
Patient able to tolerate oral or via enteral tube feeding	Sandocal “400” tablets containing 10mmol of calcium per tablet
Calcium must not be added to enteral/parenteral feeds due to stability issues. Please contact ward Dietitian or pharmacist for advice.	

Monitoring and Discharge

	<ul style="list-style-type: none"> • Monitor Potassium, Magnesium, Phosphate and Corrected Calcium (if appropriate) daily for a minimum of three days or continue until levels are within normal range • Once in normal range then three times a week for two weeks • If the patient is discharged within two weeks of being identified as a high risk or above of refeeding please ensure: <ul style="list-style-type: none"> - Appropriate biochemistry is followed up by the GP - For cost effectiveness, the refeeding vitamins are only prescribed for the remaining duration (i.e 10 days) unless otherwise indicated
--	---

Roles and Responsibilities

Doctors	<ul style="list-style-type: none"> • To ensure all appropriate bloods are ordered in the correct time frame • Action electrolyte replacement within 24 hours • Ensure correct medication is prescribed and discontinued appropriately • Please ensure the most appropriate route of administration is chosen • Ensure correct medication and dose is prescribed for the individual patient
Dietitians	<ul style="list-style-type: none"> • To determine correct level of refeeding risk • To ensure appropriate communication via medical documentation • Calculate requirements in reflection of refeeding risk • If enteral tube feeding provide a safe and clear regime • To suggest appropriate biochemistry replacement and vitamin and mineral supplementation
Pharmacist	<ul style="list-style-type: none"> • Ensure correct medication and dose is prescribed for the individual patient • Ensure refeeding medications are discontinued on discharge if appropriate
Nurses	<ul style="list-style-type: none"> • To ensure patients have an accurate weight

N.B. Dietitians are only able to advise on specific medications and doses. It is the responsibility of Doctors/Pharmacists to ensure that it is appropriate.

Refeeding Quick reminder

- 3- check MG, PS and KA for first 3/7
3 – then check 3x a week for 2 weeks

Step 1: Check the baseline bloods for: Mg^{2+} , PO_4^{3-} and K^+

Step 2: If baseline bloods are:

Mg^{2+} : <0.5 mmol/L

PO_4^{3-} : <0.4 mmol/L (not for pts with high plasma sodium or high calcium levels
- check with pharmacy)

K^+ : <2.5mmol/L (see Grey book : refeeding for renal pts)

Replace via IV

IV replacement:

Mg^{2+} : 20mmol Magnesium sulphate in 50ml sodium chloride 0.9% over 4 hours

PO_4^{3-} : Dilute one 20mL vial of Glycophos[®] in 250mL of sodium chloride 0.9% or glucose 5% and give over 6-8 hours (phosphate 20mmol, sodium 40mmol)

K^+ : Infuse potassium into a large vein at up to 20 mmol K^+ /hr (not more than 200 mmol/day). If plasma K^+ < 2 mmol/L with arrhythmia, 40 mmol K^+ may be given over 1hr . Bags for IV potassium infusion are available through Pharmacy.

Oral replacement:

Mg^{2+} : Magnesium glycerophosphate 4mmol tablets/solution . Two tablets three times a day (24mmol)

PO_4^{3-} : Phosphate Sandoz[®] Effervescent tablets (16.1mmol/tablet). Two tablets twice daily (64mmol)

K^+ : Potassium effervescent tablets (Sando-K[®] 12mmol /tablet). If >3mmol/L: Two tablets twice daily (48mmol)

If 2.5-3 mmol/L: Two tablets three times daily (72mmol)

Step 3: Prescribe:

If **Oral**: Thiamine 200-300 mg/day + Vitamin B Co strong 2-3 tablets/day + Valupac multivitamin for 10/7

If **NBM**: Pabrinex 1-2 pairs/day for 10/7 + Abidec liquid multivitamin for 4-10/7 (check with Dietitian)

Step 4: Check Mg^{2+} , PO_4^{3-} and K^+ daily for next 3/7 (or until normal range). Then 3x a week for 2/52.

If low, see above

Step 5: Ensure, if not other clinical indication, Thiamine, Vit B co strong or replacement medication is not added to the TTO's for cost efficiency

10 – only px refeeding vitamins for 10/7

Refeeding Guide for Doctors: Bloods and Prescribing

APPENDIX 4d. HPN troubleshooting algorithms: see APPENDICES on Intranet

ASSESSING METABOLIC ACIDOSIS – THE ANION GAP

Link Consultant: Dr Joyce Popoola

Metabolic acidosis, which may be fatal, will sometimes present acutely in the Emergency Department. The patient will be hyperventilating and, unusually for a 'breathless' patient, will be comfortable lying flat. The condition is characterised biochemically by a fall in arterial pH to less than 7.37 in association with a raised plasma concentration of H^+ ($>43\text{nmol/L}$) and a low plasma HCO_3^- .

Mechanisms:

- net gain of acid (increase in endogenous production or exogenous administration) *eg.* diabetic ketoacidosis, Aspirin poisoning;
- net loss of alkali *eg.* loss from intestine (diarrhoea) or renal tract (renal tubular acidosis);
- failure of renal acid excretion in patients with normal production of acids, *eg.* chronic renal failure, renal tubular acidosis.

Calculations

In health the total for the positively or negatively charged electrolytes is around 150 mmol/L. When the 4 major plasma electrolytes (sodium, potassium, chloride and bicarbonate) are considered,

$$\text{'Anion Gap'} = \text{the sum of } \{ [Na]^+ + [K]^+ \} - \{ [Cl]^- + [HCO_3]^- \}$$

The 'anion gap' is normally between 8 to 17 mmol/L, and mainly ascribable to unmeasured anions. Other 'minor' anions (sulphate, phosphate, organic compounds) and cations (magnesium, calcium, paraproteins) can be measured and both contribute a further 6mmol/L to the equation.

If metabolic acidosis is primarily the result of a loss of HCO_3^- there will be an equivalent rise in $[Cl]^-$ and the anion gap will remain normal, ie there are no unmeasured anions. If metabolic acidosis is accompanied by the presence of unmeasured anions, the gap will be increased.

Causes of Metabolic Acidosis*A) With normal anion gap:*

- Loss of HCO_3^- , as in diarrhoea, proximal renal tubular acidosis
- Decreased renal acid excretion *eg.* distal renal tubular acidosis

B) With increased anion gap

Condition	Unmeasured Anions
a) Lactic Acidosis	
<i>eg.</i> Hypoxia	Lactate, phosphate, urate
b) Ketoacidosis	
<i>eg.</i> Diabetic ketoacidosis, Starvation, Inborn enzyme defects	Ketone bodies (acetone, acetoacetate, β -hydroxybutyrate)
c) Intoxication	
Ethanol	β -hydroxybutyrate, lactate, acetoacetate
Ethylene glycol	Glycolate, oxalate
Methanol	Formate
Paraldehyde	Acetate
Salicylates	Ketone, lactate, salicylate
Uraemia	Sulphate, phosphate

It is important to realise that the ability to respond to the worsening acidosis by hyperventilation and elimination of CO₂ depends on normal lungs. Patients with lung disease are likely to become exhausted and develop severe acidosis relatively quickly.

Treatment

The treatment of metabolic acidosis varies with the underlying disorder. The therapeutic goal is to raise the arterial pH to about 7.20, a level at which arrhythmias are less likely and cardiac contractility is restored. Do not attempt to fully correct the pH as continuing hyperventilation will make the patient alkalotic and may precipitate tetany.

- In patients with renal failure who are acidotic and volume deplete, give NaHCO₃ 1.4% (regimen depending on degree of volume depletion). In contrast, patients with renal failure, acidosis and fluid overload should be referred to the on-call Renal team since they might need renal replacement therapy.

- *For treatment of patients with diabetic ketoacidosis see section on **Diabetic Ketoacidosis**.*

- In patients with lactic acidosis it is important to establish the reason for lactate accumulation (*eg.* cardiovascular compromise, ischaemic bowel) and to initiate resuscitation accordingly.

- Patients with normal anion gap metabolic acidosis secondary to profound diarrhoea or renal tubular acidosis should be treated with NaHCO₃ 1.4%.

When treating (reducing) the anion gap remember:

- Co-existing respiratory disease may lead to an inappropriately severe acidaemia and attention must be directed to the respiratory tract. The patient may even need ventilation.

- In a patient with a metabolic acidosis associated with a normal anion gap, measurement of urine pH should help distinguish between renal and non-renal causes. If the cause is renal the urine pH will be ≥ 5.4 .

CONSIDERATIONS BEFORE ATTEMPTING LUMBAR PUNCTURE

Link Consultant: Dr Niran Nirmalanathan

Lumbar puncture (LP) is potentially dangerous and should be carried out only in the presence of definite clinical indications, in the absence of any contra-indication, and if any clinical doubt, after appropriate exclusion of a space-occupying intracerebral lesion by CT or MRI scan. An LP should be performed, or supervised, by someone experienced in the technique. Unless an absolute emergency, including suspected meningitis, LP is best done during normal working hours. Make sure that samples reach the lab(s) in good time. Remember, most indications for LP are relative rather than absolute. If in doubt, contact a neurologist for advice. If the LP is done for diagnostic reasons, remember to measure the CSF pressure and to take sufficient CSF to provide for routine (bio-chemistry, microbiology) and for tests that might need to be done later (cytology, virology). Volumes greater than 10ml may be needed. When taking a CSF sample, take a 'parallel' blood sample for blood glucose estimation and oligoclonal bands.

Indications for lumbar puncture

1. To obtain CSF to help in the diagnosis of:
 - a) Infection (meningitis, encephalitis or meningovascular syphilis), but only after a CT if there are clinical features suggestive of raised intracranial pressure (see CIU guidelines).
 - b) Subarachnoid haemorrhage, but only when there is high clinical suspicion and the CT scan is negative. To avoid a false negative result or results confounded by a traumatic tap, delay the LP until at least 12 hrs after the onset of headache.
 - c) Inflammatory conditions of the peripheral nervous system eg Guillain-Barre syndrome.
The main purpose of LP is to exclude significant CSF pleocytosis; a raised protein may not be present early and is not required for the diagnosis.
 - d) Malignant meningitis.
 - e) CNS inflammatory conditions such as multiple sclerosis.
2. To introduce antimicrobials or contrast medium for myelography.
3. To measure CSF opening and closing pressure in a patient with benign intracranial pressure, but only after the presence of a mass has been excluded.

Image prior to an LP if

- History of brain mass/tumour
 - History of subdural or epidural haematoma
 - Focal neurological signs on examination
 - Altered mental status on examination
 - Papilloedema (for suspected idiopathic intracranial hypertension, contact neurologist)
- Document informed consent, explaining reasons, process, risks. If unable to, explain why.

Contraindications to lumbar puncture

- Intra-cranial mass, lesion or brain swelling causing mass effect
 - Uncontrolled prolonged or frequent epileptic seizures
 - Any possibility of intra-spinal mass lesion
 - Epidural infection of overlying cellulitis in lumbar region.
 - Anticoagulation, coagulation defect or low platelet count.
 - Where there are any concerns, including for patients on antiplatelet drugs, an individualized risk-benefit assessment should be undertaken.
- An INR of >1.4 or platelets of <50,000 are absolute contraindications. Correct and re-check before proceeding (*see also Appendix 7*).

Potential hazards of lumbar puncture

- Post LP CSF leakage through the puncture site.

This may exacerbate deterioration of brain stem or spinal cord functions (see below) or lead to 'low pressure' headache.

The risk of leakage can be reduced by using a 22g blunt-tipped needle, which carries a 1 in 8 risk of headache).

Most self-resolve, 40% in 3-4 days; 75% within a week with increased fluids and bed rest.

Others may need iv caffeine or epidural blood patch. A leak requiring surgery to fix it is extremely rare.

- Deterioration of brain stem function which may lead to death due to coning in the presence of raised intracranial pressure.
- Deterioration of spinal cord function due to an obstructive intraspinal mass lesion.
- Allergic reaction to anaesthetic
- Iatrogenic infection.
- Epidural haematoma.
- Local damage to intraspinal structures (very rare).

PROTECTING ANTICOAGULATED PATIENTS FROM BLEEDING

Link Consultant: Dr James Uprichard

Before initiating or interrupting anticoagulation, evaluation of the relative risk of thromboembolism vs bleeding is required. The risk of thrombosis varies dependent on the indication and should be carefully balanced when interruption to anticoagulation is required i.e. with active bleeding or for peri-operative management. Balancing the risk/benefit of anticoagulation in these clinical situations can often be complex and evidence based risk stratification tools should be used where appropriate to support clinical decision making.

Example risk stratification tools include:

- CHADSVASc for stroke risk in atrial fibrillation
<https://www.mdcalc.com/calc/801/cha2ds2-vasc-score-atrial-fibrillation-stroke-risk>
- HASBLED and ORBIT for bleeding risk in atrial fibrillation
<https://www.mdcalc.com/calc/807/has-bleed-score-major-bleeding-risk>
<https://www.mdcalc.com/calc/10227/orbit-bleeding-risk-score-atrial-fibrillation>
- Refer to haematology for thrombosis risk stratification (bleep 6068 or via switchboard if out of hours)

Refer to haematology (bleep 6068 or via switchboard if out of hours) and the team for the relevant organ system if bleeding. Reassessment of both risks and benefits of anticoagulation should also occur when there is any change in the clinical state of the patient. The patient and if appropriate their next of kin should be involved in the decision to initiate or stop anticoagulation.

If a decision to stop anticoagulation is made, a date for review should be agreed and documented in the medical notes. If the patient is to be discharged without recommencing anticoagulation they should have appropriate outpatient clinic follow up booked before discharge.

HEPARIN

Monitoring heparin therapy.

i) Low molecular weight heparin

Patients receiving *low molecular weight heparin* (eg dalteparin) therapy do not routinely require monitoring. However, monitoring using the anti-Xa heparin assay should be considered in:

- patients with a creatinine clearance below 30 ml/min
- patients with extremes of body weight (below 50 kg and above 150 kg)
- women who are pregnant and taking therapeutic doses

The anti-Xa assay (measured 3-4 hrs after injection) should be 0.2-0.5 units/mL for prophylaxis, or 0.5-1.0 units/mL for treatment of acute venous thromboembolism.

When therapeutic anticoagulation is required in the presence of a high bleeding risk, recent major surgery or severe renal impairment (CrCl <30ml/min), *unfractionated heparin* (UFH) should be considered as it has a half-life of 1½ hours when given by IV infusion and it can be rapidly reversed with protamine sulphate.

ii) Unfractionated heparin

Monitoring is essential in patients receiving a continuous IV infusion of *unfractionated heparin*. A scheme for instigating and monitoring use of unfractionated heparin is as follows:

1. Measure APTT ratio at start of therapy. Give a 5000 unit loading dose as a bolus IV injection.
2. **In patients who are at risk of bleeding consider omitting bolus dose.**
3. Draw 25,000 units of unfractionated heparin made up to 50mL with 0.9% sodium chloride (resulting concentration of 500 units/mL) into a 50mL syringe. Start IV infusion, at 2mL/hr.
4. The target therapeutic range for APTT ratio is 1.5-3.5. Check APTT ratio four hours after infusion started (and after any dose change) and adjust as follows:

APTT ratio	ACTION
>6	stop for 1 hr; reduce by 1mL/hr (500units/hr)
5.3-5.9	reduce by 0.6mL/hr (300units/hr)
4.7-5.2	reduce by 0.4mL/hr (200units/hr)
4.1-4.6	reduce by 0.2mL/hr (100units/hr)
3.6-4.1	reduce by 0.1mL/hr (50units/hr)
1.5-3.5	NO CHANGE
1.2-1.4	increase by 0.4mL/hr (200units/hr)
<1.2	increase by 0.8mL/hr (400units/hr)

Repeat APTT ratio daily while on IV unfractionated heparin. Check platelet count at start of therapy and after 5 days. A therapeutic dose of unfractionated heparin at the dose of 250units/kg bd can be used subcutaneously, for example in the outpatient setting. Subcutaneous unfractionated heparin has been used without APTT monitoring but it is essential these patients are discussed with Haematology (bleep 6068 or via switchboard out of hours). If required, an APTT ratio should be taken four hours after injection and this can be reduced to daily on haematology advice once the APTT ratio is in range (target 1.5-3.5).

Bleeding in a patient on heparin. Older patients on heparin for > 4 days are most at risk but bleeding can occur in anyone, from any source. Bleeding can be silent, into a “third space”, such as the retroperitoneum. A falling haematocrit, back pain or even severe anxiety in the patient, can give a clue. Arterial puncture sites should be carefully compressed and observed. Any painful swelling should be regarded as haematoma.

Action – if the patient is on continuous IV infusion of unfractionated heparin (UFH) via a pump, the UFH should be STOPPED (heparin activity will be lost from the plasma within 2 to 4 hrs). For rapid reversal, 25-50mg protamine sulphate should be given by slow IV injection at a rate not exceeding 5mg/min, and no more than 50mg should be given in any one dose.

Protamine may cause hypotension, bradycardia and anaphylaxis, so should be given when anaesthetic support is present. Protamine is less effective against LMWH but can still provide some reversal of anticoagulation. Seek *urgent* advice from the Haematology registrar (bleep 6068 or via switchboard out of hours). It may need to be repeated if bleeding persists as protamine has a shorter half-life than LMWH. Administration of plasma products will *not* reverse heparin anticoagulation.

Invasive procedures in patients on Heparin. Intravenous UFH should be stopped between 2-4 hours before undertaking an invasive procedure. In a patient on prophylaxis with LMWH, or subcutaneous UFH, the time should be extended to 12 hours. In patients on therapeutic doses of LMWH, the time should be extended to 24 hours.

WARFARIN

Tackling excessive warfarin-induced anticoagulation

INR >5.0 with no bleeding. Withdraw warfarin for 1–2 days and reduce maintenance dose. Cause of elevated INR should be investigated.

If INR >8.0 with no bleeding, give 1-5mg Vitamin K (phytomenadione) orally (the IV preparation of Konakion can be given by mouth) and repeat INR in 24 hours. Omit warfarin for 2 doses. The cause of the elevated INR should be investigated.

Minor haemorrhage, e.g. haematuria, epistaxis. Reduce dose or, if INR >4.5, withhold warfarin for one or more days. Give Vitamin K 1-3mg IV and repeat INR in 6 hours. Vitamin K administration may not be appropriate for minor bleeds in a patient with an artificial heart valve as it may induce warfarin resistance; here temporary cessation of warfarin may need cover with heparin. Seek advice from a cardiologist or haematologist. Refer for the investigation of the bleeding source. Tranexamic acid may be used topically (500mg IV preparation (one vial) should be soaked into gauze and applied to the affected area).

Major life-threatening haemorrhage. Obtain venous access and take blood for full blood count, clotting screen and cross-matching. Stop warfarin and immediately give Vitamin K 5mg by slow IV injection. Prothrombin Complex Concentrate (PCC) should be given which is stored in the Blood Transfusion Department. Contact the on-call haematology registrar (bleep 6068 or via switchboard out of hours). See the Octaplex protocol under the 'Management of bleeding' section of the [anticoagulation and thrombosis homepage](#).

Recommended doses of PCC:

INR \leq 4.0: 20units/kg;

INR >4.0: 30units/kg

The single dose should not exceed 3000units. FFP (15mL/kg) should only be infused if PCC is unavailable. Do not re-start warfarin until bleeding is controlled.

Repeat INR after 6 hours and after 24 hours; discuss with Haematology if bleeding persists. Further administration of Vitamin K may be necessary after 24 hours.

Administration of higher doses of vitamin K than advised in this guideline may result in warfarin resistance.

OTHER ANTICOAGULANTS

Fondaparinux

There is no specific antidote for fondaparinux (protamine has no effect).

Recombinant factor VIIa (*NovoSeven*) can be used for critical bleeding. Contact the on-call haematology registrar (Bleep 6068 or via switchboard out of hours).

Direct Oral Anticoagulants- DOACs (Rivaroxaban, edoxaban, dabigatran and apixaban)

The DOAC bleeding pathway should be followed:

[Document Preview - Anticoagulation and Thrombosis/Management of Haemorrhage in Patients taking DOACs \(unily.com\)](#)

If bleeding is associated with dabigatran, apixaban, edoxaban or rivaroxaban:

- Stop DOAC and inform the haematologist (bleep 6068 or via switchboard out of hours)
- Document the time of the last dose of the DOAC
- Check FBC, coagulation screen, renal function and arrange group and save

Mild bleeding: Minor bleeding may only require withholding one or two doses of the drug. Tranexamic acid 1g po/iv should be considered. Contact the on-call haematology registrar (Bleep 6068 or via switchboard out of hours).

Major bleeding: In addition to the above measures, this requires haemorrhage control (surgical or radiological), transfusion of packed red cells (aim Hb >70- 90 g/L) and platelets (aim for PLT > 50 x 10⁹/l or if CNS bleed aim PLT > 100 x 10⁹/l). Platelets should be requested if there is on-going bleeding and the platelet count has fallen below 100 x 10⁹/l. Consider tranexamic acid 1g po/iv. Consider PCC 25-50 units/kg). **Urgent Haematology advice should be sought** (bleep 6068 or via switchboard out of hours).

Dabigatran, a thrombin inhibitor, has a licensed reversal agent called Idarucizumab (Praxbind). It is stored in 2 locations at St Georges Hospital: ED Resus and Pharmacy.

Please consult the full protocol:

[Document Preview - Anticoagulation and Thrombosis / Protocol for the use of Idarucizumab \(Praxbind\) for the reversal of dabigatran etexilate / Clinical Guidelines \(unily.com\)](#)

The anti-Xa inhibitors apixaban and rivaroxaban have a licensed reversal agent called Andexanet alfa (Ondexxya). NICE has only approved this reversal agent for the indication of uncontrolled or life-threatening gastrointestinal bleeding. Andexanet alfa is stored in Lanesborough pharmacy.

Please consult the full protocol:

[Document Preview - Anticoagulation and Thrombosis/Andexanet Alfa Protocol \(unily.com\)](#)

Note: Andexanet alfa has shown to be effective in the reversal of the anti-Xa inhibitor edoxaban. However, it is not currently licensed for edoxaban due to insufficient data and is therefore not approved by NICE; Reversal of edoxaban is currently excluded from the Andexanet alfa Trust protocol.

MANAGEMENT OF MASSIVE HAEMORRHAGE

Trauma/Team Leader/Clinician must declare a 'CODE RED' and activate the Massive haemorrhage protocol if there is:

- Systolic blood pressure <90 mmHg;
- No response to fluid bolus; and,
- Suspected or confirmed haemorrhage

A nominated team member must contact the Blood Transfusion Department immediately using the dedicated telephone extension 6789 and request PACK A.

If the patient continues to bleed then PACK B should be requested through the same dedicated extension number (6789). PACK B will not be issued unless it has been requested. The Haematology SpR should be contacted on bleep 6068 or via switchboard out-of-hours. The full protocol can be found on the Trust Intranet on the Blood Transfusion Department page and in the Blood Transfusion policy:

[Document Preview - Blood Transfusion/Major Haemorrhage Protocol/SOP \(unily.com\)](#)

HIV POST-EXPOSURE PROPHYLAXIS (PEP)

Link Consultant: Dr Bojana Dragovic

Pathogenesis studies indicate that there may be a window of opportunity to avert HIV infection by inhibiting viral replication following an exposure. Once HIV crosses a mucosal barrier, it may take up to 48–72 h before HIV can be detected within regional lymph nodes and up to five days before HIV can be detected in blood. Initiation of ART has been shown to reduce dissemination and replication of virus in all tissues if initiated early after inoculation in an animal model.

1. Post-exposure prophylaxis after sexual exposure (PEPSE)

Please see the national guidelines for the use of HIV postexposure prophylaxis, 2021 <https://www.bhiva.org/file/6183b6aa93a4e/PEP-guidelines.pdf>

The recommendations for PEP after sexual exposure are summarised in Table 4 (section 6.5, page 34)

PEPSE is not routinely recommended after any type of sex with HIV-positive source on antiretroviral therapy (ART) with a confirmed and sustained (>6 months) undetectable plasma HIV viral load (VL) (<200 c/mL).

If the source is of *unknown status*:

- Proactive attempts should be made to establish the HIV status of the source.

Source individual known to be *HIV-positive*:

- Attempts should be made at the earliest opportunity to determine the HIV VL, resistance profile and treatment history.

The ED is the first point of contact for PEPSE, where following assessment, if PEPSE is required, a 30 day pack will be issued, of which the patient should take 28 days.

Patient should attend the Falcon Road Sexual Health Clinic within the next few days for follow up, further risk assessment and STI screening (Sexual Health South West London Clinic, Monday to Friday 8am - 8pm, telephone: 0333 300 2100)

2. Occupational post-exposure prophylaxis

Please see national guidelines for the use of HIV postexposure prophylaxis, 2021 <https://www.bhiva.org/file/6183b6aa93a4e/PEP-guidelines.pdf>

The recommendations for PEP following occupational exposure are summarised in Table 4 (section 6.5, page 34)

There is a small but real risk of HIV infection after accidental exposure to contaminated (HIV-containing) blood or ‘high-risk’ body fluids (amniotic, peritoneal, cerebro-spinal, synovial and pericardial fluids, breast milk, semen, vaginal secretions, body fluid that is blood-stained, saliva in association with dentistry, exudate or other fluid from a burn or other skin lesion) or unfixed tissues and organs. With prompt treatment with antiviral agents this risk can be reduced by around 80%.

The risk is greatest following a needle stick injury where the needle is blood stained, the injury is deep, the needle has a hollow bore, the source patient has a high HIV viral load,

and where the needle has been in an artery or vein. The risk is also high after percutaneous exposure from contaminated instruments or bone fragments. The risk is less after mucus membrane exposure (around a third of that after needle stick injury) or when blood or other infected body fluids contaminate broken skin. The risk is negligible where contact is with intact skin, or where there has been contamination with 'low risk' body fluids such as urine, saliva, vomit or faeces.

Please note, if the source patient is known to be HIV positive but has a confirmed and sustained (>6 months) undetectable plasma HIV viral load (<200 c/ml), occupational PEP is generally not recommended. Where there is any uncertainty about HIV VL results or adherence to ART then PEP should be given.

Act Immediately:

- Wash area liberally with soap and running water without scrubbing
- Do not use antiseptic or strong detergent washes
- Allow natural bleeding, do not suck wounds
- Cover with waterproof plaster
- Rinse eyes with water before and after removal of contact lenses
- Clean contact lenses with normal lens cleaning solution

Report:

- Contact OHD by bleep 8092
- Out of hours attend the ED and ask for immediate attention (Do not wait for triage)
- Complete an adverse incident form
- If the injury is reported to ED during out of hours, to contact OHD as soon as it opens to complete risk assessment and follow up

Immediate risk assessment:

- Normally this is done by OHD/ ED staff, but you may be asked for advice
- Source testing should be sought if BBV status is unknown

Full risk assessment

- To decide whether someone initiated on PEP should continue and complete the course.
- Normally done by occupational health up to 5 days after initial exposure, once BBV status of source patient is known

PEP prescription packs for both PEPSE and occupational PEP

Raltegravir 1200 mg (two 600mg tablets) once daily

plus

Tenofovir disoproxil 245 mg / Emtricitabine 200 mg tablet, one tablet once daily

Packs contain a 30 day supply, of which the patient should take 28 days

If the source patient is known, other combinations may be more appropriate- seek advice.

Start first dose ASAP in the department

PEP is still indicated in pregnancy but please seek advice from the Clinical Infection Unit.

Check for drug-drug interactions- NO antacids/mineral supplements/vitamins supplements

WHEN TO TEST FOR HIV INFECTION

NICE and BHIVA guidance recommends patients should be offered HIV testing when admitted to hospital or attending EDs in areas with a prevalence ≥ 2 per 1000 residents. The seroprevalence for Wandsworth in 2019 was 5.42 per 1,000. ED at St George's Hospital offers routine opt out HIV testing to all patients over the age of 16 having a blood test.

Late HIV diagnosis, however, remains a serious problem – in 2019 41% of newly diagnosed adults in the UK had CD4 < 350 cells/mm³.

HIV testing is recommended for:

While **all** general medicine admissions should be tested, HIV is particularly recommended for:

- People belonging to groups at increased risk of exposure to HIV, including men who have sex with men (MSM) and their female sexual partners, black Africans, people who inject drugs (PWID), sex workers, prisoners, trans women and people from countries with high HIV seroprevalence and their sexual partners
- People attending health services whose users have an associated risk of HIV, including sexual health services, tuberculosis (TB), hepatitis and lymphoma clinics, antenatal clinics, termination of pregnancy services and addiction and substance misuse services;
- All people presenting with symptoms and/or signs consistent with an HIV indicator condition:

Appendix 1. <https://www.bhiva.org/file/5f68c0dd7aefb/HIV-testing-guidelines-2020.pdf>

- People accessing healthcare in areas with high ($>2/1000$; if undergoing venepuncture) and extremely high ($>5/1000$; all attendees) HIV seroprevalence;
- Sexual partners of an individual diagnosed with HIV

WORKING WITH HIV POSITIVE PATIENTS

Patients infected with HIV present either with symptoms of an HIV-associated disease, or with a coincidental, unrelated problem.

The commonest presentation of AIDS itself is with *Pneumocystis carinii* pneumonia (PCP). Symptoms are usually of progressive dyspnoea, occasionally profound, with increasing severity over several days. This is often accompanied by a dry cough, fever, and less commonly chest pain.

Other severe opportunistic infections include oral and oesophageal candidiasis. Pulmonary TB is increasingly recognised as a precipitating condition of HIV infection and patients presenting with TB should be offered an HIV test. Kaposi's sarcoma is a less common presentation. Dementia is usually a late manifestation of AIDS. Features of other HIV associated diseases include skin rashes, thrombocytopenia, and a seroconversion illness

with sore throat, rash, fever, and lymphadenopathy. Chronic diarrhoea, weight loss and fevers are features of symptomatic HIV infection.

All HIV positive inpatients should have drug-drug interactions reviewed by the ward pharmacist. The CIU team should also be aware of unplanned admissions in other specialties.

Inpatients with a new diagnosis of HIV are encouraged to speak to a Health Adviser (HA) and should be seen in HIV clinic post discharge. HAs are based at the Courtyard clinic and can be contacted on ext. 3140 or 2668. Outpatient appointments can be made by calling ext. 3140.

Outpatients who are seeking HIV testing should be referred to their local sexual health clinic. In Wandsworth, this is located at 160 Falcon Rd SW11 2LN, Tel: 0333 300 2100. This is a free and confidential service which is available each weekday.

If you have patients with known HIV:

- 1) Inform CIU SpR (bleep 7568) or Courtyard clinic HIV pharmacy on ext. 1803 of all admissions of HIV positive patients even those who are admitted with non-HIV related illness.
- 2) Find out which clinic they attend and if they attend there for regular review.
- 3) Find out their latest viral load, i.e. are they "undetectable"?
- 4) Find out if their last CD4 was high (good) or low <200 (risk of opportunistic infection).
- 5) If they do not know their CD4, ask if they are on any PCP prophylaxis (e.g. cotrimoxazole or dapsone) as this will indicate someone with a CD4 <200 and at risk of opportunistic infection.
- 6) Are they on antiretrovirals medication and are they still taking it?
- 7) If yes, find out the name of the antiretrovirals and what time of day they usually take it.
- 8) Prescribe their antiretrovirals at the time at which they usually take them, as it is important that these medications are not missed.
- 9) Ensure ALL medications being prescribed are checked with pharmacy or the Liverpool HIV Drug Interactions website (<https://www.hiv-druginteractions.org/>) BEFORE they are prescribed – even simple things can interact with ARVs and be dangerous (i.e. PPIs, steroids, calcium supplements)
- 10) If your patient is going to be nil by mouth or unable to take tablets then please contact the Courtyard clinic HIV pharmacy urgently so they can advise of suitable alternatives.
- 11) It is not always necessary to do a viral load or CD4 on admission (i.e. if they have recently attended follow up and are stable on treatment and have not missed their ARVs), please discuss with CIU SPR for advice on when to order.

FIRST STEPS IN THE EVENT OF A MAJOR INCIDENT

Link Consultants: Dr Mark Haden

St Georges Hospital is a Major Trauma Centre serving both the urban population and also the rural areas to the South West of the Capital – Major Incidents have and will occur, and it is imperative that all staff know what to do.

A Major Incident is an event which requires additional resources to manage the numbers and types of patients presenting. This may be following a single event, such as a train crash or a bomb or may be following a sustained ongoing event, such as pandemic flu.

A Major Incident may be declared via the London Ambulance Service, South East Ambulance Service or self declared by the Emergency Department in conjunction with the ED Consultant.

- A hospital Command point is established in the Hospital Incident Coordination room G2.009 – Silver Command
- An ED Command point is established at the Majors Desk in ED – Bronze Command
- Patients are triaged on arrival to ED at the ambulance entrance

P1-patient actively dying now who needs immediate <C>ABC treatment– to RESUS

P2 - patient with serious injuries – to MAJORS

P3 - walking patients – to UCC

- There are command roles within Surgery, ICU, PICU and Theatres to ensure rapid damage control and ICU care

In the event of a Major Incident being declared:

1– DO NOT CALL THE HOSPITAL TO SEE IF YOUR ASSISTANCE IS REQUIRED

Hospital switch boards, emergency departments and wards will be extremely busy. You should attend your next usual shift as normal unless you are specifically called in to help. Staff who have seen media reports and want to volunteer their services should only attend hospital if they can do so and still attend their next rota'd shift. These staff should report to their usual ward, Consultant, Matron or Line Manager.

2 – CARRY YOUR ID BADGE

Hospital will be locked down by security and no staff will be admitted without an ID badge. The only entry point to the hospital will be via Lanesborough Wing.

3 – DO NOT TALK TO THE PRESS

Under no circumstances give any information out to any person.

4 – BE PREPARED

- Find the Major Incident plan on the hospital intranet and read it – especially be aware of action cards for specific roles that you may be asked to undertake.
- Undertake MAST Major Incident Training (being rolled out over 2018)
- Undergo specific Major Incident training for your role.
- Complete an HMIMMS course or similar

5 – FOLLOW YOUR ACTION CARD

If you have a specific role in a major incident you will be given an action card detailing your responsibilities, follow this card. Staff will be asked to perform tasks within their expertise and competence.

6 – LOOK AFTER YOUR SELF AND YOUR COLLEAGUES

It is normal to be affected in the aftermath of a major incident, especially if children or young people have died or suffered life changing injury. Some staff will be profoundly

affected and may need help – this applies equally to more experienced staff and to juniors. Do not suffer in silence, seek help from your partners, friends, families, colleagues, GP or occupational health.

APPENDIX 10

GUIDE TO THERAPEUTIC DRUG LEVEL MONITORING

<http://microguide.horizonsp.co.uk/viewer/sguh/adult>

Gentamicin¹ *Once-daily regimen* - sampling time: trough 18-24 hours post dose; therapeutic range: trough <1 micrograms /mL.

Conventional regimen - sampling time between 3rd and 4th dose; trough immediately prior to next dose, peaks 60min post IV dose. Therapeutic range: trough <2 micrograms/mL, peak 5-10 micrograms/mL for streptococcal or enterococcal endocarditis trough <1 microgram /mL, peak 3-5 micrograms/mL; time to steady state: 12-40 hrs (longer in renal failure).

Vancomycin¹ In patients receiving vancomycin by intermittent infusion - sampling time varies according to renal function (for further information *see*:

<http://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Vancomycin%20Guideline.pdf>

Trough levels should be taken immediately prior to next dose; therapeutic range: trough 10-15micrograms/mL, although levels of 15-20micrograms/mL may be required for deep-seated infections such as endocarditis and osteomyelitis – contact Microbiology for advice; time to steady state: 30-40 hrs (longer in renal failure). In patients receiving vancomycin by continuous infusion, sample during the infusion; therapeutic range 20-25 micrograms/mL.

Amikacin *Once-daily regimen* - sampling time: trough 18-24 hours post dose; therapeutic range: trough <5 micrograms /mL.

Carbamazepine² Sampling time: immediately prior to next dose; therapeutic range: single therapy 4-12mg/L, multiple therapy (i.e. one or more drugs used in addition to carbamazepine) 4-8mg/L; time to steady state: 2-4 weeks after start of treatment, or 4-5 days after dose change.

Digoxin² Sampling time: at least 6 hours post dose or immediately pre-dose; therapeutic range: 0.9-2.0 micrograms/L; time to steady state: 7 days (longer in renal failure).

Lithium² Sampling time 12 hours post dose; therapeutic range: as treatment - 0.4 –1.0 mmol/L, as prophylaxis - 0.5-0.8mmol/L; time to steady state: 3-7 days.

Phenytoin² Sampling time: immediately prior to next dose; therapeutic range: 5-20mg/L (interpretation difficult in renal failure, low albumin, raised bilirubin); time to steady state: 7 days or longer.

Theophylline Sampling time: liquid preps - peak 2 hours post dose, SR tablets – peak 4 hours post dose, trough immediately prior to next dose; therapeutic range: 10-20mg/L; time to steady state: 2 days.

^{1, 2} Advice on these products can be obtained from:

¹ Microbiology, ext 5685/6 or Bleep 480 or via switchboard out of hours.

² Chemical Pathology, Bleep 6032 or pager SG 138 or via switchboard out of hours.

ANTIFUNGAL MONITORING

<http://microguide.horizonsp.co.uk/viewer/sguh/adult>

AMIKACIN DOSING GUIDELINES

<http://microguide.horizonsp.co.uk/viewer/sguh/adult>

VANCOMYCIN DOSING GUIDELINES

<http://microguide.horizonsp.co.uk/viewer/sguh/adult>

APPENDIX 11

URGENT REMOVAL OF TUNNELLED OR HICKMAN/PORTACATH LINES

Link: Jackie Nicholson, Nurse Consultant Vascular Access

Patients with skin tunnelled (eg Hickman/Portacath) catheters should usually have these lines removed as an elective procedure in accordance with the management plan. However, where such lines are suspected to be contributing to or causing sepsis, they should be removed urgently following discussion with the Consultant and/or the Microbiology team (bleep 6480; or, Ext. 5676/1970).

As far as possible, urgent removal of Hickman/Portacath lines should be conducted in working hours. During weekdays, please contact the Venous Access Team on bleep 6090 (between 08.30am–4.30pm).

If a line needs to be removed urgently out-of-hours, unless someone in the team has the expertise to remove the catheter, contact the on-call Interventional Radiologist via switchboard.

Related clinical guidelines are available on the trust intranet:

<https://stginet.unilv.com/sites/venous-access-service/SitePageModern/31601/venous-access-service-home>